Constraint and divergence in the evolution of male and female recombination rates in fishes

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ABSTRACT

Recombination is a fundamental feature of sexual reproduction across eukaryotes, yet recombination rates are highly variable both within and between species. In particular, sex differences in recombination rate between males and females (heterochiasmy) is more often the rule than the exception, but despite its prevalence the ultimate causes of global patterns of heterochiasmy remain unclear. Here, we assemble a comprehensive dataset of sex-specific recombination rate estimates for 61 fish species, and combine this with information on sex determination, fertilisation mode and sexual dimorphism to test competing theories for the causes and evolution of heterochiasmy. We find that sex differences in recombination rate are evolutionary labile, with frequent shifts in the direction and magnitude of heterochiasmy. This rapid turnover does not appear to be driven by simple neutral processes and is inconsistent with non-adaptive explanations for heterochiasmy, including biological sex differences in meiosis. However, while patterns of heterochiasmy across the phylogeny indicate a potential role for adaptive processes, we are unable to directly link variation in heterochiasmy with proxies for sexual selection or sexual conflict across species, indicating that these effects – if present – are either subtle or complex. Finally, we show evidence for correlated rates of recombination rate evolution between males and females, indicating the potential for genetic constraints and sexual conflict over the recombination landscape.

INTRODUCTION

Differences in recombination rate between the sexes (heterochiasmy) were observed in early cytological studies as different chiasma frequencies in female and male meiotic cells (Burt et al. 1991). The increasing trend of estimating sex-specific recombination maps has confirmed that heterochiasmy is more often the rule than the exception, with differences typically observed in both total map length (total number of crossovers) and hotspot location for females and males (Lenormand and Dutheil 2005; Mank 2009; Brandvain and Coop 2012; Stapley et al. 2017; Brick et al. 2018; Sardell and Kirkpatrick 2020). However, despite its prevalence the ultimate causes of heterochiasmy remain unclear, and numerous non-exclusive hypotheses have been put forward.

One compelling non-adaptive argument stems from general trends observed in heterochiasmy. Linkage map length tends to be longer in females (Lenormand 2003; Lenormand and Dutheil 2005; Brandvain and Coop 2012; Sardell and Kirkpatrick 2020) and recombination hotspots tend to be located near the ends of chromosomes in males, but towards the interior portions in females (Broman et al. 1998; Brandvain and Coop 2012; Sardell and Kirkpatrick 2020). This implies that sex differences in recombination rate may simply be the result of biological differences between females and males in meiosis. For instance, the long-term arrest of vertebrate ova in the penultimate stages of meiosis may produce fundamental differences between male and female rates of double-strand breaks or other components of the recombination machinery (Gruhn et al. 2013; Gruhn et al. 2016). If biological differences in male and female meiosis are the main driver of heterochiasmy, we would expect minimal variation in rates across species with similar developmental programmes. Alternatively, other non-adaptive explanations suggest that differences in recombination rate are

the result of genetic drift (Nei 1969; Burt et al. 1991), in which case divergence in rates would be expected to accumulate gradually across species in a phylogeny, and female and male recombination might evolve largely independently depending on the degree of intersexual correlation.

On the other hand, numerous adaptive hypotheses have also been proposed. Based on observations in some species that the heterogametic sex is achiasmate (i.e. completely lacking any recombination), it has been suggested that recombination may be adaptively suppressed to varying degrees across the entire genome in the heterogametic sex, in order to prevent X-Y or Z-W crossing over (i.e. the Haldane-Huxley hypothesis) (Haldane 1922; Huxley 1928). This prediction holds across nearly all of the independent origins of achiasmy (Brandvain and Coop 2012; Vicoso and Bachtrog 2015), with very few exceptions (Dufresnes et al. 2020). However, achiasmy is an extreme and relatively rare form of heterochiasmy, and in the vast majority of species both sexes recombine to some degree. If the Haldane-Huxley hypothesis is true outside of achiasmate species, we expect relatively shorter male map lengths in male heterogametic species, and in females for female heterogametic species. Importantly, testing this hypothesis would ideally focus on relatively young sex chromosome systems, as heterochiasmy patterns might no longer be under strong selection in heteromorphic sex chromosome systems, after the Y or W has experienced significant levels of degeneration and no longer maintains significant stretches of homology to the X or Z chromosome.

Finally, Trivers (1988) proposed that sexual selection acting primarily on males would result in selection against male recombination in order to preserve favourable genetic combinations. Although Trivers' hypothesis has easily testable predictions, namely that relative map length will be shorter in males in species with pronounced sexual selection, it has not received broad empirical or theoretical support (Mank 2009), and it has been argued that the sex under more intense sexual selection would instead produce selection for increased recombination (Felsenstein 1988; Maynard 1988).

However, several other hypotheses focus on the role of sexual conflict, rather than sexual selection, in heterochiasmy. Lenormand (2003) and Lenormand and Dutheil (2005) predicted that the pattern of heterochiasmy is a function of the strength of haploid selection for female and male gametes. The potential for haploid selection on gametes is relatively limited in vertebrate females, which suggests that if haploid selection is driving heterochiasmy, it would primarily affect males. As a result, we expect that males should undergo less frequent recombination and that male recombination rates might evolve more rapidly across evolutionary time. Additional hypotheses focusing on sexual

conflict (Sardell and Kirkpatrick 2020), where recombination is suppressed in the sex subject to stronger selection, and meiotic conflict (Brandvain and Coop 2012), where meiotic drive during meiosis I or II favours increased or decreased recombination in females respectively, have also been proposed.

Testing these alternative hypotheses requires several key things. First, sex-specific recombination maps are needed, ideally across a broad sample of species that exhibit variation in recombination rate to allow for a comparative approach. Given the predicted association between heterochiasmy and sex chromosomes, it would also be beneficial if the focal clade showed rapid turn-over in sex determination, particularly a prevalence of less degenerate, homomorphic, sex chromosomes. Finally, species-specific measures of sexual selection, both in the diploid and haploid state, would also be necessary.

As a result of their large clutch size, relatively compact genomes and importance in the food chain, there has been a rapid increase in recent years in the number of high-quality linkage maps for teleost fish. Importantly, many of these maps have been calculated separately for females and males. As a group, fish are also exceptional for their diversity of sex determination mechanisms (Mank et al. 2006). Furthermore, many species have detailed spawning and morphological accounts (Breder and Rosen 1966; Froese and Pauly 2019) that make it possible to estimate the degree of sexual dimorphism and the potential for haploid selection in males. Here, we use sex-specific recombination maps for 61 teleost species, coupled with information on sex determination, fertilisation mode and sexual dimorphism, to test longstanding theories regarding the causes of global patterns of heterochiasmy. Importantly, we use a robust comparative phylogenetic framework to directly estimate male and female-specific evolutionary rates and to correct for statistical issues associated with shared ancestry.

METHODS

Linkage map data

We collected information on the total length of male and female linkage maps (cM), a proxy for recombination rate (Winckler et al. 2005; Dawson et al. 2007; Stapley et al. 2017), using data available in the primary literature for 61 species of teleost fish. For many species, there are multiple linkage mapping studies, and so to maximise data quality we chose the study for each species with

the greatest number of markers for subsequent analyses (Supplemental Table 1 for data and references). Studies varied by the number of sires and whether they were used to generate full-sib and/or half-sib families, the number of dams, the number of sons and daughters (see references in Table 1 for details).

Estimating sex differences in recombination rate

Linkage map length is widely used as a metric of overall recombination rate (Winckler et al. 2005; Dawson et al. 2007; Stapley et al. 2017). However, variation in non-biological aspects of study design (i.e. sample size, marker number) can influence the reliability of map length as a proxy for recombination, which must be considered. To investigate this in our data, we used multiple regression ('lm') to test the relationships between total map length estimates and reported values for sample sizes and marker in R v3.3.2 (Figure S1).

First, we found that map length is not correlated with the number of samples (number of progeny) used to construct the linkage map (Table S2). Therefore, for the analyses presented here we used the full dataset of 61 species. However, to ensure rigour, we repeated all of our analyses using a subset of the data, including only samples with 100 samples or more. These results are qualitatively identical to the main results and are shown in Table S3, S4, S5 and S6.

Second, we found that the number of markers is strongly correlated with linkage map length across studies (Table S2), and frequently varies between males and females even within the same study (Table S1). Therefore, to control for the effect of marker number on map length estimates, we recalculated map length for males (cM_M) and females (cM_F) as the residuals of the relationship between $\log_{10}$-transformed map lengths and marker number only (Figure S1A; $y = 2.656 + 0.194*x$, $P < 0.001$, $R^2 = 0.310$), and used this as our measure of sex-specific recombination rate in subsequent analyses.

However, in many species of fish, the recombination landscape differs between males and females. Recombination in males is often confined to the telomeres whereas recombination is more homogenous through the genome in females (Sardell and Kirkpatrick 2020). If marker number is too low, telomeric recombination events might have a lower likelihood of detection resulting in false inferences of heterochiasmy or biases in the estimated magnitude of sex differences. We took the 15 species in our dataset that are known to exhibit male-biased recombination at the telomeres (Table S1). The number of male markers in these studies ranges from 141 to 5,198. If marker density does bias estimates of heterochiasmy, we expect increasing parity between male and female

recombination rates with an increasing number of male markers. Alternatively, if marker number is sufficient, then we expect no significant relationship. Using Pearson's correlation, we found no significant relationship between male marker number and sex differences in recombination rate across these 15 studies (Figure S2A, r = 0.015, P = 0.959). This pattern holds when all species are included in the analysis (Figure S2B, r = 0.057, P = 0.662). Therefore, we are confident that we have sufficient power to accurately quantify heterochiasmy.

Finally, it is possible that variation in chromosome number or ploidy level could bias our estimates of sex-biased recombination rate. The majority of species in our dataset have a diploid chromosome number between 44 and 52 chromosomes. We repeated all of our analyses using only these species. Our results are qualitatively identical and shown in Table S3, S4, S5 and S6.

Phenotypic trait data

For each species, we collated published data on sex determination, fertilisation mode (external or internal) and presence/absence of sexual dimorphism (Supplemental Table 1 for data and references). Given the high rate of sex chromosome turnover among fish (Woram et al. 2003; Kitano and Peichel 2012; Darolti et al. 2019), it is reasonable to assume that each sex chromosome in our study has evolved independently. For instance, several of the species in our study are salmonids (Salmonidae), which are all male heterogametic, but with XY chromosomes evolving independently from different sets of autosomes (Woram et al. 2003). Therefore, it is likely that the distribution of sex determination mechanisms across our sample species represents the product of independent evolutionary events in most if not all cases. However, even if this is not the case, all of our analyses are phylogenetically controlled, which accounts for statistical non-independence among species' trait values caused by shared ancestry.

We scored presence/absence of sexual dimorphism following Mank et al. (2006) and Mank (2007). We recorded several well-established dimorphisms in fish from field guides and species accounts, including elongate or modified fins (Harrington 1997; Marcus and McCune 1999; Kuwamura et al. 2000), breeding tubercles (Kortet 2004; Kortet et al. 2004), sexual dichromatism (Reimchen 1989; Houde and Endler 1990; Stott and Poulin 1996; Amundsen and Forsgren 2001), electric mating calls (Curtis and Stoddard 2003) and size differences (van den Berghe and Gross 1989; Järvi 1990). Regarding sexual dichromatism, we did not tally counts of a particular ornament type exhibited by males, rather we recorded from published species accounts whether there were colour differences between the sexes. Due to the major differences in sensory ecology among our species, we did not

attempt to rank different types of dimorphism, but simply recorded whether species did or did not display any of these dimorphic traits.

Phylogeny

Our analyses are based on the taxonomy and phylogenies of Rabosky et al. (2018). We matched species in our dataset to tips in the full 'all-taxon assembled' phylogenies (n = 100 trees), which we pruned to leave only the species represented in our dataset. We then generated a maximum clade credibility tree based on these 100 posterior trees using the 'maxCladeCred' function in the R package 'phangorn' v2.3.1 (Schliep 2011). We note that 59 out of the 61 species (97%) in our dataset are represented by genetic data in the Rabosky phylogenies, with all 61 included in the 'all-taxon assembled' phylogenies.

Trait evolution

We estimated rates of recombination rate evolution in males (σ^2_M) and females (σ^2_F) separately using the 'variable rates' model of trait evolution (Venditti et al. 2011), implemented in the software BayesTraits v3 (http://www.evolution.rdg.ac.uk/). We used default priors and a single Markov chain Monte Carlo (MCMC) run for 20 million iterations in each case. From each chain, we sampled parameters every 1,000 iterations after removing the first 50% of each run as burn-in and checking runs for satisfactory convergence. Per-branch evolutionary rate estimates were calculated as the mean rate for each branch across all post burn-in samples.

Statistical analyses of recombination rate and phenotypic traits

To assess the relationship between sex differences in recombination rate ($cM_{M:F} = cM_M - cM_F$) and phenotypic traits (sex determination, fertilisation mode and presence/absence of sexual dimorphism), we used standard phylogenetic generalised least-squares (PGLS) regression with an optimised lambda error structure, implemented in the R package 'phylolm' v2.5 (Tung Ho and Ané 2014). PGLS models flexibly adjust regression models for the level of phylogenetic signal within the data. We used the same approach to test for relationships between predictors and sex differences in the rate of recombination rate evolution. For these analyses, we used tip (i.e. terminal branch) evolutionary rate estimates for each species ($\sigma^2_{TipM:F} = \sigma^2_{TipM} - \sigma^2_{TipF}$), which were box-cox transformed to improve normality.

Partial-R^2 values for predictor variables in PGLS models were calculated using the 'R2.lik' function in the R package 'rr2' v1.0.2 (Ives 2018) by comparing the full model including the predictor variable to

a reduced model without the predictor variable, while holding phylogenetic effects (i.e. lambda) constant.

Statistical analyses of evolutionary rate

To assess the relationship between tip rates of recombination rate evolution in males (σ^2_{TipM}) and females (σ^2_{TipF}) across species, we used phylogenetic reduced major axis (RMA) regression with optimised lambda, implemented using the function 'phyl.RMA' in the R package 'phytools' v0.6.44 (Revell 2012).

RESULTS

We compiled a comprehensive dataset of recombination rate estimates across teleost fish species to examine male and female recombination rate evolution, and to test adaptive and non-adaptive theories for the causes of heterochiasmy. In total, we identified 61 species, covering 32 families and 13 orders, for which data on linkage map length (a proxy for recombination rate) are available for both sexes. To our knowledge, this is the most comprehensive dataset to date of sex differences in recombination rate across animal species, spanning ~200 million years of evolution (Figure 1).

Recombination rate is typically female-biased and highly correlated between the sexes.

Across species, we found that females typically have a higher recombination rate (cM) than males (Figure 1; H_0: $cM_{M:F} = 0$, $P = 0.030$). Specifically, 42 of the species in our study have female-biased recombination whereas males only recombine more frequently in 19 species. Furthermore, just over half of the species with male-biased recombination are restricted to one monophyletic clade comprising the orders Anabantiformes, Cyprinodontiformes, Perciformes and Pleuronectiformes. Phylogenetic RMA regression revealed that male (cM_M) and female (cM_F) recombination rates were significantly positively correlated across species (Figure 2; $R^2 = 0.680$).

Sex biases in recombination rate evolve rapidly.

We mapped heterochiasmy (i.e. the difference in total map length between males and females) across the fish phylogeny. We found that sex biases in recombination rate ($cM_{M:F}$) are evolutionarily labile, with frequent shifts in the direction and magnitude of heterochiasmy among lineages (Figure 1). Importantly, we found that this variation was only marginally related to phylogenetic history, as the maximum-likelihood phylogenetic signal estimate for values of sex bias in recombination rate

was low, with confidence intervals that included zero (Pagel's λ = 0.196, 95% CI = 0.00 - 0.700). In other words, closely related species are no more likely to show similar levels of sex bias than more distantly related lineages.

To explore the underlying drivers of this pattern, we estimated rates of male (σ^2_M) and female (σ^2_F) recombination rate evolution across the phylogeny (Figure 3A). We found that evolutionary rates varied substantially among lineages, but shift in a correlated manner between the sexes within species (Figure 3A). Specifically, tip rates of recombination rate evolution (σ^2_{Tip}) are strongly positively correlated between males and females (Figure 3B; $R^2 = 0.828$). Furthermore, we found no evidence that evolutionary rates were consistently higher for one sex than the other (Figure 3C; H_0: $\sigma^2_{TipM:F} = 0$, $P = 0.478$). This indicates that variation in heterochiasmy is not the result of consistent evolutionary shifts in one sex (e.g. females) but emerges through sex-specific shifts in specific lineages. Indeed, we do observe certain lineages with pronounced female- or male-biased rates of recombination rate evolution (Figure 3C).

Testing adaptive hypotheses for the evolution of heterochiasmy

We used PGLS regression to test the role of lineage-specific traits in driving the evolution of heterochiasmy. First, we tested the Haldane-Huxley hypothesis (Haldane 1922; Huxley 1928) that recombination is suppressed across the entire genome in order to prevent crossing over between the sex chromosomes in the heterogametic sex. We found no strong support for this hypothesis, as there was no significant correlation between sex determination mechanism (XY, ZW, absence of sex chromosomes) and sex-bias in recombination rate ($cM_{M:F}$) across species ($R^2 = 0.016$; Table 1). Furthermore, by contrasting male and female recombination rates across XY and ZW systems, we found that sex chromosome system explains a negligible proportion of variation in heterochiasmy ($R^2 = 0.004$; $P = 0.731$).

Next, we tested hypotheses regarding the role of sexual selection and sexual conflict in the evolution of recombination rate. The Trivers (1988), Lenormand (2003) and Lenormand and Dutheil (2005) hypotheses evoke sexual selection or conflict to explain the evolution of heterochiasmy. These theories all predict that males have less frequent recombination, consistent with the patterns we observe here across fish. However, we found no significant relationship between sexual dimorphism (a proxy for the intensity of sexual selection) and sex differences in recombination rate ($cM_{M:F}$) across species ($R^2 = 0.009$; $P = 0.463$; Table 1), suggesting that sexual selection in adults is unlikely to be responsible for broad patterns of heterochiasmy. Next, we tested whether the magnitude of

haploid selection can explain variation in heterochiasmy using fertilisation mode as a proxy, where males in species with external fertilisation are subject to stronger haploid selection than those with internal fertilisation. We found no significant relationship (R^2= 0.023; P = 0.242; Table 1); however, our power is hampered by the relatively low numbers of species with internal fertilisation (4 species) for which linkage maps have been generated. The non-significant trend was for species with external fertilisation to have more female-biased recombination than those with internal fertilisation (Table 1). Whilst this pattern is consistent with Lenormand's (2003) and Lenormand and Dutheil's (2005) predictions, it clearly requires further investigation when additional data become available.

Finally, we tested for factors that could explain in variation in sex biases in evolutionary rates across species. Variation in phenotypic sexual dimorphism, sex chromosome system or fertilisation mode were not significantly correlated with sex-specific variation in the evolution of recombination rate ($\sigma^2_{TipM:F}$), and explain a very low proportion of variation (Table 2).

DISCUSSION

We assembled a comprehensive dataset of sex-specific recombination rate estimates across teleost fish species. Our dataset, spanning 61 species across 32 families and 13 orders is, to our knowledge, the most comprehensive dataset of sex differences in recombination rate to date, and we used it to test several theories about the causes of heterochiasmy.

Is heterochiasmy simply the result of differences in male and female meiosis?

There is a primary hypothesis suggesting that sex differences in recombination are largely a pleiotropic consequence of other biological phenomena. If recombination rate differences between females and males are strictly a function of sex differences in the timing or machinery of meiosis (Gruhn et al. 2013; Gruhn et al. 2016), we would expect consistent variation in relative male:female map length across fish species, as they broadly share the same developmental programme. Specifically, teleost fish all share a cell-autonomous mechanism of primordial germ cell development that is mediated by germ plasm, known as preformation (Evans et al. 2014). Although we find a tendency for fish species to exhibit female-biased recombination on average, there is a large amount of across-lineage variation in this general trend. In fact, 31% of species showed the opposite pattern of overall male-bias in recombination rate. These switches in the direction of sex-bias are particularly notable in that they show wide phylogenetic distribution across the clade (Figure 1). Together, this suggests that differences in the meiotic process between the sexes may be responsible for some of

the patterns of heterochiasmy, but are not wholly sufficient to explain recombination rate differences between males and females.

What is the potential for genetic drift and constraint?

Fully neutral explanations for heterochiasmy have also been proposed (Nei 1969; Burt et al. 1991). Specifically, the drift hypothesis predicts that recombination rates should evolve in a neutral manner across the phylogeny. However, we find that the evolution of male (cM_M), female (cM_F) and sex differences ($cM_{M:F}$) in recombination rate do not appear to evolve in a manner consistent with simple neutral processes (e.g. drift), as estimated phylogenetic inertia is low (Pagel's $\lambda = 0.196$). However, the confidence interval of this estimate ranged from 0 to 0.7, and so we cannot exclude the potential contribution of drift to patterns of heterochiasmy.

Instead, male and female recombination rates evolve in correlated shifts across species, likely due to intersexual correlations. Our findings that male and female recombination rates are tightly correlated – likely a function of chromosome structure and number – and evolve in a correlated manner suggest an intriguing potential for conflict between the sexes over optimal recombination rate. Interestingly, weaker intersexual correlations have been observed in studies that examine patterns of recombination within a single species (Fledel-Alon et al. 2011; Johnston et al. 2016; Peterson and Payseur 2020), suggesting that conflict may only manifest over long evolutionary time frames.

Is there an adaptive explanation for heterochiasmy?

The fact that recombination rate differences are not fully explained by meiotic sex differences or neutral processes leaves the potential for several adaptive hypotheses to explain this sex-specific recombination rate evolution. It has been suggested that heterochiasmy is associated with sex chromosomes type within species, with shorter linkage map lengths expected in the heterogametic sex (Haldane 1922; Huxley 1928). However, we find that in species where both sexes recombine, there is a negligible association between the direction of sex-bias in recombination rate and sex chromosome type (Table 1). Therefore, our results suggest that the Haldane-Huxley hypothesis does not hold outside of achiasmate species.

Instead, we observe that males tend to have less frequent recombination compared to females, suggesting that sexual selection may be responsible for heterochiasmy. Whilst we found that rates of recombination evolution are not consistently higher in males across all species, there are some clades where recombination evolves faster in males relative to females (Figure 3). These patterns are

consistent with Trivers (1988), Lenormand (2003) and Lenormand and Dutheil's (2005) hypotheses, as males typically experience both stronger sexual selection and stronger haploid selection.

However, we were unable to find direct associations between variation in heterochiasmy and proxies for differences in sexual selection and sexual conflict intensity across species. This indicates that these effects – if present – are either subtle or complex and clearly requires further investigation when data for more species become available. Furthermore, sexual dimorphism – the proxy for sexual selection used here – is an imperfect measure for sexual selection intensity across species as it can arise from other adaptive processes (e.g. fecundity selection on females). Therefore, future investigations would also benefit from more direct proxies for sexual selection intensity across species than used here.

Sex differences in recombination landscape

In addition to sex differences in the overall rate of recombination, males and females can differ in their recombination landscape (i.e. the location of crossovers along chromosomes) (Sardell and Kirkpatrick 2020). Unfortunately, this type of positional information was only available for 18 of the 61 species in our study, precluding us from testing hypotheses for how selection acts on the recombination landscape in males and females. Future work focusing on differences in the topology of the recombination landscape may provide important biological insights in additional to total recombination rates.

Across fish, crossovers tend to cluster near telomeres in males, but are more uniformly distributed or elevated near centromeres in females (Sardell and Kirkpatrick 2020). However, anecdotally, rapid shifts in chiasma location have been observed over short evolutionary time periods. For instance, in the 14 million years since sockeye salmon and rainbow trout diverged, recombination has switched from uniformly distributed across female chromosomes (Larson et al. 2016) to biased towards the centromeres (Sardell and Kirkpatrick 2020). It is likely that by focusing on overall levels of map length as a proxy for heterochiasmy we are missing fine-scale sex-biases that may have important biological implications for patterns of linkage and the efficacy of selection in males and females. Importantly, it has yet to be tested how quickly sexual dimorphism in fine-scale recombination patterns evolve and whether this is a consequence of sexual selection. These would be extremely interesting research avenues to pursue when additional data become available.

Concluding remarks

Overall, our study reveals broad-scale patterns in recombination rate evolution that are incompatible with neutral expectations or simple biological differences between male and female meiosis. Instead, our analysis provides evidence that sex differences in recombination may be the function of multiple, interacting factors. Although we find the potential for adaptive processes related to sexual selection or sexual conflict to explain heterochiasmy, it remains to be seen whether these trends are substantiated when more data becomes available. Nonetheless, our study provides novel insight into the evolutionary forces shaping recombination rate variation over broad timeframes and sets the stage for future analyses building on our approach.

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TABLES

Table 1. Phylogenetic linear models predicting sex-specific variation in recombination rate ($cM_{M:F}$)

Predictor	N	Level	Log (female-bias in map length)	SE	*P*	R^2
Sexual dimorphism	28	Monomorphic	0.056	0.031		
	33	Dimorphic	0.077	0.029	0.463	0.009
Sex determination	8	Sex chromosomes absent	0.109	0.053		
	30	XY	0.079	0.045	0.506	
	6	ZW	0.066	0.056	0.454	0.016
Fertilization mode	57	External	0.070	0.027		
	4	Internal	0.004	0.056	0.242	0.023

Table 2. Phylogenetic linear models predicting sex-specific variation in recombination rate evolution ($\sigma^2_{TipM:F}$)

Predictor	N	Level	Box-cox transformed (male-bias in tip rate)	SE	*P*	R^2
Sexual dimorphism	28	Monomorphic	-0.040	0.045		
	33	Dimorphic	-0.018	0.022	0.194	0.028
Sex determination	8	Sex chromosomes absent	0.009	0.047		
	30	XY	-0.005	0.029	0.618	
	6	ZW	-0.004	0.032	0.668	0.007
Fertilization mode	57	External	-0.036	0.045		
	4	Internal	0.033	0.048	0.156	0.034

FIGURES

Figure 1. Recombination rate (residual map length) estimates across fish. Black circles at the tips of the phylogenetic tree indicate male (cM_M) and female (cM_F) recombination rate across 61 species, where small circles denote short map lengths and large circles long map lengths. Coloured circles indicate sex differences in map length ($cM_{M:F} = cM_M - cM_F$), ranging from female-biased in red to male-biased in blue.

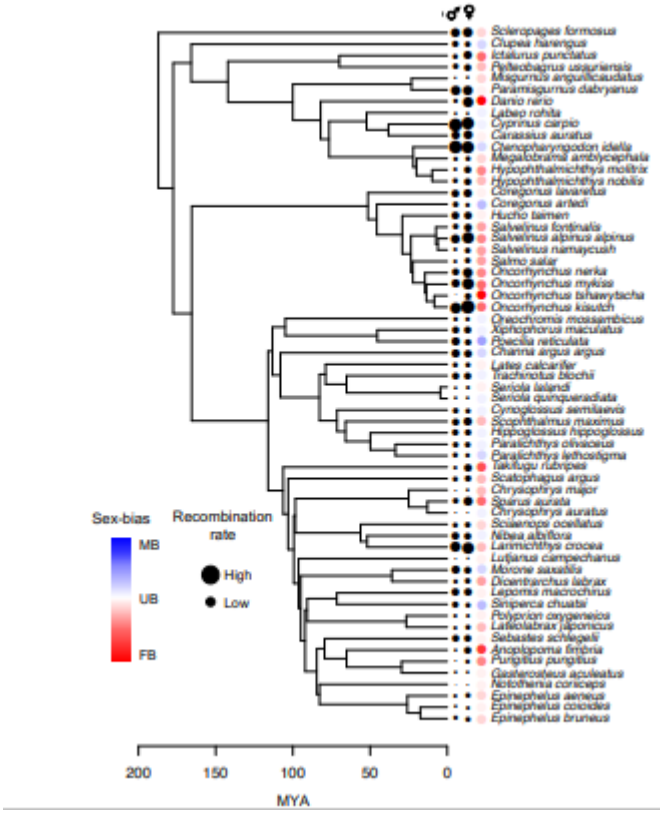

Figure 2. The relationship between female (cM_F) and male (cM_M) map length across 61 species. One-to-one (dashed) line shown. Equation of the regression line: $y = 0.066 + 0.981*x$ ($R^2 = 0.680$), which is not significantly different to a one-to-one slope ($T = 0.261$, d.f. = 46.033, $P = 0.795$).

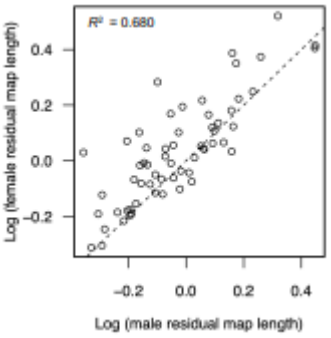

Figure 3. **The evolution of recombination rate in males and females.** Panel A) Plot showing estimates of (box-cox transformed) male (σ^2_M) and female (σ^2_F) recombination rate evolution across the phylogeny. Panel B) Relationship between (box-cox transformed) rates of recombination rate evolution in males (σ^2_{TipM}) and females (σ^2_{TipF}) across terminal branches of the phylogeny. One-to-one (dashed) line is shown. Equation of the regression line: $y = 0.031 + 1.004*x$ ($R^2 = 0.828$), which is not significantly different to a one-to-one slope ($T = 0.072$, d.f. = 43.73, $P = 0.943$). Panel C) Histogram of the distribution of sex biases in recombination rate evolution across lineages ($\sigma^2_{TipM:F} = \sigma^2_{TipM} - \sigma^2_{TipF}$), the mean of which is not significantly different to 0 ($P = 0.478$).