Disentangling Verbal Arguments: IntraLocus Sexual Conflict in Haplodiploids

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ABSTRACT: In haplodiploids, (1) alleles spend twice as many generations in females as in males, (2) males are never heterozygous and therefore express recessive alleles, and (3) males sire daughters but not sons. IntraLocus sexual conflict therefore operates differently in haplodiploids than in diploids and shares strong similarities with loci on X (or Z) chromosomes. The common co-occurrence of all three features makes it difficult to pinpoint their respective roles. However, they do not always co-occur in nature, and missing cases can be additionally studied with hypothetical life cycles. We model sexually antagonistic alleles in eight different sex determination systems and find that arguments 1 and 2 promote invasion and fixation of female-beneficial and male-beneficial alleles, respectively; argument 2 also improves prospects for polymorphism. Argument 3 harms the invasion prospects of sexually antagonistic alleles (irrespective of which sex benefits) but promotes fixation should invasion nevertheless occur. Disentangling the features helps to evaluate the validity of previous verbal arguments and yields better-informed predictions about intraLocus sexual conflict under different sex determination systems, including hitherto undiscovered ones.

Keywords: intraLocus sexual conflict, sex chromosomes, haplodiploidy, paternal genome elimination, genetic architecture, sex determination.

Introduction

Males and females typically use different strategies to survive and reproduce, with dimorphies ranging from morphology (Tarka et al. 2014) and development (Lewis et al. 2011) to behavior (Long and Rice 2007). They, however, share a large portion of their genomes, which makes it challenging to create two different optimal phenotypes from the same genome. Sexually antagonistic alleles are expressed in both sexes and have opposing effects on fitness: an allele that shifts the phenotype closer to the optimum of one sex simultaneously shifts it away from the optimum of the opposite sex (Lande 1980; van Doorn 2009).

When male and female phenotypes cannot be simultaneously optimized by evolution (Lande 1980), the scenario is called (unresolved) intraLocus sexual conflict (van Doorn 2009; Pennell and Morrow 2013). There is ample empirical evidence for unresolved (Foerster et al. 2007; Mainguy et al. 2009) and resolved (Wright et al. 2018) intraLocus sexual conflict with effects on diverse processes such as sexual dimorphism (Ingleby et al. 2015), sexual selection under female choice (Pischetta and Chippindale 2006; Brommer et al. 2007), maintenance of genetic variation (Foerster et al. 2007), and aspects of genetic architecture, including the emergence of novel Y chromosomes (Rice 1987), dosage compensation on X chromosomes (Mank et al. 2011), and genomic imprinting (Iwasa and Pomiankowski 2001; Day and Bonduriansky 2004; Dobata and Tsuji 2012).

The sex determination system affects the operation of intraLocus sexual conflict. In species with completely environmental sex determination, all alleles are inherited in an autosomal fashion. Under obligate sexual reproduction, this implies that all alleles spend equally many generations in each sex. However, if sex is determined by sex chromosomes or through haplodiploidy, then at least some alleles deviate from this expectation. For example, a Y-linked allele never finds itself in females and is therefore expected to optimize male fitness, irrespective of the optimal female phenotype. There is experimental support for the consequent masculinization of the Y chromosome as well as feminization of a novel W chromosome (in Drosophila melanogaster; Prasad et al. 2006; Rice 1992). While predictions for Y- and W-linked alleles are rather straightforward, understanding intraLocus sexual conflict on X and Z chromosomes or under haplodiploidy is more complicated: in these cases alleles are expressed in both sexes, but asymmetrically. Here, our aim is
to understand the effect of sex determination systems on intralocus sexual conflict.

There is a clear link between predictions for alleles on X chromosomes and for alleles in haplodiploid species. In both cases females are diploid at the relevant loci, while males are haploid. This analogy—that genes on X or Z chromosomes are inherited in the same fashion as genes in haplodiploid animals—will be used throughout this article. Previous theoretical work (Parsons 1961; Rice 1984) shows that sexually antagonistic alleles evolve differently on X chromosomes than on autosomes, and most predictions regarding intralocus sexual conflict in haplodiploids are derived from studying intralocus sexual conflict on X chromosomes (Kraaijeveld 2009). On autosomes, an antagonistic allele spreads when rare whenever the benefit to one sex is stronger than the detrimental effect to the other sex, but the spread of an X-linked allele depends not only on its average fitness effects but also on its dominance. If an X-linked sexually antagonistic allele is dominant, its invasion prospects are best when it has beneficial fitness effects on females; if recessive, it should be male beneficial for best prospects.

Because dominant female-beneficial and recessive male-beneficial alleles directly compete with each other, theory predicts X chromosomes to be a hot spot for sexually antagonistic fitness variation (Rice 1984). There is mixed support for this expectation (Dean and Mank 2014), with clear signs of X-linked enrichment in some taxa (Gibson et al. 2002; Reeve and Pfennig 2003; Pischke and Chippindale 2006; Foerster et al. 2007), no enrichment (Mank et al. 2005), and autosomal enrichment in other taxa (Fry 2010; Fedorka and Mousseau 2004). Fry (2010) found that these mixed results could be explained by sex-specific dominance in fitness.

While the theoretical framework appears clear at first sight, the interpretation regarding the relevant causalities presents surprising difficulties because deviations from autosomal inheritance typically combine several co-occurring asymmetries. This allows for more than one verbal argument to be seen as the main driver of the unique evolutionary patterns that occur on X chromosomes and under haplodiploidy (e.g., de la Filia et al. 2015). Below, we formulate these arguments for haplodiploid animals, but they can easily be adjusted to apply to X or Z chromosomes.

**Argument 1.** In haplodiploids, an allele finds itself twice as often in females compared with males. This argument is closely related to reproductive value. In haplodiploids, the total reproductive value of females is twice that of males: if a random allele is sampled in the distant future, there is a two-thirds probability that that allele is descended from one currently residing in a female (Gardner 2014). Consequently, intuition suggests that the fitness effect on females is twice as important for predicting spread than the allele’s effect on males.

**Argument 2.** In haplodiploids, males cannot be heterozygous. Being haploid, they always express their one allele at each locus. The sex difference in expression frequency is particularly pronounced for rare and recessive alleles, as their expression in females remains rare. This explains why a fully recessive male-beneficial allele may invade even if lethal to homozygous females.

**Argument 3.** In haplodiploids, males have only female offspring and never have sons. This forces alleles to go through at least one female generation in order to be passed from a male to his male progeny, for example, his grandsons. This might make male-beneficial alleles more prone to genetic drift and, as a corollary, extinction (de la Filia et al. 2015).

These three verbal arguments have been proposed as causal explanations for the results in intralocus sexual conflict in haplodiploids (Pamilo 1979; Rice 1984; Fry 2010), but since they all interact, their application appears somewhat post hoc. No effort appears to have been put into distinguishing between the three potential causalities, and no formal models exist to verify whether these verbal arguments have the purported consequences when examined separately from each other. The arguments are often used interchangeably (e.g., Kraaijeveld 2009; de la Filia et al. 2015; Eyer et al. 2019; Patten 2019), making it difficult to understand why exactly haplodiploidy (or a locus being on a X chromosome) affects intralocus sexual conflict.

Here, our aim is to take advantage of the fact that the three features do not show perfect covariation in nature. This helps to disentangle the causalities, especially if one additionally creates the still-missing cases by studying hypothetical life cycles that are conceivable but for which no known example exists in nature.

We want to understand whether the verbal arguments work as expected and whether they are all necessary and are together sufficient to explain the differences between haplodiploid and diploid animals regarding the evolution of sexually antagonistic alleles. We construct eight models that each represent a different combination of verbal arguments. For each of the models we analyze the conditions for invasion and fixation of sexually antagonistic alleles. We then compare these conditions to identify the effects of each verbal argument. When possible, models are based on natural species, but some sex determination systems are entirely fictional and serve the purpose of disentangling the effects of the different verbal arguments.

**Methods**

**General Approach**

That all three verbal arguments occur simultaneously in a typical comparison of diploid and haplodiploid species makes it difficult to disentangle their roles in the operation
of sexual conflict in these systems. To overcome this difficulty, our aim is to examine not only diploidy and haplodiploidy but also “intermediate” cases, where some of the verbal arguments apply while others are prevented from operating. We construct several models to which none (diploidy), one, two, or all three (haplodiploidy) verbal arguments apply. Whenever possible, models are inspired by inheritance systems found in natural organisms. If no real organism with a given combination of verbal arguments is known, we consider hypothetical species with suitable properties. We investigate a total of eight different possible combinations (fig. 1).

**Figure 1:** Illustrations of the different models. Circles and squares represent females and males, respectively, and hexagons are used to indicate that offspring can have either sex. The symbols in the right corner of each panel indicate which verbal arguments are included in the model. A, In the diploidy model, none of the effects apply. B, In the facultative sex model, females have twice the reproductive value of males; that is, the maternal alleles (orange and green) are twice as common in the offspring generation than in the paternal alleles (purple and blue). C, In the PA model, males are haploid, but the other two effects do not apply. Males have the same reproductive value as females; that is, the paternal allele (purple) is as common in the offspring generation as the two maternal alleles (green and orange) taken together. D, In the alternation model, only the alternation effect applies. E, In the shuffle model, males are haploid, and females have twice the reproductive value of males. The alternation effect does not apply. F, In the paternal genome elimination (PGE) model, alleles are transmitted similarly to the haplodiploidy model. However, males are somatically diploid in the PGE model, and therefore recessive alleles are not always expressed in males. The other two effects apply just as in the haplodiploidy model. G, In the sporic model, males are haploid and alleles alternate between males and females, but the reproductive values of males and females do not differ. H, In the haplodiploidy model, all three effects apply.
In what follows, we rephrase the third argument, of males only siring daughters, to state that alleles alternate between sexes from one generation to the other more often in haplodiploids than in diploids. Consider a haplodiploid population with a proportion $0 < y < 1$ of females. All alleles in a male are maternally derived, while half of the alleles in females are paternally derived. An allele chosen randomly from the entire population has probability $1/(1+y)$ to be maternally derived and $y/(1+y)$ to be paternally derived. Consequently, an allele drawn from the male population is more likely to be maternally derived compared with alleles chosen at random from the entire population. We show later that the rephrased verbal argument explains why strongly antagonistic alleles can fail to invade in haplodiploids.

**The Models**

All models assume nonoverlapping generations and infinite population sizes (i.e., we ignore drift). Each generation consists of a bout of selection, during which sexually antagonistic alleles are expressed, followed by a single reproductive event. We assume random mating, no mutations, and no migration. There are two competing alleles in the population: the resident allele $A$, and the invading allele $A_i$. Two models (the facultative sex model and the shuffle model) make the additional assumption that a fraction of the population reverts back to Hardy-Weinberg equilibrium (HWE) in each generation (see below).

We set the fitness of both $A,A_i$ homozygotes and $A$, hemizygous to 1. The relative fitness of $A_i$ in comparison to $A$, in homo-/hemizygous is sex specific. It is determined by parameter $a_i$ ($-1 \leq a_i < \infty$), with the subscript $s$ referring to males (m) and females (f), respectively. For example, $a_{i_{mf}} = -0.2$ implies that $A_i A_i$ females have 20% lower fitness than do $A A_i$ females. We will use the subscript $s$ for all sex-specific values ($s \in \{m, f\}$). Heterozygote fitness additionally depends on dominance factor $h$ ($0 \leq h \leq 1$), which ranges from $h = 0$ (fully recessive) to $h = 1$ (fully dominant). When both sexes are diploid, we make the simplifying assumption that $h$ is the same in both sexes.

The variables $p_i(t)$ and $q_i(t)$ denote the allele frequency of the invading and the resident allele, respectively, in a given generation $t$ after selection has taken place, such that $p_i(t) + q_i(t) = 1$ and $p_{i_m}(t) + q_{i_m}(t) = 1$. For better readability, we drop the notation $(t)$ from now on.

Table S1 (available online) lists the sex-specific expressions for genotype frequencies (in zygotes) and fitness for each model based on parameters $w_{i_m}, w_{i_f}$, and $w_{r}$, which denote the fitness of individuals with sex $s$ that are homo-/hemizygous for $A_i (A_i A_i/A)$, heterozygous ($A A_i$), and homo-/hemizygous for $A_i (A_i A_i/A)$, respectively. For seven models (all except the paternal genome elimination [PGE] model, for reasons explained below), $w_{i_m} = 1 + a_{i_m}$, $w_{i_f} = 1 + a_{i_f}$, $w_{r} = 1 + ha_{r}$, $w_{i_m} = 1 + ha_{i_m}$, and $w_{i_f} = 1 + ha_{i_f}$.

The zygotic genotype frequencies for generation $t + 1$ (i.e., zygotes produced by generation $t$) are denoted by $z_{i_m}, z_{i_f}, z_{r}$ for genotypes $A_i A_i/A_i$, $A A_i$, and $A A_i/A$, respectively. These frequencies are model-dependent functions of $p_n, p_m, q_n$ and $q_m$ (table S1). After selection, the allele frequencies are given by

$$p_{i}(t+1) = \frac{w_i z_{i_m} + (1/2) w_i z_{i_f}}{w_i z_{i_m} + w_i z_{i_f} + w_i z_{r}},$$

$$p_{m}(t+1) = \frac{w_m z_{m} + (1/2) w_m z_{m} + w_m z_{m}}{w_m z_{m} + w_m z_{m} + w_m z_{m}}. \tag{1}$$

Equation (1) applies to all models. Note that in models with haploid males, males cannot be heterozygous; therefore, $z_{m2} = 0$.

Each model is created by inserting the terms from table S1 into equation (1). For example, in diplodiploids, the allele frequencies in the next generation equal

$$p_{i}(t+1) = \frac{(1 + a_{i}) p_{i} p_{m} + (1/2)(1 + ha_{m}) p_{i} q_{m} + p_{i} q_{m}}{(1 + a_{i}) p_{i} p_{m} + (1 + ha_{m}) p_{i} q_{m} + (1 + a_{i}) p_{i} p_{m} + p_{i} q_{m} + q_{m} q_{m}}. \tag{2}$$

Below we describe and explain each model individually (see fig. 1 for a graphical summary).

**Diplodiploid Model.** Individuals of both sexes are produced sexually and receive one haploid genome from each parent (fig. 1A). To become homozygous for the invading allele ($A_i A_i$), an individual must have inherited $A_i$ from both parents. The probability of inheriting $A_i$ from the mother is $p_{i_m}$ and the corresponding probability of inheriting it from the father is $p_{i_m}$, yielding the zygotic frequency of $A_i A_i$ of $z_{i_m} = p_{i_m} p_{i_m}$. Similarly, $A_i A_i$ zygotes occur with frequency $z_{r} = q_{m} q_{m}$, and heterozygote frequencies are $z_{r} = p_{m} q_{m} + p_{m} q_{m}$.

**Haplodiploid Model.** Females are produced sexually and inherit one haploid genome from each parent. Therefore, the equations for genotype frequencies of female zygotes are identical to those in the diplodiploid model. Males inherit their haploid genome from their mother (fig. 1H). Therefore, the zygotic genotype frequency of $A_i$ males
is \( z_{m1} = p_1 \), and the zygotic frequency of \( A \), males is \( z_{m3} = q_1 \). Male heterozygotes do not exist \( (z_{m2} = 0) \).

**Facultative Sex Model.** We consider a hypothetical form of facultative sex (fig. 1B) to incorporate the effect that alleles occur twice as often in females than in males while excluding the other two verbal arguments. Both sexes are diploid, and females produce offspring both asexually and sexually. We assume that both males and females can be produced sexually and asexually and that the offspring sex ratio does not differ between sexual and asexual reproduction. This assumption ensures that the alternation effect (or a reversed alternation effect) does not apply. Note, however, that many facultatively sexual species deviate from this assumption in one way or another (e.g., sexually produced *Daphnia* and asexually produced plasmids are always female). We further assume that females produce one-third of their offspring asexually and two-thirds sexually, which ensures that the model has the desired properties: females have twice the reproductive value of males, and a randomly chosen allele is maternally derived with a probability of two-thirds.

Additionally, we wish to exclude certain properties of real automixis, such as the excess production of homozogotes. To achieve this, we assume that recombination occurs whether reproduction was sexual or asexual and that asexually produced offspring revert to HWE in each generation, yielding genotype frequencies of \( p_1^2, 2p_1q_1, \) and \( q_1^2 \) for asexually produced \( AA, Aa, \) and \( aa \) individuals, respectively.

Sexually produced offspring (with one father and one mother each) have zygotic genotype frequencies equal to the diploidiploid case. Since two-thirds of offspring are sexually produced, with the remaining proportion the result of asexual reproduction, the zygotic genotype frequency of \( AA \) individuals of both sexes is \( z_1 = 2p_1p_2/3 + p_1^2/3 \). Similar calculations yield \( z_{m1} = 2q_1q_2/3 + q_1^2/3 \) and \( z_{m3} = 2p_1q_2/3 + q_1^2/3 \).

**PA Model.** To study the effect that recessive alleles are fully expressed in males in isolation from all other effects (fig. 1C), we consider sex determination that is based on the inheritance system of the pea aphid X chromosome (Jaquiéry et al. 2013). Pea aphids have two morphs of females, sexual and asexual, that differ in anatomy, reproductive behavior, and gene expression. Therefore, alleles that improve the fitness of sexual females do not necessarily benefit asexual females. Our focus is not on this contrast but on recessive alleles being expressed in males. To differentiate our mathematical model from the natural system, we will refer to the model as the PA model and to the organisms as pea aphids.

Our simplification necessitates removing some aspects of real pea aphid biology from the model. Pea aphids complete several asexual (female-only) generations in spring and summer, followed by a single sexual generation (comprising sexual females and males) in autumn. Both sexes of the last generation are clones of their asexual mothers, apart from the X chromosomes: sons receive only one. Therefore, recessive X-linked alleles are fully expressed in males but not in females. When sexual females and males mate, their offspring all become asexual females, which inherit their father’s X chromosome and one of their mother’s X chromosomes (Jaquiéry et al. 2013). Since pea aphid males pass their single X chromosome to all of their offspring, the male X chromosomes of a population have the same total reproductive value as the X chromosomes of sexual females in that population: a random X-linked allele sampled from a distant future generation has an equal probability of its ancestor residing currently in a male or in a sexual female.

We include the unusual X inheritance patterns in the PA model but skip all asexual-sexual contrasts by simply assuming that the system bypasses all asexual generations (fig. S4; figs. S1–S4 are available online). Therefore, the model applies only to alleles that have no fitness effects on asexual females. In the PA model, females (which are always produced sexually) always receive one haploid genome from their father and one from their mother. Therefore, genotype frequencies of female zygotes equal those of the diploidiploid model. Males are haploid, and 50% of them receive their genome from their mother, the other 50% from their father (fig. 1C). In either case, the inherited genome might carry either the \( A \) or the \( a \) allele. Thus, males have a \( p_1/2 \) probability to receive \( A \) from a mother, an \( p_2/2 \) probability to receive \( A \) from a father, a \( q_1/2 \) probability to receive \( A \) from a mother, and a \( q_2/2 \) probability to receive \( A \) from a father. Therefore, the genotype frequencies of male zygotes are \( z_{m1} = (p_1 + p_2)/2 \) for \( A \), males and \( z_{m3} = (q_1 + q_2)/2 \) for \( A \), males.

**Alternation Model.** The alternation model (fig. 1D) considers the effect that alleles are more likely to be derived from a parent of the opposite sex (fig. 2). Considering this effect in isolation from all others is possible with an entirely fictional life cycle where females mate with each other to produce male offspring and males mate with each other to produce female offspring. Therefore, the zygotic genotype frequencies are entirely determined by allele frequencies in parents of the opposite sex. The zygotic genotype frequencies of females are \( z_{f1} = p_1q_2, z_{f3} = 2p_2q_2, \) and \( z_{f5} = q_2^2 \). Similarly, the zygotic genotype frequencies of males are \( z_{m1} = p_1, z_{m3} = 2p_2q_1, \) and \( z_{m5} = q_1^2 \).

While biologically unlikely, the alternation model helps to provide an extreme case of alleles alternating between the sexes over generations. Both males and females are diploid and have equal reproductive value. Therefore, recessive alleles are equally expressed in both sexes, and alleles spend as many generations in males as in females.
Because of the assumption of random mating, genotype frequencies revert to HWE in each generation.

**Shuffle Model.** The shuffle model (fig. 1E) combines the effect that alleles are fully expressed in males with alleles finding themselves in females two-thirds of the time (fig. 2). This entirely fictional model differs from the haplodiploid model in that it allows males to have sons (fig. 1E). Any allele, whether sampled from a male or a female, has a two-thirds probability to be maternally derived and a one-third probability to be paternally derived. Thus, this model lacks the tendency for alleles to alternate between the sexes. The situation envisaged by the model involves a diploid female and haploid male first producing a triploid fertilized egg that contains the full diploid genome from the mother as well as the father’s haploid genome. Either one or two haploid genomes are then randomly excluded, leaving either a haploid or a diploid zygote. Depending on ploidy, the zygote develops into a female or a male.

Two-thirds of females are produced sexually, and the remaining one-third result from female-only reproduction, where the male genome is removed as described above. The genotype frequencies for female zygotes are $z_f = 2p^m p_n/3 + p_i/3$, $z_{f1} = 2(p^m q_m + p_n q_i)/3 + 2p^f q_i/3$, and $z_{f2} = 2p^f q_m/3 + q_i/3$, as in the facultative sex model. Daughters produced under female-only reproduction revert to HWE in each generation, as in the facultative sex model.

Males are haploid and receive their genome either from their mother (probability of two-thirds) or from their father (probability of one-third). Therefore, zygotic genotype frequencies are $z_m = p_m/3 + 2p_i/3$ for $A$, males and $z_{m1} = q_m/3 + 2q_i/3$ for $A$, males.

**PGE Model.** The PGE model (fig. 1F) combines the effect that alleles spend two-thirds of all generations in females with the sex alternation effect (fig. 2). This model is based on a naturally occurring inheritance system called paternal genome elimination, specifically of a type found in human head and body lice (de la Filia et al. 2015; de la Filia 2018), where males have a diploid soma and express both maternally and paternally inherited alleles but their sperm excludes the paternally inherited genome. The route of an allele through the generations is therefore identical to those of haplodiploid organisms (fig. 1F, 1H). However, because males are diploid and somatically express both parents’ genomes, males only express fully recessive alleles if they are homozygous. Our analysis is based on somatic expression of paternal alleles; thus, it should not be applied to other systems also referred to as paternal genome elimination, where the paternal genome is silenced in males (e.g., mealybugs; de la Filia et al. 2021).

Modeling the PGE system requires tracking more components than the other models. In most models we consider, it is sufficient to keep track of allele frequencies

| (1) Often in females | (2) Haploid males | (3) Sex alternation | Model name | Invasion criterion | Fixation criterion |
|---------------------|------------------|--------------------|------------|-------------------|-------------------|
| N                   | N                | N                  | Diploidiploid | $a_m + a_f > 0$ | $a_m + a_f + 2a_m a_f > 0$ |
| F                   | N                | N                  | Facultative Sex | $a_m + 2a_f > 0$ | $a_m + 2a_f + 3a_m a_f > 0$ |
| N                   | F                | N                  | PA          | $a_m + h a_f > 0$ | $a_m + (1 - h) a_f + (2 - h) a_m a_f > 0$ |
| N                   | N                | F                  | Alternation | $a_m + a_f + h a_f a_m > 0$ | $a_m + a_f + (1 + h) a_m a_f > 0$ |
| F                   | F                | N                  | Shuffle     | $a_m + 2h a_f > 0$ | $a_m + (2 - 2h) a_f + (3 - 2h) a_m a_f > 0$ |
| F                   | N                | F                  | PGE         | $a_m + 2a_f + h a_f a_m > 0$ | $a_m + 2a_f + (2 + h) a_m a_f > 0$ |
| N                   | F                | F                  | Sporic      | $a_m + h a_f + h a_f a_m > 0$ | $a_m + (1 - h) a_f + a_f a_m > 0$ |
| F                   | F                | F                  | Haplodiploid | $a_m + 2h a_f + h a_f a_m > 0$ | $a_m + (2 - 2h) a_f + (2 - h) a_f a_m > 0$ |

**Figure 2:** Verbal arguments and invasion and fixation criteria of each model. Symbols indicate which effect applies to the model, while minus signs indicate that an effect does not apply.
in the current generation, but paternal genome elimination necessitates tracking the parental origin of alleles carried by males. We do this by making the genotype frequencies \((z_{m1}, z_{m2}, z_{m3})\) and allele frequencies \((p_m, q_m)\) of males solely refer to the maternally derived genome. The paternally derived genome is also modeled, but only to modify the phenotype and fitness \((w_m, w_m)\) of males. Following this notation, the zygotic genotype frequencies of males are \(z_{m1} = p_t\) (probability that a male receives \(A_t\) from his mother), \(z_{m2} = 0\), and \(z_{m3} = q_t\) (probability that a male receives \(A_t\) from his mother)—that is, identical to the haplodiploid case. However, the fitness of males is based on a diploid soma. Males with \(AA\), soma have fitness of 1, males with \(AA\), soma have a fitness of \((1 + a_m)\), and males with \(AA\), soma have a fitness of \((1 + ha_m)\), regardless of the parental origin of these alleles.

A male zygote that received the invading allele \(A_t\) from his mother must have received either \(A_t\) from his father, for which the probability was \(p_m\) or \(A_m\), for which the probability was \(q_m\). In the former case, fitness equals \((1 + a_m)\), the male being somatically homozygous for \(A_t\). In the latter case, the male is heterozygous, with fitness \((1 + ha_m)\). The expected fitness of a male with a maternally derived \(A_t\) allele is thus \(w_m = (1 + a_m)p_m + (1 + ha_m)q_m\) (table S1F). A similar line of reasoning yields \(w_m = (1 + ha_m)p_m + q_m\), as the expected fitness for males with a maternally derived \(A_t\) allele.

Females are produced sexually, inheriting the father’s maternally derived allele and a random allele from their mother. Therefore, the zygotic genotype frequencies in females are \(z_{f1} = p_t p_m, z_{f2} = q_t q_m, \) and \(z_{f3} = p_t q_m + p_m q_t\), as in diploid models.

**Sporic Model.** The sporic model (fig. 1G) aims to combine two effects: that alleles are fully expressed in males and the sex alternation effect. It is inspired by sporic meiosis, the life cycle of land plants and many algae. In these organisms, a haploid generation (gametophyte) alternates with a diploid generation (sporophyte). Our sporic model reinterprets sporophytes as “female” and gametophytes as “male.” Females are diploid and produce only male offspring, which inherit one of their mother’s haploid genomes. Males are haploid and produce only female offspring by mating with each other. In nature, gametophytes usually exhibit two mating types (Thornber and Gaines 2004), but here we assume that any male can mate with any other male—that is, that gametophytes are monocious with little self-fertilization, as found in some horsetail populations (Walker 1921; Korpeleinen and Kolkkala 1996). The genotype frequencies in females are as in the alternation model—\(z_{f1} = p_m^2, z_{f2} = 2p_m q_m, \) and \(z_{f3} = q_m^2\)—while the haplodiploid model genotype frequencies apply for males:

\[z_{m1} = p_t, z_{m2} = 0, \text{ and } z_{m3} = q_t.\]

**Stability Analyses**

To assess whether a sexually antagonistic allele invades when rare (called the “invasion criterion” hereafter), we derive the Jacobian matrix of the system of difference equations and focus on trivial equilibria where \(p_t = p_m = 0\). We calculated the values of the parameters \(a_m, a_t, \) and \(h\) required for the larger of the two eigenvalues of the matrix to exceed 1. At this combination of values, the trivial equilibrium is unstable, and the sexually antagonistic allele invades. We also investigated whether the invading allele reaches fixation when common (henceforth, “fixation criterion”) by performing an analogous stability analysis at the equilibrium \(p_t = p_m = 1\). As an alternative method, it is possible to derive the fixation criterion from the invasion criterion (supplemental PDF [available online], text S1). All calculations were done in Maple (ver. 2019.1).

**Results**

We structure our results as follows. We first contrast the invasion of sexually antagonistic alleles in the haplodiploid and the diplodiploid models, followed by an analysis of fixation for these two models. We thereafter discuss the effects of each verbal argument by comparing the relevant models that contain elements of the focal argument with the diplodiploid model. Subsequently, we use the shuffle model to illustrate how the three verbal arguments interact in haplodiploids. Last, the sporic and PGE models are discussed to show that the three verbal arguments are sufficient together to explain all differences between the haplodiploid model and the diplodiploid model. The invasion and fixation criteria for each model are summarized in figure 2.

**Invasion in Haplodiploids and Diplodiploids**

Our results here align with those derived by others (e.g., Rice 1984), and we therefore keep their description brief.

The diplodiploid invasion criterion is

\[a_m + a_t > 0, \quad (4)\]

that is, a sexually antagonistic allele invades when the benefit to one sex is larger than the detrimental effect to the other sex (dotted line in figs. 3, 4).

For haplodiploids, the invasion criterion depends on the dominance factor \(h\) (colored lines in fig. 4A) and equals

\[a_m + 2ha_t + ha_a_m > 0. \quad (5)\]

In the fully dominant case \((h = 1)\), equation (5) simplifies to \(a_m + 2a_t + a_a_m > 0\). Parsons (1961) and Rice (1984) make the additional assumption that the invading allele has small effects on fitness, in which case \(a_t a_m\) is negligible.
Figure 3: Invasion, fixation, and polymorphisms in the PA model (only male haploidy) and the haplodiploid model (all three effects). The parameter space shows parameter $a_f$, the fitness effect on females, on the X-axis and $a_m$, the fitness effect on males, on the Y-axis. Male-beneficial, female-harming alleles are thus in the upper left quadrants, while female-beneficial, male-harming alleles are in the lower right quadrants. The black dotted and dashed lines represent the invasion criterion and the fixation criterion of the diplodiploid model (no effects), respectively: any allele above the respective line can invade or fix in the diplodiploid model, while alleles below the respective line fail to do so. The red lines show the invasion criteria of the PA ($A-C$) and the haplodiploid ($D-F$) model for a given dominance factor $h$, while the blue lines show the corresponding fixation criteria. Yellow indicates areas of stable polymorphism, while green indicates areas where alleles both invade and fix. In some cases, the lines of the PA model overlap with those for the diplodiploid model: for $h = 0$ the PA model has the same fixation criterion as the diplodiploid model, and for $h = 1$ it has the same invasion criterion.
and equation (5) simplifies to $a_m + 2a_f > 0$. This led these authors to conclude that the effect that a dominant allele has on females is twice as important as its effect on males. However, empirical work shows that the assumption of small effects of sexually antagonistic alleles is not always true (e.g., Chippindale et al. 2001). Equation (5) shows that stronger fitness effects make it increasingly difficult for sexually antagonistic alleles to invade.

The additive case ($h = 0.5$) has $a_m + a_f + 0.5a_m a_f > 0$ as the invasion criterion, which strengthens the message that alleles with strong sexually antagonistic effects can fail to invade in haplodiploids. Because $0.5a_m a_f$ is always negative for sexually antagonistic alleles ($a_m$ and $a_f$ have different signs), equation (5) is a strictly stronger condition than the invasion criterion for diploid models (eq. [4]). This means that some alleles with $h = 0.5$ can invade a diploid but not a haplodiploid population, but the opposite (invasion under haplodiploidy but not under diploidy) is impossible. Graphically, the statement translates into the area above the haplodiploid line with $h = 0.5$, where invasion is possible, being entirely within the area above the diploid line (fig. 4A); the area between the lines describes alleles that invade only under diploidy. The difference is especially stark for alleles with strong effects on fitness; thus, these are expected to fail to invade in haplodiploids.

For a fully recessive allele with $h = 0$, equation (5) simplifies to $a_m > 0$. Thus, in a haplodiploid population any fully recessive allele with a positive effect on males invades when rare, irrespective of its effect on homozygous females. 

**Fixation in Haplodiploids and Diploidy**

Alleles reach fixation in the diploid model when

$$a_m + a_f + 2a_m a_f > 0.$$ 

(6)
In the fully recessive case, the fitness effect on females for the allele to reach fixation is twice as important as its effect on males. Three generations in females as in males, the effect an allele has on females is twice as important as its effect on males. Three generations in females as in males (eq. [7]), a sexually antagonistic allele invades (fig. 4B) when

\[ a_m + 2a_i > 0 \]

and reaches fixation when

\[ a_m + 2a_i + 3a_m a_i > 0. \]

The result is intuitive: when alleles spend twice as many generations in females as in males, the effect an allele has on females is twice as important as its effect on males. Three features of the diploid model also apply to the facultative sex model: allelic dominance does not matter (no \( h \) term in the equations above), any allele that can reach fixation when common can also invade when rare, and under weak selection, alleles that invade when rare will usually reach fixation (fig. S1).

\[ \text{Invasion When Males Are Haploid: Male-Beneficial Alleles Invade More Easily Unless They Are Fully Dominant} \]

The PA model is designed to isolate the effect that recessive alleles are fully expressed in haploid males from other features of haplodiploidy. The invasion criterion is

\[ a_m + ha_i > 0, \]

a result also found by Jaquiéry et al. (2013).

Compared with the diploid model (eq. [4]), the female-beneficial effect is now scaled with \( h \) (thus, if \( h = 1 \), the criteria become identical). As long as the sexually antagonistic allele is not fully dominant (\( h < 1 \), the difference between equations (10) and (4) makes it easier for male-beneficial alleles to invade and harder for female-beneficial alleles to invade. This sexual difference and the difference from the diploid model both increase when \( h \) decreases toward recessivity (fig. 4C). A fully recessive (\( h = 0 \)) allele invades as long as it has any positive effect on males, even if it is lethal to homozygous females (eq. [10]).
Fixation When Males Are Haploid: Large Parameter Regions with Stable Polymorphism

In the PA model, the fixation criterion for a sexually antagonistic allele is

\[ a_m + (1 - h)a_i + (2 - h)a_m a_i > 0. \]  

(11)

For fully recessive alleles, this simplifies to \( a_m + a_i + 2a_m a_i \), which is identical to the fixation criterion of diploid models (eq. [6]; fig. 3A). For \( h > 0 \), it becomes increasingly difficult for female-beneficial alleles to reach fixation when common, while it becomes easier for male-beneficial alleles to do the same. When \( h = 1 \), the fixation criterion simplifies to \( a_m(1 + a_i) > 0 \); that is, fully dominant male-harming alleles never fix.

The PA model has large parameter regions in which dominant female-beneficial and recessive male-beneficial alleles invade when rare but fail to reach fixation. The PA model differs from the haplodiploid model in that the area of polymorphism is strongly skewed toward the invasion and fixation of male-beneficial alleles. Simultaneously, it retains a similarity with the haplodiploid model: in both models there is a parameter region in which recessive female-beneficial and dominant male-beneficial alleles fail to invade when rare but could reach fixation when common.

Sex Alteration Predicts More Difficult Invasion but Easier Fixation of Sexually Antagonistic Alleles

In the alternation model, individuals exclusively produce offspring of the opposite sex, in isolation of any other verbal arguments. An antagonistic allele invades when

\[ a_m + a_i + h a_i a_m > 0 \]  

and reaches fixation when

\[ a_m + a_i + (1 + h)a_m a_i > 0. \]  

(13)

For fully recessive \((h = 0)\) alleles, the invasion criterion (eq. [12]) simplifies to \( a_m + a_i > 0 \), the same invasion criterion as in the diploid model (eq. [4]). For any other case \((h > 0)\), the term \( h a_i a_m \) is negative (sexual antagonism); therefore, equation (12) indicates that sex alternation hinders the invasion of sexually antagonistic alleles, particularly if these alleles are dominant and have a strong effect on fitness (fig. 4D).

Equation (13) replaces a factor 2 of the diploid model (eq. [6]) with \( 1 + h \), which falls below 2 except when \( h = 1 \) (in which case the alteration model has the same fixation criterion as the diploid model). Sex alternation therefore enhances the fixation of sexually antagonistic alleles, particularly if these alleles are recessive and have a strong effect on fitness.

In conclusion, in the alternation model, sexually antagonistic alleles invade less easily but reach fixation more easily than in the diploid model (fig. S2A–S2C). More specifically, dominant alleles invade less easily, while recessive alleles fix more easily. Therefore, stable genetic polymorphisms are expected to be even rarer in the alternation model than in the diploid model; alleles either reach fixation or fail to invade.

Interactions between the Three Effects: For Additive Dominance, Two Verbal Arguments Cancel Out

The shuffle model combines the effect that alleles spend two-thirds of all generations in females with the effect of haploid males. The invasion criterion is

\[ a_m + 2h a_i > 0, \]  

(14)

while the fixation criterion is

\[ a_m + (2 - 2h)a_i + (3 - 2h)a_m a_i > 0. \]  

(15)

At \( h = 0.5 \), both the invasion criterion and the fixation criterion of the shuffle model (eqq. [14], [15]) are identical to those of the diploid model (eqq. [4], [6]). Under additivity, therefore, the female-favoring effect (alleles spend more generations in females) and the male-favoring effect (alleles are fully expressed in males) cancel out exactly. As a corollary, at \( h = 0.5 \), all differences between the haplodiploid model and the diploid model should be attributed to the sex alternation effect.

The same phenomena—that is, identical invasion and fixation criteria at \( h = 0.5 \)—also apply to a comparison between the haplodiploid model (eqq. [5], [7]) and the alternation model (eqq. [12], [13]). Even though the alternation model represents an extreme case where alleles alternate sexes every single generation (stronger than the alternation tendency in haplodiploids), the sex alternation effect is as relevant in haplodiploids as in the alternation model.

All Three Verbal Arguments Are Necessary and Sufficient Together to Understand the Spread of Sexually Antagonistic Alleles in Haplodiploids

This section has two aims: first to show that the results from the single-effect models also apply to the models with two effects, and second to show that the three effects together are sufficient to explain all differences between the haplodiploid model and the diploid model regarding invasion and fixation. For this purpose, we focus on the results from two remaining models (sporic and PGE). Both models combine two verbal arguments.

For both models, we will first state the invasion and fixation criteria. Second, we show that the verbal arguments
produce the same qualitative results in these two-effect models as they do in the one-effect models discussed above. Finally, we will compare the models to the haplodiploid model to show that the models behave like a haplodiploid model from which a single effect was removed, that is, that the three arguments are sufficient together.

**PGE Model.** The PGE model combines the effect that alleles spend two-thirds of all generations in females with the sex alternation effect; therefore, we here contrast it with the facultative sex model and the alternation model. In the PGE model, the invasion criterion is

\[ a_m + 2a_f + h a_f a_m > 0 \]  

and the fixation criterion is

\[ a_m + 2a_f + (2 + h)a_f a_m > 0. \]

The invasion and fixation criteria of the PGE model closely resemble those of the facultative sex model (eqq. [8], [9]). In both models, female-beneficial alleles invade more easily than male-beneficial alleles, regardless of dominance. However, invasion is strictly more difficult, and fixation easier, in the PGE model (which includes sex alternation) than in the facultative sex model (fig. S3A–S3C). This contrast is analogous to the sex alternation effect found when comparing the alternation model to the diplodiploid model.

When comparing the haplodiploid model to the PGE model, we find that somatic haploidy in males, which is present in the haplodiploid model only, makes both invasion and fixation of male-beneficial alleles more likely. In addition, haploidy in males promotes stable polymorphisms. These qualitative results are also found when comparing the PA model (haploid males) with the diplodiploid model (diploid males). For fully dominant alleles (\( h = 1 \)), the haplodiploid model and the PGE model produce identical invasion criteria, \( a_m + 2a_f + a_f a_m > 0 \) (eqq. [5], [16]). For fully recessive alleles (\( h = 0 \)), the fixation criteria become identical, \( a_m + 2a_f + 2a_f a_m > 0 \) (eqq. [7], [17]). For any other dominance factor, male-beneficial alleles invade and reach fixation more easily in the haplodiploid model than in the PGE model, while female-beneficial alleles invade and reach fixation less easily in the former than in the latter. In the PGE model, most alleles that invade when rare will also reach fixation, meaning that stable genetic polymorphisms are rare. Conversely, in the haplodiploid model many dominant female-beneficial alleles and many recessive male-beneficial alleles invade when rare but fail to reach fixation.

**Sporic Model.** The sporic model combines the effects that alleles are fully expressed in males with the sex alternation effect; thus, the relevant comparisons are to the PA model and the alternation model. In the sporic model, an antagonistic allele invades when

\[ a_m + h a_f + h a_f a_m > 0 \]  

and reaches fixation when common if

\[ a_m + (1 - h)a_f + a_f a_m > 0. \]

The invasion and fixation criteria of the sporic model are very similar to those of the PA model (eqq. [10], [11]), with the shared assumption that alleles are fully expressed in haploid males. In both models, it is easy for recessive male-beneficial and dominant female-beneficial alleles to invade but fail to reach fixation, predictive of stable polymorphisms. The flip side—many recessive female-beneficial and dominant male-beneficial alleles fail to invade when rare but could reach fixation when common—is also shared between the two models. However, invasion is strictly more difficult (occurs over a narrower parameter range) in the sporic model than in the PA model; conversely, fixation is strictly easier in the former than in the latter. Harder invasion but easier fixation is analogous to what we found for the sex alternation effect when comparing the alternation model to the diplodiploid model.

The differences between the haplodiploid model and the sporic model are analogous to the female-favoring effect found for the facultative sex model compared with the diplodiploid model. Male-beneficial alleles invade and fix more easily in the sporic model than in the haplodiploid model, while female-beneficial alleles more easily invade and fix in the haplodiploid model. This is the case for almost every dominance factor \( h \), although there are some values at which either the fixation criterion (\( h = 1 \)) or the invasion criterion (\( h = 0 \)) of the haplodiploid model and the sporic model become identical.

**Discussion**

Our models show that the three verbal arguments that act together to produce differences in how sexual conflict operates in haplodiploids versus diplodiploids represent three distinct processes that can be disentangled. These processes are all necessary to understand the evolution of sexually antagonistic alleles in haplodiploids. Taken together, they are also sufficient to explain all previously published differences between haplodiploids and diplodiploids regarding intralocus sexual conflict.

Our models confirm the intuition that alleles “pay more attention” to female fitness when spending more time in females. Put more precisely, when alleles spend more generations in females than in males, female-beneficial alleles invade and fix more easily and male-beneficial alleles invade and fix less easily. This effect does not depend on allelic dominance, which stands in contrast to previous suggestions.
that this effect might only apply to dominant alleles (Pennel et al. 2013). The confusion arises because recessive alleles are additionally strongly affected by their expression in haploid males. Our approach allows this effect to be viewed separately from the generational asymmetries. When both act together, the net outcome depends on dominance. For the specific case of additivity ($h = 0.5$), the consequences of alleles spending two-thirds of generations in females are exactly canceled out by male haploidy. A rare additive allele is expressed twice as strongly in males than in heterozygous females but spends twice as many generations in females as in males. This is in line with Hitchcock and Gardner’s (2020) suggestion to refer to these effects as the “power” and the “agenda” of an allele. The initial invasion of a novel X-linked, recessive, male-beneficial, female-detrimental allele has been experimentally confirmed (Dean et al. 2012). The invasion of a novel dominant allele with strong detrimental effects on males and a lower beneficial effect on females remains, to our knowledge, untested.

When males are haploid, both the invasion and the fixation of sexually antagonistic alleles become skewed in favor of male-beneficial alleles. Additionally, under male haploidy both dominant female-beneficial alleles and recessive male-beneficial alleles can often invade when rare but fail to reach fixation when common. Our consequent predictions that intralocus sexual conflict should be more easily observable in species with haploid males, as well as on X chromosomes, align with earlier work (Rice 1984; but see Fry 2010). However, male haploidy can also prevent recessive female-beneficial and dominant male-beneficial alleles from invading when rare while making them more likely to reach fixation when common. This leads to positive frequency dependence, making polymorphisms less likely (Lehtonen and Kokko 2012). Empirically, X chromosomes have been found to carry a disproportional amount of sexually antagonistic fitness variation, although this trend is not ubiquitous across taxa and results differ between phenotypic and molecular studies (reviewed in Dean and Mank 2014).

The X chromosome of pea aphids is a special case, as it is haploid in males but spends equally many generations in sexual females and males. It is enriched for alleles that are expressed more strongly in males than in sexual females (Jaquiéry et al. 2013). This sex-biased gene expression can be used as a proxy for intralocus sexual conflict, based on the assumption that a sexually antagonistic allele first spread at a locus and that this locus was later silenced in the disfavored sex. Therefore, the male-biased gene expression of many X-linked genes in the pea aphid aligns with the predictions of our model that male-beneficial, female-harming alleles often invade and reach fixation when X-linked alleles are always expressed in males.

Clear examples of intralocus conflict in haplodiploids are rare, as the study of sexual conflict in haplodiploids has focused more on sex allocation and polyandry (Beukeboom et al. 2009; Shuker et al. 2009; Macke et al. 2014). A notable exception is a study of extreme intralocus sexual conflict in the invasive ant *Nylanderia fulva* (Eyer et al. 2019). Almost every male in this population carries an allele that is lethal to homoyzygous females. The great majority of females are heterozygous at that locus and in turn carry one allele that is lethal to males. A substantial region (3%) of the genome cosegregates with this locus. Because of differential mortality, daughters are highly likely to share this region with their maternal grandmothers, while sons share this region with their (obviously maternal) grandfathers. This represents a particularly extreme example of intralocus sexual conflict.

The lethality of the male-beneficial allele for males and the lethality of the male-beneficial allele for homozogous females combine to a great cost for the colonies: nearly half of all offspring, including workers, die as eggs or during their first larval stage. Our models predict this type of strongly sexually antagonistic polymorphism to be more common in haplodiploids than in diplodiploids.

We found that males having only daughters (and never sons) makes it harder for sexually antagonistic alleles to invade when rare and easier to reach fixation when common. Perhaps counterintuitively, this effect is symmetrical with respect to the sex that is benefited by the allele. Our finding stands in contrast to previous suggestions that males having no sons might make it easier for female-beneficial alleles to invade (de la Filia et al. 2015). We explain the symmetry by rephrasing the verbal argument to state that when moving from one generation to the next, alleles alternate between the sexes more frequently in haplodiploids than a baseline 1:1 placement in each sex would predict. We suspect that the sex alternation effect is often overlooked because the most cited models assume weak selection (Rice 1984; but see Pamilo 1979), which makes the sex alternation effect disappear. A recent empirical example (Eyer et al. 2019) highlights the necessity to include extreme cases in models: sexually antagonistic alleles may even be lethal to one sex. The sex alternation effect makes the invasion of such lethal alleles less likely but does not prohibit it completely. Eyer et al. (2019) show that lethal alleles can reach high frequencies despite males producing only daughters. That ant males are haploid helps to override the sex alternation effect to some degree, explaining why this system can persist.

**Model Assumptions**

Whenever possible, we based our models on naturally occurring sex determination systems. Note, however, that some of our models differ from the natural system in ways that are relevant for intralocus sexual conflict. Specifically, the facultative sex model assumes that both asexual and sexual progeny can have either sex, the PA model excludes...
asexual generations, and the PGE model applies only to some species with paternal genome elimination but not to others. As already acknowledged, some combinations of verbal arguments do not, to our knowledge, exist in nature. The reason to construct models for those too is the clarity they provide, allowing us to see how different features of a life cycle impact the prospects of invasion or fixation of antagonistic alleles in isolation from others. The shuffle model, for example, is entirely fictional but essential, because it shows that the two verbal arguments of this model will exactly cancel each other out for additive gene action and that the sex alternation effect is surprisingly strong for haplodiploids. Using only naturally occurring sex determination systems, these insights would have been much harder to find.

We assumed that X-linked inheritance is analogous to haplodiploid inheritance. It is, however, important to remember that this analogy works only under the assumption that X-linked alleles evolve independently of the autosomal genome. This is not always true, for example, if X-linked genes regulate or are regulated by autosomal or Y-linked genes (e.g., Chase et al. 2005) or if alleles translocate to another region of the genome. Our diploid and haplodiploid models are identical to the autosomal model and the X-linked model in Rice (1984). These models, although originally designed to predict the evolution of X-linked alleles, entirely ignore interactions between the autosomal genome and X chromosomes. Ironically, these models are therefore more applicable to haplodiploid animals in which the entire genome is inherited in an X-linked fashion (Kraaijeveld 2009).

Our models are agnostic as to which fitness component (survival or reproductive success) is altered by the sexually antagonistic allele. The results apply whether one envisages differential survival followed by random mating (and equal reproductive success among all females) or equal survival followed by differential mating success among males and differential fecundity among females (or vice versa should one consider a sex role–reversed species). The models do not, however, consider complications that may arise if, in eusocial species, queen fitness depends on the phenotypes of both the queen and her workers (thus, we have left intercaste conflicts outside our study; see Pennell et al. 2018), nor do we consider female choice of mates. Female choice also causes males to compete with each other, but with the added complexity that attractive traits in males coevolve with preference alleles in females. Genetic architecture can play a role in these settings (Reeve and Pfennig 2003; Albert and Otto 2005; Muralidhar 2019); for example, female preference for an X-linked attractive trait might be less advantageous, as it is inherited exclusively by her daughters.

Another common deviation from random mating, not considered here, is assortative mating by fitness, where high-fitness females mate with high-fitness males while lower-fitness females mate with lower-fitness males. Under this form of assortative mating, intralocus sexual conflict is predicted to often remain unresolved because of high frequencies of heterozygous individuals (Arnqvist 2011). A clear avenue for further work is to combine such results with the considerations of different life cycles that we have provided.

The illustrations of our life cycles (fig. 1) might give the impression that our models assume equal sex ratios. However, the mathematical formulations of the models make neither explicit nor implicit assumptions about sex ratios. Whenever using equal sex ratios to explain the logic of an argument, we did so purely for the sake of ease of understanding. This makes it necessary to emphasize that all arguments also work under unequal sex ratios, including the fact that in haplodiploids alleles spend two-thirds of all generations in females, although this is not obvious and requires additional modeling to prove (see Gardner 2014). Nonetheless, unequal sex ratios can indirectly affect intralocus sexual conflict. The more common sex often experiences stronger selection, while drift impacts the other sex more (Mullon et al. 2012).

Finally, our models are fully deterministic—that is, they ignore processes such as genetic drift. There are several reasons why future work could usefully consider genetic drift in the context of intralocus sexual conflict in haplodiploids. First, sexually antagonistic alleles are exposed to opposite selection pressures in different generations and might therefore be more affected by genetic drift and random extinction than alleles with the same average effect on fitness but benefiting both sexes equally (Connaughton and Clark 2012). Second, haplodiploidy halves the effective population sizes of alleles in males, and therefore alleles that affect male fitness might be more strongly affected by drift than similar alleles in diploid species. However, it has also been suggested that selection is stronger in haplodiploids because alleles are more frequently expressed in males and therefore exposed to selection (reviewed in Avery 1984), and hence drift would be less important for recessive alleles. Mullon et al. (2012) modeled the effect of drift on sexually antagonistic alleles on X and Z chromosomes, using the assumption that males have higher variance in reproductive output than females. The authors conclude that sexually antagonistic alleles should accumulate on X chromosomes in XY species (XY males and XX females) and on autosomes in ZW species (ZZ males and ZW females).

**Concluding Remarks**

Our models confirm two intuitive arguments that have been used in the literature for decades (Reeve 1993; Lercher et al. 2003; Jaquiéry et al. 2013; Hitchcock and Gardner 2020). Female-beneficial alleles easily invade in haplodiploids because they spend two-thirds of all generations in females.
Recessive male-beneficial alleles easily invade in haplodiploids because they are hidden from selection in heterozygous females. However, we show that another verbal argument does not work quite as intuitively. Contrary to expectation, the argument that males produce only daughters does not favor the invasion of female-beneficial alleles. Instead, this effect makes it slightly harder for any sexually antagonistic allele to invade, regardless of which sex is benefited by the allele.

By disentangling the three verbal arguments, we have contributed to a more intuitive understanding of how sex determination systems affect intralocus sexual conflict. Our findings generalize beyond haplodiploids. While haplodiploids combine all three verbal arguments into one package, the verbal arguments apply more generally. A plethora of different sex determination systems occur in nature, and often some but not all of the three verbal arguments apply to a system. We hope that this work enables more informed predictions on how intralocus sexual conflict is resolved in such species.

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Statement of Authorship

K.K. conceived the presented ideas. K.K. developed the models and performed all computations under the supervision of H.t.B. and H.K. K.K. wrote the first draft of the manuscript with significant input from H.t.B. All authors contributed to writing the final manuscript and to revisions during the review process.

Data and Code Availability

The Maple code used for this study is available at https://doi.org/10.5281/zenodo.5137212. (Klein et al. 2021). No data were used in this study, and all results should be reproducible in any analytical software.

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