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Demography can favour female-advantageous alleles

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When female fecundity is relatively independent of male abundance, while male reproduction is proportional to female abundance, females have a larger effect on population dynamics than males (i.e. female demographic dominance). This population dynamic phenomenon might not appear to influence evolution, because male and female genomes still contribute equally much to the next generation. However, here we examine two evolutionary scenarios to provide a proof of principle that spatial structure can make female demographic dominance matter. Our two simulation models combine dispersal evolution with local adaptation subjected to intralocus sexual conflict and environmentally driven sex ratio biases, respectively. Both models have equilibria where one environment (without being intrinsically poorer) has so few reproductive females that trait evolution becomes disproportionately determined by those environments where females survive better (intralocus sexual conflict model), or where daughters are overproduced (environmental sex determination model). Surprisingly, however, the two facts that selection favours alleles that benefit females, and population growth is improved when female fitness is high, together do not imply that all measures of population performance are improved. The sex-specificity of the source–sink dynamics predicts that populations can evolve to fail to persist in habitats where alleles do poorly when expressed in females.

1. Introduction

In diploid species, half of the genetic material of each offspring is provided by the male parent, the other half by the female parent. At the same time, the population dynamic properties of populations are more strongly influenced by female than by male performance. This is encapsulated in the concept of female demographic dominance [1], which refers to a set of assumptions where female fecundity is relatively independent of male abundance, while male reproduction is proportional to female abundance. While demographic dominance in this pure form is obviously a simplification (in reality males can have a multitude of effects on female fecundity [2,3]), it holds in an approximate sense widely enough to make the lack of attention to its consequences surprising. Sexual asymmetries in demographic importance are rarely taken into account when studying sexual conflict or primary sex ratios (but see [4]).

The reason why demographic dominance might be safely ignored is that each offspring inherits equally many autosomal genes from both the male and the female parent. Therefore, even if males and females differ in their life histories or reproductive roles, the overall expectation is equal male and female fitness in diploid species with a 1 : 1 primary sex ratio. As pointed out by Arnqvist [5], one should therefore express caution when interpreting claims that female evolution elevates their fitness above that of males or vice versa. There is an intuitive sense in which females or males can be argued to 'win' a conflict: consider, for example, intralocus sexual conflict. The evolved allelic values might be closer to the optimum of one sex (also often expressed as a smaller 'lag load', reviewed in [6]). Because of the equal number of

genes that pass through males and females to form the next generation, females are typically not assumed to be more likely to 'win' even though they are the main determinant of the *size* of the next generation.

Here, we build 'proof of principle' models to show that spatial variation in habitat creates scenarios where it is no longer safe to ignore female demographic dominance when arguing about sexual conflict or sex ratio dynamics. Our two models consider subpopulations that are linked via dispersal in spatially varying habitats. This creates conditions where genotype × environment interactions are important for understanding population dynamics. Local adaptation to a particular habitat can lead to a large number of propagules from that habitat; alleles carried by these propagules can come to predominate in the global population (as in source–sink theory [7]). However, this effect can be sex-specific: above-average offspring production requires that females, rather than males, are locally adapted. Therefore, female demographic dominance can, in our two models, result in: (i) more viable females than males (model 1), and (ii) female-biased sex ratios (model 2).

Perhaps surprisingly, we also show that improved female performance does not necessarily maximize global population performance: if females 'win' the conflict in one habitat but 'lose' it in another, then the population as a whole can evolve to be mainly found in habitats where 'females win'. The population will underuse habitats where 'males win', as population growth is predicted to be very poor in areas where only males can thrive. It is notable that this process, where populations evolve to thrive in one habitat only, can occur despite neither habitat being intrinsically more difficult to adapt to than the other; it arises solely owing to sexual conflict.

2. The models

Our individual-based models of sex-specific local dynamics and dispersal assume sexually reproducing diploid populations where alleles directly impact survival (intralocus sexual conflict model) or offspring sex (environmental sex determination model). Individuals in each model inhabit worlds that consist of two different environments of 50 habitat patches each, creating spatial heterogeneity in a world that totals 100 patches. Each world is initialized by placing 1000 individuals, each an adult female or an adult male (50% probability of being either), onto the patches. As there are 100 patches, each initial subpopulation has a size of approximately five males and five females. All simulations were run for 10 000 generations with 10 repetitions unless stated otherwise. In all simulations, 10 000 generations was found to be sufficient for convergence.

(a) First model: intralocus sexual conflict

There are three evolving traits in this model. One diploid locus, a, codes for the quantitative trait that impacts an individual's survival in the local patch ('survival allele' for short). This locus is expressed in both sexes as the mean of paternally and maternally inherited allelic values. The two different environments of 50 patches each, which we label environment A and B, differ in the optimal trait value a that leads to highest survival, but this is also sex-dependent (figure 1; see below). There are also two diploid loci, d_f and d_m,

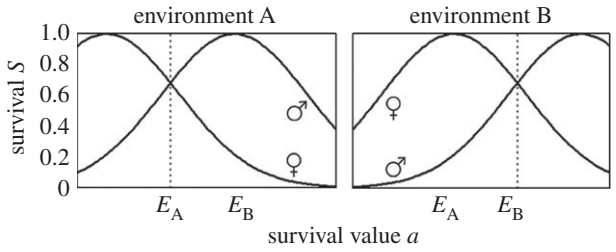

Figure 1. The relationship between the survival allele trait value, a, and survival in each environment. For females E_{opt} is lower than E_i; for males it is the opposite. All individuals start each simulation with trait values matching either environment A or B, and there is initially no sex bias in survival. Also note that individuals with $a = E_B$ survive well if dispersed to environment A but only if they are males; similarly, individuals with $a = E_A$ survive well if dispersed to environment B, but only if they are females.

which control the dispersal propensity and are expressed in females only (d_f) or males only (d_m). We assume co-dominance for each of the three diploid loci, such that phenotypes are the mean of the relevant allelic values.

When each simulation commences, individuals are assigned values of a that match the local environmental optima depending on the location of the individual but not on its sex. Individuals are also assigned values of d_f and d_m (these are initially uniformly distributed with mean d_{init} and a range $(d_{init} - \sigma_{init}, d_{init} + \sigma_{init})$ around this mean).

Each generation starts with reproduction within each subpopulation (inhabitants of a patch). We specify the number of offspring (N) produced by each subpopulation of F females and M males as follows: if $F \geq 1$ and $M \geq 1$ (at least one individual of each sex is locally present) then $N = 2 + 4Fe^{-cF}$, rounded to the nearest integer; otherwise $N = 0$. This function, where c is a constant determining the strength of local competition, has the desirable properties of female demographic dominance, in that M does not appear in the equation beyond the $M \geq 1$ requirement, as well as local competition, in that: (i) subpopulations with at least one female always produce at least two offspring (on average one of each sex); (ii) the small subpopulation's output increases if more females are added; but (iii) stronger overcrowding (large F) reduces the subpopulation's output. These rules also imply a kin-selected reason to disperse: a dispersing individual alleviates competition for its relatives (also note that our model ignores some other known reasons to disperse, e.g. inbreeding avoidance, as we assume no cost to consanguineous matings). The model then randomly selects a mother and a father among locally present individuals as parents for each offspring. The offspring inherit their genes according to Mendelian inheritance rules and each offspring has an equal probability of developing as a male or as a female.

Mutations then potentially occur at loci a, d_f and d_m, each allele doing so with probability μ_a or μ_d (the latter value is the same for both dispersal loci). If mutation occurs, the allele's value changes by an amount taken from a uniform distribution with range $[-\sigma_a, \sigma_a]$ (for a) or $[-\sigma_d, \sigma_d]$ (for either dispersal allele). Dispersal alleles that have their new values below 0 or above 1 are set to 0 or 1, respectively.

All adults die after reproduction (i.e. we assume non-overlapping generations). Thereafter, the offspring disperse based on their sex-specifically expressed dispersal probability, which is the mean of their sex-specific dispersal

alleles. Dispersing offspring land in a randomly chosen patch among all 100 patches, i.e. dispersers are as likely to experience environment A as B (note that we allow a disperser to land back on its natal patch, to keep this symmetry). Viability selection occurs after dispersal. Survival is modelled according to the conceptual model provided by Cox & Calsbeek [8]: its values are derived as $S = e^{-b(E_{opt}-a)^2}$, where b is a constant, E_{opt} the sex- and environment-specific optimal trait value and a the mean of the individual's survival alleles. Thus, an individual reaches its best survival when its alleles match perfectly the local requirements of the environment, such that $a = E_{opt}$; mismatches in either direction are associated with reduced survival. E_{opt} is assumed higher for males ($E_{opt} = E_i + k$) than for females ($E_{opt} = E_i - k$), where E_i refers to the environmental value in environment A or B. Thus, when the model is initiated ($a = E_i$), neither males nor females experience optimal survival, and their survival probabilities are equal (figure 1). This assumption reflects unresolved intralocus sexual conflict where optimal traits differ between males and females (e.g. [8–11]) as well as between environments. Offspring survival concludes a generation, and the surviving offspring become the breeders of the next generation.

(b) Second model: environmental sex determination

In the second model, we focus on a sex determining mechanism that has the potential to create biased sex ratios: temperature-dependent sex determination (TSD). In this model, the two different environments differ in climate. Environments A and B are now interpretable as 'warm' and 'cold' patches (50 each). Studies of TSD characterize a trait known as the pivotal temperature (T_{piv}), above which offspring develop mainly as one sex, and below which the other sex is overproduced [12]. Reflecting this, we state that an offspring with T_{piv} will develop as a female according to the sigmoidal probability distribution: $P_f = 1/(1 + e^{(T_{piv}-T_{env})})$, and as a male with probability $P_m = 1 - P_f$, where T_{env} is the local temperature [13]. Thus, if $T_{piv} > T_{env}$ the offspring is likely to develop as a male and conversely, if $T_{piv} < T_{env}$, as a female.

The subpopulations are initialized as in the first model, now with pivotal temperature alleles initially set to match the environment ($T_{piv} = T_{env}$, within each environment). T_{piv} is the average of the maternally and paternally inherited alleles, which are initially identical. Dispersal probability, as in the first model, is controlled by two diploid loci, one for male and another for female dispersal; an individual only expresses its sex-specific dispersal alleles. Dispersal alleles are initialized and inherited as in model 1. We assume co-dominance for the three diploid loci.

To provide another contrast to the previous model, we now assume overlapping generations. Evolution occurs as follows. Density dependence acts locally on fecundity, such that the number of offspring (N) produced by a local subpopulation is determined by the number of local females (F), $N = Fe^{-cf}$, where c is a constant (and N is rounded to the nearest integer). The function is similar in its gist but differs somewhat from that used in model 1, as there is no requirement of at least two offspring produced by a single female—the current model requires smaller fecundities to sustain a population as generations are overlapping (parents survive). Parents and the genes passed on to offspring are selected as in the first model, but the sex of each offspring is now determined via a genotype × environment interaction (P_f and P_m, see above).

Births are followed by mutation, each of the alleles present in the offspring mutate with a probability μ_d (dispersal) or μ_{piv} (pivotal temperature alleles). If mutation occurs, the allele's value changes with an amount taken from a uniform distribution within the range $[-\sigma_d, \sigma_d]$ and $[-\sigma_{piv}, \sigma_{piv}]$ for the dispersal and pivotal temperature alleles, respectively (for dispersal alleles, if the new values are below 0 or above 1, they are set to 0 or 1, respectively).

Next, there is mortality in the parental generation: each adult survives with probability $s < 1$ (i.e. we assume overlapping generations) irrespective of sex or any trait values. Thereafter, natal dispersal occurs. Dispersal is global, modelled as in model 1. Thus, a disperser has an equal probability of landing in a 'warm' or a 'cold' patch. After dispersal, all offspring become adults and are thus able to breed in the next generation together with surviving adults.

Note that even though the two models use the same dispersal rules, they differ somewhat in their costs of dispersal. Neither model assumes any other cost of dispersal than an indirect cost due to local adaptation, but in the intralocus sexual conflict model this could have a negative impact on the viability of the disperser, whereas in the current model a locally adapted individual that disperses to a novel environment and reproduces there does not experience a viability cost. Instead, it might pass on pivotal temperature genes that are maladaptive in the current climate, thus the cost is delayed by one generation.

As our aim is to provide two 'proof of principle' examples, we show outcomes based on a single set of parameter values in our figures (with the exception of initial dispersal alleles, as variation in this parameter proves important in model 2). For an additional evaluation of the generality of the results, see the electronic supplementary material.

3. Results

(a) First model: intralocus sexual conflict

Regardless of whether simulations are started with low ($d_{init} = 0.15$, first row, figure 2) or high ($d_{init} = 0.85$, second row, figure 2) dispersal, dispersal alleles evolve to be similar across environments and are somewhat male-biased (figure 2*a*,*e*; note the near identical results between the different d_{init} runs). Although the proportion of dispersing individuals remains relatively low, this gene flow is sufficient to equalize the survival alleles across environments (triangles figure 2*b*,*f*), thus the population as a whole is not locally adapted (in line with population genetic theory which predicts that relatively little gene flow is sufficient to 'swamp' local adaptation, [14,15]). The evolved survival alleles nearly match the environmental value for environment B.

Given that the allelic values conferring best viability are not only environment-specific but also sex-specific in this model, identical allelic values can produce very different male and female viabilities. The globally evolving trait values predict very high viability for males and low viability for females in environment A, while the same alleles in environment B predict higher female than male viability (figure 2*c*,*g*). This makes subpopulations in environment A unproductive (few females live to produce young), and consequently the total

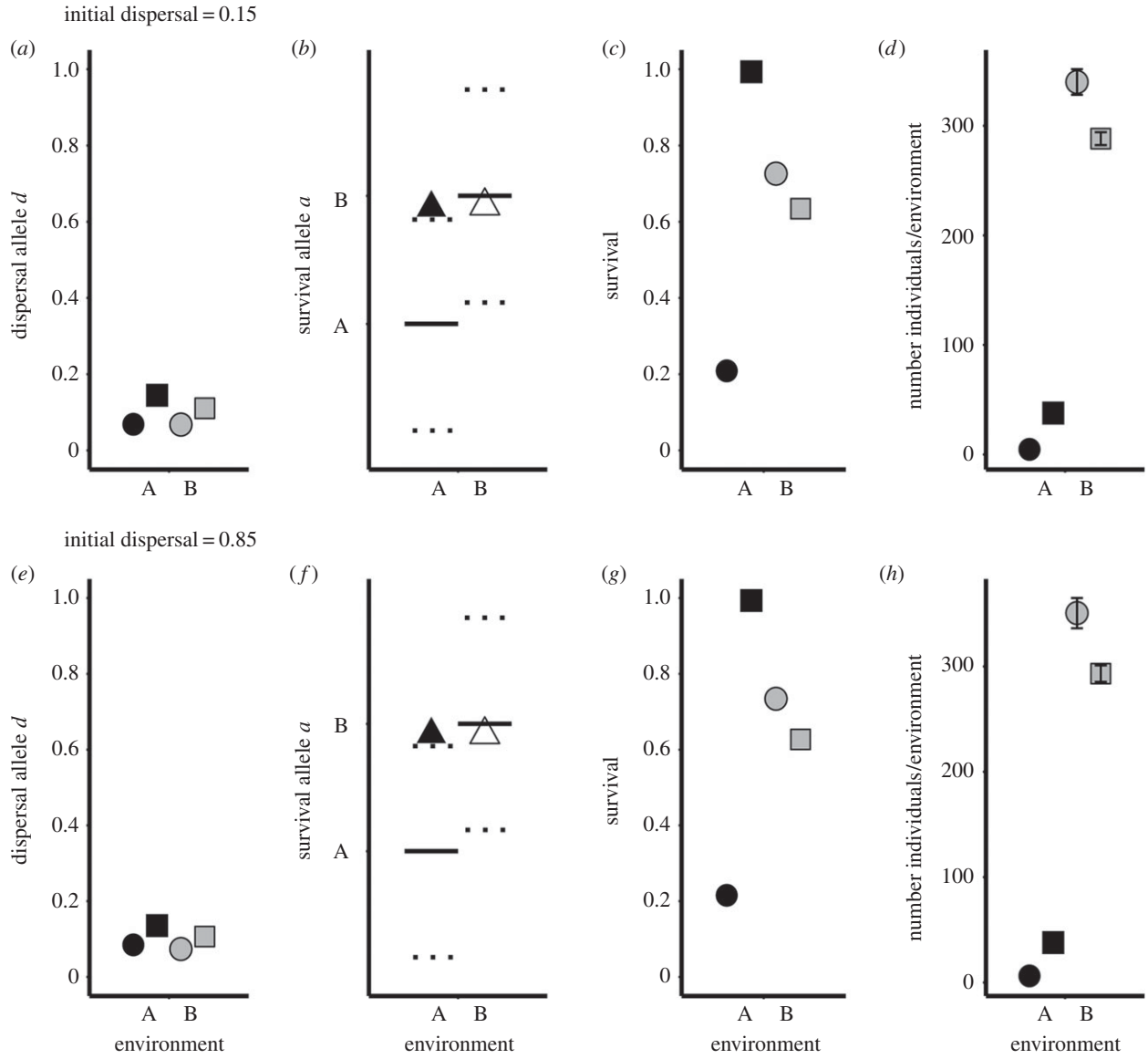

Figure 2. Evolutionary outcomes of the intralocus sexual conflict model, plotted at generation 10 000 of 10 independent simulation runs per scenario (except for mean $d_{init} = 0.15$ where one simulation resulted in extinction in both environments and points are based on nine independent runs). (*a*,*e*) Sex- and environment-specific mean of dispersal alleles, (*b*,*f*) sex- and environment-specific mean of survival alleles, (*c*,*g*) sex- and environment-specific mean of survival as predicted by figure 1, and (*d*,*h*) sex- and environment-specific number of individuals, measured after survival and before breeding. Means $\pm$ s.e. given in (*d*,*h*); in other cases the s.e. are too small to be visible and have thus been left out. Initial dispersal is $d_{init} = 0.15$ in (*a*–*d*), and $d_{init} = 0.85$ in (*e*–*f*). Black symbols refer to environment A, grey symbols to environment B; squares denote males, circles denote females and triangles denote both sexes combined. In (*b*,*f*) the solid line is the E_i value and the dotted lines indicate the sex- and environment-specific optima for *a* alleles. Also note that E_A males have near perfect survival (black squares in (*c*,*g*)). Parameter values: $E_A = 5.58$, $E_B = 6.42$, $b = 0.7$, $c = 0.05$, $a_{init} = E_{(A\ or\ B)}$, $\mu_a = \mu_d = 0.1$, $\sigma_{init} = \sigma_d = \sigma_a = 0.05$ and $k = 0.74$.

population size evolves to be far greater in environment B (figure 2*d*,*h*).

Environment A males have the highest viability of all individual categories. However, because of the low abundance of individuals in environment A, a randomly sampled individual of the global population has, on average, higher viability if it is a female (mean viability is 0.719 ± 0.005 (s.e.) for all females, and 0.676 ± 0.006 for all males when $d_{init} = 0.15$; 0.725 ± 0.005 for all females and 0.669 ± 0.006 for all males when $d_{init} = 0.85$).

(b) Second model: environmental sex determination

Unlike model 1, the environmental sex determination model features two alternative stable states for dispersal. Depending on initial dispersal alleles, the population evolves to low (approx. 10% individuals disperse, $d_{init} < 0.35$) or high dispersal (approx. 80%, $d_{init} > 0.4$) (figure 3*a*). Both evolved dispersal rates appear sufficient to prevent local adaptation, as T_{piv} evolves to the same value in both environments (figure 3*b*). However, the value of T_{piv} differs between runs that lead to low versus high dispersal: when dispersal evolves to be low, T_{piv} evolves to almost match the T_{env} of the warm environment, whereas when dispersal evolves to be high, T_{piv} evolves to the mean of the two environments (figure 3*b*).

This leads to a dichotomous pattern in the primary sex ratio produced. If dispersal is low, such that T_{piv} evolves to be near T_{env} of the warm environment, the warm environment produces a slightly female-biased sex ratio and the cold environment greatly overproduces sons. By contrast, high dispersal and the evolved intermediate T_{piv} leads to warm environments

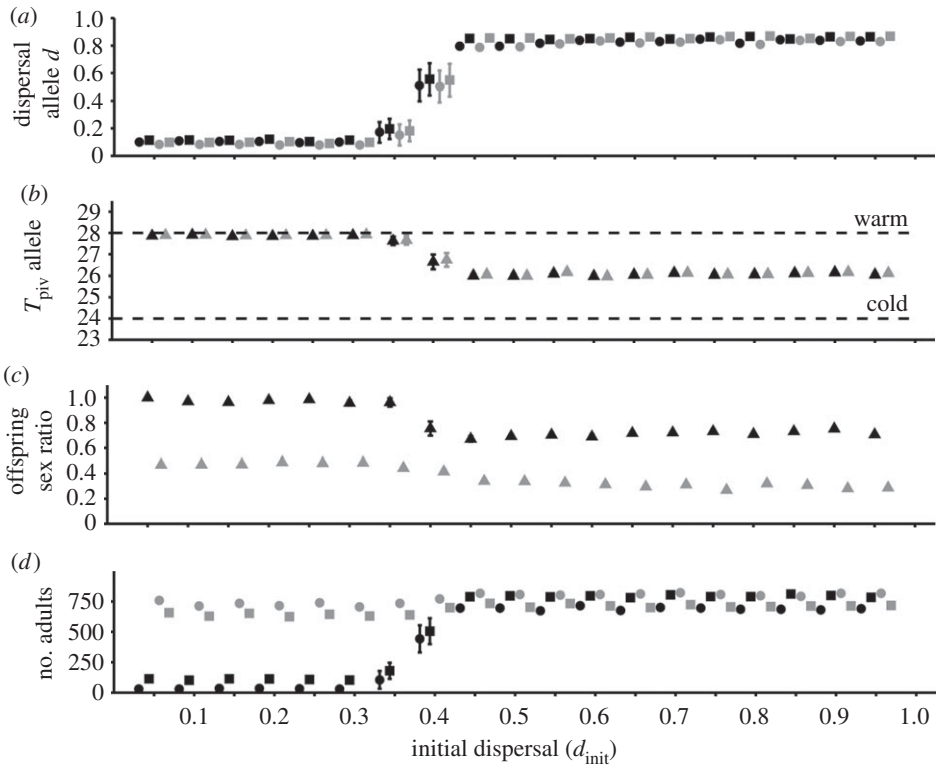

Figure 3. Evolutionary outcomes of the environmental sex determination model. The average (mean ± s.e.) at generation 10 000 of 10 independent simulation runs per scenario: (*a*) dispersal alleles, (*b*) pivotal temperature, (*c*) offspring sex ratio (males/total), and (*d*) number of adults per environment, as a function of the initial dispersal, d_{init}. Cold subpopulations in black and warm subpopulations in grey ($T_{env} = 24$ and 28, respectively). (*a*,*d*) Squares denote males and circles denote females. (*b*,*c*) Triangles denote the mean for each environment. Other parameters: $s = 0.7$, $c = 0.032$, $\sigma_{init} = \sigma_d = 0.05$, $a_{init} = T_{env}$, $\mu_{piv} = \mu_d = 0.1$, $\sigma_{piv} = 0.25$.

overproducing daughters and cold environments equally overproducing sons (figure 3*c*). The latter equilibrium leads to a relatively balanced population-wide sex ratio (figure 3*d*, high dispersal). The low-dispersal equilibrium, however, shows a similar asymmetry in environment use as was already shown for model 1: there are few females in the cold environment, which renders these subpopulations unproductive, contributing relatively little to the global gene pool.

The situation for the cold environment appears to be a vicious circle: despite being inherently equally suitable as a breeding area, any initial underproduction of daughters in this environment (owing to gene flow from warmer areas) means that few females breed locally. Therefore, the contribution of cold environments to the global gene pool remains weak, and the entire population instead adapts to the warm environment only. The situation remains more egalitarian when dispersal is high, because every population then has many females—either because of overproducing them (warm environments) or because of substantial immigration (cold environments). Under this scenario, every subpopulation continually contributes to the global gene pool.

(c) Generality

Unsurprisingly, our examples (figures 2 and 3) require a suitable combination of parameter values. To confirm that they do not represent highly unlikely special cases, we ran 600 simulation trials for the intralocus sexual conflict model and 500 simulation trials for the environmental sex determination model (owing to extinctions, we ran a higher number of simulation trials for model 1). The range of the randomly chosen parameter values for these runs is given in the electronic supplementary material.

In the intralocus sexual conflict model, these trials showed that female survival evolves to exceed male survival when the two environments vary substantially enough (see the electronic supplementary material, figure S1, for details). In the environmental sex determination model, we compared the offspring sex ratio between the cold and the warm environment (electronic supplementary material). If the environments did not differ much, high and low initial dispersal led to an identical and simple pattern with T_{piv} equal to the mean for the two environments (electronic supplementary material, figure S2). If environments were sufficiently different, we found two equilibria that depended on initial conditions, similar to our main example of figure 3. The evolutionary outcome of populations initiated with little dispersal is that the warm environment produced a relatively balanced sex ratio, and the cold populations produced a strongly male-biased sex ratio (squares in the electronic supplementary material, figure S2); high dispersal as the initial condition led to a repetition of the simpler pattern where the male bias of cold environments is approximately as strong as the female bias of warm environments (stars in the electronic supplementary material, figure S2). Thus, our findings as a whole appear to generalize, as long as there is strong enough environmental variation.

4. Discussion

Our models include no other asymmetry than the fact that local population growth depends more strongly on the number of

females than on the numbers of males (female demographic dominance). Consequently, if a population is adapted, say, to the mean of two environments, it will have some individuals residing in environments that favour females and some in environments that favour males. The former type of environment then becomes more productive as a result of females being the more important determinant of demography. Selection as a whole then becomes disproportionately driven by alleles' success in this environment, and the entire system can begin evolving in a direction where female-advantageous alleles predominate.

It is well known from source–sink theory that evolution of traits can become demographically dominated by populations with above-average productivity [16,17]. Substantial gene flow can also lead to one 'generalist' phenotype even when the selective environment differs between populations (e.g. [18,19]). We have shown that these principles have significant impacts on trait evolution when there are sex differences in the genotype × environment interaction responses. If the trait impacts survival in a sex-specific manner, or has an effect on the primary sex ratio, then a source–sink structure emerges in environments that do not differ in their suitability *a priori* but simply owing to variance among subpopulations in numbers of reproductively mature females (see also [4], for an argument of how this might favour the evolution of TSD *per se*).

Models with coevolution of local adaptation and dispersal very rarely include sexual reproduction [20,21], even though local variation in sex ratio have been identified as increasing extinction risks [22–24] and can be a significant source of selection for or against dispersal [25,26]. Conversely, models of sexual conflict rarely consider genotype × environment interactions (while mate choice studies do so more commonly, [27]). There is clearly more scope for studies linking these fields.

Both of our models show evolutionary endpoints with the following properties: there is a trait that impacts how many females are produced and/or survive to mature, and the trait is subject to a genotype × environment interaction that impacts how many mature females (versus males) will live in each environment. Given that trait evolution is disproportionately influenced by those environments where the genotype × environment interaction favours females rather than males, one might be tempted to conclude that the end result (e.g. in model 1, the average female survives better than the average male) also improves population-wide performance measures such as the ability to persist in a wide variety of environments. However, this is a premature conclusion: if one type of environment becomes disproportionately female-favouring, the importance of adapting to the male-favouring environment can become reduced to such a degree that the population barely persists in these (environment A in our intralocus sexual conflict model, and the cold environment in our environmental sex determination model). This is remarkable, given that we assumed no intrinsic quality differences of these environments: we assumed identical local density dependence across environments, therefore the same number of females led, in principle, to equally good reproduction in either environment.

Our additional result of two alternative stable states, visible in the environmental sex determination model only, is dependent on ancestral dispersal rates that evolve into high or low dispersal. This is in line with previous research, which has identified the potential for alternative stable states based on dispersal rates [28]. The mechanism operating in our model, however, differs from earlier studies with coevolution of local adaptation and dispersal [18,28]. While earlier studies document equilibria with much dispersal and little local adaptation or *vice versa*, we found no evidence of local adaptation in the strict sense (the two environments never evolved clear differences for the alleles used to determine the level of local adaptation, i.e. the survival alleles of model 1 or pivotal temperature of model 2).

Local adaptation is not, *per se*, prevented from occurring in our models. Separate computer runs with no dispersal (electronic supplementary material, figure S3) lead subpopulations in the two different environments to evolve distinct distributions of T_{piv} or a. This confirms that local adaptation is possible, and that our main results are based on dispersal being sufficient to 'swamp' local adaptation (even the lower dispersal rate of the two alternative equilibria in model 2 was able to do this). Instead, the two equilibria in model 2 reflect differences as to whether dispersal always brings enough females to every type of subpopulation so that no subpopulation ends up too small to contribute to the global gene pool, or whether the local production of females can become compromised and the global process of adaptation is no longer impacted by performance in these environments.

How general are our findings? We assumed a relatively strict form of female demographic dominance, where male availability does not constrain female reproduction unless there are no males locally. Thus, the mating system in our model is likely to be a key factor in the outcome of these models. Had we modelled a strictly monogamous mating system, instead of the polygynous mating system of our models, males and females would have been equally important for population productivity; an emergent pattern where females as a whole evolve higher viabilities is then unlikely [29]. For clarity, we also assumed clear differences between exactly two types of environment, local density dependence and global dispersal with no spatial correlation between neighbouring habitats.

Relaxing these assumptions is a clear avenue for further study. Intuitively, it appears that the scale of density dependence will matter. If it were to act on a global scale rather than the local scale as we modelled, then the dynamics would feature even more significant evolutionary effects of female demographic dominance, because highly productive sites can maintain their above-average contribution to the global gene pool. In the opposite case, stronger local density dependence than we included could result in a situation of 'soft selection' [30]. Under soft selection, improved local adaptation does not translate into higher productivity, as local density regulation equalizes productivity across habitat patches. Consequently, the differing numbers of females that reproduce in each patch have no evolutionary implications. On the other hand, our results are unlikely to depend crucially on our assumption of global dispersal. If individuals do not often disperse to the alternative habitat type, the essence of our model still applies, but with a lower effective dispersal rate. Since the striking effects of female demographic dominance were found irrespective of dispersal rates in model 1, and at low-dispersal rate in model 2, we expect the results to generalize to many spatial structures. The details of such effects, however, would be a fruitful avenue of further study.

It is important to note that dispersal in our models had no direct costs. Any selection against dispersal was based on the

possibility that moving leads to maladaptation to the new environment. This is particularly important for understanding the low-dispersal equilibrium in model 2. Dispersal is more likely to lead an individual from a warm to a cold environment than vice versa, simply because any new offspring is more likely to be born in a warm rather than a cold climate (warm patches have more females than do cold patches). Thus, it is likely to be warm-adapted in its pivotal temperature (see [31] for a more general version of this argument). In the intralocus sexual conflict model, the mechanism is similar, but impacts the disperser's own viability.

The strong demographic effects of the population sex ratio in a metapopulation could partially explain why primary sex ratios across biota (with any mechanism of sex determination) so frequently depart from 50 : 50 [32]. Even with local adaptation, biased sex ratios are predicted under some selective regimes (e.g. condition-dependent sex ratios [33,34]). However, sex ratios often seem to be even more biased than expected under adaptive explanations, particularly in species with TSD that exhibit extremely female-biased sex ratios in some populations [32,34]. Our results indicate that female-biased sex ratios can be expected across a range of dispersal levels, either owing to a lack of local adaptation or to demographic swamping by adapted populations and restricted gene flow from populations in male-producing climates, even if other suggested mechanisms such as cultural inheritance [35] are absent. Note that we have not included selection for more *variable* sex ratios, which may also impact population persistence if there is a frequent need to colonize empty patches [4].

In conclusion, our results demonstrate a potentially underappreciated role for female demographic dominance in trait evolution under sexual conflict. We have examined this principle under two very different scenarios, but we suspect that the finding can be quite general: whenever local population productivity is more dependent on the production or performance of females than of males, and there is spatial variation with different subpopulations contributing to the global gene pool, we can expect sexual asymmetries to emerge where female-beneficial alleles can be said to have an upper hand. Simultaneously, however, our results warn against any naive expectation that this necessarily maximizes global population performance: our examples also show that the conflict can cause severe failure to adapt to some habitats despite these being, in principle, adequate for breeding.

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Data accessibility. Data generated by both models is uploaded as the electronic supplementary material.

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References

1. Crowley PH. 2000 Sexual dimorphism with female demographic dominance: age, size, and sex ratio at maturation. *Ecology* **81**, 2592–2605. (doi:10.1890/0012-9658(2000)081[2592:SDWFDD]2.0.CO;2)
2. Arnqvist G, Nilsson T. 2000 The evolution of polyandry: multiple mating and female fitness in insects. *Anim. Behav.* **60**, 145–164. (doi:10.1006/anbe.2000.1446)
3. Rankin DJ, Kokko H. 2007 Do males matter? The role of males in population dynamics. *Oikos* **116**, 335–348. (doi:10.1111/j.2006.0030-1299.15451.x)
4. Freedberg S, Taylor DR. 2007 Sex ratio variance and the maintenance of environmental sex determination. *J. Evol. Biol.* **20**, 213–220. (doi:10.1111/j.1420-9101.2006.01209.x)
5. Arnqvist G. 2004 Sexual conflict and sexual selection: lost in the chase. *Evolution* **58**, 1383–1388. (doi:10.1554/03-449)
6. Kokko H, Jennions MD. In press. The relationship between sexual selection and sexual conflict. In *The genetics and biology of sexual conflict* (eds S Gavrilets, WR Rice). Cold Spring Harbor, NY: Cold Spring Harbor Press.
7. Kawecki TJ. 1995 Demography of source–sink populations and the evolution of ecological niches. *Evol. Ecol.* **9**, 38–44. (doi:10.1007/BF01237695)
8. Cox RM, Calsbeek R. 2009 Sexually antagonistic selection, sexual dimorphism, and the resolution of intralocus sexual conflict. *Am. Nat.* **173**, 176–187. (doi:10.1086/595841)
9. Foerster K, Coulson T, Sheldon BC, Pemberton JM, Clutton-Brock TH, Kruuk LEB. 2007 Sexually antagonistic genetic variation for fitness in red deer. *Nature* **447**, 1107–1110. (doi:10.1038/nature05912)
10. Fedorka KM, Mousseau TA. 2004 Female mating bias results in conflicting sex-specific offspring fitness. *Nature* **429**, 65–67. (doi:10.1038/nature02492)
11. Prassad NG, Bedhomme S, Day T, Chippendale AK. 2007 An evolutionary cost of separate genders revealed by male-limited evolution. *Am. Nat.* **169**, 29–37. (doi:10.1086/509941)
12. Bull JJ. 1980 Sex determination in reptiles. *Q. Rev. Biol.* **55**, 3–21. (doi:10.1086/411613)
13. Girondot M. 1999 Statistical description of temperature-dependent sex determination using maximum likelihood. *Evol. Ecol. Res.* **1**, 479–486.
14. Mayr E. 1963 *Animal species and evolution*. Cambridge, MA: Belknap Press of Harvard University Press.
15. Kirkpatrick M, Barton NH. 1997 Evolution of a species' range. *Am. Nat.* **150**, 1–23. (doi:10.1086/286054)
16. Ronce O, Kirkpatrick M. 2001 When sources become sinks: migrational meltdown in heterogeneous habitats. *Evolution* **55**, 1520–1531. (doi:10.1554/0014-3820(2001)055[1520:WSBSMM]2.0.CO;2)
17. Kawecki TJ, Ebert D. 2004 Conceptual issues in local adaptation. *Ecol. Lett.* **7**, 1225–1241. (doi:10.1111/j.1461-0248.2004.00684.x)
18. Kisdi E. 2002 Dispersal: risk spreading versus local adaptation. *Am. Nat.* **159**, 579–596. (doi:10.1086/339989)
19. Hendry AP, Day T, Taylor EB. 2001 Population mixing and the adaptive divergence of quantitative traits in discrete populations: a theoretical framework for empirical tests. *Evolution* **55**, 459–466. (doi:10.1554/0014-3820(2001)055[0459:PMATAD]2.0.CO;2)
20. Caswell H, Weeks DE. 1986 Two-sex models: chaos, extinction, and other dynamic consequences of sex. *Am. Nat.* **128**, 707–735. (doi:10.1086/284598)
21. Ronce O. 2007 How does it feel to be a rolling stone? Ten questions about dispersal evolution. *Annu. Rev. Ecol. Evol. Syst.* **38**, 231–253. (doi:10.1146/annurev.ecolsys.38.091206.095611)
22. Aresco MJ. 2005 The effect of sex-specific terrestrial movements and roads on the sex ratio of freshwater turtles. *Biol. Conserv.* **123**, 37–44. (doi:10.1016/j.biocon.2004.10.006)
23. Donald PF. 2007 Adult sex ratios in wild bird populations. *Ibis* **149**, 671–692. (doi:10.1111/j.1474-919X.2007.00724.x)
24. Lambertucci SA, Carrete M, Donazar JA, Hiraldo F. 2012 Large-scale age-dependent skewed sex ratio in a sexually dimorphic avian scavenger. *PLoS ONE* **7**, e46347. (doi:10.1371/journal.pone.0046347)
25. Greenwood PJ. 1980 Mating systems, philopatry and dispersal in birds and mammals. *Anim. Behav.* **28**, 1140–1162. (doi:10.1016/S0003-3472(80)80103-5)
26. Meier CM, Starrfelt J, Kokko H. 2011 Mate limitation causes sexes to coevolve towards more similar dispersal kernels. *Oikos* **120**, 1459–1468. (doi:10.1111/j.1600-0706.2011.19487.x)

27. Ingleby FC, Hunt J, Hosken DJ. 2010 The role of genotype-by-environment interactions in sexual selection. *J. Evol. Biol.* **23**, 2031–2045. (doi:10.1111/j.1420-9101.2010.02080.x)
28. Billiard S, Lenormand T. 2005 Evolution of migration under kin selection and local adaptation. *Evolution* **59**, 13–23. (doi:10.1554/04-232)
29. Legendre S, Clobert J, Møller AP, Sorci G. 1999 Demographic stochasticity and social mating system in the process of extinction of small populations: the case of passerines introduced to New Zealand. *Am. Nat.* **153**, 449–463. (doi:10.1086/303195)
30. Débarre F, Gandon S. 2011 Evolution in heterogeneous environments: between soft and hard selection. *Am. Nat.* **177**, E84–E97. (doi:10.1086/658178)
31. McNamara JM, Dall SRX. 2011 The evolution of unconditional strategies via the 'multiplier effect'. *Ecol. Lett.* **14**, 237–243. (doi:10.1111/j.1461-0248.2010.01576.x)
32. Bull JJ, Charnov EL. 1988 How fundamental are Fisherian sex ratios? *Oxf. Surv. Evol. Biol.* **5**, 96–135.
33. Charnov EL, Bull JJ. 1989 The primary sex ratio under environmental sex determination. *J. Theor. Biol.* **139**, 431–436. (doi:10.1016/S0022-5193(89)80063-3)
34. Hulin V, Girondot M, Godfrey MH, Guillon J-M. 2008 Mixed and uniform brood sex ratio strategy in turtles: the facts, the theory, and their consequences. In *Biology of turtles* (eds J Wyneken, MH Godfrey, V Bels), pp. 279–300. Chicago, IL: University Press of Chicago.
35. Freedberg S, Wade MJ. 2001 Cultural inheritance as a mechanism for population sex-ratio bias in reptiles. *Evolution* **55**, 1049–1055. (doi:10.1554/0014-3820(2001)055[1049:CIAAMF]2.0.CO;2)