Genet. Res., Camb. (2009), **91**, *pp.* 355–363.
doi:10.1017/S0016672309990255

The evolution of heterochiasmy: the role of sexual selection and sperm competition in determining sex-specific recombination rates in eutherian mammals

JUDITH E. MANK*
Edward Grey Institute, Department of Zoology, University of Oxford, South Parks Road, Oxford OX1 3PS, UK

(*Received 11 August 2009 and in revised form 14 September 2009*)

Summary

Early karyotypic work revealed that female and male recombination rates in many species show pronounced differences, and this pattern of heterochiasmy has also been observed in modern linkage mapping studies. Several hypotheses to explain this phenomenon have been offered, ranging from strictly biological mechanisms related to the gametic differences between the sexes, to more evolutionary models based on sexually antagonistic selection. However, despite the long history of interest in heterochiasmy, empirical data has failed to support any theory or pattern consistently. Here I test two alternative evolutionary hypotheses regarding heterochiasmy across the eutherian mammals, and show that sexual dimorphism, but not sperm competition, is strongly correlated with recombination rate, suggesting that sexual antagonism is an important influence. However, the observed relationship between heterochiasmy and sexual dimorphism runs counter to theoretical predictions, with male recombination higher in species with high levels of sexual dimorphism. This may be the response to male-biased dispersal, which, rather than the static male fitness landscape envisioned in the models tested here, could radically shift optimal male fitness parameters among generations.

1. Introduction

Difference in recombination rates between the sexes were observed in early cytological studies as different chiasma frequencies in female and male samples (Burt *et al*., 1991), and heterochiasmy has been subsequently noted in a diverse array of taxa (Lenormand & Dutheil, 2005). Initial explanations for this widespread phenomenon stemmed from the observation that in some species, the heterogametic sex is achiasmate, or completely lacking any recombination, and it was suggested that recombination may be suppressed to varying degrees in the heterogametic sex in all organisms in order to prevent crossing over between the different sex chromosomes (Haldane, 1922; Huxley, 1928). While this hypothesis, named the Haldane–Huxley Rule, has been carried forward and is often assumed to be true in the current literature, it only applies to the small fraction of species where one sex is achiasmate; for the majority of species where both sexes recombine, but at different rates, the Haldane–Huxley Rule is clearly untenable as many species with both male (Burt *et al*., 1991) and female (Hansson *et al*., 2005; Åkesson *et al*., 2007) heterogametic systems fail to conform to the predicted pattern.

Further fine-scale work indicates that not only overall recombination rate but also the location of recombination within the genome varies greatly between females and males (Perry & Jones, 1974; Shifman *et al*., 2006). Based on this variety, it would be reasonable to speculate that recombination is not subject to selection, but rather the patterns we observe are the result of biological differences between females and males in meiosis. Specifically, the long-term torpor of vertebrate ova in the penultimate stages of arrested meiosis may produce fundamental differences between male and female recombination rate due simply to genomic and biochemical affinities rather than evolutionary mechanisms. However, the lack of a sex-specific pattern in the data makes this explanation also somewhat unsatisfying.

Studies have shown that recombination differs not only between the sexes but also among individuals (Broman *et al*., 1998), populations (McVean *et al*.,

* Corresponding author. E-mail: Judith.Mank@zoo.ox.ac.uk

2004; Hernandez *et al*., 2007), and closely related species (Winckler *et al*., 2005), suggesting that if these differences are heritable, sufficient variation exists to provide a target for selection pressure. Recent re-examinations of the heterochiasmy phenomenon with this in mind have resulted in evolutionary theory linking recombination rate differences between males and females to differences in sexually antagonistic selection pressures. One suggestion is that, as sexual selection is typically more powerful for males, this selective regime would produce reduced recombination in males in order to preserve the favourable gene combinations that confer male reproductive success (Trivers, 1988). There is some circumstantial evidence for this, as successful male tends to produce successful sons (Wedell & Tregenza, 1990); however, mathematical treatments failed to support the conjecture (Burt *et al*., 1991). Additionally, comparative phylogenetic work across plants and animals did not support the link between sexual selection and male recombination, although this may be due to vast differences in the sexual selection regimes of plants and animals resulting from differences in haploid versus diploid selection, parental provisioning and factors contributing to fertilization success.

More recently it was suggested that the pattern of heterochiasmy is a function of sex-specific selection during the haploid phase (Lenormand & Dutheil, 2005), and results from the difference in strength of gametic selection for females and males. For vertebrates, female meiosis is typically completed at the point of fertilization, effectively eliminating any haploid selection for ova. However, haploid selection in males could theoretically result from sperm competition regimes, presumably with stronger sperm competition resulting in higher levels of heterochiasmy. Several studies have indicated that genes that function in sperm production or function are under exceptionally strong selective regimes (Aguade, 1999; Meiklejohn *et al*., 2003; Begun & Lindfors, 2005). Additionally, *Drosophila* recombination, which is limited to females, was shown to be more common in regions of male-biased gene expression, suggesting higher recombination in females for genes related to sperm function (Zhang & Parsch, 2005). Therefore, this hypothesis shows some promise in explaining heterochiasmy patterns.

Several sex-specific linkage maps and cytological screens are now available for the eutherian (placental) mammals, making it possible to quantify the degree of heterochiasmy for these lineages (Table 1). These data make tenable a rigorous test of the role of sex-specific selection in the diploid (Trivers, 1988) versus haploid (Lenormand & Dutheil, 2005) phases of the life cycle within a clade that shares many reproductive aspects. Additionally, eutherian mammals all share placental gestation, which is a major component of female reproductive fitness. Confining analysis to this clade therefore controls, at least to some extent, for variance in female-specific selection regimes, making it possible to focus on male fitness components and their relationship to recombination.

If sexually antagonistic selection, either in the diploid or haploid phase, is linked to recombination rate differences between the sexes, there should be a clear pattern in the data. Specifically if differences in sex-specific selection on the diploid phase of the life cycle resulting from sexual selection are ultimately causing the heterochiasmy under the model presented by Trivers (1988), we would expect lineages with greater variance in male mating success to have less recombination in males, therefore sexually selected lineages would show greater heterochiasmy in recombination as a function of suppression of recombination in males.

If heterochiasmy is the product of differences between females and males in haploid selection in the gametic phase, somewhat different patterns are expected. In mammals, the haploid phase of the life cycle is relatively limited in both sexes, though most so in females, where meiosis is completed at fertilization, and therefore is nearly non-existent. Haploid selection, if it is a substantive evolutionary force at all, would be observed primarily for sperm. Therefore, if heterochiasmy is a function of sexually antagonistic haploid selection, those species with more intense sperm competition should show greater differences in female and male recombination.

2. Materials and methods

I found sex-specific recombination data, either from linkage mapping studies or recombination nodule counts, for ten eutherian mammal species. Although many more eutherian mammals have been the subject of linkage mapping, the vast majority of these studies computed only a sex-averaged map. Where multiple studies existed for a single species, I selected those based on linkage mapping rather than cytology, and where multiple linkage maps existed, I chose those with greater marker density and genome coverage. The species included in this analysis (Table 1) are human (Broman *et al*., 1998), baboon (Burt *et al*., 1991), macaque (Burt *et al*., 1991), sheep (Maddox *et al*., 2001), pig (Archibald *et al*., 1995), dog (Neff *et al*., 1999), cat (Menotti-Raymond *et al*., 2009), cow (Ihara *et al*., 2004), bison (Schnabel *et al*., 2003) and mouse (Shifman *et al*., 2006). Although sex-specific maps exist for marsupial species (Zenger *et al*., 2002; Samollow *et al*., 2004), the reproductive differences resulting from the evolution of extended internal gestation and the placenta in the eutherians vastly alter the female-specific selection regime from that

https://doi.org/10.1017/S0016672309990255 Published online by Cambridge University Press

Table 1. *Eutherian taxa included in this analysis, along with information on sperm competition and sexual dimorphism*

Species	Density of map	Female crossovers per chromosome[a]	Male crossovers per chromosome[a]	Heterochiasmy[b]	Sexual dimorphism[c]	Sperm competition
Mouse (*Mus musculus*)	>10000 (Shifman *et al.*, 2006)	0·8736	0·6930	0·2306	Low	High (Dean *et al.*, 2006)
Dog (*Canis familiaris*)	276 (Neff *et al.*, 1999)	0·5074	0·3469	0·4594	Low	High (Woodall *et al.*, 1993; Nowak, 1999)
Cattle (*Bos taurus*)	2325 (Ihara *et al.*, 2004)	1·0809	1·0899	−0·0083	High[d]	Low (Van Vuure, 2005)
Cat (*Felis sylvestris*)	483 (Menotti-Raymond *et al.*, in press)	3·9108	1·8194	0·7300	Low	High (Bradshaw & Cameron-Beaumont, 2000)
Sheep (*Ovis aries*)	1093 (Maddox *et al.*, 2001)	1·1681	1·4173	−0·1927	High	High (Coltman *et al.*, 1999)
Human (*Homo sapiens*)	>8000 (Broman *et al.*, 1998)	1·9466	1·1778	0·4921	Low	Low (Anderson, 2006)
Bison (*Bison bison*)	358 (Schnabel *et al.*, 2003)	1·0979	0·8473	0·2576	High	Low (Nowak, 1999; Roden *et al.*, 2003)
Pig (*Sus scrofa*)	239 (Archibald *et al.*, 1995)	1·3538	0·9798	0·3206	Low	Low (Delgado *et al.*, 2008)
Baboon (*Papio papio*)	Crossover frequency (Burt *et al.*, 1991)	1·5850	1·9800	−0·2216	High	Low (Nowak, 1999; Maestripieri *et al.*, 2007)
Macaque (*Macaca mulatta*)	Crossover frequency (Burt *et al.*, 1991)	1·9800	2·0750	−0·0469	High	High (Bercovitch & Nurnberg, 1997; Widdig *et al.*, 2004)

[a] Corrected for variation in marker density (Chakravarti *et al.*, 1991) and undetected crossover events in unmapped regions (Hall & Willis, 2005).
[b] (Female recombination per chromosome-male recombination per chromosome)/average recombination per chromosome.
[c] High levels of sexual dimorphism is defined as difference in body weight >30%, or the presence of a sex-specific sexual ornament, such as horns or antlers. All estimates taken from (Nowak, 1999) unless otherwise noted.
[d] (Van Vuure, 2005)

of the non-placental clades, therefore these linkage maps were not included in this analysis.

For each of these species, I also searched the current literature for information about mating system in order to assign the degree of dimorphism and sperm competition. The effort and expense of linkage map analysis means that, until recently, most efforts were either confined to those animals of medical (mouse and macaque), commensal (dog and cat) or agricultural (sheep, pig and cattle) importance, and for these species I either searched for mating system information on the wild (macaque and mouse), ancestral (cattle) or feral (cat, dog, sheep and pig) populations. Based on mating system assessments (Woodall *et al.*, 1993; Nowak, 1999; Bradshaw & Cameron-Beaumont, 2000; Maestripieri *et al.*, 2007) and genetic paternity analyses (Bercovitch & Nurnberg, 1997; Coltman *et al.*, 1999; Nowak, 1999; Roden *et al.*, 2003; Widdig *et al.*, 2004; Anderson, 2006; Dean *et al.*, 2006; Delgado *et al.*, 2008), I determined whether lineages were likely to experience relatively high or low sperm competition. It is important to note that a certain degree of sperm competition is possible in virtually every type of mating system, given sufficient time and scrutiny; however, some mating systems are far more likely to contain higher levels of sperm competition than others. Specifically mate guarding in conjunction with pair-bonded and haremic mating systems will generally result in less sperm competition than more promiscuous behavioural ecologies.

Sexual dimorphism can be used to assess the degree to which pre-copulatory sexual selection defines the majority of variance in male mating success, which is predicted to correlate with heterochiasmy under diploid models (Trivers, 1988). Sexual dimorphism is present to some extent in all eutherians due to the physiological and anatomic requirements of internal gestation and lactation, therefore the strict presence or absence of sexual dimorphism alone is not helpful in this analysis. However, the degree of sexual dimorphism can be used as an indicator, therefore, I assessed whether species were strongly sexually dimorphic, exhibiting either large body size differences (>30%) or sexually selected traits such as horns in sheep (Nowak, 1999).

(i) *Recombination data*

Linkage mapping data must be corrected mathematically for variation in marker density and coverage in order to make comparable mapping efforts made with different panels of genetic markers. I first corrected the linkage mapping data for variance in marker density using the equations in Chakravarti *et al.* (1991), as greater marker density is more likely to recover double crossing over. I then corrected for undetected crossover events on unmapped portions according to Hall

& Willis (2005). Once these corrections were made, the sex-averaged cytological data from macaque and baboon matched well with the sex-averaged linkage maps of these and closely related species (Rogers *et al*., 2000, 2006; Cox *et al*., 2006), therefore recombination nodule information was included in further analyses. For all species, I only analysed the autosomal portions of the genome, as the sex chromosomes do not recombine along the majority of their length in the heterogametic sex.

The size and number of chromosomes can influence recombination (de Villena & Sapienza, 2001), therefore I corrected for this by calculating the number of crossover events per chromosome, defined here as homologous autosomes for females, males and averaged across sexes. I also calculated a measure of heterochiasmy, or the difference in recombination between the sexes, defined as $(\text{recombination}_{\text{female}} - \text{recombination}_{\text{male}})/\text{recombination}_{\text{average}}$.

(ii) *Comparative analysis*

Recombination hotspots change rapidly among related species (Winckler *et al*., 2005) and within populations of the same species (McVean *et al*., 2004; Hernandez *et al*., 2007), and a strong case can be made that the millions of years that separate the species in this study would obliterate any phylogenetic signal in recombination rate. Therefore I first examined the data regardless of shared ancestry by computing the average female, male and sex-averaged crossovers per chromosome for species with high and low sexual dimorphism, and high and low sperm competition. I also computed the average degree of heterochiasmy and assessed whether this was related to dimorphism or sperm competition. Significant difference was assessed with a two-tailed *t* test in each case.

Despite the potential for a quickly evolving trait to change more rapidly than can be mapped onto a sparse topology, it is important to test for phylogenetic signal as shared ancestry can create spurious associations. I therefore used the complete mitochondrial coding content of the species in this analysis to construct a neighbour-joining phylogeny in MEGA (Tamura *et al*., 2007), using the PAM Dayhoff Matrix of amino acid substitution, and assuming a difference rate of substitution among sites. Significance was assessed with 1000 bootstrap replicates. The branch lengths and topology from this tree were used to identify whether the continuous characters of female crossovers per chromosome and male crossovers per chromosome evolve in accordance with the phylogenetic history, or whether shared ancestry is not relevant to the distribution of the trait. This was done with the CoMET module (Lee *et al*., 2006) in Mesquite 2.6 (Maddison & Maddison, 2009). Both male and female chiasmata per chromosome fit best to the non-phylogenetic, equal matrix model, suggesting that phylogenetic history could be ignored for these characters.

Interestingly, the heterochiasmy score did follow a phylogenetic distribution (pure phylogenetic model). I therefore divided the heterochiasmy data into two categories, those taxa with higher recombination in females and those with higher recombination in males, and then tested the relationship between direction of heterochiasmy (male or female) and sexual dimorphism (high or low) and sperm competition (high or low) using the MESQUITE implementation of Pagel's maximum likelihood estimation of discrete character evolution (Pagel, 1994), using 1000 simulations to assess significance in each case.

3. Results

Recombination rates varied widely over the taxa assessed here, and the sex-averaged recombination rate per chromosome was lowest in dog (0·5371), where the long stretches of linkage disequilibrium (Karlsson & Lindblad-Toh, 2008) have proved ideal for haplotype mapping of breed-specific traits and diseases (Drogemuller *et al*., 2008; Wiik *et al*., 2008). Average recombination was highest in cat (2·8651).

Male and female recombination also varied widely, both within and between sexes. Heterochiasmy estimates show that while many species have higher recombination in females, cattle have slightly more recombination in males, and sheep, baboons and macaques have significantly male-biased recombination rates. Therefore, heterogamety cannot explain heterochiasmy as all eutherian mammals share a homologous system of male heterogamety, and the Haldane–Huxley Rule fails to explain 30–40 % of the data.

Assessing all data irrespective of phylogenetic history showed a strong overall relationship between heterochiasmy and sexual dimorphism ($P < 0{\cdot}005$, Fig. 1), though neither male nor female crossovers per chromosome differed significantly by themselves by sexual dimorphism class ($P > 0{\cdot}13$ in both cases). Sperm competition was not significantly associated with sex-specific recombination or heterochiasmy rates ($P > 0{\cdot}5$, Fig. 2).

The mitochondrial coding content was used to derive a phylogenetic reconstruction of these taxa, shown in Fig. 3. This phylogeny is topologically identical to recent mitochondrial (Arnason *et al*., 2008) and genomic (Prasad *et al*., 2008) phylogenies of the mammals. As all branches were supported by at values >95 % (based on 1000 bootstrap replicates), there was no need to collapse any branches into polytomies to indicate phylogenetic uncertainty.

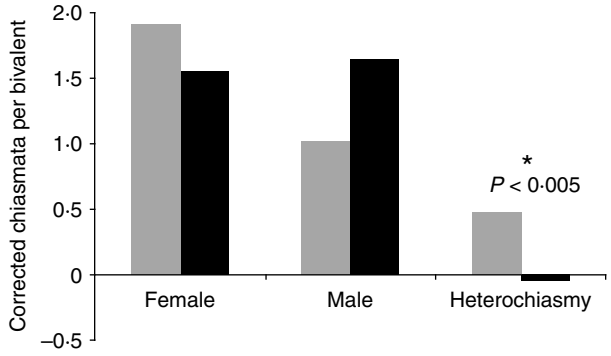

Fig. 1. Average number of crossovers per chromosome for taxa with high and low levels of sexual dimorphism. The comparison of overall heterochiasmy was significantly different (two-tailed *t* test). Grey bars indicate taxa with low sexual dimorphism, black bars indicate high sexual dimorphism.

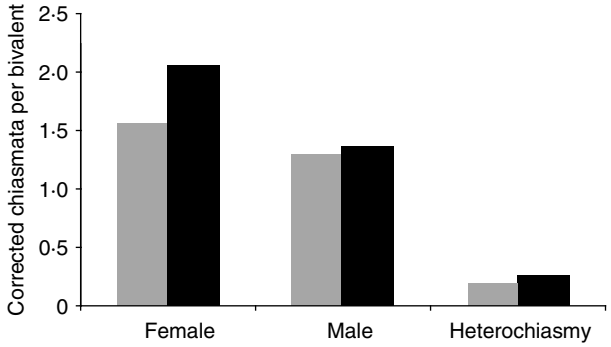

Fig. 2. Average number of crossovers per chromosome for taxa with low (grey) and high (black) levels of sperm competition, based on mating system and/or genetic parentage analysis. No comparison was significant (two-tailed *t* test).

I used this phylogeny to determine whether the recombination traits assessed here show an associated due to shared ancestry, or whether phylogenetic history can be safely ignored. Model testing (Lee *et al*., 2006) indicated that while sex-specific estimates of chiasmata per chromosome did not show a phylogenetic distribution greater than that expected by chance (for reasons discussed later), the direction of heterochiasmy did show some phylogenetic signal in the continuous data.

Due to this phylogenetic signal, I also performed assessments of the relationship between heterochiasmy and sexual dimorphism and sperm competition using maximum likelihood models of discrete character evolution (Pagel, 1994), incorporating the topology and branch-length estimates from the phylogeny in Fig. 1. For this analysis, I assigned the heterochiasmy data into two states; taxa either showed greater recombination in females or males. The results from this analysis are convergent with those counting species independently. Specifically, lineages with high

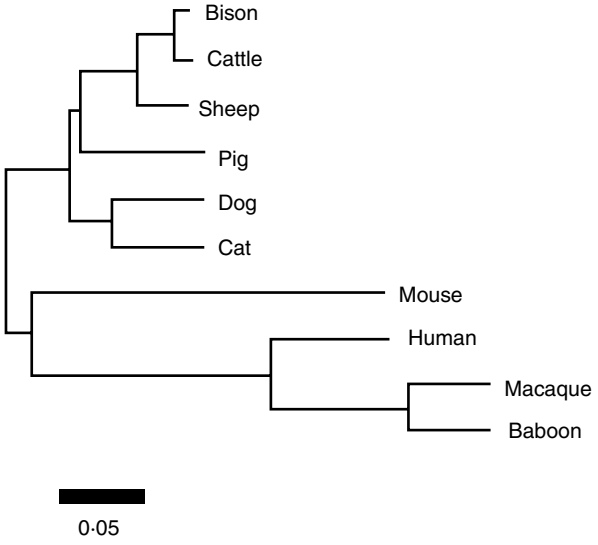

Fig. 3. Neighbour-joining reconstruction of the phylogenetic relationships among the taxa in this study based on the full protein-coding complement of the mitochondrial genome. In all cases, bootstrap values of significance exceeded 95%. Relative branch lengths are indicated, indicated by the scale bar.

levels of sexual dimorphism were more likely to experience greater relative recombination in males (1000 simulations, $P = 0{\cdot}02$). There was no relationship between heterochiasmy and sperm competition (1000 simulations, $P = 0{\cdot}45$).

4. Discussion

In this study, I tested two alternative hypotheses related to the difference in recombination between the sexes. Previous studies of this phenomenon (Burt *et al*., 1991; Lenormand & Dutheil, 2005) attempted to circumvent the paucity of data by pooling across diverse clades with vastly different fertilization and maternal provisioning characteristics. By employing information solely from the eutherian mammals, a clade defined by internal fertilization and placental gestation, this analysis controls for variation in female-specific fitness related to maternal–foetal conflict and internal fertilization, both of which can significantly influence the female fitness topology.

I found sex-specific recombination data for ten eutherian species distributed throughout the mammalian phylogeny, including eight molecular linkage maps that I corrected for marker density (Chakravarti *et al*., 1991) and genome coverage (Hall & Willis, 2005), and two visual cytological scans for recombination nodules. All of these assessments were corrected for variation in chromosome number, as this can affect rates of recombination (de Villena & Sapienza, 2001). The inclusion of these latter karyotypic studies is not expected to be problematic for two primary reasons. Most importantly, the majority of

https://doi.org/10.1017/S0016672309990255 Published online by Cambridge University Press

this study is concerned with relative within-species differences between the sexes, and while there may be differences in overall measurement between the different types of analysis used here, there is no reason to suspect a sex-specific bias, therefore the use of a heterochiasmy ratio would not be affected by the use of different types of recombination data. Additionally, the karyotypic studies showed similar numbers of chiasmata per chromosome to corrected sex-averaged linkage maps for these and closely related taxa (Rogers *et al*., 2000, 2006; Cox *et al*., 2006).

Interesting, female- and male-specific estimates of crossovers per chromosome did not show a phylogenetic signal in this study, which differs from work done on a similar set of taxa (Dumont & Payseur, 2007). This may reflect the difference in marker density in the data of Dumont & Payseur (2007) and that employed here. While these studies used overlapping taxon samples, the requirement of sex-specific maps in this analysis necessitated the inclusion of some maps constructed from far fewer markers (Archibald *et al*., 1995; Neff *et al*., 1999; Maddox *et al*., 2001). However, there is no reason to expect a sex-specific bias, and hence different linkage data is not a problem in this analysis as the majority of the study is concerned with the ratio between the sexes, rather than overall chiasmata numbers. This is supported by the recovery of a phylogenetic signal from the heterochiasmy ratio itself.

These data made it possible to test two alternative hypotheses proposed to explain the pattern of heterochiasmy. Lenormand & Dutheil (2005) showed mathematically that differences in sex-specific selection at the haploid level, presumably due to sperm competition, should predict recombination rate differences between the sexes, and I used information on mating system and genetic paternity analyses to infer the potential for sperm competition. However, there was no association in either the phylogenetically independent or controlled data between sperm competition and heterochiasmy. This may be because the haploid phase in males as well as females is functionally limited. While female haploid selection is very brief at best due to arrested meiosis, which is only completed at fertilization, the male haploid phase may also be limited due to limited gene expression in the spermatazoa.

An alternative hypothesis does not require haploid selection, as Trivers (1988) posited that sexual selection, in general, would select for reduced recombination in males in order to preserve the gene combinations that conferred high fitness in the father for his sons. I tested this by measuring the degree of heterochiasmy and relating it to the degree of sexual dimorphism, which is a measure of sexual selection and reproductive skew in males. Sexual dimorphism was significantly associated with heterogamety, both in the general analysis ($P<0{\cdot}005$) and once controlled for phylogeny ($P=0{\cdot}02$). However, the observed pattern did not fit the prediction, with male-biased recombination accompanying high levels of sexual dimorphism.

This raises the question of why males in systems with high levels of sex-specific fitness variance recombine more. The answer to this may reside in the prevalence in these mammals of male dispersal. Previous models assumed a static sex-specific fitness landscape (Trivers, 1988; Lenormand & Dutheil, 2005), assuming the factors that predict reproductive fitness in one generation would also hold in subsequent generations. However, the tendency of males to disperse more often and farther than females in eutherian mammals (Pusey & Packer, 1986; Gates & Larter, 1990; Thomson *et al*., 1992; Pal *et al*., 1998; Turner & Bateson, 2000; Truve & Lemel, 2003) could produce a shifting male fitness topology.

In the absence of mutual mate choice, the main determinants of the female-specific fitness landscape at the diploid stage are linked to gestation and postnatal care, and these factors do not, in theory, change greatly over the generations. By contrast, the male-specific fitness landscape involves elements of female-choice and male–male competition. With the added component of male dispersal, this diploid landscape has the potential to shift among generations, and the genetic combinations that ensured the reproductive success of the father may no longer predict the reproductive success of his sons due to differences in female preference and adaptations of rival males. The temporal shifting of the male-specific fitness topology would potentially select for increased recombination in males, as changing ecologies have been shown to select for increased recombination (Charlesworth, 1976; Otto & Nuismer, 2004). Therefore, differences in sex-specific selection pressures, in conjunction with changing fitness topologies for one sex alone, could very well exert a strong antagonistic evolutionary pressure to increase recombination in males and decrease it in females.

Additionally, theoretical studies have shown that higher rates of recombination may result from higher selection pressures (Felsenstein & Yokoyama, 1976; Lenormand & Otto, 2000; Otto & Barton, 2001) in order to reduce the problems associated with the loss of genetic variation due to linkage disequilibrium. This is in accordance with the analysis presented here, as recombination rate in males under higher sex-specific selection pressures, show higher rates of recombination.

The increase in male recombination for lineages under greater levels of sexual selection provides an interesting potential solution to the lek paradox. Continuous and powerful selection for male traits theoretically depletes the genetic variability for male

sexually selected traits, therefore reducing the heritability (Reynolds & Gross, 1990; Ritchie, 1996). Despite these predictions, systems with strong sexual selection often contain significant levels of heritability for male sexually selected traits (Norris, 1993; Kruuk *et al*., 2002), and female choice for male traits persists in these lineages, creating a paradox that has proved difficult to explain in a population genetic context. The process of recombination has been suggested to be mutagenic (Lercher & Hurst, 2002; Hellmann *et al*., 2003), creating the possibility that the accelerated recombination in males provides a continuously renewing source of genetic variation to counter the depletions resulting from sexual selection.

The generation of additional high-density sex-specific linkage maps in the future will make it possible to test this hypothesis in several ways. Specifically, the inclusion of maps from a greater variety of eutherians will facilitate the study of the evolutionary forces exerted by mating system, including polyandry and monogamy, and female and male philopatry on recombination rate differences between the sexes. Additionally, maps of greater marker density will make it possible to pinpoint regions of heterochiasmy, and relate these to reproductive fitness components via gene function and expression.

This work was supported in part by a grant from the Royal Society. The helpful comments of two anonymous reviewers are gratefully acknowledged.

References

Aguade, M. (1999). Positive selection drives the evolution of the *Acp29AB* accessory gland protein in *Drosophila*. *Genetics* **152**, 543–551.

Åkesson, M., Hansson, B., Hasselquist, D. & Bensch, S. (2007). Linkage mapping of AFLP markers in a wild population of great reed warblers: importance of heterozygosity and number of genotyped individuals. *Molecular Ecology* **16**, 2189–2202.

Anderson, K. G. (2006). How well does paternity confidence match actual paternity? *Current Anthropology* **47**, 513–520.

Archibald, A. L., Haley, C. S., Brown, J. F., Couperwhite, S., McQueen, H. A., Nicholson, D., Coppieters, W., Vandeweghe, A., Stratil, A., Wintero, A. K., Fredholm, M., Larsen, N. J., Nielsen, V. H., Milan, D., Woloszyn, N., Robic, A., Dalens, M., Riquet, J., Gellin, J., Caritez, J. C., Burgaud, G., Ollivier, L., Bidanel, J. P., Vaiman, M., Renard, C., Geldermann, H., Davoli, R., Ruyter, D., Verstege, E. J. M., Groenen, M. A. M., Davies, W., Hoyheim, B., Keiserud, A., Andersson, L., Ellegren, H., Johansson, M., Marklund, L., Miller, J. R., Dear, D. V. A., Signer, E., Jeffreys, A. J., Moran, C., Letissier, P., Muladno, Rothschild, M. F., Tuggle, C. K., Vaske, D., Helm, J., Liu, H. C., Rahman, A., Yu, T. P., Larson, R. G. & Schmitz, C. B. (1995). The PigMap consortium linkage map of the pig (*Sus Scrofa*). *Mammalian Genome* **6**, 157–175.

Arnason, U., Adegoke, J. A., Gullberg, A., Harley, E. H., Janke, A. & Kullberg, M. (2008). Mitogenomic relationships of placental mammals and molecular estimates of their divergences. *Gene* **421**, 37–51.

Begun, D. J. & Lindfors, H. A. (2005). Rapid Evolution of Genomic Acp complement in the melanogaster subgroup of *Drosophila*. *Molecular Biology and Evolution* **22**, 2010–2021.

Bercovitch, F. B. & Nurnberg, P. (1997). Genetic determination of paternity and variation in male reproductive success in two populations of rhesus macaques. *Electrophoresis* **18**, 1701–1705.

Bradshaw, J. & Cameron-Beaumont, C. (2000). The signalling repertoire of the domestic cat and its undomesticated relatives. In *The Domestic Cat: The Biology of its Behaviour*, pp. 67–94. Cambridge: Cambridge University Press.

Broman, K. W., Murray, J. C., Sheffield, V. C., White, R. L. & Weber, J. L. (1998). Comprehensive human genetic maps: Individual and sex-specific variation in recombination. *American Journal of Human Genetics* **63**, 861–869.

Burt, A., Bell, G. & Harvey, P. H. (1991). Sex differences in recombination. *Journal of Evolutionary Biology* **4**, 259–277.

Chakravarti, A., Lasher, L. K. & Reefer, J. E. (1991). A maximum likleihood method for estimating genome length using genetic linkage data. *Genetics* **128**, 175–182.

Charlesworth, B. (1976). Recombination modification in a fluctuatung evironment. *Genetics* **83**, 181–195.

Coltman, D. W., Bancroft, D. R., Robertson, A., Smith, J. A., Clutton-Brock, T. H. & Pemberton, J. M. (1999). Male reproductive success in a promiscuous mammal: behvaioural estimates compared with genetic paternity. *Molecular Ecology* **8**, 1199–1209.

Cox, L. A., Mahaney, M. C., VandeBerg, J. L. & Rogers, J. (2006). A second-generation genetic linkage map of the baboon (*Papio hamadryas*) genome. *Genomics* **88**, 274–281.

de Villena, F. P. M. & Sapienza, C. (2001). Recombination is proportional to the number of chromosome arms in mammals. *Mammalian Genome* **12**, 318–322.

Dean, M. D., Ardlie, G. & Nachman, M. W. (2006). The frequency of multiple paternity suggests sperm competition is common in house mouse. *Molecular Ecology* **15**, 4141–4151.

Delgado, R., Fernandez-Llario, P., Azevedo, M., Beja-Pereira, A. & Santos, P. (2008). Paternity assessment in free-ranging wild boar (*Sus scrofa*) – Are littermates full-sibs? *Mammalian Biology* **73**, 169–176.

Drogemuller, C., Karlsson, E. K., Hytonen, M. K., Perloski, M., Dolf, G., Sainio, K., Lohi, H., Lindblad-Toh, K. & Leeb, T. (2008). A mutation in hairless dogs implicates FOXI3 in ectodermal development. *Science* **321**, 1462–1462.

Dumont, B. L. & Payseur, B. A. (2007). Evolution of the genomic rate of recombination in mammals. *Evolution* **62**, 276–294.

Felsenstein, J. & Yokoyama, S. (1976). Evolutionary advantage of recombination. II. Individual selection for recombination. *Genetics* **83**, 845–859.

Gates, C. C. & Larter, N. C. (1990). Growth and dispersal of an erupting large herbivore population in northern Canada: The Mackenzie wood bison. *Arctic* **43**, 231–238.

Haldane, J. (1922). Sex ratio and unisexual sterility in hybrid animals. *Journal of Genetics* **12**, 101–109.

Hall, M. C. & Willis, J. H. (2005). Transmission ratio distortion in intraspecific hybrids of Mimulus gutatus: implications for genomic divergence. *Genetics* **170**, 375–386.

Hansson, B., Åkesson, M., Slate, J. & Pemberton, J. M. (2005). Linkage mapping reveals sex-dimorphic map distances in a passerine bird. *Proceedings of the Royal Society B-Biological Sciences* **272**, 2289–2298.

Hellmann, I., Ebersberger, I., Ptak, S. E., Pääbo, S. & Przeworski, M. (2003). A neutral explanation for the correlation of diversity with recombination rates in humans. *American Journal of Human Genetics* **72**, 1527–1535.

Hernandez, R. D., Hubisz, M. J., Wheeler, D. A., Smith, D. G., Ferguson, B., Rogers, J., Nazareth, L., Indap, A., Bourquin, T., McPherson, J., Muzny, D., Gibbs, R., Nielsen, R. & Bustamante, C. D. (2007). Demographic histories and patterns of linkage disequilibrium in Chinese and Indian rhesus macaques. *Science* **316**, 240–243.

Huxley, J. (1928). Sexual differences in linkage in *Gammarus chevruxi*. *Journal of Genetics* **20**, 145–156.

Ihara, N., Takasuga, A., Mizoshita, K., Takeda, H., Sugimoto, M., Mizoguchi, Y., Hirano, T., Itoh, T., Watanabe, T., Reed, K. M., Snelling, W. M., Kappes, S. M., Beattie, C. W., Bennett, G. L. & Sugimoto, Y. (2004). A comprehensive genetic map of the cattle genome based on 3802 microsatellites. *Genome Research* **14**, 1987–1998.

Karlsson, E. K. & Lindblad-Toh, K. (2008). Leader of the pack: gene mapping in dogs and other model organisms. *Nature Reviews Genetics* **9**, 713–725.

Kruuk, L. E. B., Slate, J., Pemberton, J. M., Brotherstone, S., Guinness, F. & Clutton-Brock, T. (2002). Antler size in red deer: Heritability and selection but no evolution. *Evolution* **56**, 1683–1695.

Lee, S., Blay, S., Mooers, A. O., Singh, A. & Oakley, T. H. (2006). CoMET: A Mesquite package for comparing models of continuous character evolution on phylogenies. *Evolutionary Bioinformatics Online* **2**, 193–196.

Lenormand, T. & Dutheil, J. (2005). Recombination difference between sexes: A role for haploid selection. *Plos Biology* **3**, 396–403.

Lenormand, T. & Otto, S. P. (2000). The evolution of recombination in a heterogeneous environment. *Genetics* **156**, 423–428.

Lercher, M. J., Hurst, L. D. (2002). Human SNP variability and mutation rate are higher in regions of high recombination. *Trends in Genetics* **18**, 337–340.

Maddison, W. P. & Maddison, D. R. (2009). Mesquite: a modular system for evolutionary analysis. 2.6 http://mesquiteproject.org ed.

Maddox, J. F., Davies, K. P., Crawford, A. M., Hulme, D. J., Vaiman, D., Cribiu, E. P., Freking, B. A., Beh, K. J., Cockett, N. E., Kang, N., Riffkin, C. D., Drinkwater, R., Moore, S. S., Dodds, K. G., Lumsden, J. M., van Stijn, T. C., Phua, S. H., Adelson, D. L., Burkin, H. R., Broom, J. E., Buitkamp, J., Cambridge, L., Cushwa, W. T., Gerard, E., Galloway, S. M., Harrison, B., Hawken, R. J., Hiendleder, S., Henry, H. M., Medrano, J. F., Paterson, K. A., Schibler, L., Stone, R. T. & van Hest, B. (2001). An enhanced linkage map of the sheep genome comprising more than 1000 loci. *Genome Research* **11**, 1275–1289.

Maestripieri, D., Mayhew, J., Carlson, C. L., Hoffman, C. L. & Radtke, J. M. (2007). One-male harems and female social dynamics in Guinea baboons. *Folia Primatologica* **78**, 56–68.

McVean, A. T., Myers, S. R., Hunt, S., Deloukas, P., Bentley, D. R. & Donnelly, P. (2004). The fine-scale structure of recombination rate variation in the human genome. *Science* **304**, 581–584.

Meiklejohn, C. D., Parsch, J., Ranz, J. M. & Hartl, D. L. (2003). Rapid evolution of male-biased gene expression in *Drosophila*. *Proceedings of the National Academy of Sciences, USA* **100**, 9894–9899.

Menotti-Raymond, M., David, V. A., Schaffer, A. A., Tomlin, J. F., Eizirik, E., Phillip, C., Wells, D., Pontius, J. U., Hannah, S. S. & O'Brien, S. J. (2009). An autosomal genetic linkage map of the domestic cat, *Felis sylvestris catus*. *Genomics* **93**, 305–313.

Neff, M. W., Broman, K. W., Mellersh, C. S., Ray, K., Acland, G. M., Aguirre, G. D., Ziegle, J. S., Ostrander, E. A. & Rine, J. (1999). A second-generation genetic linkage map of the domestic dog, *Canis familiaris*. *Genetics* **151**, 803–820.

Norris, K. (1993). Heritable variation in a plumage indicator of variability in male great tits - *Parus major*. *Nature* **362**, 537–539.

Nowak, R. M. (1999). *Walker's Mammals of the World*. Baltimore, MD: Johns Hopkins University Press.

Otto, S. P. & Barton, N. H. (2001). Selection for recombination in small populations. *Evolution* **55**, 1921–1931.

Otto, S. P. & Nuismer, S. L. (2004). Species interactions and the evolution of sex. *Science* **304**, 1018–1020.

Pagel, M. (1994). Detecting correlated evolution on phylogenies, a general method for the comparative analysis of discrete characters. *Proceedings of the Royal Society of London, B* **255**, 37–45.

Pal, S. K., Ghosh, B. & Roy, S. (1998). Dispersal behaviour of free-ranging dogs (*Canis familiaris*) in relation to age, sex, season, and dispersal distance. *Applied Animal Behaviour Science* **61**, 123–132.

Perry, P. E. & Jones, G. H. (1974). Male and female meiosis in grasshoppers. I. Stethophyma grossum. *Chromosoma* **47**, 227–236.

Prasad, A. B., Allard, M. W., Green, E. D. & Program, N. C. S. (2008). Confirming the phylogeny of mammals by use of large comparative sequence data sets. *Molecular Biology and Evolution* **25**, 1795–1808.

Pusey, A. E. & Packer, C. (1986). *Dispersal and Philopatry*. Chicago: Primate societies. University of Chicago Press.

Reynolds, J. D. & Gross, M. R. (1990). Costs and benefits of female mate choice - is there a lek paradox. *American Naturalist* **136**, 230–243.

Ritchie, M. G. (1996). The shape of female mating preferences. *Proceedings of the National Academy of Sciences, USA* **93**, 14628–14631.

Roden, C., Vervaecke, H., Mommens, G. & van Elsacker, L. (2003). Reproductive success of bison bulls (*Bison bison*) in semi-natural conditions. *Animal Reproductive Science* **79**, 33–43.

Rogers, J., Mahaney, M. C., Witte, S. M., Nair, S., Newman, D., Wedel, S., Rodriguez, L. A., Rice, K. S., Slifer, S. H., Perelygin, A., Slifer, M., Palladino-Negro, P., Newman, T., Chambers, K., Joslyn, G., Parry, P. & Morin, P. A. (2000). A genetic linkage map of the baboon (*Papio hamadryas*) genome based on human microsatellite polymorphisms. *Genomics* **67**, 237–247.

Rogers, J., Garcia, R., Shelledy, W., Kaplan, J., Arya, A., Johnson, Z., Bergstrom, M., Novakowski, L., Nair, P., Vinson, A., Newman, D., Heckman, G. & Cameron, J. (2006). An initial genetic linkage map of the rhesus macaque (*Macaca mulatta*) genome using human microsatellite loci. *Genomics* **87**, 30–38.

Samollow, P. B., Kammerer, C. M., Mahaney, S. M., Schneider, J. L., Westenberger, S. J., VendeBerg, J. L. & Robinson, E. S. (2004). First-generation linkage map of the gray, short-tailed opossum, *Monodelphis domestica*,

reveals genome-wide reduction in female recombination rates. *Genetics* **166**, 307–329.
Schnabel, R. D., Taylor, J. F. & Derrm, J. N. (2003). Development of a linkage map and QTL scan for growth traits in North American bison. *Cytogenetic and Genome Research* **102**, 59–64.
Shifman, S., Bell, J. T., Copley, R. R., Taylor, M. S., Williams, R. W., Mott, R. & Flint, J. (2006). A high-resolution single nucleotide polymorphism genetic map of the mouse genome. *PLoS Biology* **4**, 2227–2237.
Tamura, K., Dudley, J., Nei, M. & Kumar, S. (2007). MEGA4: Molecular Evolutionary Genetics Analysis (MEGA) software version 4.0. *Molecular Biology and Evolution* **24**, 1596–1599.
Thomson, P. C., Rose, K. & Kok, N. E. (1992). The behavioural ecology of dingoes in northwestern Australia. VI. Temporary extraterritorial movements and dispersal. *Wildlife Research* **19**, 585–595.
Trivers, R. (1988). Sex differences in rates of recombination and sexual selection. In *The Evolution of Sex* (ed. R. Michod & B. R. Levin), pp. 270–286. Sunderland, MA: Sinauer Press.
Truve, J. & Lemel, J. (2003). Timing and distance of natal dispersal for wild boar *Sus scrofa* in Sweden. *Wildlife Biology* **9**, 51–57.
Turner, D. C. & Bateson, P. (2000). *The domestic cat: the biology of its behaviour*. Cambridge: Cambridge University Press.
Van Vuure, C. (2005). *Retracing the aurochs: History, morphology and ecology of an extinct wild ox*. Sofia: Pensoft.
Wedell, N. & Tregenza, T. (1990). Successful fathers sire successful sons. *Evolution* **53**, 620–625.
Widdig, A., Bercovitch, F. B., Streich, W. J., Sauermann, U., Nurnberg, P. & Krawczak, M. (2004). A longitudinal analysis of reproductive skew in male rhesus macaques. *Proceedings of the Royal Society, B* **271**, 819–826.
Wiik, A. C., Wade, C., Biagi, T., Ropstad, E. O., Bjerkas, E., Lindblad-Toh, K. & Lingaas, F. (2008). A deletion in nephronophthisis 4 (NPHP4) is associated with recessive cone-rod dystrophy in standard wire-haired dachshund. *Genome Research* **18**, 1415–1421.
Winckler, W., Myers, S. R., Richter, D. J., Onofrio, R. C., McDonald, G. J., Bontrop, R. E., McVean, G. A. T., Gabriel, S. B., Reich, D., Donnelly, P. & Altshuler, D. (2005). Comparison of fine-scale recombination rates in humans and chimpanzees. *Science* **308**, 107–111.
Woodall, P. F., Pavlo, P. & Tolley, L. K. (1993). Comparative dimensions of testes, epididymides and spermatozoa of Australian dingoes (*Canis familiaris dingo*) and domestic dogs (*Canis familiaris familiaris*): Some effects of domestication. *Australian Journal of Zoology* **41**, 133–140.
Zenger, K. R., McKenzie, L. M. & Coop, D. W. (2002). The first comprehensive linkage map of a marsupial: The tammar wallaby (*Macropus eugenii*). *Genetics* **162**, 321–330.
Zhang, Z. & Parsch, J. (2005). Positive correlation between evolutionary rate and recombination rate in *Drosophila* genes with male-biased expression. *Molecular Biology and Evolution* **22**, 1945–1947.