# Sexual conflict explains diverse patterns of transgenerational plasticity

Nathan W Burke*[1], Shinichi Nakagawa[1], Russell Bonduriansky[1]

*Corresponding author

[1]Evolution & Ecology Research Centre, School of Biological, Earth and Environmental Sciences, University of New South Wales, Sydney, Australia

## ABSTRACT

Transgenerational plasticity (TGP) occurs when the environment experienced by parents induces changes in offspring traits[1]. Such effects can be adaptive or non-adaptive[2,3] and are increasingly recognised as key determinants of health[4], cognition[5], development[6] and performance[7–9] across a wide range of taxa, including humans. While the conditions that favour maternal TGP are well understood[10,11], rapidly accumulating evidence indicates that TGP can be maternal or paternal [12–15], and offspring responses can be sex-specific[12–18]. However, the evolutionary mechanisms that drive this diversity are unknown. Here we use individual-based models to show that diverse patterns of TGP can evolve when the sexes experience different environments. We find that non-adaptive patterns of TGP result when alleles at loci that determine offspring responses to environmental information originating from the mother and father are subject to sexually antagonistic selection. By contrast, a variety of sex-specific responses evolve via duplication and sex-limitation of loci responsive to parental information, including non-adaptive TGP when sexual selection is strong. Sexual conflict can therefore explain why adaptive TGP evolves in some species but not others, why sons and daughters respond to parental signals in different ways, and why complex patterns of sex-specific TGP may often be non-adaptive.

# INTRODUCTION

Theory predicts the evolution of anticipatory TGP when environmental conditions experienced by parents and offspring are correlated[10,11]. Environmental predictability across generations is thought to allow parents experiencing conditions that fluctuate in space or time to adaptively match the phenotype of their offspring to expected conditions through the transmission of relevant environmental information[8,19]. However, like other forms of plasticity, TGP is not always adaptive. Anticipatory TGP can be costly if the environment of offspring fails to match the parental environment[20], and some environmental factors appear to induce phenotypic changes that are invariably maladaptive or pathological[21,22]. Furthermore, mother-offspring conflict can result in induced phenotypes that optimise mothers' long-term fitness but are suboptimal for individual offspring[23,24]. Although this diversity of effects might explain why evidence of adaptive TGP across studies is weak[3], existing explanations do not account for other important patterns of variation in TGP.

An increasing number of studies suggests that TGP is often sex-specific, both in terms of the parent that transfers environmental information and the offspring that responds. Paternal and maternal environments are known to have contrasting effects on offspring[12–15], and sons and daughters often respond differently to maternal versus paternal information[16–18], leading to diverse sex-specific patterns of TGP, such as mother-daughter, father-son, mother-son and/or father-daughter effects. For example, early-life smoking in human fathers induces larger body mass in sons, but not in daughters[17]. While there is growing interest in the importance of sex-specific TGP in ecology[25], conservation biology[26], gerontology[27], psychology[28], neurology[29], and epidemiology[4], existing models consider only maternal TGP (i.e., environment-induced maternal effects) and typically ignore fathers and sons (e.g., see[23,24]). Thus, it remains unclear why maternal and paternal environments often have contrasting phenotypic effects on offspring, and why sons and daughters often respond differently depending on the sex of the parent that transfers environmental information.

The sex-specificity of many observed instances of TGP suggests that sexually antagonistic selection could play a role in the evolution of these effects. Many genetically determined traits expressed in both sexes have separate male and female fitness optima[30]. This form of genetic conflict between the sexes ("intralocus sexual conflict") can result in either an evolutionary stalemate, where each sex expresses the trait sub-optimally[31], or a resolution,

where sex-linked modifiers or duplicated genes[32,33] allow for optimal, sex-specific trait expression (i.e., optimal sexual dimorphism)[34]. Loci that control offspring responses to parental information may be subject to intralocus sexual conflict if the sexes consistently experience different environments. In dioecious organisms, males and females are often selected to utilise environments in different ways[35], which can lead to sex differences in aggregation[36], dispersal[37], foraging[38], predation[39], parasitism[40], nutrient intake[41] and physiological stress[42]. Environmental change can have sex-specific effects as well[43], and such sex-differences are expected to be consistent across generations. Thus, environmental changes experienced by mothers (fathers) are likely to predict changes experienced by daughters (sons). Although such parent-offspring correlations are a key condition for the evolution of adaptive TGP[11], the implications of sex-specific correlation of environments across generations have not been considered previously.

Patterns of sex-specific selection that are stable over many generations are expected to drive the evolution of genetically based sexual dimorphism. However, predictable environmental fluctuations that affect the sexes differently could favour the evolution of sex-specific TGP through females and males transmitting contrasting information to their offspring. If the sexes share a common genetic architecture for response to parental information, progeny that pay attention to their same-sex parent may gain a fitness benefit because their induced phenotype will match the expected environment for their sex, but progeny that pay attention to their opposite-sex parent are likely to pay a fitness cost resulting from a mismatch between their phenotype and environment. Whether this conflict plays out in stalemate or resolution could have profound consequences for resulting patterns of TGP.

## THE MODEL

Using a spatially explicit, individual-based model, we investigated the role of sexually antagonistic selection in the evolution of TGP under two scenarios, where loci responsive to environmental information originating from parents are (1) subject to persistent intralocus sexual conflict (‘ongoing conflict’ model); and (2) sex-specific and sex-limited and therefore not subject to sexually antagonistic selection (‘resolved conflict’ model). Each version of the model considers a scenario typical of organisms that show anticipatory TGP in which periodic environmental change (such as higher predation risk[7], increased temperature[15], or

greater food scarcity[44]) causes costly physiological stress. We assume that stressed individuals cannot respond directly to environmental change but transfer information about the changed environment to their offspring via an acquired epigenetic mark that can induce development of a stress-resistant phenotype in the offspring. We model scenarios where environmental change affects the sexes in either similar or different ways by independently manipulating the sexes' experience of stress (for a full description of the lifecycle, see Supplementary Methods and Supplementary Figure S1). Existing models of anticipatory TGP assume periodic shifts from one environmental state to another[23,24]. For simplicity, we consider a single period of environmental change, but simulations with multiple periodic shifts produce very similar results (see Supplementary Figure S2).

Our model is based on an "offspring's eye view" of TGP, whereby selection acts on offspring responses to information received from the father and mother (i.e., whether offspring "listen" to parental information). This reflects the fact that offspring may be able to optimise the use of parental information based on their own sex and the sex of parents, whereas parents may have a more limited ability to optimise information transmitted to male versus female offspring. In our model, offspring that listen to epigenetic marks transmitted by stress-exposed parents develop a stress-resistant phenotype that is adaptive in stressful conditions but costly and maladaptive in benign conditions. Offspring that do not inherit epigenetic marks, or inherit a mark but do not listen to it, adopt a default phenotype that is adaptive in benign conditions but maladaptive in stressful conditions. Whether or not offspring listen to parentally transmitted epigenetic marks is determined by alleles at "listening" loci (i.e., loci responsive to environmental information; see Supplementary Methods).

Mating and reproduction occur once at the end of life. The proportion of the lifetime spent phenotypically matched to the environment ($\partial_{ind}$) affects resource accumulation which determines condition ($C_{ind}$). In males, condition determines competitiveness ($Q_{ind} = C_{ind}$), whereas in females condition determines fecundity ($W_{ind} = C_{ind}$). These positive linear relationships reflect the strong condition dependence of male secondary sexual traits[45] and female fecundity[46] in natural systems. At the end of each generation, females mate once with the male in their neighbourhood that has the highest $Q_{ind}$ value ('best male'), or, as a control, a random male from the same neighbourhood ('random male'). To investigate the complex dynamics that can result from strong sexual and sexually antagonistic selection[47,48], we alter

the strength of sexual selection on males by varying the size of the mating neighbourhood, with larger neighbourhoods generating a larger skew in male mating success.

# RESULTS & DISCUSSION

## *Ongoing conflict*

We first assume a diploid bi-sexual organism with two autosomal listening loci that control embryos' ability to respond to the epigenetic signal from their father (locus *A*) and mother (locus *B*), and which have sex-independent effects on offspring. The introduction of listening alleles at these loci leads to four evolutionary outcomes: paternal TGP (allele *A* fixes), maternal TGP (allele *B* fixes), both types of TGP (alleles *A* and *B* fix), or no TGP (neither *A* nor *B* fixes) (see Supplementary Table S1). The relative level of stress experienced by each sex largely determines the probability of these outcomes (see Figure 1(a)). When males experience higher stress than females, allele *A*, which controls listening to fathers, is favoured in sons but disfavoured in daughters because the stress-induced phenotype that listening permits improves the phenotypic match of sons but decreases the match of daughters. However, allele *B*, which controls offspring listening to mothers, is neutral, since mothers rarely experience stress. Despite selection on sons to pay attention to paternal information under these settings, paternal TGP does not readily evolve because the benefit to sons of carrying allele *A* is not enough to overcome the cost to daughters of carrying the same allele (hence, the widespread lack of evolution of paternal TGP in the 'best male' graphs in Figure 1(a)). A similar but converse situation occurs when mothers experience higher stress than fathers (see 'best male' graphs in Figure 1(a)). Thus, despite the benefit of listening for the stressed sex, intralocus sexual conflict at listening loci constrains the evolution of TGP because of the costs that listening imposes on the opposite sex. Sex-differences in environmental stress[42] (and the conflict that this engenders) could therefore explain the heterogeneous patterns of TGP seen in nature, including why such effects appear to be non-adaptive and are often not found despite their predicted adaptive benefit[3].

Listening alleles mediate the level of match ($\partial_{ind}$) between an offspring's phenotype and its environment, which in turn determines its fitness. Thus, the effect that sexual selection on males and fecundity selection on females has on offspring fitness can be observed in distributions of $\partial_{ind}$ (see Supplementary Figure S3(a)), and has important consequences for

listening outcomes. In the 'random mating' version of the model, where selection on males is absent (Figure 1(a)), offspring consistently evolve to listen to mothers when females experience higher stress than males because fecundity selection favours listening in daughters. However, when males experience higher stress than females in the absence of sexual selection, fecundity selection on females disfavours listening to maladaptive paternal information, thereby preventing the evolution of any TGP (see 'random mating' graphs in Figure 1(a)). However, the presence of sexual selection on males provides a counterbalance to fecundity selection and largely prevents optimal listening. Males gain an ever-larger edge in the conflict as the intensity of sexual selection increases, as evidenced by the reduced number of simulations ending in maternal TGP, and the increased number of simulations ending in paternal TGP (see 'best male' graphs in Figure 1(a)). When sexual selection and fecundity selection are similar in strength but opposite in sign, stable sex-specific listening polymorphisms evolve (Supplementary Figure S4). These intermediate allele frequencies are associated with bimodal distributions of $\partial_{ind}$, whereby each sex exhibits both high and low matching (see Supplementary Figure S3(a)), reflecting an evolutionary stalemate between the sexes. The evolution of listening polymorphisms suggests that intralocus sexual conflict could potentially explain why support for adaptive TGP is generally weak[3]. Indeed, the ubiquity of sex-specific ecologies in natural systems[36–43], and the conflicting signals that parents in such ecologies are likely to send to offspring, may often prevent the evolution of adaptive TGP.

In simulations in which neither parent is stressed, listening alleles are neutral and there is no selection for listening (Supplementary Figure S3(a)). However, in cases where mothers and fathers are both stressed, listening to either parent is favoured in both sons and daughters (Supplementary Figure S3(a)), and the particular form of TGP that evolves (paternal, maternal or both) is determined by whichever allele happens to spread fastest (Figure 1(a)). This suggests that adaptive, sex-independent TGP[12,15] is likely to evolve only when the stress ecologies of the sexes are similar and conflict is absent.

### *Resolved conflict*

When intralocus sexual conflict is resolved through the duplication and sex-limitation of listening loci[33], the sexes can pursue optimal strategies free from the constraints of a shared genetic architecture. This resolution allows for simulations of the resolved conflict model to

end in any combination of sex-specific maternal and paternal TGP (see Supplementary Table S1).

The resolution of intralocus conflict is characterised by high values of $\partial_{ind}$ for both sexes (see 'best male' graphs in Supplementary Figure S3(b)) and sex-specific listening outcomes (see 'best male' graphs in Figure 1(b)). When females experience higher stress than males, fecundity selection drives daughters to listen exclusively to mothers (see 'best male' graphs in Figure 1(b)). Conversely, when males experience higher stress than females, sexual selection drives sons to evolve to listen exclusively to fathers (see 'best male' graphs in Figure 1(b)). This suggests that sex-specific TGP, where offspring listen only to their same-sex parent[16–18], could reflect intralocus sexual conflict that has been resolved by sex-limited listening loci. The evolution of sex-specific listening has the potential to mitigate intralocus sexual conflict in a similar way to parent-of-origin effects on gene expression[49].

A diversity of listening combinations evolves when both parents experience stress (see 'best male' graphs in Figure 1(b)). This diversity results from the random fixation of equally beneficial alleles: if both parents send identical stress signals, sons and daughters can reap identical benefits by listening to either parent. Very few simulations in this parameter space end in only one sex listening because sexual selection on males and fecundity selection on females favour sex-specific listening. By contrast, when both sexes experience stress but sexual selection is absent, listening evolves only in daughters (see 'random mating' graphs in Figure 1(b)). These results suggest that sex-specific TGP where sons and daughters both listen to the same-sex or opposite-sex parent can be favoured when both sexes initially experience similar stresses and sexual conflict has been resolved but where the sexes no longer experience sex-specific ecologies—a situation that may be uncommon in nature.

We also find that strong sexual selection can lead to stochastic patterns of sex-specific listening. As expected, no TGP evolves if both parents are unstressed and sexual selection is weak (see 'best male' graphs in Figure 1(b)). However, as sexual selection intensifies, a diversity of combinations of sex-specific TGP evolves. Indeed, 11 of the 15 possible listening combinations evolve in our simulations at the highest sexual selection intensity (see 'best male' graphs in Figure 1(b)). This diversity occurs because the skew in male fitness generated by intense sexual selection substantially reduces effective population size, causing neutral alleles carried by the most successful males to reach fixation. The diversity of

combinations that evolve stochastically without selection in the resolved conflict model suggests that many of the sex-specific parental effects seen in nature[16–18] may be non-adaptive. Rather, diverse combinations of sex-specific TGP could result from neutral listening alleles hitchhiking to fixation via linkage disequilibrium with alleles that affect fitness.

Our model suggests a number of novel predictions. First, species in which the sexes experience substantially different ecologies may be more likely to exhibit non-adaptive TGP due to ongoing intralocus conflict at listening loci. However, such species may also be more prone to sex-specific TGP if conflict is resolved through the evolution of sex-specific listening loci, with mother-daughter (father-son) effects most likely when females (males) experience greater stress. Second, sex-independent TGP may be common in species in which males and females experience similar ecologies and no sexual conflict. Although our model suggests that sex-specific TGP can result from sex-independent ecologies as well, the conditions that generate such outcomes may be uncommon. Third, complex patterns of sex-specific TGP are likely to be non-adaptive, and may be most common in species in which males are subject to strong sexual selection. Future studies could test these predictions by investigating the genetic architecture of loci involved in TGP and selection on expression of parentally transmitted information in both sexes. The diversity of listening outcomes generated by our model is consistent with the diverse patterns of TGP seen in nature across a wide range of taxa[5,6,17,18,22,7–9,12–16]. Our model therefore provides a unifying framework for understanding the origin and maintenance of diversity in observed patterns of TGP.

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## DATA AVAILABILITY

The simulation data that support the findings of this study are available on Dryad Data Repository:

https://datadryad.org/stash/share/a_c-2-t-hugOmUf0IdBKmw6gcL8ujoC_eDKVovj39fA

## CODE AVAILABILITY

The IBM code that generated the data is available on Dryad Data Repository:

https://datadryad.org/stash/share/a_c-2-t-hugOmUf0IdBKmw6gcL8ujoC_eDKVovj39fA

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## SUPPLEMENTARY METHODS

Our individual-based model considers a dioecious, bi-sexual population with discrete generations distributed over 21 x 21 patches in a torus-shaped world with no limitation on the number of individuals per patch. Modeling individuals within an explicit spatial structure allows us to investigate the consequences of variation in sexual selection intensity (which we model by varying the number of patches in an individual's mating neighbourhood: 1 patch, 9 patches, 25 patches, 49 patches), and to assess the contribution of stochastic processes that

often occur in finite populations. Patches vary in the amount of food they contain, which is determined at the start of each generation by assigning patches a randomly sampled value from the discrete uniform distribution $\mathrm{U}\{0, 2\}$. Food resources are fixed for the duration of each generation and cannot be reduced or depleted. Random movement of individuals between patches every timestep generates spatial and temporal variation in within-patch population density, within and between generations. In each generation, individuals perform the following ordered tasks: moving, eating, encountering, mating, reproducing, dying. Males vary in competitive ability ($Q_{ind}$), and this results in sexual selection at the mating stage. Fecundity selection on female reproductive output ($W_{ind}$) occurs at the reproduction stage. In both cases, selection acts on phenotypic match ($\partial_{ind}$), which affects condition ($C_{ind}$), $Q_{ind}$ and $W_{ind}$ (see below).

In the ongoing conflict model, two loci (*A* and *B*) determine offspring responses to information from stress-exposed fathers and mothers, respectively. Two alleles segregate at each locus (wildtype: *a* and *b*; mutants: *A* and *B*), with additive effects on the probability of listening (0% chance of listening: *aa* and *bb*; 50% chance of listening: *Aa* and *Bb*; 100% chance of listening: *AA* and *BB*). In the resolved conflict model, listening loci are duplicated and sex-limited: locus *C* controls sons' listening to fathers, locus *D* controls sons' listening to mothers, locus *E* controls daughters' listening to fathers, and locus *F* controls daughters' listening to mothers. Two alleles segregate at each locus (wildtype: *c*, *d*, *e*, and *f*; mutant: *C*, *D*, *E*, and *F*). with additive effects on the probability of listening (0% chance of listening: *cc*, *dd*, *ee* and *ff*; 50% chance of listening: *Cc*, *Dd*, *Ee* and *Ff*; 100% chance of listening: *CC*, *DD*, *EE* and *FF*).

The life cycle initially starts with the emergence of adult individuals (see Supplementary Figure S1). These individuals roam randomly from patch to patch encountering conspecifics and acquiring resources. Adults experience stress when interacting with conspecifics, and males and females can experience different levels of stress when one sex is more likely to interact with surrounding individuals. We assume that the rate of encounter is random, but the rate of interaction with encountered individuals can be sex-specific. Stress is triggered when an individual's cumulative count of intra-patch encounters ($K_{ind}$) surpasses a threshold $\alpha$, that is, when $K_{ind} \geq \alpha_m$ or $K_{ind} \geq \alpha_f$, where $\alpha_m$ ($\alpha_f$) is the number of encounters that males (females) can withstand without becoming stressed, given their sex-specific rates of

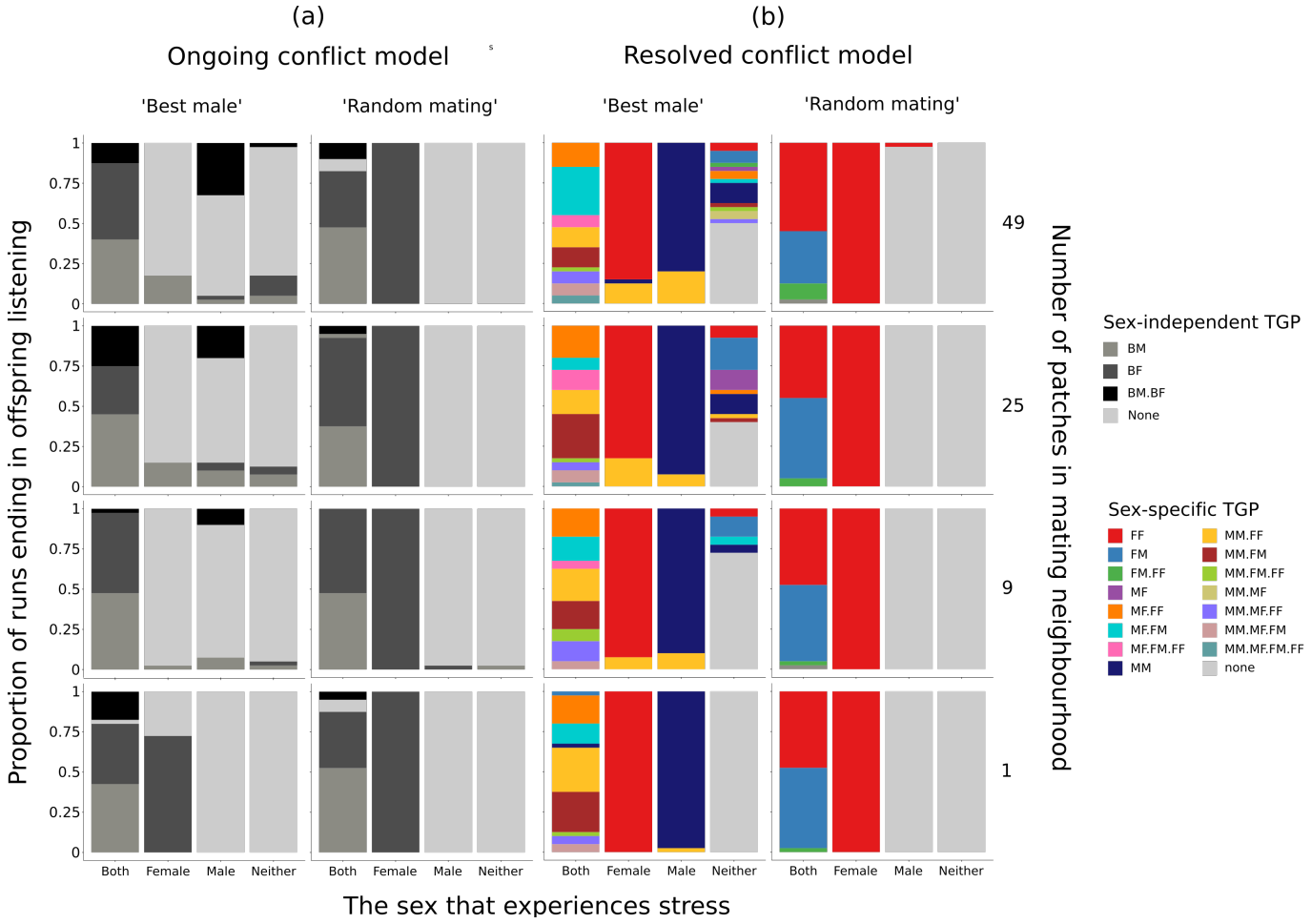
interaction with encountered individuals. $\alpha_m$ and $\alpha_f$ are allowed to vary for a fixed high or low value (80 and 8, respectively), such that the sexes can have a similar or different experience of stress. Thus, a high value of $\alpha_m$ and low value of $\alpha_f$ would represent a scenario where males rarely interact with surrounding conspecifics and therefore experience their environment as benign, and females regularly interact with conspecifics and therefore experience their environment as stressful. Adults cannot plastically change their own phenotype to cope with elevated stress. However, stress induces an epigenetic mark in the germ-line via which information about the stressfulness of the environment is passed on to progeny. Progeny utilise this information to develop a stress-adapted phenotype if they carry appropriate listening alleles. While we focus on TGP triggered by high rates of interaction with conspecifics, our model generalizes to any type of adaptive TGP in response to a sex-specific environmental challenge ("stress"), such as social interactions, predation, parasitism, thermal stress, diet change, or starvation.

Individuals with the default (non-stress-resistant) phenotype are considered well-matched if they experience benign conditions (i.e., if $K_{ind} < \alpha_m$ or $K_{ind} < \alpha_f$) and mismatched if they experience stressful conditions (i.e., if $K_{ind} \geq \alpha_m$ or $K_{ind} \geq \alpha_f$). Conversely, individuals with the stress-resistant phenotype are considered well-matched if they experience stressful conditions (i.e., if $K_{ind} \geq \alpha_m$ or $K_{ind} \geq \alpha_f$) and mismatched if they experience benign conditions (i.e., if $K_{ind} < \alpha_m$ or $K_{ind} < \alpha_f$). Phenotypic match determines an individual's ability to accumulate resources, and therefore its condition at reproduction ($C_{ind}$), such that $C_{ind} = \partial_{ind}.\varphi_{ind}$, where $\partial_{ind}$ is the proportion of the lifetime (fixed at 20 timesteps) spent matched to the environment, and $\varphi_{ind}$ is the total quantity of resources encountered during the lifetime while moving randomly through the environment. The greater the proportion of time spent matched, the greater the amount of resources that an individual is able to accumulate, and the higher its condition at reproduction. Condition at reproduction determines female fecundity ($W_{ind}$) and male competitiveness ($Q_{ind}$). A male's mating success is determined by his value of $Q_{ind}$ relative to that of competitors in his neighbourhood.

At the end of each generation, females mate once with the male in their neighbourhood that has the highest condition ($C_{ind}$) ('best male' model), or a random male ('random mating' model). We assume no male contribution to fecundity. Reproduction occurs immediately

following mating, within the same timestep. Females produce a total of $W_{ind} = C_{ind}$ offspring (to the nearest integer), with sons and daughters equally likely to be produced. Both parents faithfully pass on their experience of stress to the zygote via the gametes. Zygotes that carry the epigenetic mark induced by parental stress and alleles coding for a developmental response to this epigenetic information (i.e., listening alleles) develop a stress-resistant phenotype. Zygotes that do not carry listening alleles cannot alter their development in response to epigenetic information, and instead develop the default phenotype. The phenotypic effect of receiving the mark from both parents is the same as receiving it from one parent. Offspring that receive conflicting information listen to the signal of the stressed parent. The total number of offspring, $n_{gen}$, produced at the end of a generation is always more than can survive (i.e., greater than the global carrying capacity, $P$). To maintain a stable population size, $n_{gen} - P$ offspring are killed randomly before eggs hatch. We assume $P = 1000$ for all simulations. The lifecycle repeats following the death of all parents and emergence of hatchlings.

In both the ongoing conflict and resolved conflict models, mutations at listening loci are introduced haphazardly at the start of generation 25 following a short burn-in period to allow population dynamics to stabilise, such that wildtype alleles are replaced by listening alleles, and *vice versa*, at an ongoing per-locus per-timestep rate $r$ (fixed at $r = 0.001$). To assess the role of sexual conflict in the evolution of listening alleles, we varied parameters controlling male and female sensitivity to conspecific encounter, $\alpha_m$ and $\alpha_f$, and the intensity of sexual selection on males. We ran 40 simulations per parameter combination for 1000 generations and recorded allele frequencies at each listening locus at the end of each run. We considered listening to have evolved if the frequency of the listening allele was $\geq 0.95$. To understand how selection was acting on the sexes, we also recorded the sex-specific distribution of phenotypic matching for each parameter combination.

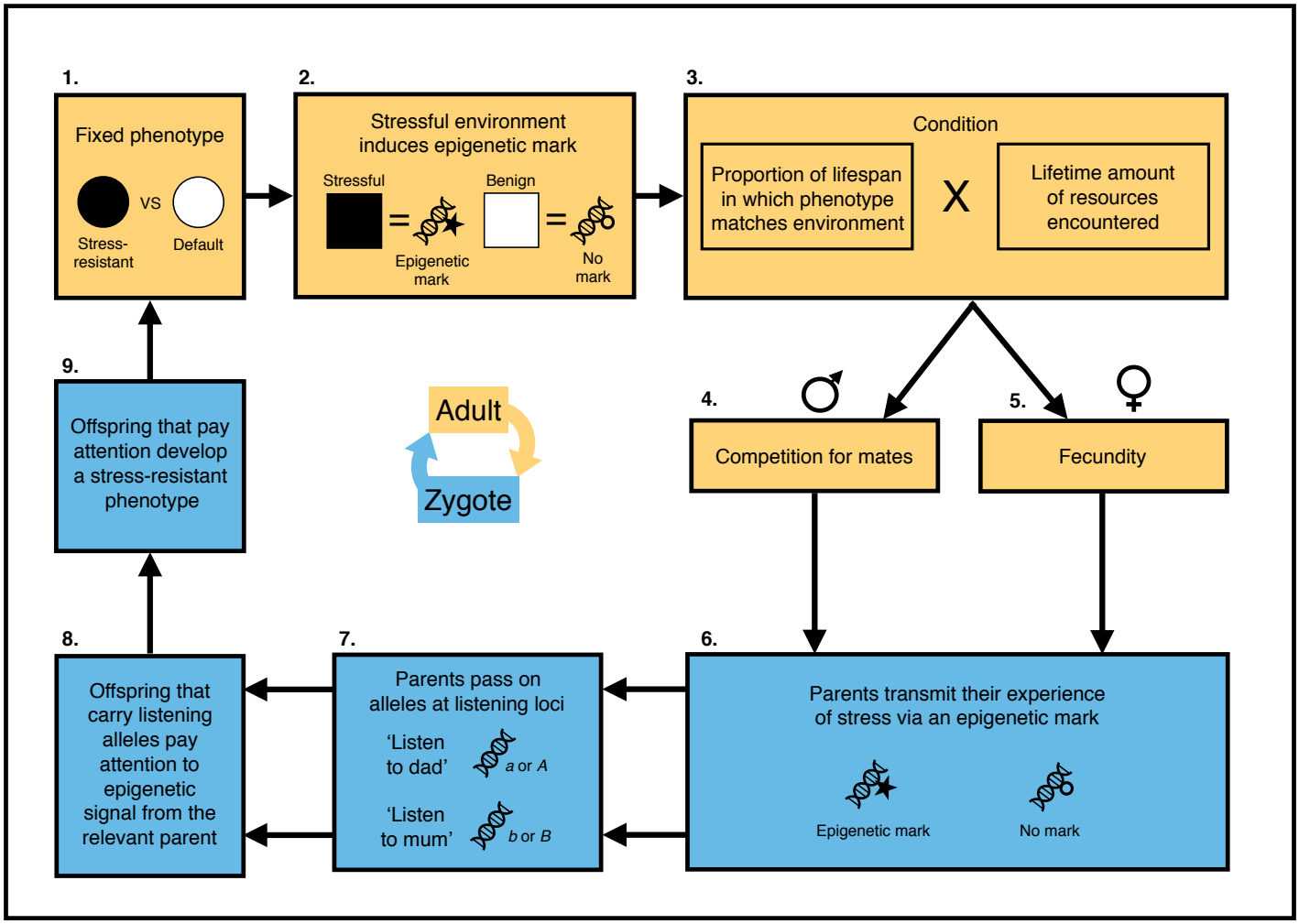



# FIGURE 1

**Sex-independent listening (a) and sex-specific listening (b)**

Graphs on the left show the proportion of runs of the ongoing conflict model ending in sex-independent paternal TGP (dark grey), maternal TGP (medium grey), both types of TGP (black), and no TGP (light grey) at generation 1000. Graphs on the right show the proportion of simulation runs of the resolved conflict model ending in sex-specific TGP at generation 1000. Colours in the legend represent all possible combinations of listening. The first letter in each double-letter code represents the sex of the offspring that does the listening (M = males; F = females; B = both sexes), whereas the second letter represents the sex of the parent that is listened to (M = males; F = females). For example, BM indicates a sex-independent paternal effect (i.e., sons and daughters both listening to fathers), and MF indicates a son-specific maternal effect (i.e., sons listening to mothers). Colours with more than one double-letter code indicate runs where more than one combination of sex-independent or sex-specific TGP

evolves. Runs were counted as ending in TGP if listening allele frequencies were $\geq 0.95$. In (a), when one parent experiences higher stress than the other, intralocus conflict is generated between sons and daughters over whether to pay attention to information received from the stressed or unstressed parent. In (b), sex-specific listening loci allow sexual conflict to be resolved via the evolution of sex-specific TGP.

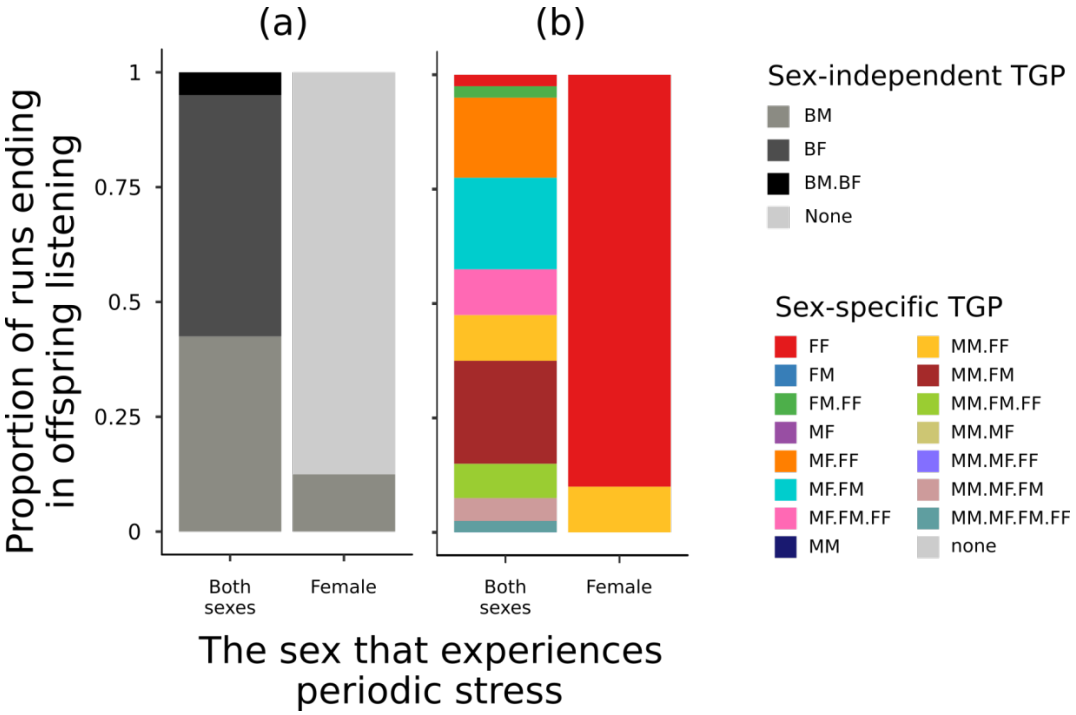



## SUPPLEMENTARY FIGURE S1

**Lifecycle of organisms in the ongoing conflict model**

At the start of the lifecycle, adult individuals emerge with a default phenotype suited to benign conditions that is fixed for the lifetime (1). A stress-resistant phenotype is also possible but must be induced transgenerationally. Individuals experience stress when they interact with too many conspecifics. Stress induces an epigenetic mark (2), which is later transferred to offspring. The extent to which an individual's phenotype matches its experience of stress modulates the amount of resources it accumulates in its lifetime to determine condition at reproduction (3). For males, condition determines mating success (4), whereas for females, condition determines fecundity (5). Females mate with the most competitive male in their mating neighbourhood, or with a random male as a control. At reproduction, both parents faithfully pass on their epigenetic marks (6) as well as listening alleles (7). Zygotes that carry listening alleles utilise their parents' epigenetic signals (8) to

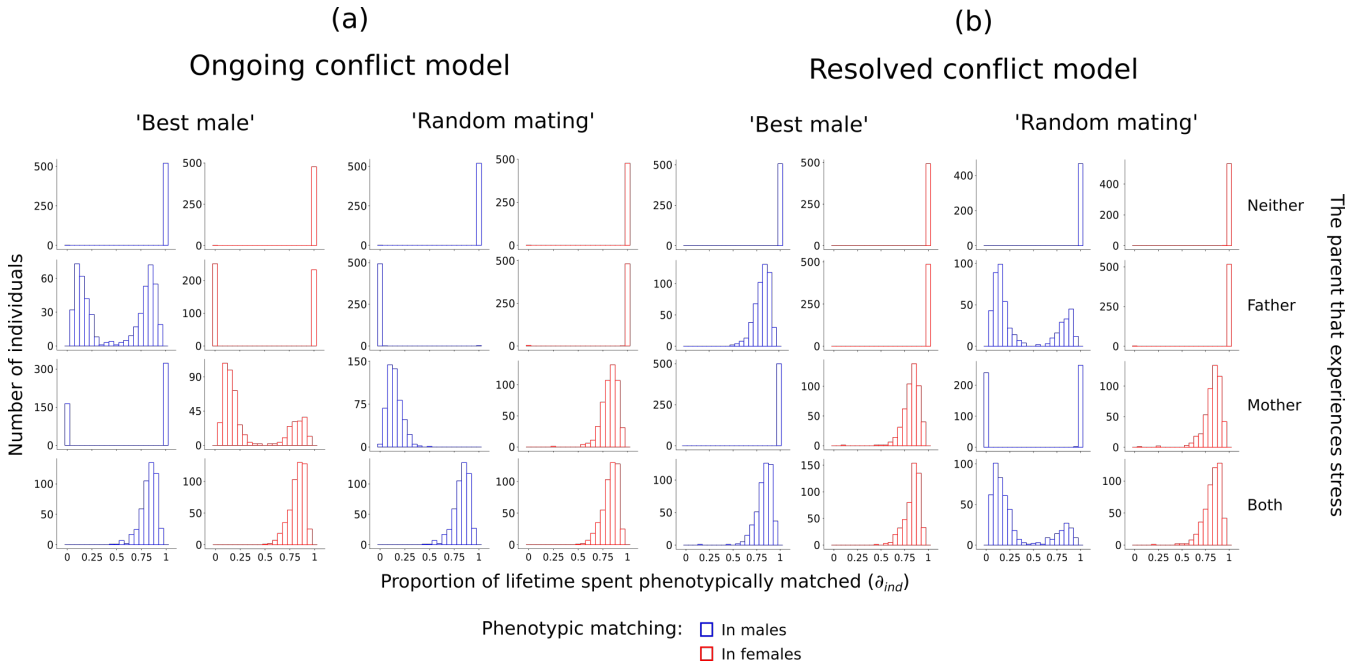
develop a stress-resistant phenotype (9). Zygotes without listening alleles ignore parental information and develop a default phenotype suited to benign conditions (1). The same lifecycle applies to the resolved conflict model except that sex-specific listening loci *C*, *D*, *E* and *F* replace loci *A* and *B*.




## SUPPLEMENTARY FIGURE S2

**Sex-independent listening (a) and sex-specific listening (b) when environmental stress is periodic**

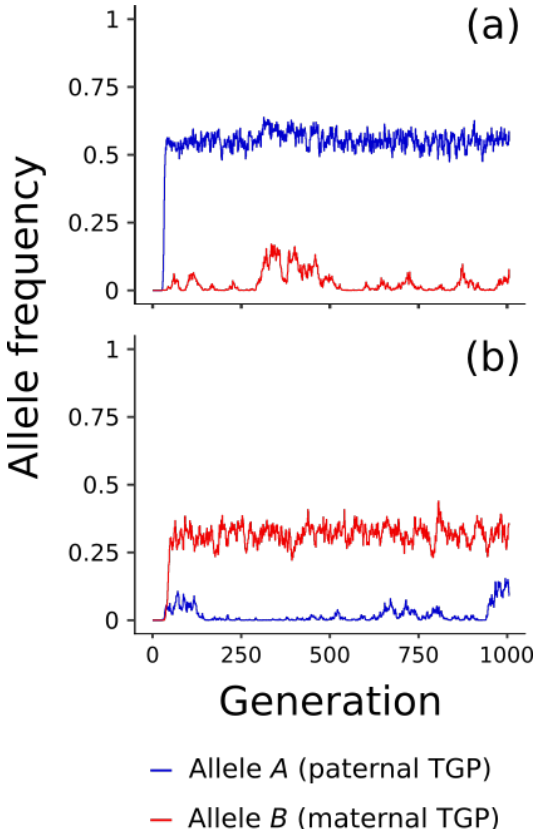
Fluctuating stress generates patterns of TGP that are very similar to those resulting from a single, extended bout of elevated stress (see main text). The plot shows the proportion of 40 simulation runs ending in listening outcomes at generation 1000 when conditions alternate between benign and stressful every 25 generations, causing females or both sexes to experience fluctuating stress. Other settings are: 'best male' version of each model; mating neighbourhood size = 25 patches.




# SUPPLEMENTARY FIGURE S3

**Patterns of phenotypic matching ($\partial_{ind}$)**

Histograms show the outcome of selection on males (blue) and females (red) at the end of simulation runs of the ongoing conflict model (a) and resolved conflict model (b) at generation 1000. In (a), divergence in sex-specific matching is indicative of intralocus sexual conflict and occurs when one parent experiences higher stress than the other. These divergent distributions become bimodal under 'best male' settings due to the opposing action of sexual selection on males and fecundity selection on females. By contrast, the absence of sexual selection on males under 'random mating' allows fecundity selection to maximise female matching regardless of which parent experiences stress. The resolution of sexual conflict in (b) allows each sex to achieve maximal matching at the same time. However, the absence of selection on males under 'random mating' limits maximal matching in males. For the unstressed sex in both (a) and (b), distributions of $\partial_{ind}$ show little variation around the peak because individuals of this sex rarely encounter more conspecifics than the high encounter-sensitivity setting, and so are almost always uniformly well-matched. Other settings: mating neighbourhood size = 25 patches.




## SUPPLEMENTARY FIGURE S4

**Opposing selection on the sexes generates listening polymorphisms**

Graphs show the frequencies of allele *A* (paternal TGP; blue) and allele *B* (maternal TGP; red) for a single simulation run of the 'best male' version of the ongoing conflict model when only males experience stress (a) and only females experience stress (b). Listening polymorphisms are consistently maintained for hundreds of generations. Other settings: mating neighbourhood size = 25 patches.

# SUPPLEMENTARY TABLE S1

**Phenotypic effects of listening genotypes**

When sexual conflict is ongoing, listening alleles have sex-independent effects on offspring responses to maternal and paternal information; whereas when sexual conflict is resolved, listening alleles have sex-specific effects on offspring responses. Note that an individual's overall genotype is determined by the alleles it carries at each locus.

| | Locus | Genotype | Phenotype |
|---|---|---|---|
| Ongoing conflict | *A* | *aa* | Offspring do not listen to father's information |
| | | *Aa* | Offspring listen to father's information 50% of the time |
| | | *AA* | Offspring listen to father's information 100% of the time |
| | *B* | *bb* | Offspring do not listen to mother's information |
| | | *Bb* | Offspring listen to mother's information 50% of the time |
| | | *BB* | Offspring listen to mother's information 100% of the time |
| Resolved conflict | *C* | *cc* | Sons do not listen to father's information |
| | | *Cc* | Sons listen to father's information 50% of the time |
| | | *CC* | Sons listen to father's information 100% of the time |
| | *D* | *dd* | Sons do not listen to mother's information |
| | | *Dd* | Sons listen to mother's information 50% of the time |
| | | *DD* | Sons listen to mother's information 100% of the time |
| | *E* | *ee* | Daughters do not listen to father's information |
| | | *Ee* | Daughters listen to father's information 50% of the time |
| | | *EE* | Daughters listen to father's information 100% of the time |
| | *F* | *ff* | Daughters do not listen to mother's information |
| | | *Ff* | Daughters listen to mother's information 50% of the time |
| | | *FF* | Daughters listen to mother's information 100% of the time |