On the origins of Y and W chromosomes as an outcome of sex allocation evolution

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Abstract. A great many plants and animals have evolved separate sexes from hermaphroditism. In species with separate sexes, the development of an individual as male or female is often controlled by a diallelic sex-determining locus (XY and ZW systems). Transitions from hermaphroditism to separate sexes must therefore have often entailed the emergence of such a locus. However, the evolutionary mechanisms governing the emergence of XY and ZW systems in ancestral hermaphroditic populations, and in particular the mechanisms leading some species to acquire an XY rather than a ZW system, remain elusive. Here, we model the co-evolution of resource allocation to male and female functions (sex allocation) with the genetic architecture of sex determination, and show that gradual evolution readily leads to the emergence of XY and ZW systems. Our model also reveals a strong influence of the shape of the relationship between resource allocation and fecundity in each sex (male and female gain curves) on whether an XY or a ZW system evolves. This is because gain curves indicate the intensity of competition for reproduction through each sex, which in turn affects selection on the genetic architecture of sex allocation. Taken together, our results advance the understanding of sexual systems by uncovering a hitherto unappreciated link between the ecology and economics of sex allocation and the genetic basis of sex determination.

Keywords. Sex chromosomes, quantitative traits, genetic architecture, separate sexes, sex determination.

Introduction

A great many plants and animals have evolved separate sexes from hermaphroditism (Renner, 2014; Leonard, 2018). These transitions to dioecy are thought to have occurred either to avoid self-fertilisation and the accompanying deleterious effects of inbreeding depression (Charlesworth and Charlesworth, 1978, 1981), or because individuals specialising in one sexual function achieve a greater reproductive success than if they allocate resources to both (Charnov et al., 1976; Charlesworth, 1999). How resource allocation affects reproductive success is usefully understood in terms of the economics of fitness gained through male and female functions, which are influenced by a number of ecological, behavioural and physiological factors (Givnish, 1982; Lloyd, 1982; Renner and Ricklefs, 1995; Freeman et al., 1997; Iyer and Roughgarden, 2008; Eppley and Jesson, 2008; Schärer, 2009; Pannell and Jordan, 2022). In sex allocation theory (Charnov et al., 1976; Charnov, 1982), the joint effect of these factors is encapsulated in the shape of the male and female 'gain curves' (Figure 1A). Hermaphroditism is predicted to be favoured by saturating gain curves, whereby individuals accrue diminishing fitness returns on investment so that sexual specialisation is disfavoured. Conversely, dioecy is favoured by accelerating gain curves, which reflect increasing fitness returns on investment and therefore advantages of sexual specialisation (Charnov et al., 1976; Charnov, 1982).

When selection favours sexual specialisation, the potential for a population to reach dioecy ultimately depends on the genetic basis of sex allocation (Charlesworth, 1999; Beukeboom and Perrin, 2014). The development of an individual as male or female is often determined at a locus where one sex is heterozygous and the other is homozygous (XX/XY or ZW/ZZ females and males, Bachtrog et al., 2014; Beukeboom and Perrin, 2014). Thus, the transition from hermaphroditism to dioecy must often have entailed the emergence of such a locus. Population genetic models invoking inbreeding avoidance suggest that a sex-determining locus can emerge from the sequential invasion of male and female sterility mutations at two linked genes (the two-factor model, Charlesworth and Charlesworth, 1978; Charlesworth et al., 2005; Olito and Connallon, 2019). Sex allocation theory, on the other hand, has so far remained mute to the sex determination system most likely to evolve when dioecy is the outcome of selection for sexual specialisation. Here, we show that a heterogametic sex-determining locus readily emerges from the co-evolution of sex allocation with the genetic architecture of sex determination. Moreover, whether selection favours an XY or a ZW system depends on the shapes of the male and female gain curves and

thus ultimately on the economics of sex allocation.

Results

Model. We consider a large population of diploid individuals that allocate a proportion x of their reproductive resources to their female function and $1-x$ to their male function. In turn, strategy x results in realised female and male fecundities $F(x) = F_0 x^{\gamma_♀}$ and $M(x) = M_0(1-x)^{\gamma_♂}$, respectively, where F_0 and M_0 correspond to the maximum achievable fecundity, and exponents $\gamma_♀$ and $\gamma_♂$ control the shape of the female and male gain curves, thus determining the fitness effects of sexual specialisation (we assume that fitness gain curves are power functions in the main text, but many of our results in our appendix hold for more general functions; Charnov et al., 1976; Charnov, 1982; Charlesworth, 1999). Following gamete production (or pollen and ovule production), male and female gametes fuse randomly and a new generation is formed from the resulting zygotes (Figure 1B; Appendix A.1 for details). Note that we picture mating here as the union of free-swimming gametes, as in broadcast spawners, but the model equally applies to random mating by union of randomly dispersed pollen with ovules in plants, or to analogous cases of internal fertilisation in animals.

Gradual evolution of sexual systems. We initially assume that the sex allocation strategy, x, is encoded by a quantitative trait locus subject to mutations of weak and additive effects ('continuum of alleles' model; Fig. 1C), in which case evolution occurs in two steps (Appendix A.2). First, the population gradually converges to express the intermediate strategy $x^* = \gamma_♀/(\gamma_♀ + \gamma_♂)$, with all individuals being hermaphrodites (Appendix A.2.3). Second, the population experiences either stabilising selection and therefore remains hermaphroditic, or disruptive selection resulting in the gradual differentiation of two alleles encoding increasingly male and female strategies. Which of these two outcomes unfolds depends on the gain curves, with disruptive selection requiring at least one of them to be sufficiently accelerating (specifically that $2\gamma_♀\gamma_♂ > \gamma_♀ + \gamma_♂$; Fig. 2A; Appendix A.2.4). When both gain curves are accelerating ($\gamma_♀ > 1$ and $\gamma_♂ > 1$), disruptive selection leads to the co-existence of two alleles: one that encodes a pure male ($x = 0$) and another that encodes a pure female ($x = 1$) strategy (Fig. 2C). When only one curve is accelerating, one allele encodes a unisexual strategy (female or male), while the other encodes a hermaphroditic strategy, albeit biased towards the opposite sex (Fig. 2D,E; Appendix A.3.3 for details

on the analysis, and Appendix A.3.4 for connection between our results on evolutionary dynamics and classical optimality models, Charnov et al., 1976; Charnov, 1982).

Emergence of XY and ZW sex determination systems. Disruptive selection on sex allocation leads to a polymorphic population with three genotypes at equilibrium: two homozygotes that express female- and male-biased sex allocation strategies, and a heterozygote with an intermediate hermaphroditic strategy. These intermediate heterozygotes are less fit than their homozygous counterparts, yet are produced each generation by mating among the three genotypes. Under these circumstances, selection should favour mechanisms that modify the sex allocation strategy expressed by heterozygotes (Van Dooren, 1999; Rueffler et al., 2006). To examine how this might occur, we extended our model to include co-evolution of sex allocation with its underlying genetic basis. The evolving locus is now composed of two linked elements, a sex allocation gene where, as before, alleles encode a sex allocation strategy, and its promoter where alleles determine the level of expression of their linked sex allocation allele (Figure 3A). Variation at the promoter leads to variation in allelic expression through *cis* effects, which in turn determine the dominance relationships among sex allocation alleles. We let the sex allocation gene and its promoter co-evolve through the spread of recurrent mutations of weak effect (Appendix B.1 for details on the model).

We first investigated this model using simulations under conditions predicted to lead to pure male and female alleles (so when $\gamma_{♀} > 1$ and $\gamma_{♂} > 1$). We found that complete dominance of one sex allocation allele always evolves, so that eventually dioecy is complete and the population comprises only males and females (Figure 3B-C). Remarkably, whether the male or the female allele becomes dominant depends strongly on the shape of the male and female gain curves (Figure 3D). When fitness returns increase more steeply via female function (i.e., when $\gamma_{♀} > \gamma_{♂}$), the male allele is more likely to become dominant so that females are homozygotes and males are heterozygotes. In other words, the evolution of dominance via *cis* regulation leads to the emergence of an XY system here. Conversely, when fitness returns increase more steeply via male function (i.e., when $\gamma_{♂} > \gamma_{♀}$), the female allele most often evolves to be dominant, leading to a ZW system where males are homozygotes and females are heterozygotes. We also simulated scenarios predicted to lead to gyno- and androdioecy, where pure females and pure males coexist with hermaphrodites, respectively (i.e. with either $\gamma_{♀} > 1$ or $\gamma_{♂} > 1$). In these cases, the allele encoding the unisexual strategy most often becomes dominant (Figure 3E), so that the population typically ends

up being composed of either heterozygote (XY) males and homozygote (XX) hermaphrodites (when $\gamma_{♀} > 1$), or heterozygote (ZW) females and homozygote (ZZ) hermaphrodites (when $\gamma_{♂} > 1$).

A positive feedback causes heterozygotes to replace the less competitive homozygotes. To better understand the nature of selection on dominance, we analysed mathematically a version of the model where only dominance evolves. We assume that two sex allocation alleles segregate, $x_{♀}$ and $x_{♂}$, where one encodes a more female strategy than the other ($x_{♀} > x_{♂}$, hereafter referred to as 'female' and 'male' alleles for brevity). The dominance of the female over the male allele is denoted h, so that the phenotype of a $x_{♂}/x_{♀}$ heterozygote is $h\ x_{♀} + (1-h)\ x_{♂}$. This h is controlled by a quantitative trait locus that is unlinked to the sex allocation gene and evolves through recurrent mutations of small effect, allowing us to consider mechanisms broader than *cis* regulation that could influence dominance (e.g. *trans* effects). Our analysis reveals that selection depends on the current level of dominance h (Figure 4A, Appendix B.2 for detailed analysis). When h is above some threshold h^* (i.e., when $h > h^*$), selection systematically favours an increase in dominance, thus leading to complete dominance of the female allele ($h \to 1$). Conversely, when $h < h^*$, selection favours a decrease in dominance, resulting in complete recessivity of the female allele ($h \to 0$). It is difficult to derive the threshold value h^* explicitly, but we show that $h^* < 1/2$ when the male gain curve is more accelerating ($\gamma_{♀} < \gamma_{♂}$), whereas $h^* > 1/2$ when the female gain curve is more accelerating ($\gamma_{♀} > \gamma_{♂}$; Appendix B.2.3.2). This means that, where alleles are initially additive ($h = 1/2$), selection favours complete dominance of the allele associated with the sex exhibiting the less accelerating gain curve, in agreement with our simulations.

The positive feedback that causes selection to favour either ever higher or ever lower levels of dominance can be understood by considering that selection on dominance acts only among hermaphroditic heterozygotes. These heterozygotes can invest more heavily in male or female function, through an increase or decrease in dominance. However, whatever the change in dominance, these individuals will always achieve lower female fecundity than female homozygotes and lower male fecundity than male homozygotes. This situation causes selection in heterozygotes to favour allocating more to the sex function for which the competitive edge of homozygotes is the smallest. In other words, selection here favours heterozygotes that "make the best of a bad job". Whether this is becoming more female- or male-biased depends on the current level of dominance and on the shape of gain curves (Figure 4B-C). In any case, once selection favours a change in dominance such that heterozygotes allocate more to one

sex, the competitive edge of the corresponding homozygote is reduced, which in turn favours further change in dominance in that same direction (and so on). This positive feedback gradually leads to complete dominance of one sex allocation allele, so that heterozygotes eventually replace the homozygotes that were initially less competitive. In short, our model shows that selection can act on the dominance of alleles at nascent sex-determining loci, and favours the emergence of either an XY or a ZW system depending on the strength of competition for reproduction through male and female function.

Discussion

Our analyses characterise a path for the evolution of dioecy that invokes selection for sexual specialisation and that leads to the emergence of XY or ZW sex determination involving a single gene. Sexual specialisation has long been considered a potential reason for the evolution of separate sexes, and previous modelling has shown that dioecy should be maintained over hermaphroditism when individuals maximise their fitness by allocating to only one sex (Charnov et al., 1976; Charnov, 1982). Our model advances this earlier work by considering the path by which transitions from hermaphroditism to dioecy might occur. We find that separate sexes can evolve via gradual divergence of increasingly male and female phenotypes and the concomitant evolution of dominance at a sex-determining locus.

The evolution of dioecy through divergence at a single gene contrasts with predictions of the classical 'two-factor model', in which sex determination evolves through the consecutive invasion of male and female sterility mutations at two linked loci (Charlesworth and Charlesworth, 1978). Sex determination involving two linked loci has been identified or inferred in a few flowering plant species, consistent with classical theory (Harkess et al., 2020; Akagi et al., 2019, reviewed in Renner and Müller, 2021), but sex determination involving a single gene has also been documented in a number of lineages (Akagi et al., 2014; Henry et al., 2018; Müller et al., 2020, reviewed in Renner and Müller, 2021). Although single-locus sex determination could derive from an initial two-locus system, e.g., via gene loss or replacement (Charlesworth, 2002, 2019), it is clear from our results that single-locus sex determination can also evolve directly from hermaphroditism without an intermediate two-locus stage.

In our model, single-locus sex determination arises through the evolution of dominance via modifiers of allelic expression with *cis* or *trans* effects on the sex-determining locus. Crucially, whether the

male or female allele becomes dominant, and therefore whether XY or ZW sex determination evolves, depends on the fitness returns of allocating resources to male *vs* male function and thus on the ecology of reproduction. In general, dioecy should evolve when the benefits of sexual specialisation lead to increasing returns (accelerating gain curves, Charnov et al., 1976). Our model further demonstrates that selection favours the evolution of dominance of the allele promoting the sex function in which the increase in fitness returns is the weakest. For instance, a dominant Y should evolve more frequently when the male gain curve accelerates less steeply than the female one. The observation that a majority of dioecious flowering plant species have XY sex determination (about 85%, Ming et al., 2011) thus suggests that, to the extent that dioecy has evolved in response to selection for sexual specialisation in these species, the female gain curve in plants may often be more accelerating than the male one. Although we have few empirical estimates of these curves, an accelerating female gain curve can result from the benefits of seed predator satiation (Janzen, 1971; Lloyd, 1982), or from benefits associated with increased attractiveness to seed dispersers of larger crops of fleshy fruits (Givnish, 1982; Vamosi et al., 2007; Biernaskie, 2010). We demonstrate this in the context of our model in Appendix C.

Most empirical evidence for transitions from hermaphroditism to dioecy comes from flowering plants, where such transitions have occurred thousands of times independently (Renner, 2014; Henry et al., 2018). In animals, our results are directly relevant to a number of taxa in which separate sexes have evolved from hermaphroditism, e.g., the *Ophryotrocha* genus in polychaete annelids or flatworms of the *Schistosoma* genus (Ramm, 2016; Picchi and Lorenzi, 2018; Leonard, 2018; Wang et al., 2022). Our model may also be useful to understand 'split sex-ratios' in ants and other social Hymenoptera, where colonies produce either male or female sexuals leading to a form of colony-level dioecy (Meunier et al., 2008; Kuemmerli and Keller, 2009). In *Formica glacialis*, split sex-ratios is determined by a single non-recombining region acting like a W chromosome (Lagunas-Robles et al., 2021), suggesting interesting parallels with the scenario we establish here.

In conclusion, our model widens our understanding of sexual system evolution beyond the abiding view that separate sexes evolve from hermaphroditism via sterility mutations and selection for inbreeding avoidance (Charlesworth and Charlesworth, 1978). Dioecy can also be the outcome of the gradual co-evolution of sex allocation and its genetic basis. This process readily leads to the emergence of a sex-determining locus and nascent sex chromosomes, setting the stage for major genetic changes such as

recombination suppression, genetic degeneration and dosage compensation (e.g., Bachtrog et al., 2014; Charlesworth, 2019; Lenormand and Roze, 2022). Finally, by linking the economics of sex allocation with the evolution of sex determination, our model exposes a selective mechanism for the evolution of XY versus ZW sex chromosomes, illustrating the potential relevance of ecology for the way traits are genetically assembled.

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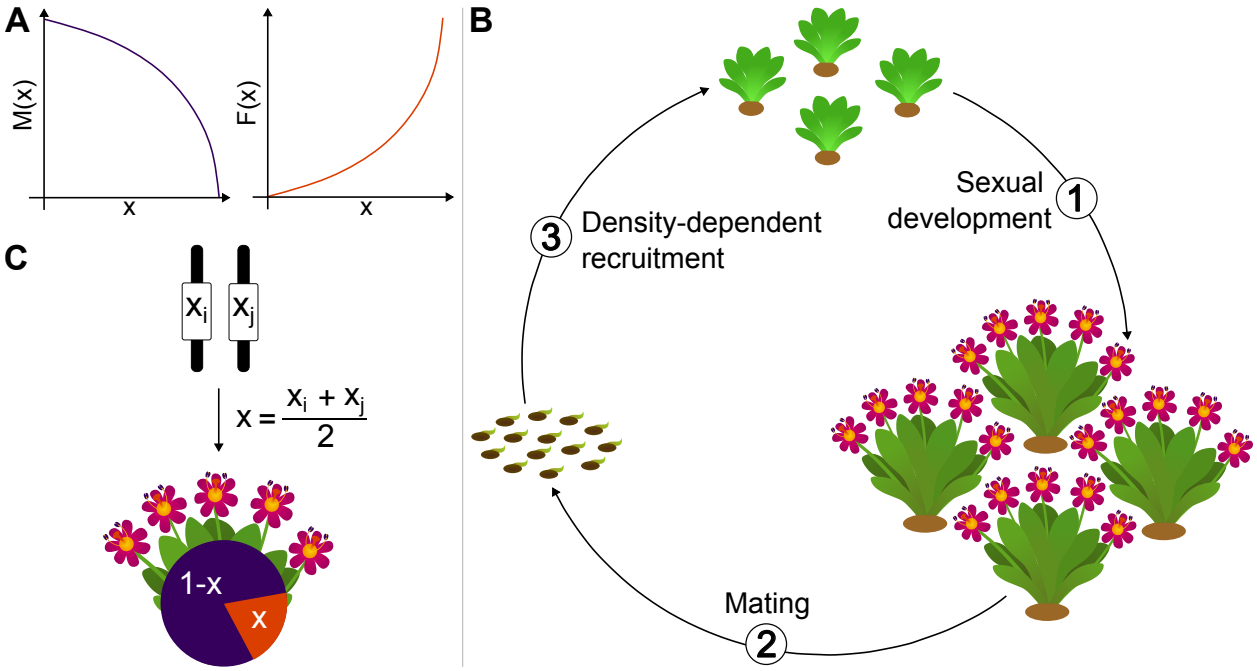

Figure 1: Life cycle and genetic architecture of sex allocation. **A** Male ($M(x)$, dark purple) and female ($F(x)$, orange) gain curves as functions of the amount x of resources allocated to female function. In this example, the male gain curve is saturating, reflecting diminishing fitness returns through male function, whereas the female gain curve is accelerating, reflecting increasing fitness returns through female function. **B** Life cycle assumed in the model. (1) Juveniles develop into mature adults and allocate resources to their female and male functions in proportions x and $1 - x$, respectively, resulting in female and male fecundities $F(x)$ and $M(x)$. (2) Individuals mate randomly to produce a large number of offspring. (3) Adults die and are replaced by juveniles sampled uniformly from the offspring pool. Note that plants are used for illustration purposes only in this figure, as our model is not limited to a particular taxonomic group. **C** Genetic architecture of sex allocation in our baseline model. The sex allocation strategy x expressed by an individual is determined by its genotype at a quantitative trait locus where alleles are additive.

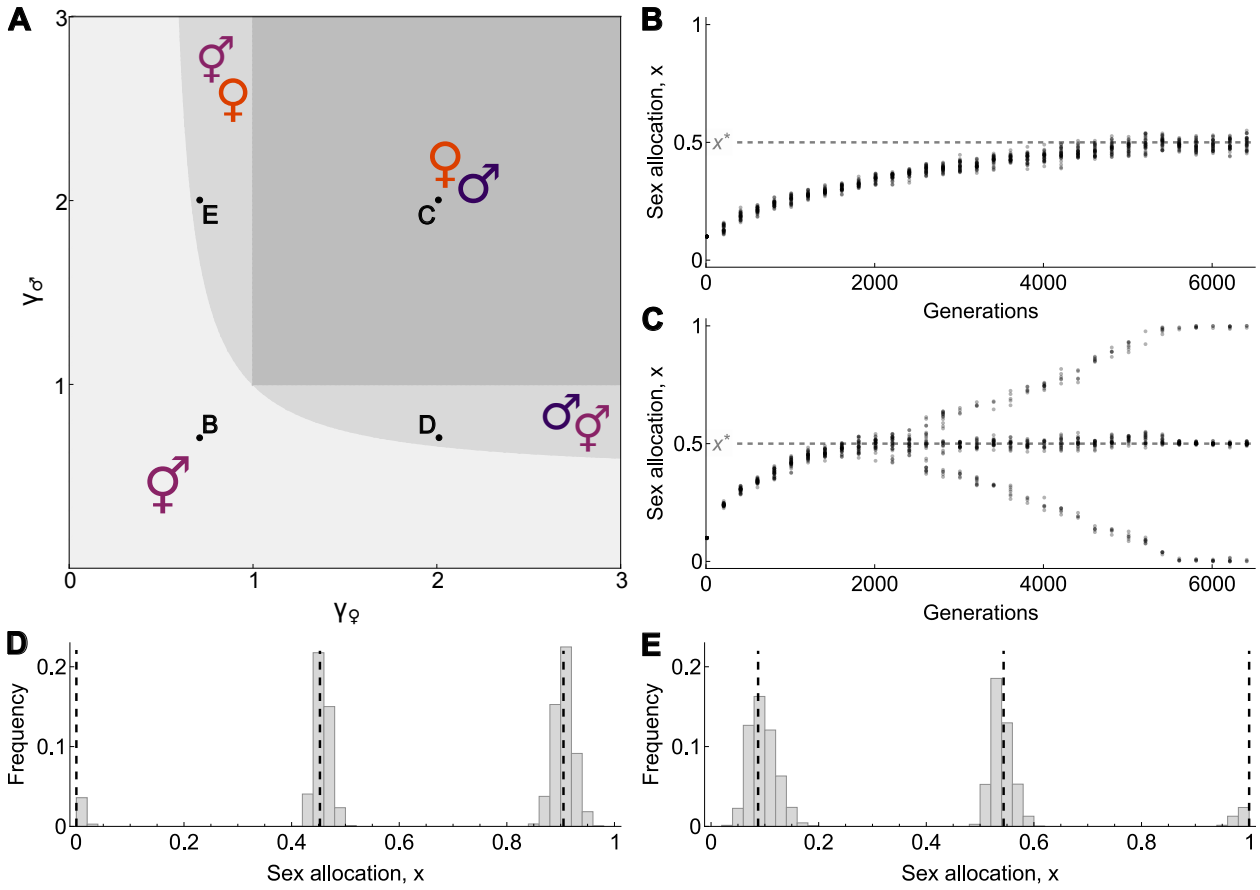

Figure 2: The gradual evolution of sex allocation. **A** The four outcomes of evolution according to $\gamma_♀$ and $\gamma_♂$ (Appendix A.2 for analysis): (i) hermaphroditism (light grey, B); (ii) dioecy (dark grey, C); (iii) androdioecy (medium light gray, D) and (iv) gynodioecy (medium light gray, E), where pure males and males coexist with hermaphrodites, respectively. **B** Phenotypes expressed by 30 randomly sampled individuals every 200 generations in a simulation under conditions predicted to lead to hermaphroditism (with $\gamma_♀ = \gamma_♂ = 1/\sqrt{2}$). The population converges to express the equilibrium strategy $x^* = \gamma_♀/(\gamma_♀ + \gamma_♂)$, indicated by the light grey dashed line (Appendix A.4 for simulation details). **C** Same as B under conditions predicted to favour dioecy (with $\gamma_♀ = \gamma_♂ = 2$). The population first converges to x^* and then experiences disruptive selection, leading to pure male ($x = 0$) and female ($x = 1$) alleles. At equilibrium, the population is composed of males, females and hermaphrodites. **D** Distribution of phenotypes at equilibrium in a simulation where androdioecy evolves (with $\gamma_♀ = 2$ and $\gamma_♂ = 1/\sqrt{2}$). Dashed vertical lines indicate the equilibrium strategies computed numerically in Appendix A.3.3. **E** Same as D where gynodioecy evolves (with $\gamma_♀ = 1/\sqrt{2}$ and $\gamma_♂ = 2$). Other parameters used in all simulations: $N = 10^4$, $\mu = 5 \times 10^{-3}$ and $\sigma = 10^{-2}$ (Appendix A.4 for description of parameters).

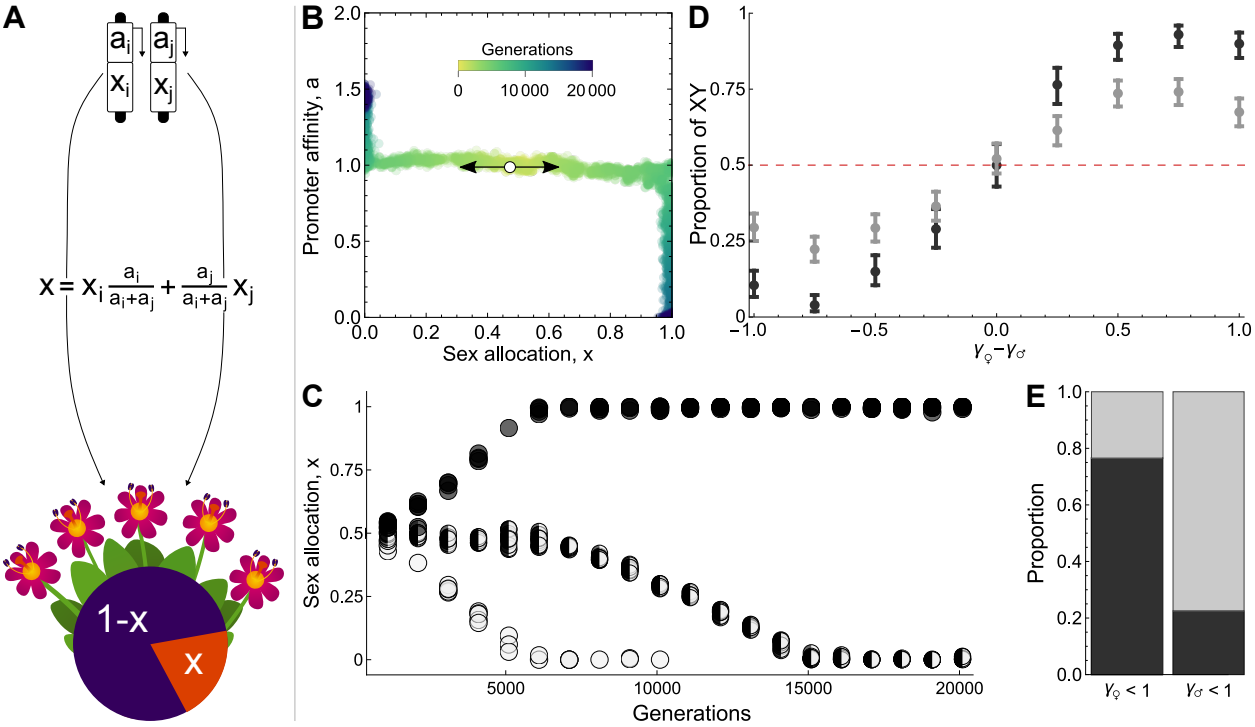

Figure 3: Co-evolution of sex allocation and dominance. A Genetic architecture of sex allocation. The sex allocation locus is composed of a sex allocation gene and its promoter. Transcription factors must bind to the promoter for the sex allocation gene to be expressed, which they do at a rate that depends on the promoter's affinity, a. Consequently, sex allocation alleles are expressed in proportion to their promoter's affinity, and promoter affinities encode the dominance relationship between sex allocation alleles. In this example, alleles x_i and x_j are associated with promoters with affinities a_i and a_j, so that they contribute in proportions $a_i/(a_i + a_j)$ and $a_j/(a_i + a_j)$ to the expressed sex allocation strategy x. **B** Phase diagram of sex allocation and promoter affinity when the two co-evolve in a simulation under conditions predicted to lead to dioecy ($\gamma_♀ = \gamma_♂ = 2$). Each dot depicts an allele, characterised by the sex allocation strategy it encodes and its promoter's affinity. Colour indicates time since the start of the simulation (in generations), with darker colours indicating later times. The population is initially monomorphic with $x_0 = 0.5$ and $a_0 = 1$ (white circle). Here, the male allele becomes associated with an increasingly high affinity promoter while the female allele becomes associated with an increasingly low affinity one, leading to complete dominance of the male allele and the emergence of XY sex determination. (Parameters: $N = 10^4$, Appendix B.1 for simulation details). **C** Phenotypes expressed by individuals as a function of time for the same simulation as figure B. Each circle depicts an individual. Fully black and white circles depict homozygotes for female- and male-biased alleles, respectively, whereas half black and white circles depict heterozygotes (defined as individuals bearing two alleles that are more different than the average difference between two alleles within the same individual). As sex allocation alleles diverge and dominance evolves, heterozygotes gradually become more male-biased, and eventually replace male homozygotes, thereby achieving dioecy with XY sex determination. **D** Proportion of XY systems (with binomial 95% confidence intervals) evolving out of 200 and 400 simulations for population sizes $N = 3,000$ and $N = 300$, respectively, as a function of $\gamma_♀ - \gamma_♂$ (when $\gamma_♀ - \gamma_♂ < 0$, $\gamma_♂ = 2$ and $\gamma_♀$ varies between 1 and 2; when $\gamma_♀ - \gamma_♂ > 0$, $\gamma_♀ = 2$ and $\gamma_♂$ varies between 1 and 2). XY and ZW systems are equally likely to emerge when $\gamma_♀ = \gamma_♂$, whereas XY systems are more prevalent where $\gamma_♀ > \gamma_♂$ and ZW systems where $\gamma_♀ < \gamma_♂$. This difference is less pronounced in smaller populations (light grey), reflecting the influence of genetic drift (Appendix B.2.5 for details). **E** Proportion of cases where the male-biased allele (light grey) or female-biased allele (dark grey) became dominant in the gynodioecious ($\gamma_♀ = 1/\sqrt{2}$ and $\gamma_♂ = 2$) and androdioecious ($\gamma_♀ = 2$ and $\gamma_♂ = 1/\sqrt{2}$) cases, out of 200 simulations with $N = 3,000$. In both cases, the allele encoding unisexuality is more likely to become dominant than the one encoding hermaphroditism. Parameters used in all simulations: $\mu = 5 \times 10^{-3}$, $\sigma = 10^{-2}$.

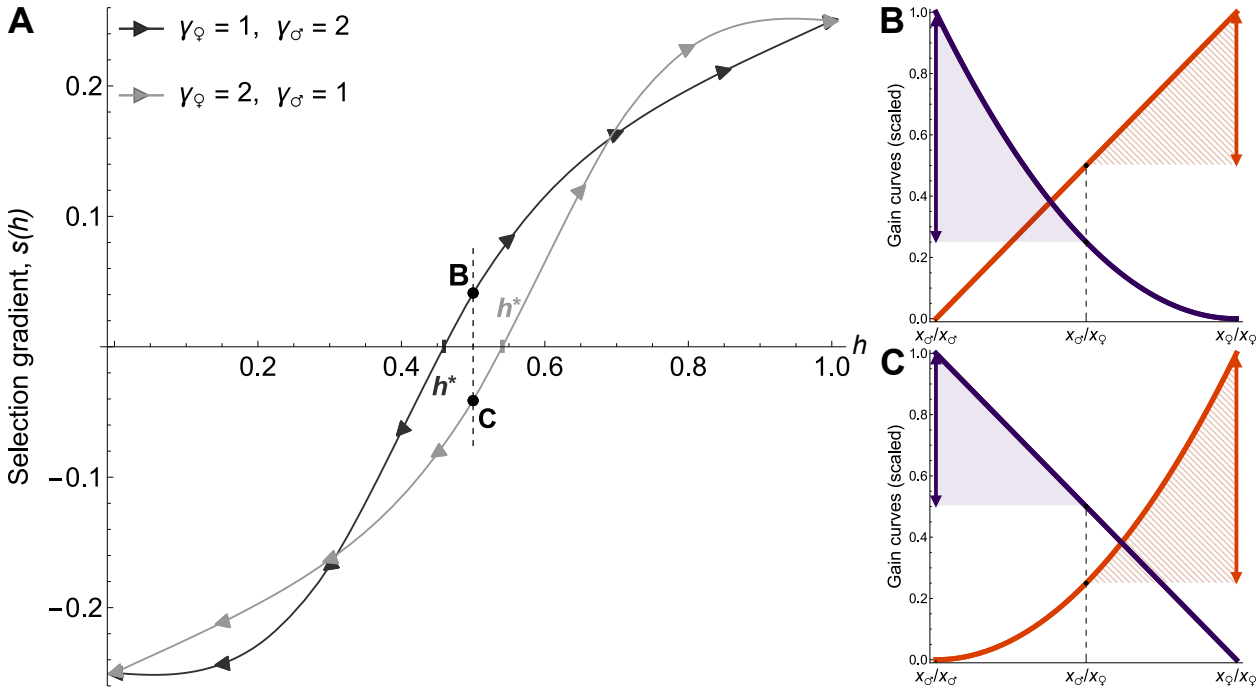

Figure 4: Selection on dominance. **A** Selection gradient on dominance h of a female allele $x_♀ = 1$ over a male allele $x_♂ = 0$ when the male gain curve is the more accelerating ($\gamma_♀ = 1$ and $\gamma_♂ = 2$, dark grey) and when the female gain curve is the more accelerating ($\gamma_♀ = 2$ and $\gamma_♂ = 1$, light grey; Appendix B.2.3.2 for how to compute this gradient). Selection favours an increase in h when $s(h)$ is positive, and a decrease in h when it is negative. The selection gradient $s(h)$ is negative when h is smaller than h^* and positive above (as defined in eq. B49). This threshold is $h^* < 1/2$ when $\gamma_♂ > \gamma_♀$ and $h^* > 1/2$ when $\gamma_♂ < \gamma_♀$, so that starting from $h = 1/2$ (dashed line), selection favours dominance of male allele $x_♂$ ($h \to 0$) and XY sex determination when $\gamma_♂ < \gamma_♀$ (light grey), and dominance of female allele $x_♀$ ($h \to 1$) and ZW sex determination when $\gamma_♂ > \gamma_♀$ (dark grey). **B**-**C** Female (orange) and male (purple) gain curves corresponding to the two cases presented in figure A. The two homozygotes $x_♀/x_♀$ and $x_♂/x_♂$ and heterozygotes $x_♀/x_♂$ are positioned along the x-axis, depicting the sex allocation strategy they express under additive gene action ($h = 1/2$). The competitive edge held by male and female homozygotes over heterozygotes is indicated by vertical arrows, and the filled and hatched areas under the male and female gain curves, respectively. In B, which corresponds to a case where the male gain curve is more accelerating than the female one ($\gamma_♀ < \gamma_♂$, in dark grey in fig. A), the male homozygotes holds a larger competitive edge over heterozygotes than female homozygotes, so that selection favours heterozygotes becoming more female and thus dominance of the female allele (i.e. an increase in h). In C, the female gain curve is more accelerating than the male one ($\gamma_♀ > \gamma_♂$, in light grey in fig. A), so that the competitive advantage held by female homozygotes is larger, which favours dominance of the male allele (i.e. a decrease in h).