LETTER

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Mating preferences of selfish sex chromosomes

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The evolution of female mating preferences for harmful male traits is a central paradox of sexual selection[1–9]. Two dominant explanations for this paradox[8,10] are Fisher's runaway process, which is based on genetic correlations between preference and trait[1,3,4], and Zahavi's handicap principle, in which the trait is an honest costly signal of male quality[2,6,8,11]. However, both of these explanations require the exogenous initial spread of female preferences before harmful male traits can evolve[1–4,6,8,11]. Here I present a mechanism for the evolution of female mating preferences for harmful male traits that is based on the selfish evolutionary interests of sex chromosomes. I demonstrate that female-biased genetic elements—such as the W and X sex chromosomes—will evolve mating preferences for males who display traits that reduce their fitness and/or that of their male offspring, but increase fitness in female offspring. In particular, W-linked preferences can cause nearly lethal male traits to sweep to fixation. Sex-linked preferences can drive the evolution of traits such as ornamental handicaps and male parental care, and can explain variation in ornamentation and behaviour across taxa with divergent sex-determining mechanisms.

Female mating preferences should evolve to maximize total offspring fitness[7]. Intra-genomic conflict complicates this picture, because females can carry multiple genetic elements that have sex-biased transmission[12,13]. This is clearest for the W chromosome in female-heterogametic (ZW) species, such as birds: autosomes spend as many generations in males as in females, but the W chromosome is only ever carried by females[12–14]. A preference encoded on the W chromosome should therefore evolve to maximize the total fitness of daughters, with no regard for the fitness of sons (to whom it is not transmitted).

Traits that increase the fitness of daughters at the expense of the fitness of fathers or sons can take many forms. One major category is sexually antagonistic traits, which increase fitness in one sex but reduce it in the other[13,15]. Such traits are common in natural populations[16–19]. Usually, to avoid elimination by natural selection, a sexually antagonistic trait must either confer a fitness advantage when averaged across the sexes or be sex-linked[13,15]. In previously studied scenarios, these conditions limit the fitness cost that can be imposed on the sex for which the trait is deleterious; here, I show that this is not true when mating preferences for sexually antagonistic traits are encoded on a sex chromosome.

Previous theoretical work has separately considered the roles of sexual antagonism[9,20], sex linkage[21], sex determination[22,23] and reinforcing female preferences[5,20] in sexual selection. However, to my knowledge, no previous model has examined the co-evolution of sex-linked female preferences for autosomal, sexually antagonistic traits.

To examine this process, I considered a two-locus population genetic model of a ZW species, with an autosomal 'trait' locus and a W-linked 'preference' locus (for full details, see Methods). Z-linked and X-linked preferences are discussed below. In this model, two alleles segregate at the trait locus: the wild-type allele (t) and the mutant allele (T), which increases female viability (by a factor $1 + s_f$ for TT homozygotes and $1 + h_T s_f$ for Tt heterozygotes) but reduces male viability (by $1 - s_m$ for TT and $1 - h_T s_m$ for Tt). s_f is the strength of the viability advantage of the T allele in females; s_m is the strength of its viability disadvantage in males. h_T is the dominance of the T allele with respect to the t allele. The alleles mutate from one to the other at a symmetrical rate u per replication. I assume that $s_m > s_f$, so that T is selected against in the absence of other forces. Two alleles segregate at the W-linked preference locus: the wild-type allele p and the mutant allele P, the bearers of which (always female) have a greater propensity to mate with trait-expressing males (by a factor $\alpha > 1$ for TT males and α^{h_T} for Tt males, where α is the strength of the preference). Here I assume $h_T = 1/2$ (co-dominance), although the qualitative features of the results do not depend on this assumption (see Extended Data Fig. 1).

It can be proven (Supplementary Information) that the P allele increases in frequency as long as the trait locus is polymorphic. Therefore, the P allele will fix if there is a source of persistent trait polymorphism, such as recurrent mutation or migration from a population with reduced selection against the trait (Fig. 1). This positive selection arises indirectly. The P allele generates a positive genetic correlation between itself and the T allele by inducing its bearers to preferentially mate with males that bear the T allele. Because the T allele increases fitness in females (and the P allele is present only in females), this positive association causes the frequency of the P allele to rise.

The strength of positive selection acting on the P allele depends on several factors. For example, it increases with the strength of the preference induced by the P allele, and with the fitness advantage conferred by the T allele in females. To investigate the strength of selection in favour of the P allele, I compared the strengths observed in several configurations of the model to those observed in the standard two-locus autosomal model of Fisherian sexual selection[4]. In this model, selection for low-frequency W-linked preferences is consistently stronger—often by orders of magnitude—than selection for analogous autosomal preferences, even when the latter start at the high frequencies required for the trait to spread (Supplementary Information).

Selection on the T allele depends on its cost to males, its benefit to females and the proportion of females that carry the P allele. If the strength of the preference is sufficiently large ($\alpha \gtrsim 1/[(1 - s_m)(1 + s_f)]$, Supplementary Information), selection favours the T allele for frequencies of the P allele above a certain threshold. Because the P allele inevitably rises to fixation, this threshold is eventually exceeded and the T allele spreads. The resultant equilibrium is one in which many males exhibit a trait that severely impairs their survival, and all females exhibit a strong mating preference for these low-viability males (Fig. 1, Extended Data Fig. 2b). This can occur even for traits that are nearly lethal to males but that confer only a small advantage to females (Fig. 1d). If $\alpha \lesssim 1/[(1 - s_m)(1 + s_f)]$ instead, the T allele remains at low frequency, even after the P allele has fixed. In this equilibrium, all females prefer low-viability males despite these males being nearly absent from the population (Fig. 1, Extended Data Fig. 2a).

In this model, the spread of the harmful male trait does not require initial neutral drift of—or exogenous selection for—the mutant preference, unlike in analogous two-locus models of Fisher's runaway process[4,8] and Zahavi's handicap model[6,8,11]. By extension, preferences that impose fitness costs on females (for example, by reducing their probability of finding a mate) can invade from low frequency in this model, unlike in comparable major-effect runaway and handicap models (which are very sensitive to costs of female preferences[8]).

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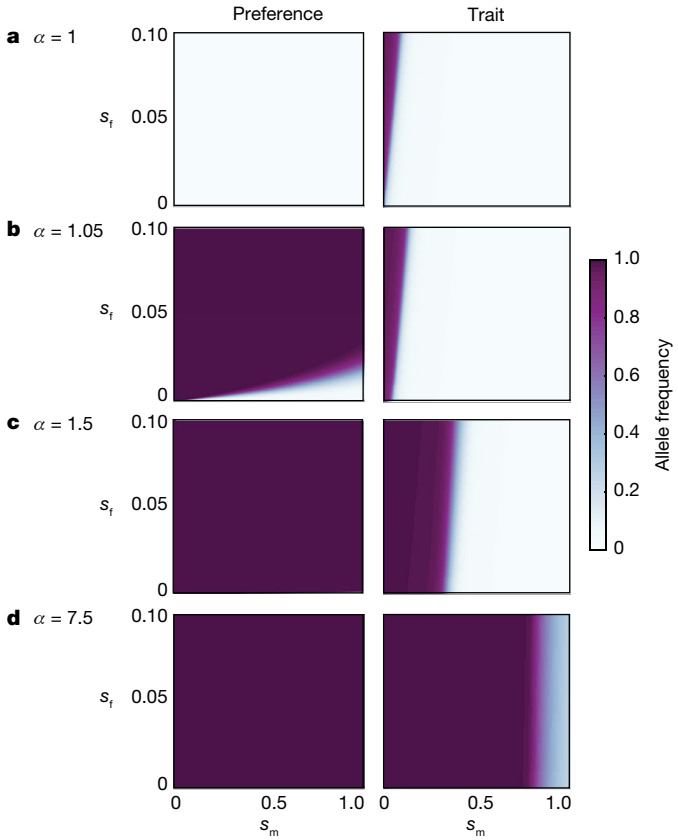

Fig. 1 | Evolution of W-linked preferences for sexually antagonistic traits. Long-run frequencies of the W-linked *P* allele and the autosomal sexually antagonistic *T* allele after 5×10^6 generations, each having started at 1% frequency. The *T* and *t* alleles mutate from one to the other at a rate of 10^{-3} per replication. **a**, When the *P* allele induces no preference ($\alpha = 1$), the sexually antagonistic *T* allele reaches high frequency only when it increases viability on average across the sexes (that is, when $(1 + s_f)(1 - s_m) > 1$). **b**, Even when the preference encoded by the *P* allele is weak ($\alpha = 1.05$), the *P* allele is positively selected for, and fixes in a large region of the parameter space in which the sex-averaged viability effect of the *T* allele is negative (that is, where $(1 + s_f)(1 - s_m) < 1$). Fixation of the *P* allele pushes the *T* allele to high frequency over a small region of parameter space, in which the cost of the trait to males (s_m) is not too large compared to the benefit of the trait to females (s_f). **c**, For slightly higher strengths of the preference encoded by the *P* allele ($\alpha = 1.5$), the allele always fixes and the *T* allele attains high frequency in regions of parameter space where male costs are very high. **d**, When the preference is strong ($\alpha = 7.5$), the *T* allele attains high frequency even when it is nearly lethal in homozygous male bearers, imposing an 80% survival cost on them.

One way to resolve sexual antagonism is to restrict the expression of a trait to the sex it benefits[13,15,19]. Counterintuitively, this is not necessarily the expected outcome for sexually antagonistic traits when they are subject to sex-linked mating preferences. For instance, the presence at high frequency of the W-linked *P* allele can select against modifiers that restrict expression of the *T* allele to females, because female-specific expression, although it increases the viability of males that bear the *T* allele, also decreases their mating success. Sex-linked preferences can thus impede the evolution of sex-specific expression and, by extension, sexual dimorphism[15].

I have thus far limited the discussion to classical sexually antagonistic traits. However, the model applies more generally to three categories of costly male-specific traits: those that (i) increase the fitness of daughters; (ii) have no effect on the fitness of daughters; or (iii) act as an indicator of 'good genes' (for example, classical handicap traits). For category (i), costly male traits that increase offspring fitness are functionally identical to sexually antagonistic traits in my model. Such traits include male parental care[24], which is more common in ZW than XY species[25]. For category (ii), W-linked preferences for traits with no effect on females ($s_f = 0$) but large costs in males ($s_m \gg 0$) evolve neutrally. Such preferences can therefore drift to high frequency, which could possibly drive the evolution of exaggerated male-specific phenotypes that have previously been assumed to be the result of Fisherian runaway processes[2,6,7].

For category (iii), if a male-specific handicap signals intrinsic sex-independent quality[2,6], then a W-linked mating preference for handicapped males is favoured irrespective of the costs of the handicap, because daughters enjoy higher quality without suffering the handicap[22]. An analogous autosomal preference is transmitted to sons half of the time, so the higher quality of the offspring of its bearers must be offset by fitness costs in their handicapped sons. If the handicap is too costly, an autosomal mating preference for it will not spread—although a W-linked preference will. The handicap then signals a 'sexually antagonistic genome': good in females (because of the high quality it imparts) but bad in sons (because of the severe cost of the handicap). Formal modelling of this process (Supplementary Information) reveals: (i) that the W-linked preference is always favoured under the standard 'Spence condition'[6,26] that the viability cost of the handicap is proportionally lower in higher-quality males; (ii) that more stringent conditions are required for the analogous autosomal preference to be favoured; and (iii) that the handicap must be heritable for these differences to hold.

In the above model, the selfish W-linked *P* allele can drive to high frequency a trait that severely impairs male survival. This might create selection for autosomal suppression of the preference encoded by the *P* allele. To study this possibility, I considered an augmented model with a third locus that is autosomal but is not linked to the trait locus. At this locus, there segregates a mutant allele *S* that suppresses the effect of the *P* allele, such that its female bearers are indiscriminate in mate choice (see Methods). Simulations reveal that the *S* allele invades only when the strength of the preference that it suppresses is weak, and when the trait carries a high net cost (Extended Data Figs. 3, 4). Thus, strong W-linked preferences appear to be robust to suppression.

Sex-specific chromosomes (the W or Y chromosomes) are often stereotyped as degraded and gene-poor, which would seem to diminish the possibility of their carrying preference genes. However, although the sex-specific chromosomes of therian mammals and neognath birds are indeed gene-poor, in other clades the sex-specific chromosome can vary widely in size and gene content[14,27]. In addition, sex-specific chromosomes usually contain a non-degraded 'pseudo-autosomal region' that recombines in the heterogametic sex[28]. Simulations reveal that preferences similar to those modelled above can fix in the pseudo-autosomal region, although only if they arise close to the border between this region and the sex-determining region (Extended Data Fig. 5).

The logic articulated above for the W chromosome applies to other genetic elements with exclusive or predominantly maternal transmission. These include mitochondria and other cytoplasmic factors[12,13], intracellular parasites such as *Wolbachia*[29] as well as microbiota, which often show vertical maternal transmission[30] and are known to influence behaviour—including mate choice—in a number of taxa[31].

Although the W chromosome is sex-specific, the Z and X chromosomes are only partially sex-biased, as they are borne twice as often by one sex (males for the Z chromosome and females for the X chromosome). These transmission biases—together with recent discoveries of X- and Z-linked genes that influence mate choice (Supplementary Information)—raise the possibility that the Z and X chromosomes can shape the evolution of preferences for sexually antagonistic traits; the Z chromosome for male-beneficial, female-costly traits and the X chromosome for male-costly, female-beneficial traits. The evolution of X- and Z-linked preferences for costly male-limited traits has previously been considered[21].

Modifying the model for X- and Z-linked preferences (Methods), I find that—in both cases—preference and trait alleles can co-evolve to high frequency (Fig. 2). This effect is stronger for the Z chromosome, despite the 'biases' of the X and Z chromosomes being symmetric.

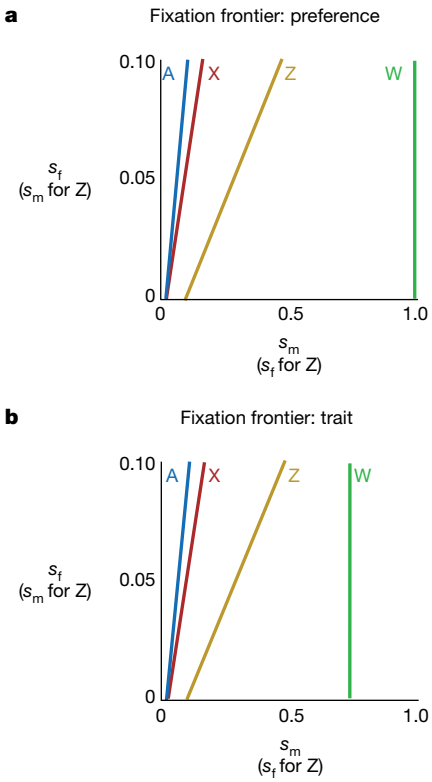

Fig. 2 | Relative propensities of the W, Z and X chromosomes to evolve female mating preferences for males that exhibit sexually antagonistic traits. a, **b**, The preference strength is $\alpha = 5$ in all cases. Each line is a frontier between a parameter region in which the preference (**a**) or trait (**b**) allele attains high frequency (region to the left of the line), and a parameter region in which it does not (region to the right of the line). The frontier for autosomes (labelled A) is displayed for reference. Note that Z-linked preferences are for male-beneficial, female-costly traits (contrary to the W- and X-linked preferences), so the axes are reversed for the Z chromosome. W-linked preferences for males displaying female-beneficial, male-costly traits fix for any degree of sexual antagonism. Z-linked preferences for males displaying male-beneficial, female-costly traits fix even with substantially female-costly traits, although the parameter range over which they fix is smaller than for W-linked preferences. X-linked preferences for female-beneficial, male-costly traits fix only when the degree of sexual antagonism is relatively small, although they nonetheless fix in regions in which autosomal preferences cannot. Note that Z- and X-linked preferences (unlike W-linked preferences) fix only when they also drive their preferred traits to high frequency.

To understand this, consider sex-chromosome transmission from ZW and XX females to offspring. A Z-linked allele that encodes a mating preference for a male-beneficial trait is passed on by a mother only to her sons, and thus gains an immediate advantage. By contrast, an X-linked preference allele is transmitted equally to sons and daughters, and thus immediately experiences both the cost and benefit of the trait. In fact, the pedigree transmission profiles of X- and Z-linked preference alleles, starting in females, are symmetric, except for the initial sons-only generation of the Z-linked allele (Supplementary Information), which explains why Z-linked mating preferences for sexually antagonistic traits evolve more readily. As expected, the effect is weaker for both the Z and X chromosomes than for the W chromosome (Fig. 2).

I have considered a population in which mate choice is practised exclusively by females, but the model also applies to male mate choice, which recent work has suggested is more common than has previously been recognized[32].

To investigate the empirical possibility of sex-linked preferences, I collected a list of known genomic locations of mate-preference genes (Supplementary Information). Sex chromosomes are substantially over-enriched for preference genes across a variety of heterogametic species. Sex-specific chromosomes do not feature prominently, probably because they are highly degenerate in the majority of species in the list. Indeed, one of the major goals of the theoretical work presented here is to point genomic research on mate preferences towards species with gene-rich sex-specific chromosomes.

The model described here predicts different outcomes for XY and ZW systems when mate choice is practised predominantly by females. In ZW species, the female-specific W chromosome is a very strong attractor of preferences for male-costly, female-beneficial traits, whereas the male-biased Z chromosome attracts preferences for male-beneficial, female-costly traits. By contrast, XY species have no female-specific chromosome and the X chromosome attracts preferences more weakly than does the Z chromosome (Fig. 2). Therefore, ZW species are particularly prone to the evolution of sex-linked preferences for sexually antagonistic traits. This is consistent with the phylogenetic association between ZW heterogamety and greater male ornamentation in vertebrates[23], although this relationship is ambiguous within some clades[33]. Further comparative research—especially in clades with rapid heterogametic transitions—would be useful in clarifying this relationship[14].

Online content

Any methods, additional references, Nature Research reporting summaries, source data, statements of data availability and associated accession codes are available at https://doi.org/10.1038/s41586-019-1271-7.

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Competing interests The author declares no competing interests.

Additional information
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METHODS

In all versions of the model considered here, the population is assumed to be infinite, with non-overlapping generations in which the order of events is: viability selection, mating, reproduction and death, followed by viability selection among the offspring, and so on. The organism is diploid with heterogametic sex determination. Mendelian segregation operates among all loci.

The mate choice model is one of fixed relative preferences[4,20]. In general, if there are n types of male (each expressing a different degree of some trait) in proportions $p_1, p_2 \ldots p_n$ at the time of mating (after viability selection), and a given female has relative preference strengths $\alpha_1, \alpha_2 \ldots \alpha_n$ over the male types, then the probability that her next mate is of type i is $\alpha_i p_i / \sum_{k=1}^{n} \alpha_k p_k$. If this female is of type j among m female types (each expressing a different set of preferences over the male types), with female types in proportions $q_1, q_2 \ldots q_m$ after viability selection, then the fraction of all mating events in the population that are between type j females and type i males is $q_j \alpha_i p_i / \sum_{k=1}^{n} \alpha_k p_k$.

In the case of W-linked preferences for autosomal traits, at the W-linked preference locus there segregate the wild-type p allele and the mutant P allele, while at the autosomal trait locus there segregate the wild-type t allele and mutant T allele. The T allele encodes a trait that is costly in males but beneficial in females: tt males and females have a baseline relative viability of 1; Tt males and females have viabilities of $1 - h_T s_m$ and $1 + h_T s_f$, respectively; and TT males and females have viabilities of $1 - s_m$ and $1 + s_f$, respectively. A female bearing the p allele has equal preferences over the three male genotypes, whereas a female bearing the P allele has relative preferences $1, \alpha^{h_T}$ and α over the male genotypes tt, Tt and TT, respectively. The results discussed in the main text (Figs. 1, 2) assume $h_T = 1/2$; results for $h_T = 0$ and $h_T = 1$ are given in Extended Data Fig. 1.

The justification for the specific form of the relative preference of the females bearing the P allele for Tt males (α^{h_T}) is as follows: when $h_T = 0$, such that the T allele is recessive and the trait is not expressed by Tt males, a female that bears the P allele cannot distinguish tt and Tt males—her relative preference for Tt males should therefore be 1 (α^0). When $h_T = 0$, such that that the T allele is dominant, the female cannot distinguish between Tt and TT males; her relative preference for Tt males should therefore be α (α^1). Finally, in the case of exactly intermediate dominance of the T allele ($h_T = 1/2$), the preference of a female that bears the T allele for TT males over Tt males should equal the strength of her preference for Tt males over tt males; this requires that her relative preference for Tt males be $\sqrt{\alpha}$ (that is, $\alpha^{1/2}$). A similar logic will govern the choice of intermediate relative preferences in the case of Z-linked and X-linked preferences.

In the case of X-linked preferences, the viability effects of the T and t alleles in males and females are as for the case of W-linked preferences described above. The dominance of the P allele in females is denoted by h_P: pp females have equal preferences for the three male genotypes tt, Tt and TT; Pp females have relative preferences $1, \alpha^{h_T h_P}$ and α^{h_P}; and PP females have relative preferences $1, \alpha^{h_T}$ and α. The results discussed in the main text (Fig. 2) assume $h_T = h_P = 1/2$; the results for other possibilities are displayed in Extended Data Fig. 1.

For Z-linked preferences, the mutant T allele encodes a trait that is beneficial in males but costly in females: tt males and females have baseline relative viability 1; Tt males and females have viabilities $1 + h_T s_m$ and $1 - h_T s_f$, respectively; and TT males and females have viabilities $1 + s_m$ and $1 - s_f$, respectively. The Z-linked mutant P allele encodes a mating preference for males that bear the T allele in the same way as the W-linked preference described above.

Finally, for the case in which the preference locus is pseudo-autosomal in a ZW system, the viability effects of the T allele and preference effects of the P allele (now at a diploid locus) are as in the case of X-linked preferences, and the preference locus recombines with the sex-determining locus in a fraction r of gametes.

In the simulations, the results of which are displayed in Figs. 1, 2 and Extended Data Fig. 1, the population starts off with initial low frequencies of the mutant P and T alleles (1% each), with the loci in Hardy–Weinberg equilibrium when diploid, and in linkage equilibrium with each other. I assume that the two alleles at the trait locus mutate from one to the other at a symmetrical rate of $u = 10^{-3}$ per replication; there is no mutation at the preference locus (see Supplementary Information for a discussion of the effects of different mutation rates). From this starting configuration in each case, the population model was simulated for 5×10^6 generations (Figs. 1, 2) or 10^6 generations (Extended Data Fig. 1), and the final frequencies of the mutant P and T alleles recorded.

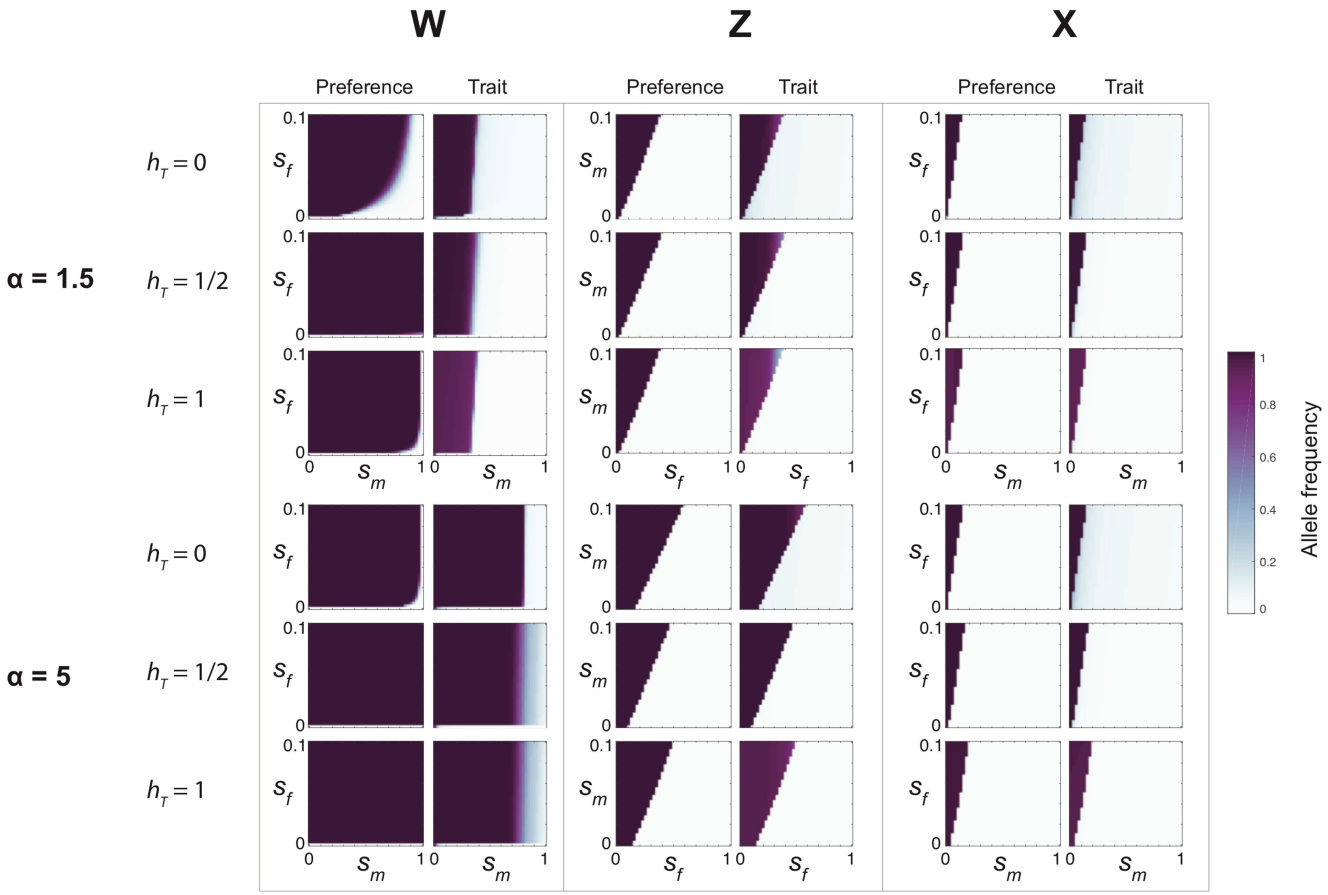
Reporting summary. Further information on research design is available in the Nature Research Reporting Summary linked to this paper.

Data availability

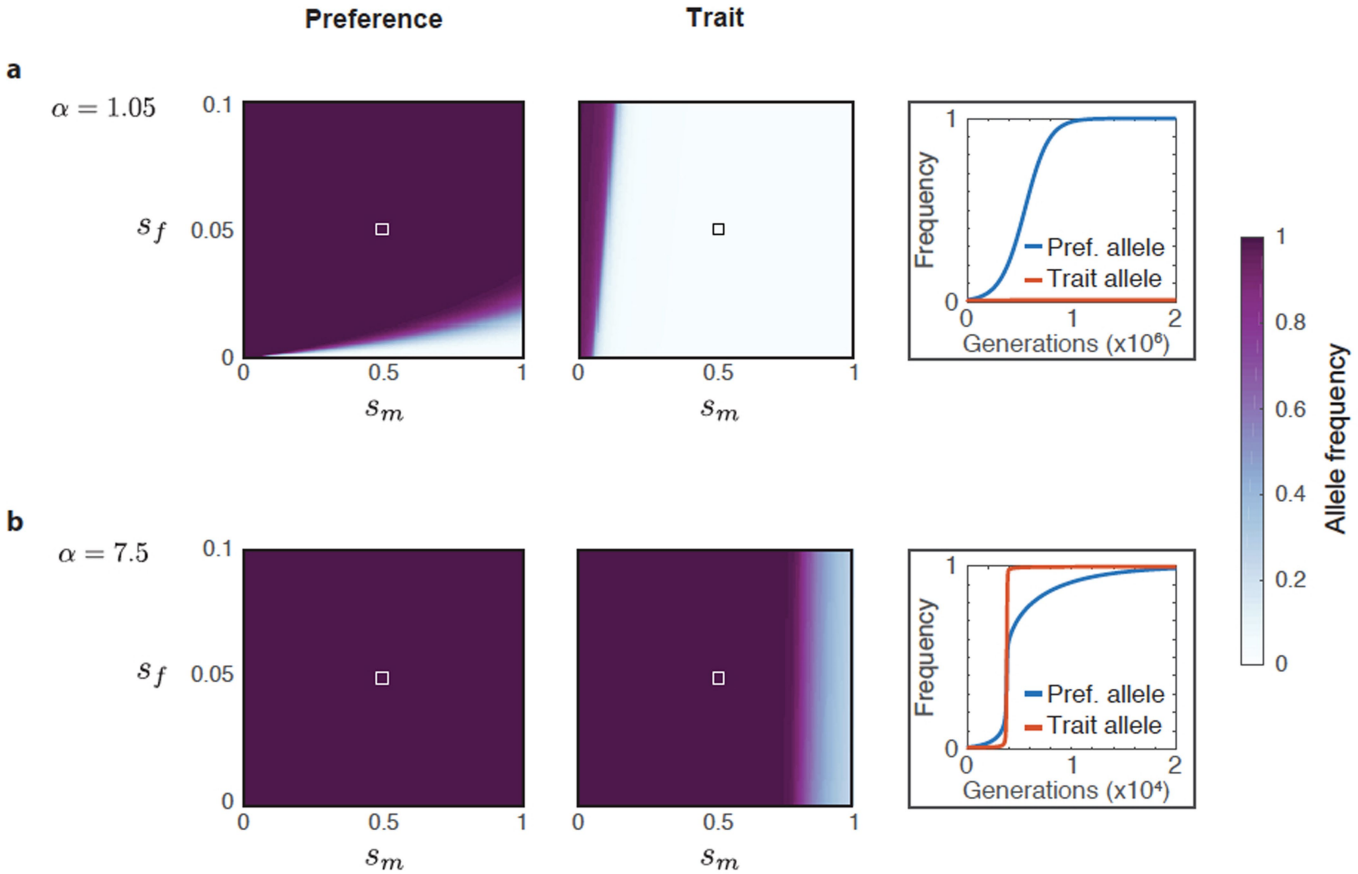
No datasets were generated or analysed in this study.

Code availability

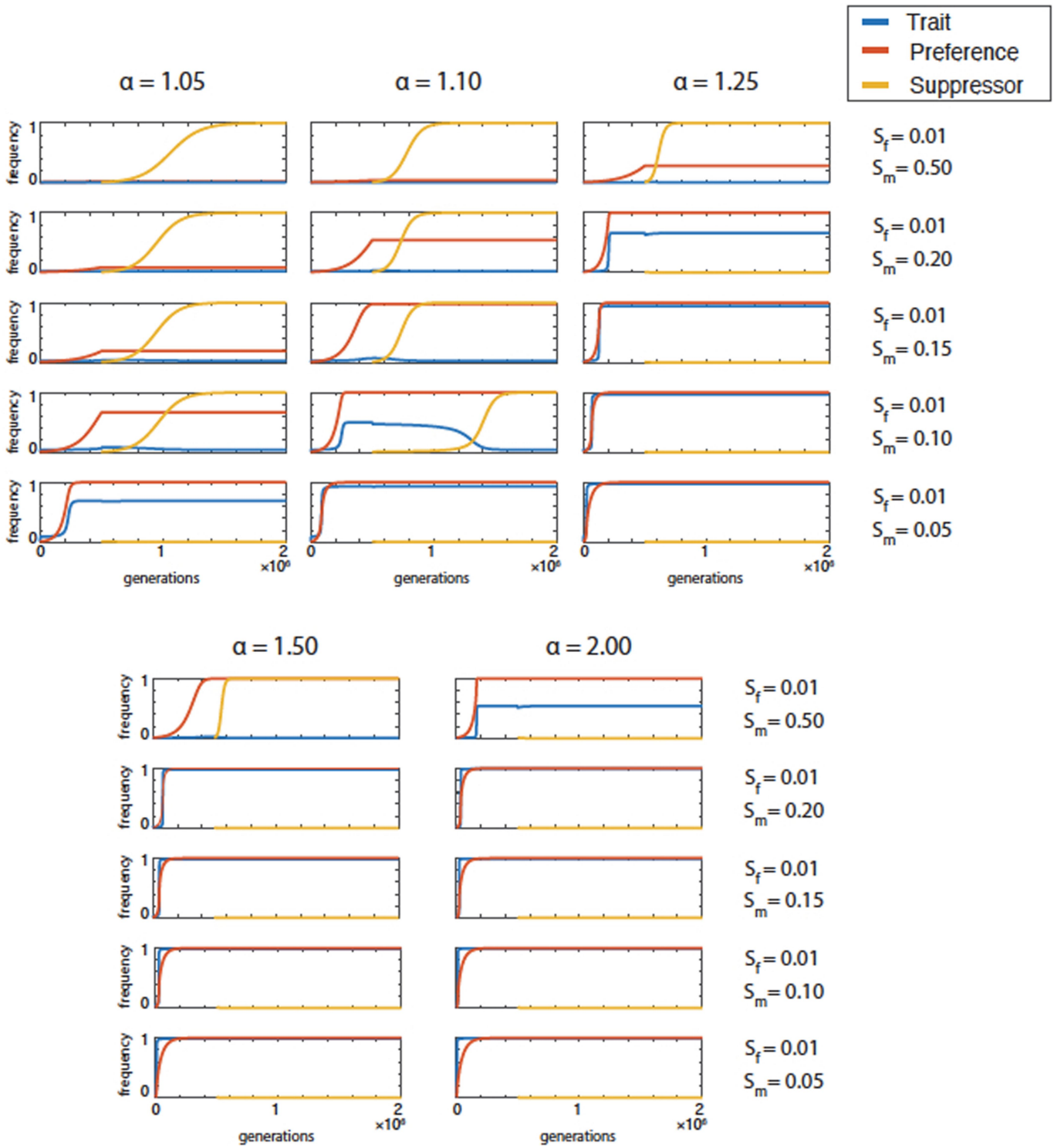
Simulation code is available upon request.



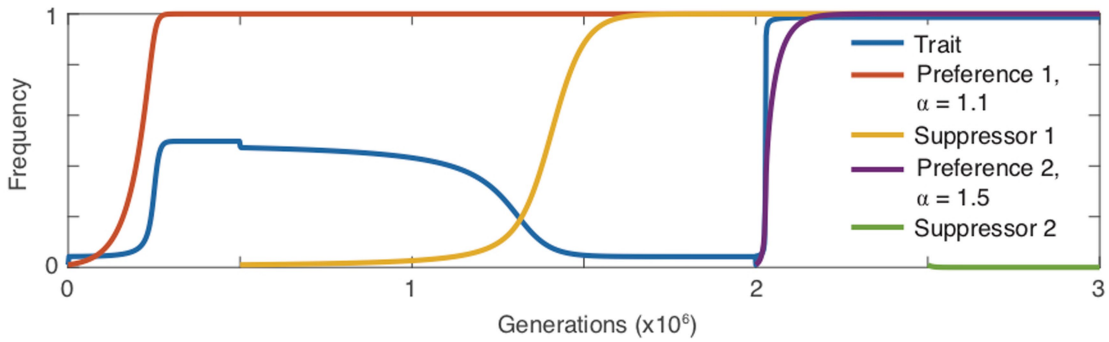
Extended Data Fig. 1 | Long-term frequencies of sex-linked preferences and traits. Frequencies of a W-linked, Z-linked and X-linked mutant *P* allele and an autosomal mutant *T* allele after 10^6 generations, each having started at 1% frequency, in Hardy–Weinberg and linkage equilibrium. The strength of the preference is $\alpha = 1.5$ (top) or $\alpha = 5$ (bottom). h_T is the dominance of the *T* allele with respect to the wild-type *t* allele; h_P is the dominance of the *P* allele with respect to the wild-type *p* allele. In the case of an X-linked preference, I assume that $h_P = h_T$; in the case of W-linked and Z-linked preferences, h_P is not applicable, as both W- and Z-linked preferences are hemizygous in females. Note that in the case of a W-linked preference, the *P* allele will eventually attain high frequency in parameter regions in which it does not appear to do so here; for example, compare the results here for a W-linked preference of strength $\alpha = 1.5$ with Fig. 1c, in which frequencies after 5×10^6 generations are reported.



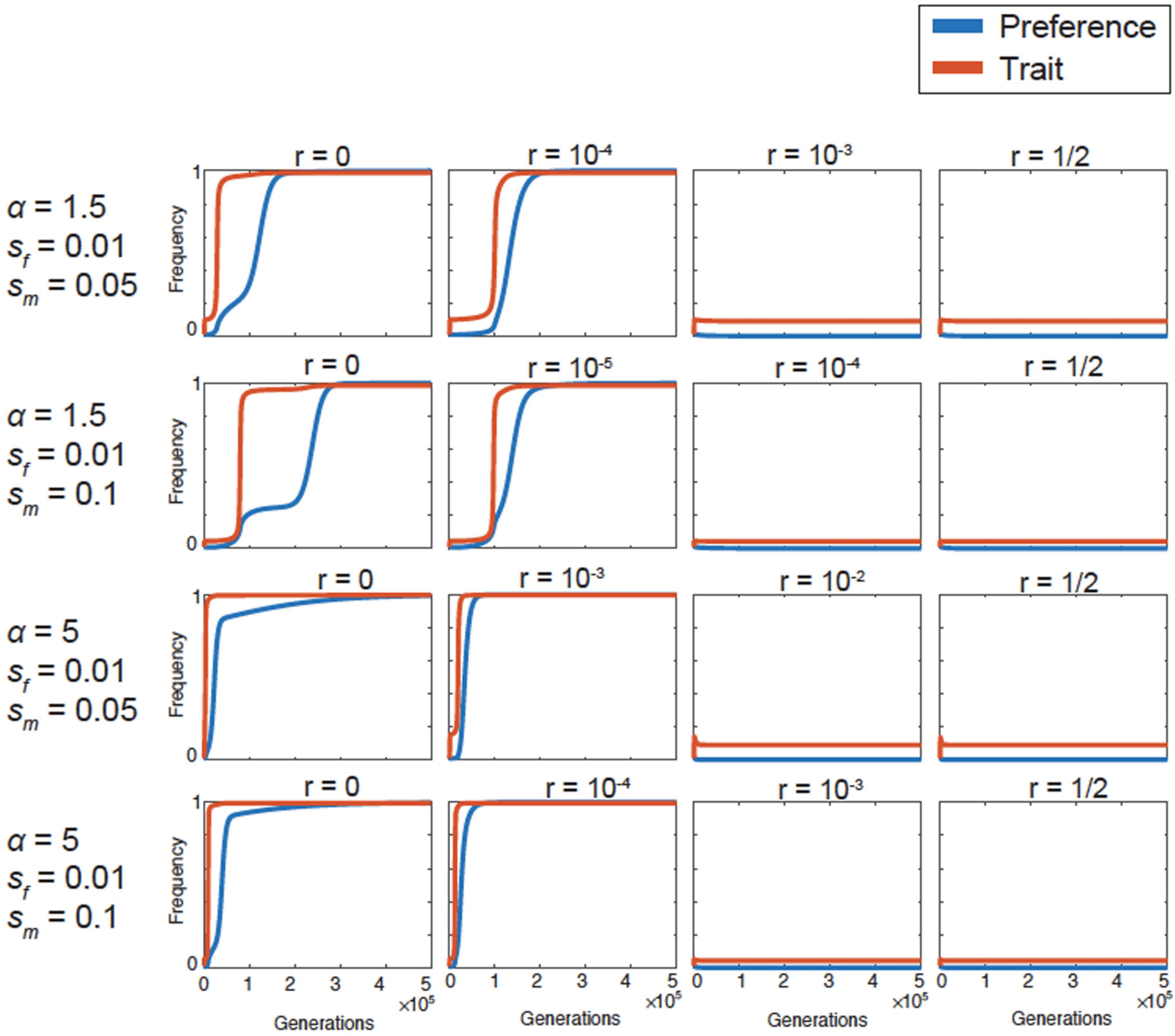
Extended Data Fig. 2 | Two equilibria for W-linked preferences for sexually antagonistic traits. Frequency trajectories of the W-linked *P* allele and an autosomal, male-costly female-beneficial *T* allele under two strengths of the preference. The heat maps displayed here are also shown in Fig. 1b (top) and Fig. 1d (bottom), and their details are described in the Methods. **a**, Sample trajectories of *P* and *T* alleles when the preference is weak and the cost of the trait to males is large. The *P* allele fixes but the *T* allele remains at a low-frequency mutation–selection balance: the equilibrium is one in which all females prefer males that display the costly trait, but very few males display it. **b**, Sample trajectories of *P* and *T* alleles when the preference is strong. The *P* allele fixes and the *T* allele attains a very high-frequency mutation–selection balance: the equilibrium is one in which almost all males have low viability, and all females strongly prefer the low-viability males.



Extended Data Fig. 3 | Suppression of selfish W-linked preferences for sexually antagonistic traits. Trajectories of the W-linked *P* allele, the autosomal, male-costly female-beneficial *T* allele and an autosomal *S* allele that suppresses the preference allele, across various fitness effects of the trait. The mutation rate at the trait locus is 10^{-3} and the *T* allele is co-dominant ($h_T = 1/2$). In each simulation, at generation 0 the *T* and *P* alleles are introduced into the population. After 5×10^5 generations, a mutant *S* allele appears at an autosomal locus. The simulation is run for an additional 1.5×10^6 generations. Each allele is introduced at frequency 1%, in Hardy–Weinberg equilibrium if at a diploid locus, and in linkage equilibrium with respect to the other loci. The autosomal suppressor locus is unlinked to the trait locus, and the *S* allele is co-dominant ($h_S = 1/2$), so that a female bearing the *P* allele and a single *S* allele has preferences 1, $\alpha_i^{1/4}$ and $\alpha_i^{1/2}$ for *tt*, *Tt* and *TT* individuals, respectively, whereas a female with the *P* allele and no *S* allele has preferences 1, $\alpha_i^{1/2}$ and α_i for *tt*, *Tt* and *TT* individuals. Suppression is more likely to evolve when the strength of the W-linked preference is weak, and the average fitness cost of the trait across males and females is high.



Extended Data Fig. 4 | Arms-race dynamics between W-linked preferences and their suppressors. A weak preference allele P_1 initially invades and fixes, which pushes the sexually antagonistic T allele ($s_f = 0.01$, $s_m = 0.1$) to intermediate frequency. At 5×10^5 generations, a mutant allele that suppresses the action of P_1 appears at an unlinked locus. The suppressor invades and fixes, which eliminates the effect of P_1 so that the T allele decreases to a low frequency. At 2×10^6 generations, a medium-strength preference allele P_2 invades and fixes, which pushes T back to a high frequency. An unlinked suppressor of P_2 appears at 2.5×10^6 generations, but immediately goes extinct: the medium-strength preference is evolutionarily resistant to suppression.



Extended Data Fig. 5 | Dynamics of preferences in the pseudo-autosomal region. Trajectories of the pseudo-autosomal mutant *P* allele and autosomal mutant *T* allele in a ZW system, for various strengths of the preference, fitness effects of the trait (always costly in males and beneficial in females) and recombination rates between the preference locus and the sex-determining locus. The mutant trait allele is co-dominant ($h_T = 1/2$). In each case, there is some (low) threshold recombination rate, below which the preference and trait can evolve to high frequency and above which they cannot.

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