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E-Article

Genomic Imprinting and Sex Allocation

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abstract: Genomic imprinting allows maternally and paternally derived alleles to have different patterns of expression (one allele is often silent). Kin selection provides an explanation of genomic imprinting because conflicts of interest can arise between paternally and maternally inherited alleles when they have different probabilities of being present in other individuals. Our aim here is to examine the extent to which conflicts between paternally and maternally inherited alleles could arise over the allocation of resources to male and female reproduction (sex allocation), for example, conflict over the offspring sex ratio. We examine the situations in which sex allocation is influenced by competitive or cooperative interactions between relatives: local resource competition, local mate competition, and local resource enhancement. We determine solutions for diploids and haplodiploids when either the mother or the offspring controls sex allocation. Our results suggest that the greatest conflict between paternally and maternally inherited alleles and therefore the strongest selection for genomic imprinting will occur in haplodiploid species where the offspring can control sex allocation, such as the social hymenoptera and the polyembryonic parasitoid wasps. Within the social hymenoptera, we expect especially strong selection for genomic imprinting in species subject to local resource competition, such as honeybees and army ants.

Keywords: conflict, direct fitness, kin selection, Hymenoptera, inclusive fitness, sex ratio.

Introduction

Genomic imprinting occurs when maternally and paternally derived alleles have different patterns of expression (Burt and Trivers 2006). Usually one allele is silent and the other active, although the difference in levels of expression can be more subtle. The clearest examples of genomic imprinting were first described in insects, but this phenomenon has since been most studied in plants and mammals, especially mice and humans (Field et al. 2004; Burt and Trivers 2006).

Kin selection theory provides a possible explanation for the selective advantage of genomic imprinting. Very simply, kin-selected behaviors strike a balance among the competing interests of genetically related individuals (Hamilton 1964). In some cases, paternally and maternally inherited genes in one individual have different probabilities of also being present in other individuals, and so genetic relatedness differs depending on which point of view (paternally or maternally inherited genes) we adopt. In these same cases, maternally and paternally inherited genes naturally disagree over how a kin-selected balance among competing interests is to be struck (Haig 2000, 2002, 2004). This has been termed the kinship theory of imprinting.

The classic example of the kinship theory of genomic imprinting is overparental investment (Haig 2002; Burt and Trivers 2006). Assuming a large outbred population, a gene derived from the father will be unrelated to the mother and so will be selected to maximize the amount of resources obtained from the mother. In contrast, maternal genes have a kin-selected (indirect) interest in the mother's survival and production of further (related) offspring. Consistent with this prediction, a high proportion of the genes that are imprinted in the mammalian genome are involved in fetal growth, with paternal imprinting leading to greater growth and hence greater resource acquisition from the mother. Although the kinship theory of genomic imprinting has proved popular, other explanations have been suggested, and a potential limitation is that the theory has been developed to explain the data rather than data collected to test a priori predictions of theory (Wilson and Burley 1983; Hurst and McVean 1998; Wilkins and Haig 2003; Badcock and Crespi 2006; Wood and Oakley 2006).

Sex allocation has provided some of the clearest support for kin selection and social evolution theory (Charnov

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1982; Hamilton 1996; West et al. 2005; Meunier et al. 2008). Sex allocation theory predicts that under certain circumstances, the marginal fitness benefit of allocating resources to male or female reproduction differs, selecting for biased sex allocation. In particular, there is a huge theoretical literature predicting when sex allocation should be manipulated in response to environmental conditions and a rich empirical literature proving a wealth of qualitative and in some cases quantitative support for the predictions of theory (Charnov 1982). Indeed, it has been suggested that sex allocation theory is the area of evolutionary theory that best proves the power of the Neo-Darwinian paradigm and can have a predictive power almost comparable to that of the "hard" sciences such as physics or chemistry (Hamilton 1996).

Given these past successes of sex allocation theory, we suggest that it is useful to examine the extent to which sex allocation can provide testable predictions for genomic imprinting. The possibility of genomic imprinting influencing sex allocation has attracted only limited attention. It is well accepted that there are a number of cases in which we might expect conflict over sex allocation between mothers and their offspring (Trivers and Hare 1976; Werren and Hatcher 2000; Beukeboom et al. 2001; Werren et al. 2002; Gardner et al. 2007; Uller et al. 2007) or between parents (Trivers 1974; Charnov 1982; Pen and Weissing 2002; Wild and Taylor 2005; Pen 2006; Wild 2006). In situations where there are conflicts between parents, this opens the possibility for selection for genomic imprinting. The area in which this possibility has been most explicitly developed is in social insects, where the haplodiploid genetics means that fathers make a genetic contribution only to daughters. Consequently, if we consider the behaviors of the workers, paternally inherited alleles favor a more female-biased sex allocation (Haig 1992; Queller 2003). Conflicts between parents and the subsequent selection for genomic imprinting have also been suggested to be important in the evolution of sex determination systems, including the evolution of haplodiploidy (Werren and Beukeboom 1998; Normark 2003, 2006).

Our aim here is to examine when genomic imprinting in sex allocation is selectively advantageous. The kinship theory of genomic imprinting suggests that genomic imprinting can be favored when the genetic relatedness between social interactants differs from the point of view of maternal and paternal genes (Haig 2000, 2002, 2004). An area of sex allocation in which this could be important is that in which competitive or cooperative interactions between relatives drive the pattern of sex allocation: local resource competition (LRC) among related females (Clark 1978), local mate competition (LMC) among related males (Hamilton 1967), and local resource enhancement (LRE; Taylor 1981). Here, we investigate these three scenarios, considering the influence of mating system (monogamy, polygyny, or polyandry), genetics (diploid or haplodiploid), and who has control of sex allocation (parents or offspring; e.g., Trivers and Hare 1976). In order to provide a treatment that is amenable to both empirical and theoretical workers, we describe our predictions conceptually and graphically, based on a formal theoretical analysis that is presented in appendixes A–E. In the majority of situations we shall discuss, there is a complete lack of data on whether genomic imprinting occurs in the relevant species, let alone whether it occurs in genes involved in sex allocation. However, we see this as an advantage because it allows us to make clear predictions before the data are available. The increasing interest in the genetic mechanisms underlying sex allocation and especially how these may be influenced by conflict will provide data that could be used to test our predictions qualitatively.

Where Could Conflict Occur?

Consider a dioecious species, with separate sexes, where two parents produce offspring and the relevant sex allocation problem is the sex ratio of those offspring. Genomic imprinting could influence the sex ratio of those offspring in at least three ways (fig. 1). First, the maternally and parentally inherited alleles in the parents (derived from the grandparents) could favor a different sex ratio (fig. 1*a*). In this case, there could be imprinting on the genes that control offspring sex or genes involved in any parental behavior that differentially influences sex allocation. This could occur in any species where one or both of the parents are able to control the offspring sex ratio (primary or secondary).

The second and third cases both occur when the maternally inherited and paternally inherited genes in the offspring (derived from the parents) favor a different sex ratio. The second case is when sex determination occurs in the offspring, as with mechanisms such as environmental sex determination (fig. 1*b*). In this case, there could again be imprinting on the genes involved with sex determination. This could occur in species where the offspring control their own sex, such as environmental sex determination, sex change, or simultaneous hermaphrodites who can alter their relative allocation to male and female reproduction. The third possibility, when sex determination is not under the control of the offspring, is when the offspring has some mechanism to alter the sex ratio during development (Trivers and Hare 1976), for example, through influencing larval mortality. This would lead to a difference between the sex ratio at conception (primary sex ratio) and the sex ratio when reaching maturity (secondary sex ratio). In this case, there could be imprinting on the genes involved with interactions with

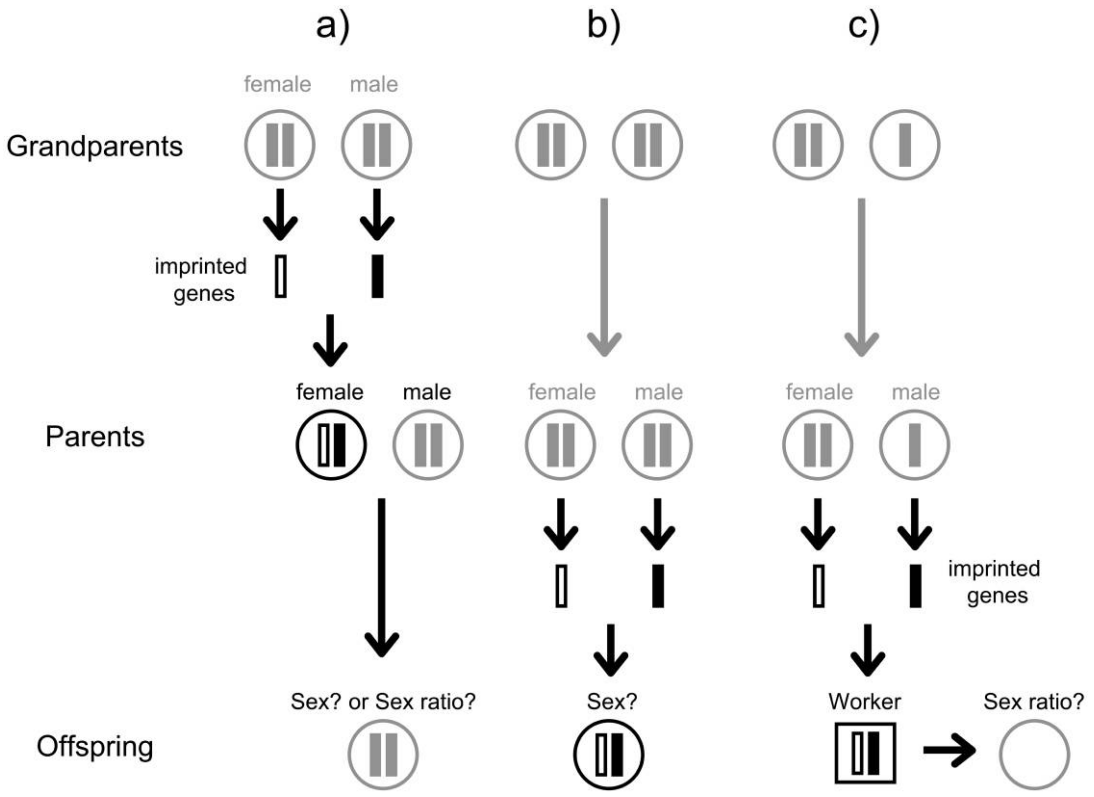

Figure 1: We will consider the effects of genomic imprinting under the assumption that there is either maternal control of the sex ratio or offspring control of the sex ratio. *a*, Under maternal control, imprinted alleles in the actor (*black*) influence the offspring sex and/or any behavior that affects sex allocation (diploid or haplodiploid). *b*, Under diploid offspring control, imprinted alleles in the actor (*black*) influence offspring sex. *c*, For haplodiploids, we consider the possibility that an offspring actor (e.g., a member of a worker caste; *black*) influences the sex ratio of a brood produced by its parents.

relatives. This could potentially occur in cooperative social species, where offspring help rear their siblings and other relatives (e.g., fig. 1*c*), in polyembryonic parasitoid wasps with a sterile soldier caste (Grbic et al. 1992), or when there is asymmetric larval competition for resources (Sykes et al. 2007).

The potential for genomic imprinting over sex allocation has been analyzed in the case of haplodiploid social species. Assuming that she has mated only once, a queen favors equal investment in the sexes because she is equally related to sons and daughters ($r = 0.5$ in both cases). In contrast, the workers are three times more related to sisters ($r = 0.75$) than to brothers ($r = 0.25$) and so are selected to invest three times as much resources in sisters, leading to a 0.75 allocation in females (Trivers and Hare 1976). Haig (1992) pointed out that conflict could occur over the sex allocation between paternal and maternal genes because the 0.75 investment was an average of the optimum from the point of view of a maternal gene ($r = 0.5$ to both brother and sisters, favoring an investment ratio of 0.5) and the paternal gene ($r = 1$ to sisters and $r = 0$ to brothers, favoring an investment ratio of 1.0 or a sex ratio of 0). A variety of factors such as queen mating frequency and the number of queens in the nest can alter the genetic relatedness structure of social insect populations, leading to more complicated patterns (Chapuisat and Keller 1999; Mehdiabadi et al. 2003; Meunier et al. 2008), but there has been a lack of further attention to the consequences of genomic imprinting (but see Queller 2003).

Local Resource Competition

We report results for a variety of different LRC models (see app. A). To help the reader, we summarize all LRC results in table 1.

Diploidy with Maternal Control of the Sex Ratio

Female Monogamy. We start by considering the simplest possible model of LRC among diploid females. We assume

Table 1: Summary of the equilibrium sex ratios for local resource competition models

Model, control, and mating system	Sex ratio		
	No imprinting	Paternally inherited	Maternally inherited
Diploidy:			
Maternal:			
Female monogamy	$\frac{N(3N+1)}{3N(2N-1)+1}$	$\frac{2N^2}{4N^2-3N+1}$[a]	$\frac{2N^2(3N+1)}{(4N^2-N+1)(3N-1)}$
Polyandry	$\frac{MN(3N+1)}{6MN^2-2MN-N+1}$	$\frac{2MN^2}{2MN(2N-1)-N+1}$[a]	$\frac{2MN^2(3N+1)}{2MN[3N(2N-1)+1]-(N-1)^2}$
Polygyny	$\frac{3N+1}{5N-1}$	$\frac{2N}{3N-1}$	$\frac{2N(3N+1)}{11N^2-4N+1}$[a]
Offspring:			
Female monogamy	$\frac{3N+1}{6N}$	$\frac{2N}{4N-1}$[a]	$\frac{2N(3N+1)}{12N^2-N+1}$
Polyandry	$\frac{M(3N+1)}{6MN+M-1}$	$\frac{2MN}{4MN-1}$[a]	$\frac{2MN(3N+1)}{12MN^2-N+1}$
Polygyny	$\frac{3N+1}{5N+1}$	$\frac{2}{3}$	$\frac{2(3N+1)}{11N+1}$[a]
Haplodiploidy:			
Maternal:			
Female monogamy	$\frac{N(3N+1)}{2(3N^2-2N+1)}$	$\frac{N^2}{2N^2-2N+1}$	$\frac{N^2(3N+1)}{6N^3-4N^2+3N-1}$[a]
Polyandry	$\frac{MN(3N+1)}{2(3MN^2-MN-N+1)}$	$\frac{MN^2}{MN(2N-1)-N+1}$[a]	$\frac{MN^2(3N+1)}{MN[3N(2N-1)+1]-(N-1)^2}$
Polygyny	$\frac{3N+1}{4N}$	1	$\frac{3N+1}{5N-1}$[a]
Offspring:			
Female monogamy	$\frac{3N+1}{4(3N-2)}$	0	$\frac{N(3N+1)}{2(3N^2-2N+1)}$[a]
Polyandry	$\frac{M(3N+1)}{2[M(3N-1)+3(N-1)]}$	0	$\frac{MN(3N+1)}{2(3MN^2-MN-N+1)}$[a]
Polygyny	$\frac{3N+1}{4N}$	…	$\frac{3N+1}{4N}$

Note: Recall that N refers to the number of females breeding on a patch and M refers to the number of mates chosen by each female in polyandrous systems.

[a] The more moderate (i.e., less biased) allele-specific perspective.

nonoverlapping generations, and we assume that each generation proceeds as follows. (1) An amount N of singly mated (i.e., monogamous) females breed on a patch and produce a large number of offspring. On average, a fraction z of the offspring are sons (z is called the sex ratio). We assume that male and female offspring are equally costly to produce, and so z mirrors sex allocation decisions. (2) Following birth, males—and only males—disperse completely before the next round of mating occurs. We assume that males disperse infinitely far so that they do not compete against relatives. (3) After the dispersal phase, intrasexual competition occurs at random (among males, competition is for mates; among females, competition is for breeding sites) and the next generation begins.

If the sex ratio is under maternal control and if there is (for the moment) no genomic imprinting, then it is possible to show that the population is at an equilibrium with respect to the evolution of z when z itself is equal to no imprinting,

$$z^* = \frac{N(3N+1)}{3N(2N-1)+1}, \tag{1}$$

which is, in turn, greater than 1/2.

Under LRC, the production of daughters carries with it an inclusive fitness penalty (Clark 1978; Taylor 1981); hence, selection favors sex ratios—such as z^* in equation (1)—that are male biased. The extent of the bias depends on the severity of the inclusive fitness penalty, which, in turn, depends on $\overline{R}$, the average relatedness among competing females. When patch size is small, $\overline{R}$ is quite large and equilibrium sex ratios become strongly biased toward males (the inclusive fitness penalty for production of daughters is relatively large). In contrast, when patch size is very large, $\overline{R}$ becomes small and equilibrium sex ratios tend toward 1/2 (the inclusive fitness penalty for production of daughters is relatively small). As we shall see, thinking about how $\overline{R}$ (or the appropriate analogue to $\overline{R}$)

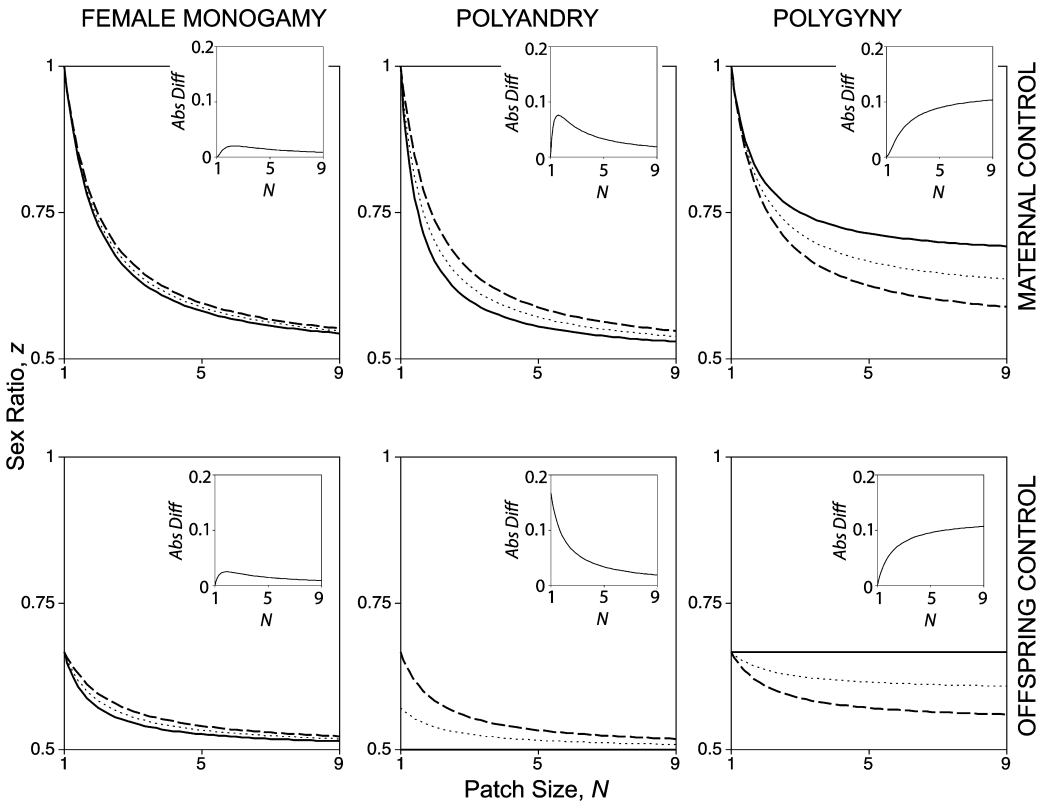

Figure 2: Graphical depiction of the relationship between the stable sex ratio and patch size for diploid local resource competition models. We show results for both maternal and offspring control and for each of the three different mating systems (in the polyandry case, the large M approximation is given). Solid curves refer to sex ratios "preferred" by paternally inherited alleles (i.e., grandpaternal alleles in mother or paternal alleles in offspring), dashed curves refer to sex ratios "preferred" by maternally inherited alleles (i.e., grandmaternal alleles in mother or maternal alleles in offspring), and dotted curves refer to standard "no imprinting" results. Insets depict how the absolute difference (*Abs Diff*) between sex ratios favored by paternally inherited and maternally inherited alleles changes with patch size.

changes as we vary model assumptions can effectively guide our intuition about z^*.

We now shift focus and consider the point of view of a maternally or paternally inherited allele in a breeding female. In this case, we are therefore considering the effect of which grandparent the allele in a parent comes from (fig. 1). Clearly, the LRC inclusive fitness penalty (as measured by the appropriate analogue of $\overline{R}$) will be judged to be less severe by the paternally inherited allele (from the grandfather of fig. 1). Imprinting acts as a cue that "reminds" paternally inherited alleles that they are relative newcomers to the patch and much less likely than their maternally inherited counterparts (from the grandmother of fig. 1) to randomly encounter genetically identical copies of themselves during competition.

Using information presented in appendixes A–E, it can be shown that the population is at evolutionary equilibrium from the perspective of the paternally inherited allele when

$$z^* = \frac{2N^2}{4N^2 - 3N + 1}, \tag{2}$$

and at evolutionary equilibrium from the perspective of the maternally inherited allele when

$$z^* = \frac{2N^2(3N + 1)}{(4N^2 - N + 1)(3N - 1)}. \tag{3}$$

For simplicity, we refer to equations (2) and (3) as the sex ratios "preferred" or "favored" by the paternally and maternally inherited alleles, respectively.

Equations (2) and (3) show that both alleles favor male-biased sex ratios ($z^* > 1/2$; fig. 2). However, in keeping with our intuition, maternally inherited alleles prefer sex ratios that are more strongly male biased than those favored by paternally inherited alleles—although the difference in the preferred sex ratios from the perspective of

paternally and maternally inherited alleles is relatively minor (fig. 2). The "no imprinting" result reported in equation (1) lies between the allele-specific preferences (in fact, eq. [1] is the harmonic mean of eqq. [2], [3]).

Polyandry. The size of the LRC inclusive fitness penalty (i.e., the size of the relevant $\overline{R}$) can also be adjusted by changing the assumptions one makes about the mating system (Pen 2006). We assumed that each female mates with exactly one male. Relaxing this assumption and allowing females to mate with exactly M males (polyandry) increases the genetic diversity of a brood and decreases $\overline{R}$. Polyandry, then, promotes the evolution of more even sex ratios in the absence of imprinting (table 1).

As was the case with female monogamy, both maternally and paternally inherited genes favor male-biased sex ratio, but the bias preferred by the maternally inherited gene (from the grandmother of fig. 1) is stronger than that preferred by the paternally inherited one (from the grandfather of fig. 1). A plot of the absolute difference between sex ratios preferred by paternal alleles and those preferred by maternal alleles (a variable we call "Abs Diff") shows that polyandry exacerbates the conflict between paternally and maternally inherited alleles (fig. 2). Multiple matings reduce the likelihood that sisters (i.e., female parents of fig. 1 who compete for the same patch) share the same father (i.e., the same grandfather of fig. 1). This leads to a reduction in the relatedness between paternally inherited alleles of broodmates and hence a greater difference in relatedness between the maternally and the paternally inherited alleles.

Polygyny. If we now assume that one male monopolizes all matings on the patch (polygyny), then $\overline{R}$ increases (a consequence of the fact that all female patchmates in this scenario are at least half-sibs). A larger $\overline{R}$ means larger LRC inclusive fitness penalties. Larger penalties are reflected in equilibrium sex ratios that are more strongly male biased than those found with other model mating systems (fig. 2).

Interestingly, we find that in a polygynous mating system, it is the paternally inherited allele that prefers the more strongly biased sex ratio. Although this result provides a stark contrast to the results obtained under assumptions of monogamy and polyandry, it is hardly surprising. All female patchmates have the same father but may not have the same mother. As a result, females are more closely related to one another through patrilines than through matrilines. A high degree of relatedness through patrilines means that—relative to maternally inherited alleles—paternally inherited alleles incur larger penalties for producing daughters. To avoid the more substantial penalties, then, paternally inherited alleles favor greater investment in sons.

Diploidy with Offspring Control of the Sex Ratio

Under maternal control of the sex ratio, inclusive fitness penalties are balanced against fitness gains made by the actor's own offspring. When we give control of the sex ratio to offspring (in this case, an offspring determines its own sex), penalties become balanced against fitness gains made by the actor itself. Because actors, in this case, are always more closely related to themselves than they are to the offspring produced by their parent, the scales tip more heavily away from sex ratio bias. In short, all else being equal, handing control of the sex ratio to offspring acts as an "effective" reduction in $\overline{R}$. In addition to an effective reduction, we see in appendix D that there is an "actual" reduction in $\overline{R}$ brought about by the arrival of foreign paternal genes.

Given that $\overline{R}$ is both "actually" and "effectively" smaller, we expect equilibrium sex ratios under offspring control to be less biased than those found with maternal control. Indeed, this is what we observe (fig. 2). Note that a shift toward less biased strategies is the only substantive difference between maternal and offspring control. The qualitative effects of changing mating system remain unchanged (fig. 2). However, in this case, the conflict between paternally and maternally inherited alleles is now played out in the offspring between alleles that came from the mother and father, as opposed to in the parent between alleles that came from the grandfather and grandmother (fig. 1).

Haplodiploidy

The main qualitative differences between haploid and diploid models occur in cases with offspring control of the sex ratio (in this case, "offspring" are assumed to be sterile female workers). First, equilibrium sex ratios are not always male biased. If there is no imprinting, then offspring-controlled equilibrium sex ratios tend toward the well-known value $z^* = 1/4$ (i.e., the 3 : 1 female : male ratio predicted by Trivers and Hare 1976) as patch size N becomes large. Of course, in haplodiploids, paternally inherited alleles in worker-offspring have no genetic interest in workers' brothers, and so these alleles prefer extreme female bias (fig. 3). The "extreme" point of view of paternally inherited alleles also means that the conflict with maternally inherited homologues can be substantial (fig. 3, *insets*). In addition, we find that under offspring control with polygyny, the paternally inherited allele has no preferred sex ratio strategy. From the point of view of the paternally inherited allele in this case, LRC exactly cancels any fitness gained through the production of daughters.

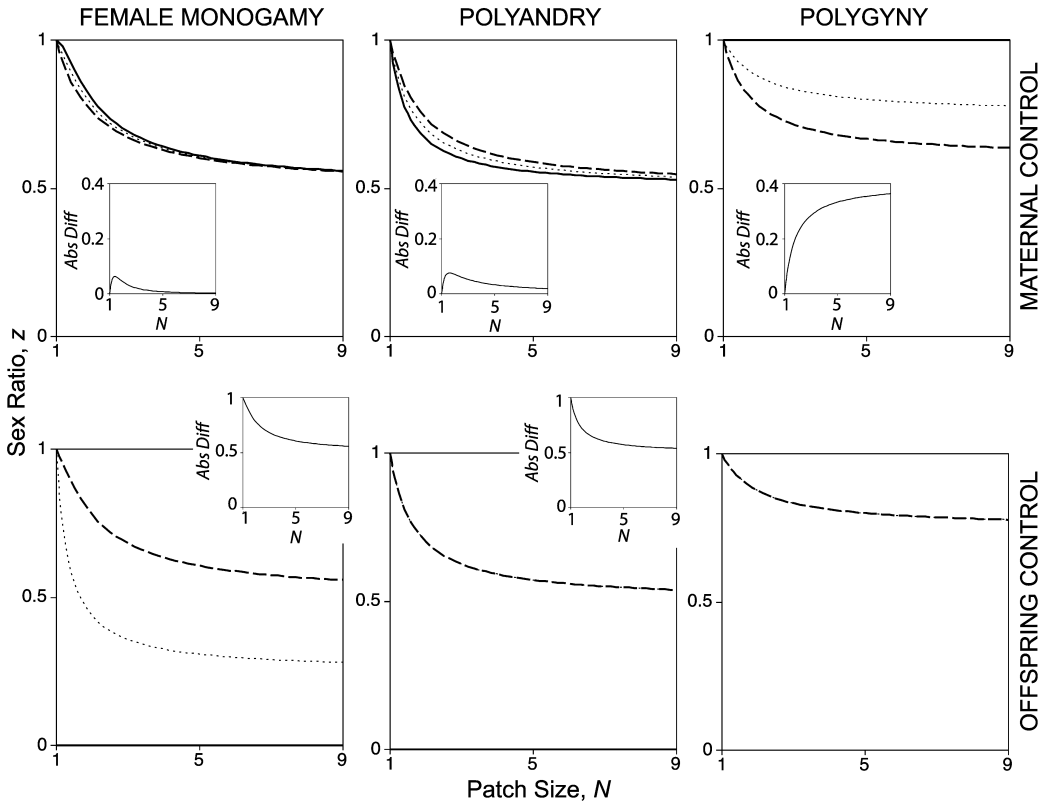

Figure 3: Graphical depiction of the relationship between the stable sex ratio and patch size for haplodiploid local resource competition models. We show results for both maternal and offspring control and for each of the three different mating systems (in the polyandry case, the large M approximation is given). Solid curves refer to sex ratios "preferred" by paternally inherited alleles (i.e., grandpaternal alleles in mother, or paternal alleles in offspring), dashed curves refer to sex ratios "preferred" by maternally inherited alleles (i.e., grandmaternal alleles in mother or maternal alleles in offspring), and dotted curves refer to standard "no imprinting" results. Where appropriate, insets depict how the absolute difference (*Abs Diff*) between sex ratios favored by paternally inherited and maternally inherited alleles changes with patch size. Note that the dotted and dashed curves of the bottom middle panel coincide and that there is only a dashed curve in the bottom right panel.

The paternally inherited allele is equally indifferent to workers' brothers because these brothers are not, from the perspective of the allele, relatives.

Local Mate Competition

We report results for a variety of different LMC models (app. B). To help the reader, we summarize all results in table 2.

Diploidy with Maternal Control of the Sex Ratio

We first consider the classic LMC model constructed by Hamilton (1967): (1) N singly mated females lay eggs on a patch. (2) The offspring develop and mate. Males die. (3) Following mating, fertilized daughters disperse.

Under these conditions, Hamilton showed that the equilibrium sex ratio from the perspective of a mother (i.e., under maternal control) is given by $z^* = (N - 1)/2N < 1/2$, a result that holds for all mating systems we consider here (table 2). Taylor (1981) emphasized that the female bias in Hamilton's z^* occurs because there are both inclusive fitness penalties for production of sons (sons compete for mates against related males) and inclusive fitness bonuses for producing daughters (daughters provide new mating opportunities for related males).

In diploids, LMC does not lead to a conflict that favors genomic imprinting over the sex ratio, simply because male and female genes disperse together (unlike LRC) and are transmitted to offspring in a symmetric manner (unlike haplodiploidy). Therefore, grandpaternal and grandmaternal genes are equally likely to be in grandoffspring of either sex, and so the genes suffer inclusive fitness penalties or enjoy inclusive fitness bonuses to the same extent.

Changing the mating system has no effect on our basic conclusion: in diploid LMC models with polygynous or

Table 2: Summary of the equilibrium sex ratios for local mate competition models

Model, control, and mating system	Sex ratio		
	No imprinting	Paternally inherited	Maternally inherited
Diploidy:			
Maternal:			
All mating systems	$\frac{N-1}{2N}$	$\frac{N-1}{2N}$	$\frac{N-1}{2N}$
Offspring:			
Monogamy/polygyny	$\frac{N-1}{2N-1}$	$\frac{N-1}{2N-1}$	$\frac{N-1}{2N-1}$
Polyandry	$\frac{2M(N-1)}{(4N-3)M+1}$	$\frac{(N-1)[4MN+(M-1)]_a}{2N[(4N-3)M+1]}$	$\frac{(N-1)[4MN-(M-1)]}{2N[(4N-3)M+1]}$
Haplodiploidy:			
Maternal:			
All mating systems	$\frac{(2N-1)(N-1)}{N(4N-1)}$	$\frac{(2N-1)(N-1)_a}{2N(2N-1)+1}$	$\frac{N-1}{2N+1}$
Offspring:			
Monogamy/polygyny	$\frac{N-1}{4N-1}$	$\frac{N-1}{N(4N-1)}$	$\frac{(2N-1)(N-1)_a}{N(4N-1)}$
Polyandry	$\frac{2MN(N-1)}{(4N-1)[N(M+1)+M-1]}$	$\frac{M(N-1)}{(4N-1)(N+M-1)}$	$\frac{(2N-1)(N-1)_a}{N(4N-1)}$

Note: Recall that N refers to the number of females breeding on a patch, and M refers to the number of mates chosen by each female in polyandrous systems.

[a] The more moderate (i.e., less biased) allele-specific perspective.

polyandrous mating systems, imprinting continues to have no consequences for equilibrium sex ratios when these are under maternal control. As we will see, however, imprinting does introduce some complications when offspring have control.

Diploidy with Offspring Control of the Sex Ratio

Female Monogamy or Polygyny. In monogamous or polygynous mating systems, when control of the sex ratio is given to offspring, we calculate the equilibrium value to be $z^* = (N - 1)/(2N - 1)$. Just like LRC, the LMC equilibrium sex ratios become less biased when control is taken away from parents and given to offspring (Werren and Hatcher 2000; Beukeboom et al. 2001). Furthermore, because both maternally and paternally inherited alleles (1) have arrived together on a foreign patch and (2) are equally likely to be transmitted to offspring of either sex, there is no conflict between parental genes in offspring. Consequently, imprinting has no effect on z^*.

Polyandry. When we assume that each female mates with exactly M males, bias in the offspring-controlled equilibrium sex ratio changes. Specifically, in the absence of bias, z^* is reduced as M increases (table 2).

Because males and females now have unequal genetic shares in broods, we expect imprinting to have an effect on equilibrium sex ratios. Indeed, this is what we observe (table 2). While both paternally and maternally inherited alleles favor a female-biased sex ratio, that favored by the maternally inherited allele (the allele that stands to lose more through inclusive fitness penalties and that stands to gain more from inclusive fitness bonuses) prefers the stronger bias (fig. 4*a*). However, this difference is not substantial.

Haplodiploidy with Maternal Control of the Sex Ratio

With LMC and maternal control of the sex ratio, the equilibrium trait values are the same for all mating systems considered (table 2). If we allow imprinting, we find that paternally and maternally inherited genes come into conflict: paternally inherited genes favor a more even sex ratio (table 2; fig. 4*b*). The effect of imprinting here is counterintuitive at first glance. Both alleles in the mother have an equal chance of being transmitted to sons and daughters, and both alleles find themselves on a nonnative patch—so why do they have different perspectives?

To answer this question, we must consider what happens over three generations, beginning with the parents of the focal actor (i.e., the grandparents of fig. 1). Fixing attention on a single grandmaternal allele, it becomes clear that there are two ways that this allele can possibly find its way into granddaughters: (1) through daughters or (2) through sons. The same cannot be said for the grandpaternal allele. The only way that the grandpaternal allele finds its way into a granddaughter is through daughters. Overall, the "echo" of the asymmetric modes of transmission in hap-

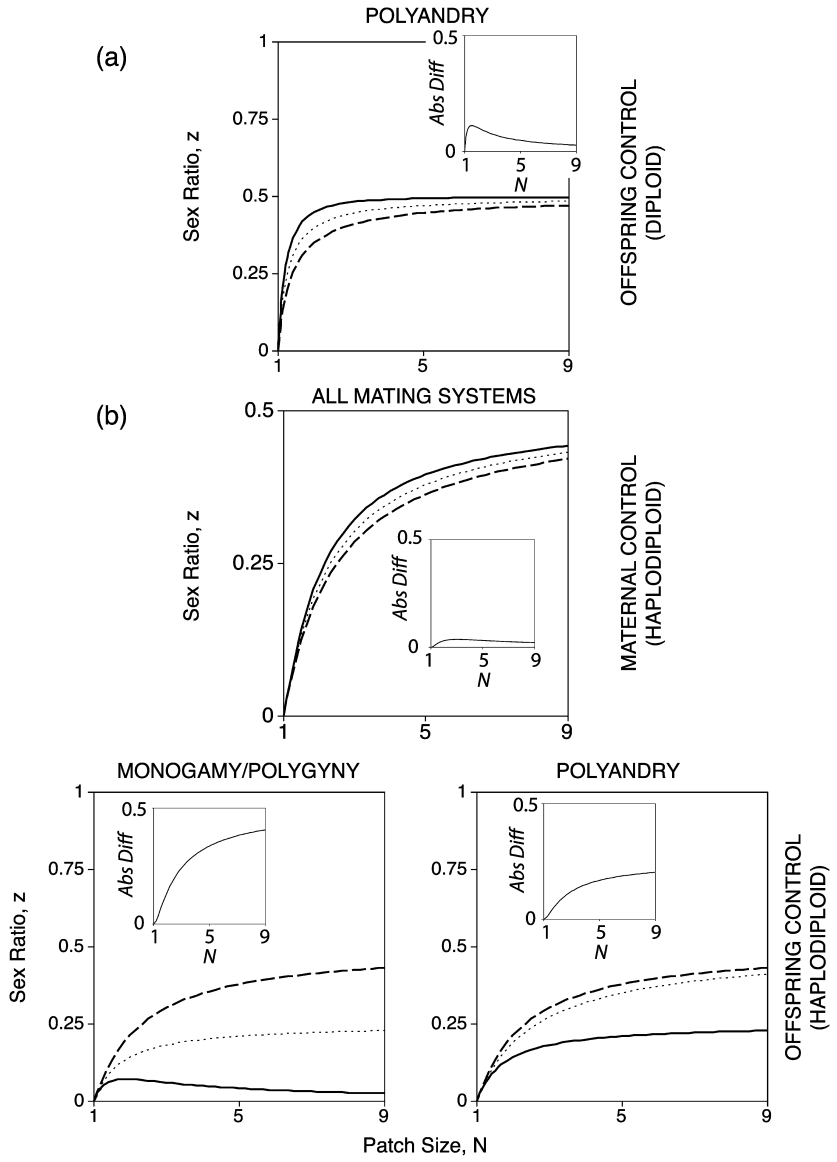

Figure 4: Graphical depiction of the relationship between the stable sex ratio and patch size for diploid local mate competition models. *a*, We show results for diploid offspring control with polyandry (large *M* approximation) only because conflict does not occur with diploid maternal control or other diploid offspring control models. *b*, We show the results for haplodiploid models (again, large *M* approximation is given polyandry models). In both *a* and *b*, solid curves refer to sex ratios "preferred" by paternally inherited alleles (i.e., grandpaternal alleles in mother or paternal alleles in offspring), dashed curves refer to sex ratios "preferred" by maternally inherited alleles (i.e., grandmaternal alleles in mother or maternal alleles in offspring), and dotted curves refer to standard "no imprinting" results. Insets depict how the absolute difference (*Abs Diff*) between sex ratios favored by paternally inherited and maternally inherited alleles changes with patch size.

lodiploids means that, in LMC models, the maternally inherited allele is more closely related to daughters than is the paternally inherited allele. The fact that the maternally inherited allele places greater genetic value on daughters is reflected in its preference for a stronger female bias.

Haplodiploidy with Offspring Control of the Sex Ratio

Female Monogamy or Polygyny. Now we give control of the sex ratio to female offspring (e.g., members of a worker caste). In the absence of imprinting, we find that, relative

to maternal control, there is a stronger female bias to the equilibrium sex ratio under offspring control, provided that $N > 1$ (table 2). The stronger bias is due to the fact that the paternally inherited allele in female offspring has less genetic interest than the maternally inherited alleles in that offspring's brothers. In fact, when we allow imprinting, we find that the sex ratio preferred by the paternally inherited allele is more strongly biased than that favored by the maternally inherited allele (table 2; fig. 4*b*). Note that previous models (Haig 1992; Queller 2003) consider one queen, singly mated (Queller's table 3), and correspond to the case in which there is no LMC (i.e., $N \to \infty$). In these models, the paternally inherited allele favors $z^* = 0$, and the maternally inherited allele favors $z^* = 0.5$ (fig. 4*b*). Note also that the sex ratio conflict that exists between the parental alleles also increases with increasing patch size (fig. 4*b*, *inset*).

Polyandry. The relatedness between a worker female and her reproductive sister decreases with increasing *M*. However, because males arise from unfertilized eggs, changing *M* does not change the relatedness between a worker female and her brother. Intuitively, there is a reduced inclusive fitness incentive in a polyandrous system for workers to invest in sisters. We therefore expect less biased sex ratios at equilibrium with increasing *M*, an expectation that is supported by analytical results for the "no imprinting" case (table 2; fig. 4*b*).

When imprinting is allowed, we find that, from the perspective of the maternally inherited allele, polyandry has changed nothing (cf. final two entries in the rightmost column of table 2). As suggested, though, polyandry means that paternally inherited alleles have less interest in daughters. We still see that the sex ratio preferred by the paternally inherited allele is the one that is more strongly biased toward daughters (fig. 4*b*).

Local Resource Enhancement

Sometimes the production of one sex increases the overall productivity of a breeding pair (LRE), for example, in a range of cooperatively breeding vertebrates and bees (Griffin et al. 2005; West et al. 2005). In species that exhibit sex-specific helping behavior, sex ratios are often predicted to favor the more helpful sex (Emlen et al. 1986; Pen and Weissing 2002). However, in diploids, LRE alone will not promote conflict over the sex ratio, unless we build in additional assumptions. There is a wide variety of additional assumptions available and an equally wide variety of ways in which they can be added to LRE models (e.g., Pen and Weissing 2002; Wild 2006). Our goal then is to stimulate future work with a brief introduction of how intragenomic conflict might arise when LRE models incorporate assumptions of the LRC models.

We modify the basic LRC model by adding the assumption that the brood size of a given female, *K*, increases with the number of daughters produced on its natal patch. To be clear, we posit a maximum brood size that decreases at a rate proportional to the patch-average sex ratio, *y*. Mathematically, $dK/dy = -\alpha K$, where $\alpha > 0$ describes the strength of the LRE effect (larger α indicates stronger effect of LRE). In this very simple model, we find that LRC and LRE exert opposing pressure on the sex ratio (app. C). As we outlined, LRC favors male-biased sex ratios, and LRE favors (in this case) the "more helpful" sex, females. Although an analytical expression for the equilibrium sex ratio is not available, we do find that there is a threshold value of $\alpha = 4$ below which equilibrium sex ratios are male biased (LRC dominates). Of course, above the threshold value, the reverse is true (LRE dominates).

When we allow imprinting, we find that (1) both maternally and paternally inherited alleles agree on the direction of sex ratio bias (again the direction of bias depends on α) and (2) the paternally inherited allele never favors a sex ratio bias that is more extreme than that favored by the maternally inherited allele. Of course, these results depend on the assumption that the nondispersing sex (females) is also the more helpful sex. In many species with juvenile helpers, the nondispersive sex is indeed more helpful (Griffin et al. 2005). When, instead, the dispersive sex is more helpful, biased investment in this sex is promoted by both LRE and LRC. In these cases, we expect the conflict between maternally inherited alleles found in models with LRC alone to be maintained.

Discussion

Theoretical Predictions

We have shown that in a variety of sex allocation scenarios where relatives can interact, the favored sex allocation can differ between paternally and maternally inherited alleles. This conflict, which could select for genomic imprinting, can occur either when parents control the sex of their offspring (though we have considered only mothers) or when offspring control sex allocation. Specifically, genomic imprinting can be selected for when (1) there are competitive interactions between relatives (LRC or LMC; figs. 2–4; tables 1, 2), (2) there are cooperative interactions between relatives (LRE), or (3) sex allocation is determined by offspring (siblings) in haplodiploid species (as occurs in the social hymenoptera and polyembryonic wasps with soldier castes; Haig 1992; Queller 2003).

Intuitively, the extent of the conflict between allele-specific perspectives reflects the strength of selection for

imprinting. To be precise, increasing the distance between the population-average sex ratio (i) and the equilibrium value (z^*) favored by a particular allele will increase the selective pressure on that allele to regulate its activity when information about the sex of the parent from which it originated is available. Having said this, we predict that the greatest selection for genomic imprinting will occur when sex allocation is controlled by offspring in haplodiploid species (case 3). If we consider cases involving diploid species with parental or offspring control of sex allocation and haplodiploids with parental control of sex allocation (cases 1 and 2), LRC and LRE can lead to noteworthy conflict between maternally and paternally inherited genes (figs. 2, 3), but there seems to be relatively minimal scope for such conflict under LMC (fig. 4). In contrast, in haplodiploids with offspring control (case 3), there can be considerable conflict with both LRC and LMC. In these cases, LRC increases the extent of conflict, and LMC decreases the extent of conflict (cf. insets on bottom panels of figs. 3, 4*b*).

Genomic imprinting at a sex allocation locus could also be selected for in the absence of kin interactions such as LRC, LMC, or LRE. If females mate with different males in different years and the survival cost (to the female) of producing a son or daughter differ, then females will be selected to produce a lower proportion of the costly sex than their mates (Trivers 1974; Charnov 1982; Leigh et al. 1985; Pen and Weissing 2002). We expect that this conflict will promote the evolution of genomic imprinting when offspring have control of parental sex allocation decisions. However, the importance of such conflicts is not clear. In particular, in species where sex differential survival effects may occur, such as vertebrates, sex allocation appears to be controlled by parents. In addition, in species where interactions between relatives are unimportant, the primary reason for conditional adjustment of sex allocation is when the environment has different effects on the fitness of male and female offspring (Trivers and Willard 1973), which would tend to align the strategies favored by males and females and hence reduce selection for genomic imprinting. Explicit theory is needed to examine these issues.

Application to the Real World

One way to test our predictions would be to look for genomic imprinting at the level of the genes involved in sex allocation. This could be done with either molecular or quantitative genetic approaches (Burt and Trivers 2006). As discussed in "Introduction," an advantage of this study is that the data to test it do not yet exist. We are therefore in a position to make a priori predictions over what situations and species are most likely to involve imprinting on genes that influence sex allocation (see also Queller and Strassman 2002; Queller 2003). The greatest candidates are clearly haplodiploid species in which offspring can control sex allocation. The most obvious group here is the social hymenoptera (ants, bees, and wasps), where workers have been shown to manipulate sex allocation (of their reproductive siblings) in the nest, through mechanisms such as male killing or biasing the final caste (queen or worker) of developing females (Sundstrom et al. 1996; Hammond et al. 2002). In this case, we would expect imprinting on genes that influenced these behaviors, such as paternally inherited alleles leading to higher levels of male killing or a higher queen : worker caste ratio (Queller 2003). Similar conflicts could potentially occur in the haplodiploid eusocial thrips (Kranz et al. 1999).

Within the social hymenoptera, our models suggest that selection for genomic imprinting would be increased in species where there is LRC (fig. 3; table 1) and reduced in species where there is LMC (fig. 4). LRC can occur as a result of colony fission, colony budding, or when nests recruit their own daughters as reproductive queens (Trivers and Hare 1976; Bourke and Franks 1995; West et al. 2005). Another complication in the social hymenoptera that we have not considered is that there is often selection for split sex ratios, where some colonies produce predominantly male sexuals and others produce predominantly female sexuals. This can be selected for in response to variation in relatedness asymmetry, arising from factors such as variation in mating frequency and queen number (Boomsma and Grafen 1990, 1991; Boomsma 1991) or variation in the extent of LRC (Ward 1983; Brown and Keller 2000; Kummerli et al. 2005). Further theory is required to determine the consequences of split sex ratios for selection on genomic imprinting.

The other haplodiploid taxa where offspring can have a large influence on sex allocation are the polyembryonic wasps (Strand and Grbic 1997). In these species, the offspring have control over sex allocation, both because they control the proliferation of embryos and because, in some species, some larvae develop as sterile soldier larvae. These sterile soldiers are predominantly females, who preferentially kill the males (brothers; Grbic et al. 1992; Giron et al. 2004). The production of the these soldiers appears to result from sex ratio conflict under LMC because the females would prefer a more female-biased sex ratio than would the males (Gardner et al. 2007). Consequently, genomic imprinting could be selected for in genes that influence the relative proliferation rate of the two sexes or the development and behavior of the sterile soldier caste.

In species where sex allocation is controlled by parents, selection for genomic imprinting is weaker but can still occur with LRC between females and LRE (figs. 2, 3; table 1). LRC and LRE have been suggested to occur in a range of organisms where parents appear to control offspring

sex ratios, including cooperative breeding birds and mammals (Komdeur et al. 1997; Griffin et al. 2005), cooperative breeding bees (Schwarz 1988; Stark 1992; Martins et al. 1999), rodents (Lambin 1994), lions (Packer and Pusey 1987), mealybugs (Varndell and Godfray 1996), primates (Clark 1978), marsupials (Cockburn 1990; Johnson et al. 2001), ungulates (Clutton-Brock et al. 1982), aphids (Dagg and Vidal 2004), thrips (Kranz et al. 1999), and plants (de Jong et al. 2002). Even when there is relatively little sex allocation conflict, this could lead to substantial differences in gene activity, leaving evolutionary footprints in sex-determining mechanisms (Pen 2006; Uller et al. 2007). A general issue here is that the ability to test our models will depend on the taxonomic distribution of genomic imprinting, about which relatively little is known (Burt and Trivers 2006). Particularly exciting from this perspective are recent molecular and genetic advances suggesting the possibility for genomic imprinting in both the social (honeybee; Wang et al. 2006) and the parasitic hymenoptera (*Nasonia vitripennis*; Beukeboom and van den Assem 2001, 2002; Beukeboom et al. 2007).

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Literature Cited

Badcock, C., and B. J. Crespi. 2006. Imbalanced genomic imprinting in brain development: an evolutionary basis for the etiology of autism. Journal of Evolutionary Biology 19:1007–1032.

Beukeboom, L. W., and J. van den Assem. 2001. Courtship and mating behavior of interspecific *Nasonia* hybrids (Hymenoptera, Pteromalidae): a grandfather effect. Behavior Genetics 31:167–177.

———. 2002. Courtship displays of introgressed interspecific hybrid *Nasonia* males: further investigations into the "grandfather effect." Behaviour 139:1029–1042.

Beukeboom, L. W., T. J. de Jong, and I. Pen. 2001. Why girls want to be boys. Bioessays 23:477–480.

Beukeboom, L. W., A. Kamping, and L. van de Zande. 2007. Sex determination in the haplodiploid wasp *Nasonia vitripennis* (Hymenoptera: Chalcidoidea): a critical consideration of models and evidence. Seminars in Cell and Developmental Biology 18:371–378.

Boomsma, J. J. 1991. Adaptive colony sex ratios in primitively eusocial bees. Trends in Ecology & Evolution 6:92–95.

Boomsma, J. J., and A. Grafen. 1990. Intraspecific variation in ant sex ratios and the Trivers-Hare hypothesis. Evolution 44:1026–1034.

———. 1991. Colony-level sex ratio selection in the eusocial Hymenoptera. Journal of Evolutionary Biology 4:383–407.

Bourke, A. F. G., and N. R. Franks. 1995. Social evolution in ants. Princeton University Press, Princeton, NJ.

Brown, W. D., and L. Keller. 2000. Colony sex ratios vary with queen number but not relatedness asymmetry in the ant *Formica exsecta*. Proceedings of the Royal Society B: Biological Sciences 267:1751–1757.

Burt, A., and R. L. Trivers. 2006. Genes in conflict: the biology of selfish genetic elements. Harvard University Press, Cambridge, MA.

Chapuisat, M., and L. Keller. 1999. Testing kin selection with sex allocation data in eusocial Hymenoptera. Heredity 82:473–478.

Charnov, E. L. 1982. The theory of sex allocation. Princeton University Press, Princeton, NJ.

Clark, A. B. 1978. Sex ratio and local resource competition in a prosimian primate. Science 201:163–165.

Clutton-Brock, T. H., S. D. Albon, and F. E. Guiness. 1982. Competition between female relatives in a matrilocal mammal. Nature 300:178–180.

Cockburn, A. 1990. Sex ratio variation in marsupials. Australian Journal of Zoology 37:467–479.

Dagg, J. L., and S. Vidal. 2004. Sex ratio adjustment and maternal condition in two aphid species. Behavioral Ecology and Sociobiology 55:231–235.

de Jong, T. J., F. H. D. E. van Batenburg, and J. V. van Dijk. 2002. Seed sex ratio in dioecious plants depends upon relative dispersal of pollen and seeds: an example using a chessboard simulation model. Journal of Evolutionary Biology 15:373–379.

Emlen, S. T., J. M. Emlen, and S. A. Levin. 1986. Sex-ratio selection in species with helpers-at-the-nest. American Naturalist 127:1–8.

Field, L. M., F. Lyko, M. Madrioli, and G. Prantera. 2004. DNA methylation in insects. Insect Molecular Biology 13:109–115.

Gardner, A., I. C. W. Hardy, P. D. Taylor, and S. A. West. 2007. Spiteful soldiers and sex ratio conflict in polyembryonic parasitoid wasps. American Naturalist 169:519–533.

Giron, D., D. W. Dunn, I. C. W. Hardy, and M. R. Strand. 2004. Aggression by polyembryonic wasp soldiers correlates with kinship but not resource competition. Nature 430:676–679.

Grafen, A. 1985. Hamilton's rule OK. Nature 318:310–311.

Grbic, M., P. J. Ode, and M. R. Strand. 1992. Sibling rivalry and brood sex-ratios in polyembryonic wasps. Nature 360:254–256.

Greenwood-Lee, J., P. D. Taylor, and D. Haig. 2001. The inclusive fitness dynamics of genomic imprinting. Selection 2:101–116.

Griffin, A. S., B. C. Sheldon, and S. A. West. 2005. Cooperative breeders adjust offspring sex ratios to produce helpful helpers. American Naturalist 166:628–632.

Haig, D. 1992. Intragenomic conflict and the evolution of eusociality. Journal of Theoretical Biology 156:401–403.

———. 1997. The social gene. Pages 284–304 *in* J. R. Krebs and N. B. Davies, eds. Behavioural ecology: an evolutionary approach. Blackwell, London.

———. 2000. The kinship theory of genomic imprinting. Annual Review of Ecology and Systematics 31:9–32.

———. 2002. Genomic imprinting and kinship. Rutgers University Press, New Brunswick, NJ.

———. 2004. Genomic imprinting and kinship: how good is the evidence? Annual Review of Genetics 38:553–585.

Hamilton, W. D. 1964. The genetical evolution of social behavior. I, II. Journal of Theoretical Biology 7:1–52.

———. 1967. Extraordinary sex ratios. Science 156:477–488.

———. 1996. Narrow roads of gene land. I. Evolution of social behaviour. W. H. Freeman, Oxford.

Hammond, R. L., M. W. Bruford, and A. F. G. Bourke. 2002. Ant workers selfishly bias sex ratios by manipulating female development. Proceedings of the Royal Society B: Biological Sciences 269:173–178.

Hurst, L. D., and G. T. McVean. 1998. Do we understand the evolution of genomic imprinting? Current Opinion in Genetics and Development 8:701–708.

Johnson, C. N., M. Clinchy, A. C. Taylor, C. J. Krebs, P. J. Jarman, A. Payne, and E. G. Ritchie. 2001. Adjustment of offspring sex ratios in relation to availability of resources for philopatric offspring in the common brushtail possum. Proceedings of the Royal Society B: Biological Sciences 268:2001–2006.

Komdeur, J., S. Daan, J. Tinbergen, and C. Mateman. 1997. Extreme modification of sex ratio of the Seychelles warbler's eggs. Nature 385:522–525.

Kranz, B. D., M. P. Schwarz, L. A. Mound, L. A., and B. J. Crespi. 1999. Social biology and sex ratios of the eusocial gall inducing thrips *Kladothrips hamiltoni*. Ecological Entomology 24:432–442.

Kummerli, R., K. R. Helms, and L. Keller. 2005. Experimental manipulation of queen number affects colony sex ratio investment in the highly polygynous ant *Formica exsecta*. Proceedings of the Royal Society B: Biological Sciences 272:1789–1794.

Lambin, X. 1994. Sex ratio variation in relation to female philopatry in Townsend's voles. Journal of Animal Ecology 63:945–953.

Lehmann, L. 2007. The evolution of trans-generational altruism: kin selection meets niche construction. Journal of Evolutionary Biology 20:181–189.

Leigh, E. G., E. A. Herre, and E. A. Fischer. 1985. Sex allocation in animals. Experientia 41:1265–1276.

Martins, R. P., Y. Antoni, F. A. da Silveira, and S. A. West. 1999. Seasonal variation in the sex allocation of a Neotropical solitary bee. Behavioral Ecology 10:401–408.

Maynard Smith, J., 1982. Evolution and the theory of games. Cambridge University Press, Cambridge.

Mehdiabadi, N. J., H. K. Reeve, and U. G. Mueller. 2003. Queens versus workers: sex-ratio conflict in eusocial Hymenoptera. Trends in Ecology & Evolution 18:88–93.

Meunier, J., S. A. West, and M. Chapuisat. 2008. Split sex ratios in the social Hymenoptera: a meta-analysis. Behavioral Ecology 19: 382–390.

Michod, R. E., and W. D. Hamilton. 1980. Coefficients of relatedness in sociobiology. Nature 288:694–697.

Normark, B. B. 2003. The evolution of alternative genetic systems in insects. Annual Review of Entomology 48:397–423.

———. 2006. Maternal kin groups and the origins of asymmetric genetic systems: genomic imprinting, haplodiploidy, and parthenogenesis. Evolution 60:631–642.

Packer, C., and A. E. Pusey. 1987. Intrasexual competition and the sex ratio in African lions. American Naturalist 130:636–642.

Pen, I. 2006. When boys want to be girls: effects of mating system and dispersal on parent-offspring sex ratio conflict. Evolutionary Ecology Research 8:103–113.

Pen, I., and F. J. Weissing. 2002. Optimal sex allocation: steps toward a mechanistic theory. Pages 266–286 *in* I. C. W. Hardy, ed. Sex ratios: concepts and research methods. Cambridge University Press, Cambridge.

Price, G. R. 1970. Selection and covariance. Nature 227:520–521.

Queller, D. C. 2003. Theory of genomic imprinting conflict in social insects. BMC Evolutionary Biology 3:15.

Queller, D. C., and J. E. Strassman. 2002. The many selves of social insects. Science 296:311–313.

Rousset, F. 2004. Genetic structure and selection in subdivided populations. Princeton University Press, Princeton, NJ.

Schwarz, M. P. 1988. Local resource enhancement and sex ratios in a primitively social bee. Nature 331:346–348.

Shaw, R. F., and J. D. Mohler. 1953. The selective advantage of the sex ratio. American Naturalist 87:337–342.

Stark, R. E. 1992. Sex ratio and maternal investment in the multivoltine large carpenter bee *Xylocopa sulcatipes*. Ecological Entomology 17:160–166.

Strand, M. R., and M. Grbic. 1997. The development and evolution of polyembryonic insects. Current Topics in Developmental Biology 35:121–159.

Sundstrom, L., M. Chapuisat, and L. Keller. 1996. Conditional manipulation of sex ratios by ant workers: a test of kin selection theory. Science 274:993–995.

Sykes, E. M., T. M. Innocent, I. Pen, D. M. Shuker, and S. A. West. 2007. Asymmetric larval competition in the parasitoid wasp *Nasonia vitripennis*: a role in sex allocation. Behavioral Ecology 61: 1751–1758.

Taylor, P. D. 1981. Intra-sex and inter-sex sib interactions as sex ratio determinants. Nature 291:64–66.

———. 1988. An inclusive fitness model for dispersal of offspring. Journal of Theoretical Biology 130:363–378.

———. 1989. Evolutionary stability in one-parameter models under weak selection. Theoretical Population Biology 36:125–143.

———. 1994. Sex ratio in a stepping-stone population with sex-specific dispersal. Theoretical Population Biology 45:203–218.

Taylor, P. D., and S. A. Frank. 1996. How to make a kin selection model. Journal of Theoretical Biology 180:27–37.

Trivers, R. L. 1974. Parent-offspring conflict. American Zoologist 14: 249–264.

Trivers, R. L., and H. Hare. 1976. Haplodiploidy and the evolution of the social insects. Science 191:249–263.

Trivers, R. L., and D. E. Willard. 1973. Natural selection of parental ability to vary the sex ratio. Science 179:90–92.

Uller, T., I. Pen, E. Wapstra, L. W. Beukeboom, and J. Komdeur. 2007. The evolution of sex ratios and sex-determining systems. Trends in Ecology & Evolution 22:292–297.

Varndell, N. P., and H. C. J. Godfray. 1996. Facultative adjustment of the sex ratio in an insect (*Planococcus citri*, Pseudococcidae) with paternal genome loss. Evolution 50:2100–2105.

Wang, Y., M. Jorda, P. L. Jones, R. Maleszka, X. Ling, H. M. Robertson, C. A. Mizzen, M. A. Peinado, and G. E. Robinson. 2006. Functional CpG methylation system in a social insect. Science 314: 645–647.

Ward, P. S. 1983. Genetic relatedness and colony organization in a species complex of ponerine ants. Behavioural Ecology and Sociobiology 12:301–307.

Werren, J. H., and L. W. Beukeboom. 1998. Sex determination, sex ratios and genetic conflict. Annual Review of Ecology and Systematics 29:233–261.

Werren, J. H., and M. J. Hatcher. 2000. Maternal-zygotic conflict

over sex determination: effects of inbreeding. Genetics 155:1469–1479.

Werren, J. H., M. J. Hatcher, and H. C. J. Godfray. 2002. Maternal-offspring conflict leads to the evolution of dominant zygotic sex determination. Heredity 88:102–111.

West, S. A., D. M. Shuker, and B. C. Sheldon. 2005. Sex ratio adjustment when relatives interact: a test of constraints on adaptation. Evolution 59:1211–1228.

Wild, G. 2006. Sex ratios when helpers stay at the nest. Evolution 60:2012–2022.

Wild, G., and P. D. Taylor. 2004. Kin selection models for the co-evolution of the sex ratio and sex-specific dispersal. Evolutionary Ecology Research 6:481–502.

———. 2005. A kin-selection approach to the resolution of a sex-ratio conflict between mates. Journal of Theoretical Biology 236: 126–136, doi:10.1016/j.jtbi.2005.03.003.

Wilkins, J. F., and D. Haig. 2003. What good is genomic imprinting: the function of parent specific gene expression. Nature Reviews Genetics 4:1–10.

Wilson, M. F., and N. Burley. 1983. Mate choice in plants. Princeton University Press, Princeton, NJ.

Wood, A. J., and R. J. Oakley. 2006. Genomic imprinting in mammals: emerging themes and established theories. PLoS Genetics 11:1677–1685.

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Appendix A from G. Wild and S. A. West, "Genomic Imprinting and Sex Allocation"

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Kin selection analysis is a powerful method for addressing theoretical questions about the evolution of social traits. The method itself is based on a genetic difference equation usually attributed to Price (1970). Basically, "kin selection analysis" is just a name we give to the process of constructing an approximation of allele frequency change as it is described by Price's equation. The particular approximation we use is valid as long as selection is weak (Grafen 1985; Taylor 1989) but does not, in this case, require deviant or "mutant" phenotypes to be rare (Rousset 2004). The latter point, especially, distinguishes kin selection methods we apply here from standard game theoretic ones that consider the success of a mutant phenotype at vanishingly small frequencies (Maynard Smith 1982).

Greenwood-Lee et al. (2001) have derived a general kin selection framework for the evolution of an imprinted social trait. We apply their framework in appendixes A–C in order to study the various models introduced in the main text. The reader should note that our models always assume that the production of sons and the production of daughters are equally costly; consequently, we use the terms "sex ratio" and "sex allocation" interchangeably.

Local Resource Competition

We focus on models of sex ratio evolution with local resource competition (LRC) described in the main text. Most of the following information is devoted to diploid models with maternal control of the sex ratio. Later we discuss how analyses change for haplodiploid species and/or offspring control.

The Standard "No Imprinting" Result

To begin, we suppose that the sex ratio strategy is the phenotype of a singly mated adult female. That is to say, the actor is the adult female itself. We identify recipients as fertilized adult females. Note that there is a subtle difference between the actor and the recipient in this model. The actor is an individual, whereas the recipient is effectively two individuals—a diploid female carrying both copies of her mate's genes in the form of a large amount of stored sperm.

Fix attention on one patch (the focal patch) and on one recipient breeding on this patch (the focal recipient). We define neighbor-modulated recipient fitness, W, as simply a count of the expected number of next-generation fertilized females produced by the focal recipient, weighted by genetic contribution. Let x denote the sex ratio "strategy" used by the "female half" of focal recipient, let y denote the sex ratio strategy of the average female breeding on the focal patch, and let z denote the population-average strategy.

We assume that the focal recipient produces a very large number of offspring, K. Using the notation, we write the number of daughters produced by the focal recipient as $K(1 - x)$. The total number of daughters produced on the focal patch is $NK(1 - y)$. In LRC models with female monogamy, each daughter chooses exactly one mate (fig. A1*a*), and then each daughter competes at random on its natal patch for access to one of the N breeding sites found there. It follows that any given daughter produced on the focal patch is successful with probability $1/K(1 - y)$, and so the fitness gained by the focal recipient through daughters is

$$\frac{1}{2} \times \frac{1 - x}{1 - y}.$$

The factor 1/2 reflects the fact that the focal recipient directly contributes only half of the genetic material found in the "offspring"-fertilized female.

The focal recipient also produces Kx sons. Each son disperses to a different patch, on which we find a total of

$NK(1-z)$ daughters and NKz sons. With female monogamy, each son expects $(1-z)/z$ matings (=fertilized females). As above, each of fertilized females competes successfully for one of N sites with probability $1/(1-z)$; hence, fitness through sons is

$$\frac{1}{2} \times \frac{x}{z}.$$

We can now express W as fitness through sons plus fitness through daughters:

$$W(x, y, z) = \frac{1}{2} \times \frac{x}{z} + \frac{1}{2} \times \frac{1-x}{1-y}.$$

It turns out that we arrive at the same fitness function when we replace the assumption of female monogamy, in the derivation given above, with either polygyny or polyandry. Note that recipient fitness, in all three cases, has the familiar Shaw-Mohler (1953) form.

To find the equilibrium level of investment in sons, z^*, we employ a direct fitness argument (Taylor and Frank 1996). We think of W, temporarily, as a function of a single variable g, the genotypic value of the recipient. Specifically,

$$W(g) \equiv W(x(g), y(g), z).$$

The analysis of W is now quite straightforward. We choose an allele at random from the focal recipient and increase its genic value by a small amount. The resulting marginal change in W (in a population experiencing weak selection) tells us whether selection favors increased or decreased production of sons:

$$\left.\frac{dW}{dg}\right|_{x=y=z} \begin{cases} > 0 \text{ then selection increases } z \\ < 0 \text{ then selection decreases } z \end{cases}.$$

Of course when the marginal change in W vanishes, the population is at equilibrium with respect to the evolution of z.

Applying the Chain Rule, we find

$$\left.\frac{dW}{dg}\right|_{x=y=z} = \frac{1}{2} \times \frac{dx/dg}{z} - \frac{1}{2} \times \frac{dx/dg - dy/dg}{1-z}.$$

Our weak selection assumption allows us to replace the derivatives dx/dg and dy/dg with coefficients of relatedness (Taylor and Frank 1996). Now

$$\left.\frac{dW}{dg}\right|_{x=y=z} \propto \Delta W(z) \stackrel{\text{def}}{=} \frac{R}{z} - \frac{R-\overline{R}}{1-z},$$

where R is the relatedness of the recipient (a pair) to its own "female half" and $\overline{R}$ is the relatedness of the recipient (a pair) to the average actor (an individual) on the patch. We call $\Delta W(z)$ the inclusive fitness effect of increasing z.

Calculations presented in appendix D give us $R = 1/2$ and $\overline{R} = (5N-1)/[2N(3N+1)]$. Using these relatedness coefficients, we find that the evolutionary equilibrium proportion of sons in a brood is given by

$$z^* = \frac{N(3N+1)}{3N(2N-1)+1}. \tag{A1}$$

Note that the result has been recently reported by Wild and Taylor (2005) and Pen (2006), though these authors use a slightly different direct fitness argument. The unusual approach we adopt here (two individuals = a single recipient) was previously used by Taylor (1994) and will facilitate modeling the evolution of z with imprinting.

Evolution with Imprinting

We use the same basic setup as before, with some small modifications. We still consider actors to be adult females, and we still consider recipients to be fertilized females. However, unlike the previous case, we now assume that realized sex ratio decisions are mean averages of the "strategies" that are "implemented" by alleles derived from an individual's male and female parent, respectively. In other words, we adopt the "strategic gene" metaphor (Haig 1997).

To reflect the differing perspectives, we will attach subscripts "m" and "f" to sex ratio strategies x, y, and z. Naturally, subscripts refer to the strategies implemented by the allele inherited from the male (m) or female (f) parent.

Understanding the evolution of z with genomic imprinting actually requires us to track the evolution of two separate traits, z_{m} and z_{f}. As before, the direction of evolution favored by selection will be determined by the sign of a derivative, but now we use a different notational trick.

To describe the action of selection on z_{m}, we begin by writing

$$W_{\mathrm{m}}(g) \equiv W_{\mathrm{m}}(x_{\mathrm{m}}(g), y_{\mathrm{m}}(g), z_{\mathrm{m}}, z_{\mathrm{f}}) = \frac{1}{2} \times \frac{(x_{\mathrm{m}}(g) + z_{\mathrm{f}})/2}{(z_{\mathrm{m}} + z_{\mathrm{f}})/2} + \frac{1}{2} \times \frac{1 - (x_{\mathrm{m}}(g) + z_{\mathrm{f}})/2}{1 - (y_{\mathrm{m}}(g) + z_{\mathrm{f}})/2}.$$

We use this function to construct the inclusive fitness effect, ΔW_{m}. We differentiate W_{m} with respect to g and replace the derivates dx_{m}/dg and dy_{m}/dg with so-called patrilineal relatedness coefficients (Greenwood-Lee et al. 2001) R_{m} and $\overline{R}_{\mathrm{m}}$, respectively. Ignoring a positive multiplier, we get

$$\Delta W_{\mathrm{m}}(z_{\mathrm{m}}, z_{\mathrm{f}}) \stackrel{\mathrm{def}}{=} \frac{R_{\mathrm{m}}}{z} - \frac{R_{\mathrm{m}} - \overline{R}_{\mathrm{m}}}{1 - z},$$

where $z = (z_{\mathrm{m}} + z_{\mathrm{f}})/2$ is the realized sex ratio. To be clear, R_{m} is the patrilineal relatedness of a recipient (again, a pair of individuals) to its own "female half," and $\overline{R}_{\mathrm{m}}$ is the patrilineal relatedness of a recipient to the average female actor (again, an individual) breeding on the same patch. In appendix D we show that

$$R_{\mathrm{m}} = \frac{1}{2},$$

$$\overline{R}_{\mathrm{m}} = \frac{1}{2N}\left(1 + \frac{N - 1}{2N}\right)$$

under LRC.

To describe the action of selection on z_{f}, we use

$$W_{\mathrm{f}}(g) \equiv W_{\mathrm{f}}(x_{\mathrm{f}}(g), y_{\mathrm{f}}(g), z_{\mathrm{m}}, z_{\mathrm{f}}) = \frac{1}{2} \times \frac{(z_{\mathrm{m}} + x_{\mathrm{f}}(g))/2}{(z_{\mathrm{m}} + z_{\mathrm{f}})/2} + \frac{1}{2} \times \frac{1 - (z_{\mathrm{m}} + x_{\mathrm{f}}(g))/2}{1 - (z_{\mathrm{m}} + y_{\mathrm{f}}(g))/2}$$

to construct

$$\Delta W_{\mathrm{f}}(z_{\mathrm{m}}, z_{\mathrm{f}}) = \frac{R_{\mathrm{f}}}{z} - \frac{R_{\mathrm{f}} - \overline{R}_{\mathrm{f}}}{1 - z}.$$

We calculate the matrilineal relatedness coefficients R_{f} and $\overline{R}_{\mathrm{f}}$ in appendix D. For the reader's convenience,

$$R_{\mathrm{f}} = \frac{1}{2},$$

$$\overline{R}_{\mathrm{f}} = \frac{1}{2N}\left(1 + \frac{N-1}{2N}\frac{5N-1}{3N+1}\right).$$

Observe that $\overline{R}_{\mathrm{m}} \leq \overline{R}_{\mathrm{f}}$ with equality if and only if $N = 1$: the relatedness of the recipient to the average actor on the same patch through their patriline never exceeds that through their matriline.

Solving $\Delta W_{\mathrm{m}} = 0$, we find that the realized sex ratio preferred by the paternally inherited allele is

$$z = \frac{2N^2}{4N^2 - 3N + 1}, \tag{A2}$$

and solving $\Delta W_{\mathrm{f}} = 0$, we find that the realized sex ratio preferred by the maternally inherited allele is

$$z = \frac{2N^2(3N+1)}{(4N^2 - N + 1)(3N-1)}. \tag{A3}$$

Intuitively, the standard result (eq. [A1]) lies somewhere between the allele-specific perspectives. Our consideration of genomic imprinting highlights the different perspectives and brings our attention to the question of how the different perspectives are combined to yield equation (A1). Simply put, equation (A1) is exactly the harmonic mean of equations (A2) and (A3) (appropriate because the standard result represents a "harmony" of sorts between alleles?), a fact that can be easily reckoned by substituting $R = R_{\mathrm{f}} = R_{\mathrm{m}} = 1/2$ and $\overline{R} = (\overline{R}_{\mathrm{m}} + \overline{R}_{\mathrm{f}})/2$ into $\Delta W(z)$ and applying some simple algebraic manipulation.

As mentioned in the main text, we should not assume that a compromise between the paternally and the maternally inherited alleles is guaranteed. In fact, in the models we construct here, the perspective of one allele always "wins out."

Conflict resolution can be a theoretically complicated (and mathematically challenging) issue. Luckily, for our models, a simple sketch of the selection nullclines, $\Delta W_{\mathrm{m}} = 0$ and $\Delta W_{\mathrm{f}} = 0$ in the z_{m}, z_{f} plane, tells the story (see app. E). Figure E1 is the "simple sketch" that tells the story

In figure E1, we see that both nullclines lie above the antidiagonal (indicated by a dashed line); hence, both the paternally and the maternally derived alleles "favor" a realized sex ratio that is male biased. Still, the maternally inherited allele favors a larger male bias than that favored by the paternally inherited one (geometrically, the $\Delta W_{\mathrm{f}} = 0$ nullcline in fig. E1 lies above the $\Delta W_{\mathrm{m}} = 0$ nullcline).

Recall that the sign of ΔW_X ($X = \mathrm{m}, \mathrm{f}$) tells us the direction in which selection is pushing z_X: if ΔW_X is positive, for example, then selection encourages movement of z_X in the positive direction (Greenwood-Lee et al. 2001). With this in mind, we see that over evolutionary time, it is the perspective of the paternally inherited allele that wins out (follow the arrows). Under genomic imprinting, then, the stable level of investment in sons is given by equation (A2) and is less male biased than the stable sex ratio predicted by the "no imprinting" model.

Effects of Polyandry

Genomic imprinting is often thought to be significant in cases of multiple paternity of the same brood. We can investigate the effects of polyandry by assuming that each female mates with exactly M males (fig. A1*b*). We will assume further that the female stores a large amount of sperm from each mate and fertilizes her oocytes from a gamete taken at random from these reserves.

Interestingly, the only things that differ from the analysis above are the formulas for the various relatedness coefficients. We still have $R = 1/2$, but now

$$\overline{R} = \frac{N(4M+1) - 1}{2MN(3N+1)}.$$

Substituting these values into $\Delta W(z) = 0$ and solving for z, we identify

$$z^* = \frac{MN(3N+1)}{6MN^2 - 2MN - N + 1}$$

as the equilibrium sex ratio strategy. This sex ratio strategy is still male biased; however, it is less biased than that computed under female monogamy ($M = 1$). In the limit of large M,

$$z^* = \frac{3N+1}{2(3N-1)}, \tag{A4}$$

as $M \to \infty$, which is the result reported by Pen (2006).

To understand the simultaneous effects of polyandry and imprinting, we first observe

$$R_{\mathrm{m}} = \frac{1}{2},$$

$$R_{\mathrm{f}} = \frac{1}{2},$$

$$\overline{R}_{\mathrm{m}} = \frac{2N(M+1) - 1}{4MN^2},$$

$$\overline{R}_{\mathrm{f}} = \frac{2MN(5N-1) + (N-1)^2}{4MN^2(3N+1)}.$$

These relatedness coefficients can be used to show that the paternally inherited allele of a female actor "prefers"

$$z = \frac{2MN^2}{2MN(2N-1) - N + 1} \tag{A5}$$

and the maternally inherited allele of a female actor "prefers"

$$z = \frac{2MN^2(3N+1)}{2MN[3N(2N-1)+1] - (N-1)^2}. \tag{A6}$$

Once again, it is easy to see that (i) equation (A5) is less male biased than equation (A6) and (ii) equation (A4) is simply the harmonic mean of equations (A5) and (A6). Perhaps more interesting is the observation that if each female is fertilized by a large number of males, then the paternally inherited allele favors

$$z \to \frac{N}{2N-1}$$

as $M \to \infty$ and the maternally inherited allele favors

$$z \to \frac{N(3N+1)}{3N(2N-1)+1}.$$

That is to say, in the limit of M large, the sex ratio strategies "preferred" by the paternally and maternally inherited alleles tend toward the strategies that would be favored by a father or a mother, respectively, if either of these had control of the sex ratio (see Wild and Taylor 2005). In short, the conflict between alleles tends toward a conflict between mates.

Returning to the case with finite M, we ask, "Which allelic perspective wins out?" An analysis similar to that illustrated by figure E1 reveals that, once again, it is the (less biased) perspective of the paternally inherited allele that wins out. Though polyandry does not appear to change the qualitative predictions of the model, it does change the qualitative predictions (albeit only slightly). A simple calculation shows that the "winning

perspective" of the paternally inherited allele under polyandry is never more biased than the corresponding "winning perspective" in the case of female monogamy.

Effects of Polygyny

Now we suppose that males compete at random for the opportunity to fertilize all females born on a given patch (fig. A1*c*). Note that under LRC, this implies that all females breeding on the same patch have been fertilized by the same male.

We still have $R = 1/2$, but now

$$\overline{R} = \frac{1}{2N} + \frac{(N-1)(N+1)}{N(6N+2)}$$

(which is never less than the value of $\overline{R}$ under female monogamy). Substituting these into the inclusive fitness effect, we recover Pen's (2006) result:

$$z^* = \frac{3N+1}{5N-1}.$$

One striking feature here is that $z^* \to 3/5$ as $N \to \infty$, in contrast to female monogamy and polyandry.

With imprinting, we find that

$$R_{\mathrm{m}} = R_{\mathrm{f}} = \frac{1}{2},$$

$$\overline{R}_{\mathrm{m}} = \frac{N+1}{4N},$$

$$\overline{R}_{\mathrm{f}} = \frac{(N+1)(N-1)+8N}{4N(3N+1)}.$$

We see that, now, in contrast to the previous situations, matrilinial relatedness never exceeds patrilinial relatedness. The consequence of this is that under polygyny, the paternally inherited allele favors a stronger male bias than that favored by its maternally inherited homologue. Specifically, we find that the paternally inherited allele favors

$$z = \frac{2N}{3N-1}$$

and the maternally inherited allele favors

$$z = \frac{2N(3N+1)}{11N^2-4N+1}.$$

As shown in the main text, the difference between the allelic perspective in this case grows with increasing N, in contrast to previous findings.

Because the maternally inherited allele now has the strategy with the smaller bias, selection acts to promote the interests of this allele. Although polygyny changes our conclusions about the resolution of the intragenomic conflict, polygyny does not (in this case) change the implications of imprinting itself for sex ratio evolution.

Sex Ratio Control by Offspring (Diploidy)

We briefly consider what happens to the analysis when the roles are recast. Suppose that offspring have control over the sex ratio (now interpreted as the probability with which an individual develops into a male). Although

offspring are both actors and recipients in this case, neighbor-modulated recipient fitness maintains its previous form. For example, under offspring control, the "no imprinting" result is recovered by analyzing

$$w(x, y, z) = \frac{1}{2} \times \frac{x}{z} + \frac{1}{2} \times \frac{1 - x}{1 - y}.$$

We use w as a reminder that (i) the recipient is an individual offspring and (ii) x, y, and z are now strategies that belong to offspring fulfilling their roles as actors. Later, we will introduce r's to denote the analogues of R, $\overline{R}$, R_x, and so on.

Haplodiploidy

In a haplodiploid model with maternal control of the sex ratio, we return to the roles assigned at the beginning of this appendix: actors are again adult females and recipients are again fertilized females (effectively two individuals). Despite the fact that roles have been restored, the fitness function changes in two important ways. Consider the "no imprinting" fitness function:

$$W(x^{(\mathrm{f})}, x^{(\mathrm{f+m})}, y^{(\mathrm{f+m})}, z) = \frac{1}{3} \times \frac{x^{(\mathrm{f})}}{z} + \frac{2}{3} \times \frac{1 - x^{(\mathrm{f+m})}}{1 - y^{(\mathrm{f+m})}}.$$

The coefficients 2/3 and 1/3 reflect the fact that, under haplodiploidy, the total reproductive value of males is less than the total reproductive value of females (see Taylor and Frank 1996). The superscripts attached to x, y, and z remind us which relatedness coefficient to use when carrying out the direct fitness calculation. In the "no imprinting" model, we replace (1) $dx^{(\mathrm{f})}/dg$ with $R^{(\mathrm{f})}$, the relatedness of an actor to herself; (2) $dx^{(\mathrm{f+m})}/dg$ with $R^{(\mathrm{f})}$, the relatedness of an actor to her own pair (herself + her mate); (3) $dy^{(\mathrm{f+m})}/dg$ with $R^{(\mathrm{f})}$, the relatedness of an actor to the average recipient on her patch, to arrive at

$$\Delta W(z) = \frac{R^{(\mathrm{f})}}{z} - 2\frac{R^{(\mathrm{f+m})} - \overline{R}^{(\mathrm{f+m})}}{1 - z}. \tag{A7}$$

The need for these relatively complicated relatedness coefficients stems from the fact that haploid males make no genetic contribution to their sons (or rather the sons produced by their mates).

Our haplodiploid offspring control models assume that the control over the sex ratio decision of a reproductive female is exerted by the female worker caste. Formally, the actors in this model are still a (reproductive) adult female, and the recipients are still fertilized females. However, following Taylor (1988), relatedness coefficients must now be constructed in a manner that respects the fact that the individual with control and the actor are not the same individual. To denote this important change, we will continue to use R but write it in boldface, e.g., $\boldsymbol{R}$ (see app. D). When we allow imprinting, we simply substitute the appropriate matrilineal or patrilineal relatedness for $R^{(\mathrm{f})}$, $R^{(\mathrm{f+m})}$, and $\overline{R}^{(\mathrm{f+m})}$ (or their boldface analogues) in equation (A7).

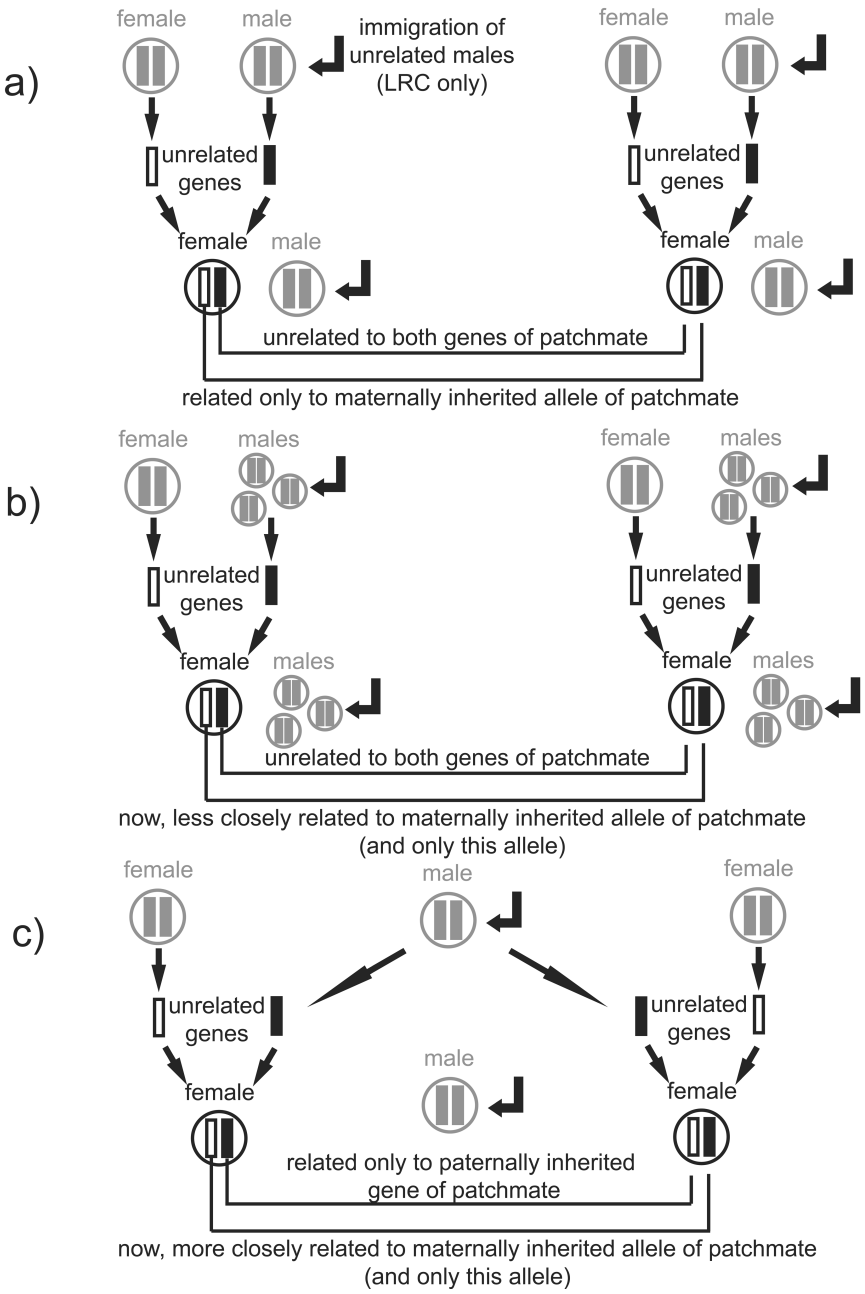

Figure A1: In models of local resource competition (LRC), females mate with unrelated males following long-distance male dispersal. Once fertilized, a female remains on its natal patch to breed. *a*, Under female monogamy, each female mates with exactly one male. In this case, the sex-specific pattern of dispersal means that the maternally inherited gene of any diploid offspring (we depict female offspring) is related to nonsibs born on the same patch—a consequence of the shared matriline. In contrast, the paternally inherited gene of any diploid offspring is unrelated to nonsibs born on the same patch. *b*, Under polyandry, each female mates with a fixed number of males, $M > 1$, and so not all offspring born to the same mother are full sibs. As a result, the relatedness between the maternally inherited gene of a diploid offspring and the average nonsib born on the same patch is reduced relative to the case illustrated in *a*. *c*, Under polygyny, one immigrant male monopolizes all matings on a given patch. Now, all offspring born on the same patch are at least half sibs. Relative to the case

illustrated in *a*, then, we observe an increase in both the relatedness between the maternally inherited gene of a diploid offspring and the average offspring born on the same patch and between the paternally inherited gene of a diploid offspring and the average offspring born on the same patch. In fact, we find that the latter exceeds the former in this case.

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Appendix B from G. Wild and S. A. West, "Genomic Imprinting and Sex Allocation"

(Am. Nat., vol. 173, no. 1, p. E1)

Local Mate Competition

We turn our attention to the local mate competition (LMC) models discussed in the main text. First we discuss diploid species and then the changes one must make to cope with haplodiploids.

"No Imprinting" Results

For a diploid species under LMC, the fitness function we work with is $W(x, y, z)$, or

$$w(x, y, z) = \frac{1}{2} \times \frac{x}{y} \times \frac{1 - y}{1 - z} + \frac{1}{2} \times \frac{1 - x}{1 - z}.$$

In this case, fitness through daughters is written $(1/2) \times (1 - x)/(1 - z)$ because daughters always compete on nonnatal patches. Fitness through sons is written $(1/2) \times x/y \times (1 - x)/(1 - z)$ because a son competes on its natal patch and because its mate later competes on a patch that is not the natal patch of the son. As with local resource competition, we can use the same fitness function to study each of the three mating systems described in the main text.

Using the fitness function, we can recover Hamilton's (1967) result by solving $\Delta W(z) = 0$, which is equivalent to

$$\frac{1 - z}{z} = \frac{R + \overline{R}}{R - \overline{R}}.$$

Doing so yields

$$z^* = \frac{N - 1}{2N} \tag{B1}$$

as the equilibrium stable sex ratio for the case of no imprinting (under female monogamy, polyandry, and polygyny). If we allow imprinting to occur in a mother, then nothing changes. However, we might expect imprinting to occur in offspring under polyandry, as we now show.

With offspring control in a diploid species, we recover

$$\Delta w(z) = \frac{r - \bar{r}}{z} - \frac{r + \bar{r}}{1 - z}.$$

To be clear, r is the relatedness of a diploid offspring (as actor) to itself (as recipient), and $\overline{R}$ is the relatedness of a diploid offspring to a random offspring (actually, random male offspring) born on the same patch. Of course $r = 1$, and it is easy to show that $\bar{r} = 1/(2N - 1)$ (app. D). Given these coefficients of relatedness, we can calculate

$$z^* = \frac{N - 1}{2N - 1}, \tag{B2}$$

which is less female biased than the sex ratio preferred by the parent (eq. [B1]).

Equation (B2) is also the stable offspring sex ratio strategy under polygyny. If we assume a polyandrous mating system where each female is fertilized by M males, then $r = 1$, $\bar{r} = (M + 1)/(4NM - 3M + 1)$, and

$$z^* = \frac{2M(N-1)}{4NM - 3M + 1}. \tag{B3}$$

Note that z^* increases with M, becoming less biased. As $M \to \infty$,

$$z^* \to \frac{2(N-1)}{4N-3} < \frac{1}{2}.$$

Imprinting and Polyandrous Mating Systems

We now allow imprinting to occur in a polyandrous mating system with offspring control of the sex ratio. In this case, we have $r_m = r_f = 1$, where r_m and r_f are, respectively, the patrilineal and the matrilineal relatedness of an individual to itself. We also have

$$\bar{r}_m = \frac{2N + (M-1)}{N(4MN - 3M + 1)},$$

$$\bar{r}_f = \frac{2MN - (M-1)}{N(4MN - 3M + 1)},$$

where $\bar{r}_m$ and $\bar{r}_f$ are, respectively, the patrilineal and the matrilineal relatedness of an individual to a random male competing on the patch. If we look at these relatedness coefficients for the extreme case of M large, we begin to suspect that intragenomic conflict

$$\bar{r}_m \to \frac{1}{N(4N-3)},$$

$$\bar{r}_f \to \frac{2N-1}{N(4N-3)}$$

as $M \to \infty$. Clearly, $r_f \geq r_m$ with equality if and only if $N = 1$.

We again construct

$$\Delta w_m(z_m, z_f) = \frac{r_m - \bar{r}_m}{z} - \frac{r_m + \bar{r}_m}{1-z},$$

$$\Delta w_f(z_m, z_f) = \frac{r_f - \bar{r}_f}{z} - \frac{r_f + \bar{r}_f}{1-z}.$$

Recall that $z = (z_m + z_f)/2$. Setting Δw_m equal to 0 and solving for z, we see that the paternally inherited allele favors

$$z^* = \frac{(N-1)[4MN + (M-1)]}{2N(4MN - 3M + 1)} \to \frac{(N-1)(4N+1)}{2N(4N-3)} \tag{B4}$$

as $M \to \infty$, and doing the same for Δw_f, we see that the maternally inherited allele favors

$$z^* = \frac{(N-1)[4MN - (M-1)]}{2N(4MN - 3M + 1)} \to \frac{(N-1)(4N-1)}{2N(4N-3)} \tag{B5}$$

as $M \to \infty$. Both alleles favor a female-biased sex ratio; however, the bias favored by the maternally inherited allele is stronger than that favored by the paternally inherited allele. In fact, it is easy to show that the "no imprinting" result, equation (B3), is the arithmetic mean of the strategy favored by the paternal allele, equation (B4), and that favored by the maternal allele, equation (B5).

Haplodiploidy

With haplodiploid species,

$$W(x^{(\mathrm{f})}, x^{(\mathrm{f+m})}, y^{(\mathrm{f})}, y^{(\mathrm{f+m})}, z) = \frac{1}{3} \times \frac{x^{(\mathrm{f})}}{y^{(\mathrm{f})}} \times \frac{1 - y^{(\mathrm{f})}}{1 - z} + \frac{2}{3} \times \frac{1 - x^{(\mathrm{f+m})}}{1 - z},$$

and so, ignoring a positive multiplier,

$$\Delta W(z_{\mathrm{m}}, z_{\mathrm{f}}) = \frac{R^{(\mathrm{f})} - \overline{R}^{(\mathrm{f})}}{z} - 2\frac{R^{(\mathrm{f+m})} + \overline{R}^{(\mathrm{f})}/2}{1 - z}$$

in the case of maternal control and

$$\Delta W(z_{\mathrm{m}}, z_{\mathrm{f}}) = \frac{\boldsymbol{R}^{(\mathrm{f})} - \overline{\boldsymbol{R}}^{(\mathrm{f})}}{z} - 2\frac{\boldsymbol{R}^{(\mathrm{f+m})} + \overline{\boldsymbol{R}}^{(\mathrm{f})}/2}{1 - z}$$

in the case of offspring (worker) control. Of course, with imprinting we use appropriate matrilineal or patrilineal relatedness coefficients in the inclusive fitness effects.

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Appendix C from G. Wild and S. A. West, "Genomic Imprinting and Sex Allocation"

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Local Resource Enhancement

We construct a model of local resource enhancement (LRE). Recall that we suppose the production of females increases the success of offspring from that patch or possibly the overall productivity of the patch.

LRE is a process that involves interactions between generations, and we would like to build this idea, in a biologically reasonable manner, into the framework that assumes nonoverlapping generations. We will assume, then, that brood size of each fertilized female, K, depends in a positive way on how many daughters were produced on its own natal patch (the idea here could be that male offspring exert some negative effect on their female counterparts, the consequences of which are not felt until adulthood). Mathematically, we consider K to be a decreasing function of y', the average sex ratio strategy on the natal patch of the "female half" of the focal recipient. This scenario is reminiscent of transgenerational interactions studied by Lehmann (2007).

With global mixing and maternal control of the sex ratio, we have

$$W(x, y', z) = \frac{K(y')}{K(z)}\left(\frac{1}{2} \times \frac{1-x}{1-z} + \frac{1}{2} \times \frac{x}{z}\right),$$

which yields

$$\Delta W(z) = \frac{K'(z)}{K(z)}\overline{R}' + \frac{1}{2}\left(\frac{1}{z} - \frac{1}{1-z}\right)R,$$

where $R = 1/2$ is the relatedness of the diploid female actor to its own tetraploid pairing and $\overline{R}' = 1/4N$ is the relatedness of the diploid female actor to the average next-generation mated pair whose "female half" was born on the actor's patch.

A convenient model for the relationship between brood size and sex ratio is $K(z) = K_{\max}e^{-\alpha z}$, where the constant $K_{\max}$ denotes the maximum brood size and $\alpha > 0$ is a constant related to the deferred (i.e., transgenerational) fecundity cost associated with the production of males. As expected (e.g., Emlen et al. 1986), the numerical solution of the equation $\Delta W = 0$ indicates that the stable sex ratio strategy is biased toward the sex that contributes toward productivity.

LRE will not promote genomic imprinting without sex-specific dispersal patterns. Under both LRE and local resource competition (LRC), we use

$$W(x, y, y', z) = \frac{1}{2}\frac{(1-x)}{(1-y)} + \frac{1}{2}\frac{K(y')x}{K(z)z}$$

to derive the inclusive fitness effect,

$$\Delta W(z) = \frac{R}{z} - \frac{R-\overline{R}}{1-z} - \alpha\overline{R}' = R\left(\frac{1}{z} - \frac{1}{1-z}\right) + \overline{R}\left(\frac{1}{1-z} - \frac{\alpha}{2}\right).$$

The second equality follows from the fact that with complete outbreeding, we have $\overline{R}' = \overline{R}/2$.

With this simple model, we see that the effect that LRC has on sex ratio evolution dominates that of LRE,

provided that the negative "transgenerational" effect of male offspring is not too large. That is to say, the numerical solution of $\Delta W = 0$ shows us that when $\alpha < 4$, the stable sex ratio is male biased. When the negative effect of male offspring is large (i.e., when $\alpha > 4$), the reverse is true: LRE dominates LRC, and stable sex ratios are biased toward females.

If we allow genomic imprinting, we see that when $\alpha = 4$, there is no conflict between the paternally and the maternally inherited allele: both favor an unbiased sex ratio. More generally, the perspective of the paternally inherited allele is never more extreme than that of its maternally inherited homologue.

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Appendix D from G. Wild and S. A. West, "Genomic Imprinting and Sex Allocation"

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Calculation of Relatedness Coefficients

The relatedness coefficients used in the previous appendixes are constructed using coefficients of consanguinity (CCs). If we draw an allele at random from one individual (individual I) and an allele at random from an second individual (individual J), then the CC between I and J is simply the probability that the chosen alleles have descended from a common ancestor without mutation. Simply put, CCs are probabilities of identity by descent.

Following Michod and Hamilton (1980), we express relatedness as the quotient of two CCs. Following Taylor (1988), our relatedness coefficients will, in general, make reference to three individuals, the actor (individual I), the recipient (individual J), and the individual with control of the actor's phenotype (individual K). The third individual is sometimes also the actor, but this is not always the case. For the moment, if $G_{J,K}$ is the CC between the recipient and the individual with control of the actor's phenotype and if $G_{I,K}$ is the CC between the actor and the individual with control of the actor's phenotype, then the relatedness between I and J from K's perspective is

$$\frac{G_{J,K}}{G_{I,K}}.$$

Because we use a weak selection approximation of Price's equation, it is enough to calculate CCs and relatedness coefficients under the assumption that selection does not operate in the population. All calculations therefore assume that the population is monomorphic with respect to sex allocation strategy.

Diploidy

Female Monogamy

Let G_n denote the CC between two offspring born on the same patch in generation n. A standard conditioning argument yields the following recursive expression:

$$G_{n+1} = \frac{1}{N}\frac{1 + 3k_{\mathrm{m}}G_n}{4} + \frac{N-1}{N}\frac{k_{\mathrm{f}}^2(1 + k_{\mathrm{m}})^2 G_n}{4}, \tag{D1}$$

where k_{m} and k_{f} denote the probability that male and female offspring, respectively, compete successfully on their natal patch. To understand equation (D1), recall that male dispersal precedes male-male competition for mates. Recall also that mating precedes dispersal by females, which in turn precedes female-female competition for breeding sites. This is the so-called disperse-mate-disperse model that has been studied by Taylor (1994) and Wild and Taylor (2004). When $k_{\mathrm{m}} = 1$ and $k_{\mathrm{f}} = 0$, we recover the local mate competition (LMC) life cycle studied by Hamilton (1967), and when $k_{\mathrm{m}} = 0$ and $k_{\mathrm{f}} = 1$, we recover the local resource competition (LRC) life cycle discussed by Clark (1978).

As $n \to \infty$, we see that G_n converges to the equilibrium value

$$G = \frac{1}{4N - (N-1)k_{\mathrm{f}}^2(1 + k_{\mathrm{m}})^2 - 3k_{\mathrm{m}}}. \tag{D2}$$

If F denotes the well-known coefficient of inbreeding, then at equilibrium, $F = k_{\mathrm{m}}G$. That is to say,

$$F = \frac{k_{\mathrm{m}}}{4N - (N-1)k_{\mathrm{f}}^2(1+k_{\mathrm{m}})^2 - 3k_{\mathrm{m}}}. \tag{D3}$$

Now, let H_{XY} denote the CC between a sex X adult and a sex Y adult breeding on the same patch (i.e., a "neighbor") and chosen with replacement where appropriate. It is easy to see that

$$H_{\mathrm{ff}} = \frac{1}{N}\frac{1+F}{2} + \frac{N-1}{N}k_{\mathrm{f}}^2 G, \tag{D4}$$

$$H_{\mathrm{mm}} = \frac{1}{N}\frac{1+F}{2} + \frac{N-1}{N}k_{\mathrm{f}}^2 k_{\mathrm{m}}^2 G, \tag{D5}$$

$$H_{\mathrm{fm}} = \frac{1}{N}k_{\mathrm{m}} G + \frac{N-1}{N}k_{\mathrm{f}}^2 k_{\mathrm{m}} G. \tag{D6}$$

It is worth noting that

$$H_{\mathrm{mm}} \leq H_{\mathrm{ff}}.$$

The previous inequality holds as an equality whenever $k_{\mathrm{f}} = 0$ (complete female dispersal) or $k_{\mathrm{m}} = 1$ (complete male philopatry). In particular $H_{\mathrm{mm}} = H_{\mathrm{ff}}$ under LMC.

In the case where there is maternal control (actor = individual with control) and no imprinting, we express the relatedness of an adult female (as actor) to its own mated pair (as recipient) as

$$R = \frac{1}{2}(1) + \frac{1}{2}\frac{k_{\mathrm{m}}G}{(1+F)/2} = \begin{cases} 1/2 & \text{LRC} \\ N/(2N-1) & \text{LMC} \end{cases},$$

where we have measured relatedness from the perspective of the actor (=individual with control). Similarly, the relatedness of an adult female to the average mated pair breeding on her patch (taken from the perspective of the adult female herself) is

$$\overline{R} = \frac{(1/2)H_{\mathrm{ff}} + (1/2)H_{\mathrm{fm}}}{(1+F)/2} = \begin{cases} (5N-1)/2N(3N+1) & \text{LRC} \\ 1/(2N-1) & \text{LMC} \end{cases}.$$

When imprinting occurs, our relatedness calculation changes slightly. If sex Y is the actor and the gene derived from the actor's sex Z parent has control of actor's phenotype, we have

$$R_Z = \frac{1}{2} + \frac{1}{2}\frac{k_{\mathrm{m}}(H_{\mathrm{f}Z} + H_{\mathrm{m}Z})}{1+F} = \begin{cases} 1/2 & \text{LRC, } Z = \mathrm{m}, \mathrm{f} \\ N/(2N-1) & \text{LMC, } Z = \mathrm{m}, \mathrm{f} \end{cases}.$$

Note that for LRC and LMC, $R_Z = R$.

To state average relatedness coefficients, we need to introduce $H_{X,Y|Z}$, the CC between a random allele residing in a sex X adult and the allele residing in the average sex Y neighbor that was derived from the neighbor's own sex z parent. Now,

$$H_{\mathrm{f,f|f}} = \frac{1}{N}\frac{1+F}{2} + \frac{N-1}{N}k_{\mathrm{f}}^2\left(\frac{1}{2}H_{\mathrm{ff}} + \frac{1}{2}H_{\mathrm{fm}}\right),$$

$$H_{\mathrm{f,f|m}} = \frac{1}{N}\frac{1+F}{2} + \frac{N-1}{N}k_{\mathrm{f}}^2\left(\frac{1}{2}H_{\mathrm{fm}} + \frac{1}{2}H_{\mathrm{mm}}\right),$$

$$H_{\mathrm{f,m|f}} = H_{\mathrm{m,f|f}} = \left(\frac{1}{N}k_{\mathrm{m}} + \frac{N-1}{N}k_{\mathrm{f}}^2k_{\mathrm{m}}\right)\left(\frac{1}{2}H_{\mathrm{ff}} + \frac{1}{2}H_{\mathrm{fm}}\right),$$

$$H_{\mathrm{f,m|m}} = H_{\mathrm{m,f|m}} = \left(\frac{1}{N}k_{\mathrm{m}} + \frac{N-1}{N}k_{\mathrm{f}}^2k_{\mathrm{m}}\right)\left(\frac{1}{2}H_{\mathrm{fm}} + \frac{1}{2}H_{\mathrm{mm}}\right),$$

$$H_{\mathrm{m,m|f}} = \frac{1}{N}\frac{1+F}{2} + \frac{N-1}{N}k_{\mathrm{m}}^2k_{\mathrm{f}}^2\left(\frac{1}{2}H_{\mathrm{ff}} + \frac{1}{2}H_{\mathrm{fm}}\right),$$

$$H_{\mathrm{m,m|m}} = \frac{1}{N}\frac{1+F}{2} + \frac{N-1}{N}k_{\mathrm{m}}^2k_{\mathrm{f}}^2\left(\frac{1}{2}H_{\mathrm{fm}} + \frac{1}{2}H_{\mathrm{mm}}\right),$$

and we note that in each case,

$$H_{XY|\mathrm{m}} \leq H_{XY|\mathrm{f}},$$

with equality now implying at least one of $k_{\mathrm{f}} = 0$, $k_{\mathrm{m}} = 0$, $k_{\mathrm{m}} = 1$. Interestingly, $H_{X,Y|\mathrm{m}} = H_{X,Y|\mathrm{f}}$ under LMC, a consequence of the fact that, under LMC, $H_{\mathrm{ff}} = H_{\mathrm{mm}}$.

The Z-line relatedness between a sex $Y = \mathrm{f}$ actor and the average mated pair breeding on the same patch can now be expressed as

$$\overline{R}_Z = \frac{H_{\mathrm{m,f}|Z} + H_{\mathrm{f,f}|Z}}{1+F} = \begin{cases} (11N^2 - 4N + 1)/4N^2(3N+1) & \text{LRC, } Z = \mathrm{f} \\ (3N-1)/4N^2 & \text{LRC, } Z = \mathrm{m} \\ 1/(2N-1) & \text{LMC, } Z = \mathrm{m, f} \end{cases}.$$

Observe that $\overline{R}$ used in the "no imprinting" models is simply the mean average of $\overline{R}_{\mathrm{m}}$ and $\overline{R}_{\mathrm{f}}$. In addition, we note that with LMC, $\overline{R}_{\mathrm{f}} = \overline{R}_{\mathrm{m}} = \overline{R}$. We have already seen that imprinting does not change the R coefficients used in "no imprinting" models of LMC. It stands to reason, then, that the inclusive fitness calculation for the LMC/maternal control/diploid yields the same equilibrium phenotype in both the "imprinting" and "no imprinting" cases.

If we allow offspring control of the sex ratio, then the relevant relatedness coefficients include (1) the relatedness of a diploid offspring (as actor) to itself, $r = 1$; (2) the relatedness of a diploid offspring (as actor) to the average offspring born on the same patch,

$$\bar{r} = \frac{2G}{1+F} = \begin{cases} 2/(3N+1) & \text{LRC} \\ 1/(2N-1) & \text{LMC} \end{cases};$$

(3) the Z-line relatedness of an offspring to itself, $r_Z = r = 1$; and (4) the Z-line relatedness of an offspring to the average offspring born on the same patch,

$$\bar{r}_Z = \frac{H_{\mathrm{f}Z} + H_{\mathrm{m}Z}}{1+F} = \begin{cases} (5N-1)/2N(3N+1) & \text{LRC, } Z = \mathrm{f} \\ 1/2N & \text{LRC, } Z = \mathrm{m} \\ 1/(2N-1) & \text{LMC, } Z = \mathrm{m, f} \end{cases}.$$

Again we note that imprinting has no effect on the value taken by the relatedness coefficients used to calculate inclusive fitness effects under LMC.

Polyandry

Now we suppose that each female mates with exactly M males, storing a large amount of sperm from each, with which she fertilizes her oocytes. In this case,

$$G_{n+1} = \frac{1}{N}\left[\frac{1}{4}\frac{1+k_{\mathrm{m}}G_n}{2} + \frac{1}{2}k_{\mathrm{m}}G_n + \frac{1}{4}\left(\frac{1}{M}\frac{1+k_{\mathrm{m}}G_n}{2} + \frac{M-1}{M}k_{\mathrm{m}}^2G_n\right)\right] + \frac{N-1}{N}\frac{k_{\mathrm{f}}^2(1+k_{\mathrm{m}})^2G_n}{4}. \tag{D7}$$

Note that when $M = 1$, equation (D7) reduces to equation (D1).

From equation (D1), we see that G_n converges to

$$G = \frac{M+1}{8NM - 2(M-1)k_{\mathrm{m}}^2 - 2(N-1)k_{\mathrm{f}}^2(1+k_{\mathrm{m}})^2M - (5M+1)k_{\mathrm{m}}},$$

and hence

$$F = \frac{k_{\mathrm{m}}(M+1)}{8NM - 2(M-1)k_{\mathrm{m}}^2 - 2(N-1)k_{\mathrm{f}}^2(1+k_{\mathrm{m}})^2M - (5M+1)k_{\mathrm{m}}}.$$

Following the procedure we adopted in the case of female monogamy, we compute

$$H_{\mathrm{ff}} = \frac{1}{N}\frac{1+F}{2} + \frac{N-1}{N}k_{\mathrm{f}}^2G, \tag{D8}$$

$$H_{\mathrm{mm}} = \frac{1}{N}\left(\frac{1}{M}\frac{1+F}{2} + \frac{M-1}{M}k_{\mathrm{m}}^2G\right) + \frac{N-1}{N}k_{\mathrm{f}}^2k_{\mathrm{m}}^2G, \tag{D9}$$

$$H_{\mathrm{fm}} = \frac{1}{N}k_{\mathrm{m}}G + \frac{N-1}{N}k_{\mathrm{f}}^2k_{\mathrm{m}}G \tag{D10}$$

and use these, in turn, to compute

$$H_{\mathrm{f,f|f}} = \frac{1}{N}\frac{1+F}{2} + \frac{N-1}{N}k_{\mathrm{f}}^2\left(\frac{1}{2}H_{\mathrm{ff}} + \frac{1}{2}H_{\mathrm{fm}}\right),$$

$$H_{\mathrm{f,f|m}} = \frac{1}{N}\frac{1+F}{2} + \frac{N-1}{N}k_{\mathrm{f}}^2\left(\frac{1}{2}H_{\mathrm{fm}} + \frac{1}{2}H_{\mathrm{mm}}\right),$$

$$H_{\mathrm{f,m|f}} = H_{\mathrm{m,f|f}} = \left(\frac{1}{N}k_{\mathrm{m}} + \frac{N-1}{N}k_{\mathrm{f}}^2k_{\mathrm{m}}\right)\left(\frac{1}{2}H_{\mathrm{ff}} + \frac{1}{2}H_{\mathrm{fm}}\right),$$

$$H_{\mathrm{f,m|m}} = H_{\mathrm{m,f|m}} = \left(\frac{1}{N}k_{\mathrm{m}} + \frac{N-1}{N}k_{\mathrm{f}}^2k_{\mathrm{m}}\right)\left(\frac{1}{2}H_{\mathrm{fm}} + \frac{1}{2}H_{\mathrm{mm}}\right),$$

$$H_{\mathrm{m,m|f}} = \frac{1}{N}\left(\frac{1}{M}\frac{1+F}{2} + \frac{M-1}{M}k_{\mathrm{m}}^2\frac{H_{\mathrm{fm}}+H_{\mathrm{ff}}}{2}\right) + \frac{N-1}{N}k_{\mathrm{f}}^2k_{\mathrm{m}}^2\left(\frac{H_{\mathrm{fm}}+H_{\mathrm{ff}}}{2}\right),$$

$$H_{\mathrm{m,m|m}} = \frac{1}{N}\left(\frac{1}{M}\frac{1+F}{2} + \frac{M-1}{M}k_{\mathrm{m}}^2\frac{H_{\mathrm{fm}}+H_{\mathrm{mm}}}{2}\right) + \frac{N-1}{N}k_{\mathrm{f}}^2k_{\mathrm{m}}^2\left(\frac{H_{\mathrm{fm}}+H_{\mathrm{mm}}}{2}\right).$$

Using calculating formulas introduced above, we find

$$R = \begin{cases} 1/2 & \text{LRC} \\ \{M(2N-1) + 1/[M(4N-3)+1]\} & \text{LMC'} \end{cases}$$

$$\overline{R} = \begin{cases} [(4M+1)N-1]/2MN(3N+1) & \text{LRC} \\ M(2N-1)+1]/N[M(4N-3)+1] & \text{LMC} \end{cases}$$

(for use in maternal control models without imprinting);

$$R_Z = \begin{cases} 1/2 & \text{LRC, } Z = \text{m, f} \\ [(4N^2-3N+1)M+3N-1]/2N[(4N-3)M+1] & \text{LMC, } Z = \text{m} \\ [4N^2-N-1)M+N+1]/2N[(4N-3)M+1] & \text{LMC, } Z = \text{f} \end{cases},$$

$$\overline{R}_Z = \begin{cases} [(2M+1)N-1]/4MN^2 & \text{LRC, } Z = \text{m} \\ [2MN(5N-1)+(N-1)^2]/4MN^2(3N+1) & \text{LRC, } Z = \text{f} \\ [(4N^2-3N+1)M+3N-1]/2N^2[(4N-3)M+1] & \text{LMC, } Z = \text{m} \\ [(4N^2-N-1)M+N+1])/2N^2[(4N-3)M+1] & \text{LMC, } Z = \text{f} \end{cases}$$

(for use in maternal control models with imprinting), $r = 1$ and

$$\bar{r} = \begin{cases} (M+1)/M(3N+1) & \text{LRC} \\ (M+1)/[M(4N-3)+1] & \text{LMC} \end{cases}$$

(for use in offspring control models without imprinting); and finally $r_Z = 1$ and

$$\bar{r}_Z = \begin{cases} 1/2MN & \text{LRC, } Z = \text{m} \\ [(4M+1)N-1]/2MN(3N+1) & \text{LRC, } Z = \text{f} \\ (2N-1+M)/N[M(4N-3)+1] & \text{LMC, } Z = \text{m} \\ [(2N-1)M+1]/N[M(4N-3)+1] & \text{LMC, } Z = \text{f} \end{cases}$$

(for use in offspring control models with imprinting).

Polygyny

Now assume a polygynous mating system in which the same male fertilizes all females born on a given patch. Under LMC, polygyny is clearly equivalent to female monogamy, and so we report only LRC relatedness coefficients.

Under the assumption of polygyny, the general recursion for G_n becomes

$$G_{n+1} = \frac{1}{N}\frac{1+3k_{\text{m}}G_n}{4} + \frac{N-1}{N}\left(\frac{1}{4}k_{\text{f}}^2 G_n + \frac{1}{2}k_{\text{f}}^2 k_{\text{m}} G_n + \frac{1}{4}k_{\text{f}}^2\frac{1+k_{\text{m}}G_n}{2}\right), \tag{D11}$$

and over time G_n goes to

$$G = \frac{2+k_{\text{f}}^2(N-1)}{8N-(N-1)k_{\text{f}}^2(2+5k_{\text{m}})-6k_{\text{m}}},$$

$$F = \frac{[2+k_{\text{f}}^2(N-1)]k_{\text{m}}}{8N-(N-1)k_{\text{f}}^2(2+5k_{\text{m}})-6k_{\text{m}}}.$$

We will also need

$$H_{\mathrm{ff}} = \frac{1}{N}\frac{1+F}{2} + \frac{N-1}{N}k_{\mathrm{f}}^2 G, \tag{D12}$$

$$H_{\mathrm{mm}} = \left(\frac{1}{N} + \frac{N-1}{N}k_{\mathrm{f}}^2\right)\frac{1+F}{2}, \tag{D13}$$

$$H_{\mathrm{fm}} = \frac{1}{N}k_{\mathrm{m}} G + \frac{N-1}{N}k_{\mathrm{f}}^2 k_{\mathrm{m}} G. \tag{D14}$$

The calculating formulas for $H_{XY|Z}$ that change are those for $H_{\mathrm{mm}|Z}$. Now, $H_{\mathrm{mm}|\mathrm{m}} = H_{\mathrm{mm}|\mathrm{f}} = H_{\mathrm{mm}}$. The calculating formulas for $H_{\mathrm{ff}|Z}$ and $H_{\mathrm{fm}|Z}$ are as they were under polyandry and female monogamy.

Using previous calculating formulas, we find that in LRC models of sex ratio evolution with polygyny and maternal control,

$$R = \frac{1}{2},$$

$$\overline{R} = \frac{N+3}{2(3N+1)},$$

$$R_Z = \frac{1}{2},$$

$$\overline{R}_Z = \begin{cases} (N+1)/4N & Z = \mathrm{m} \\ (N^2+8N-1)/4N(3N+1) & Z = \mathrm{f} \end{cases}.$$

With LRC and offspring control, we have

$$r = 1,$$

$$\bar{r} = \frac{N+1}{3N+1},$$

$$r_Z = 1,$$

$$\bar{r}_Z = \begin{cases} 1/2 & Z = \mathrm{m} \\ (N+3)/2(3N+1) & Z = \mathrm{f} \end{cases}.$$

Haplodiploidy

Female Monogamy

Now we assume that males arise from a mother's unfertilized gametes. Because of sex-specific ploidies, it is more straightforward to express the next-generation recursions for H_{ff}, H_{fm}, and H_{mm}, respectively:

$$H_{\mathrm{ff},n+1} = \frac{1}{N}\frac{1 + k_{\mathrm{m}}(H_{\mathrm{ff},n} + H_{\mathrm{fm},n})/2}{2} + \frac{N-1}{N}k_{\mathrm{f}}^2\left(\frac{H_{\mathrm{ff},n} + 2H_{\mathrm{fm},n} + H_{\mathrm{mm},n}}{4}\right), \tag{D15}$$

$$H_{\mathrm{fm},n+1} = \left(\frac{1}{N}k_{\mathrm{m}} + \frac{N-1}{N}k_{\mathrm{m}}k_{\mathrm{f}}^2\right)\left(\frac{1}{2}H_{\mathrm{ff},n} + \frac{1}{2}H_{f\mathrm{m},n}\right), \tag{D16}$$

$$H_{\mathrm{mm},n+1} = \frac{1}{N} + \frac{N-1}{N}k_{\mathrm{f}}^2 k_{\mathrm{m}}^2 H_{\mathrm{ff},n}. \tag{D17}$$

The equilibrium solution of equation (D15) is a bit cumbersome for arbitrary k_{m} and k_{f}. For simplicity, then, we present only the solutions for LRC ($k_{\mathrm{m}} = 0$, $k_{\mathrm{f}} = 1$) and LMC ($k_{\mathrm{m}} = 1$, $k_{\mathrm{f}} = 0$). For LRC,

$$H_{\mathrm{ff}} = \frac{3N-1}{N(3N+1)},$$
$$H_{\mathrm{mm}} = \frac{1}{N},$$
$$H_{\mathrm{fm}} = 0,$$
$$F = 0,$$

and for LMC,

$$H_{\mathrm{ff}} = \frac{2N-1}{N(4N-3)},$$
$$H_{\mathrm{mm}} = \frac{1}{N},$$
$$H_{\mathrm{fm}} = \frac{1}{N(4N-3)},$$
$$F = \frac{1}{4N-3}.$$

If G_{XY} denotes the CC between a sex X and sex Y offspring born on the same patch, then at equilibrium,

$$G_{\mathrm{ff}} = \frac{H_{\mathrm{mm}}}{4} + \frac{H_{\mathrm{fm}}}{2} + \frac{H_{\mathrm{ff}}}{4}, \tag{D18}$$
$$G_{\mathrm{fm}} = \frac{H_{\mathrm{ff}} + H_{\mathrm{fm}}}{2}, \tag{D19}$$
$$G_{\mathrm{mm}} = H_{\mathrm{ff}}, \tag{D20}$$
$$F = k_{\mathrm{m}} G_{\mathrm{fm}}, \tag{D21}$$

and we can define

$$G_{\mathrm{f,f|}Z} = \frac{H_{\mathrm{f}Z} + H_{\mathrm{m}Z}}{2}, \tag{D22}$$
$$G_{\mathrm{m,f|}Z} = H_{\mathrm{f}Z}. \tag{D23}$$

Recall that haplodiploid models use the coefficient $R^{(\mathrm{f+m})}$, that is, the relatedness of the fertilized recipient to its own "female half" (actor). A simple calculation yields

$$R^{(\mathrm{f+m})} = \frac{(1/2)k_{\mathrm{m}}G_{\mathrm{fm}} + (1/2)(1+F)/2}{(1+F)/2} = \frac{1}{2}\frac{1+3F}{1+F}$$

or

$$R^{(\mathrm{f+m})} = \begin{cases} 1/2 & \mathrm{LRC} \\ N/(2N-1) & \mathrm{LMC} \end{cases}.$$

Notice that when we compute $R^{(\mathrm{f+m})}$, we choose the male and female portion of the fertilized recipient with equal probability, even though males are haploid. This is a reflection of the fact that mothers and fathers make equal genetic contributions to daughters.

The coefficient $R^{(f)}$ denotes the relatedness of the "female half" of a fertilized female to itself. Of course $R^{(f)} = 1$ in both the LRC and the LMC cases.

The coefficient $\overline{R}^{(f+m)}$ denotes the relatedness of the diploid female actor to the average member of the fertilized female recipient breeding on the same patch. Now,

$$\overline{R}^{(f+m)} = \frac{1}{N}R^{(f+m)} + \frac{N-1}{N}\frac{(1/2)k_m k_f^2 G_{fm} + (1/2)k_f^2 G_{ff}}{(1+F)/2} = \frac{H_{ff} + H_{fm}}{1+F}$$

or

$$\overline{R}^{(f+m)} = \begin{cases} (3N-1)/N(3N+1) & \text{LRC} \\ 1/(2N-1) & \text{LMC} \end{cases}.$$

The coefficient $\overline{R}^{(f)}$ denotes the relatedness of the diploid female actor to the average (diploid) female recipient breeding on the same patch. Now,

$$\overline{R}^{(f)} = \frac{H_{ff}}{(1+F)/2} = \frac{1}{N}(1) + \frac{N-1}{N}\frac{k_f^2 G_{ff}}{(1+F)/2} = \begin{cases} 2(3N-1)/N(3N+1) & \text{LRC} \\ 1/N & \text{LMC} \end{cases}.$$

If we allow genomic imprinting, then the general form of the relevant relatedness coefficients is, in general,

$$\begin{aligned} R_Z^{(f)} &= 1, \\ R_Z^{(f+m)} &= \frac{(1/2)k_m G_{m,f|Z} + (1/2)(1+F)/2}{(1+F)/2}, \\ \overline{R}_Z^{(f)} &= \frac{1}{N}(1) + \frac{N-1}{N}\frac{k_f^2 G_{f,f|Z}}{(1+F)/2}, \\ \overline{R}_Z^{(f+m)} &= \frac{1}{N}R_Z^{(f+m)} + \frac{N-1}{N}\frac{(1/2)k_m k_f^2 G_{m,f|Z} + (1/2)k_f^2 G_{f,f|Z}}{(1+F)/2}. \end{aligned}$$

In special cases we find

$$R_Z^{(f+m)} = \begin{cases} 1/2 & \text{LRC, } Z = m, f \\ (2N^2 - N + 1)/2N(2N-1) & \text{LMC, } Z = m \\ (N+1)/2N & \text{LMC, } Z = f \end{cases},$$

$$\overline{R}_Z^{(f)} = \begin{cases} (2N-1)/N^2 & \text{LRC, } Z = m \\ (6N^2 - 3N + 1)/N^2(3N+1) & \text{LRC, } Z = f \\ 1/N & \text{LMC, } Z = m, f \end{cases},$$

$$\overline{R}_Z^{(f+m)} = \begin{cases} (2N-1)/2N^2 & \text{LRC, } Z = m \\ (6N^2 - 3N + 1)/2N^2(3N+1) & \text{LRC, } Z = f \\ (2N^2 - N + 1)/2N^2(2N-1) & \text{LMC, } Z = m \\ (N+1)/2N^2 & \text{LMC, } Z = f \end{cases}.$$

Recall that in haplodiploid "offspring control" models, we formally consider mothers as actors, but we give control of maternal sex ratio decisions to female offspring. Let $\boldsymbol{R}^{(f)}$ denote the relatedness of mother (actor) to her own "female half" (recipient), now measured from the perspective of the female offspring of the mother herself (individual with control). From the definition given at the beginning of this appendix, we now normalize CCs using

$$\frac{(1/2)(1+F)}{2} + \frac{1}{2}k_m G_{fm} = \begin{cases} 1/4 & \text{LRC} \\ N/(4N-3) & \text{LMC'} \end{cases}$$

the CC of mother (actor) and daughter (individual with control). It follows that

$$\boldsymbol{R}^{(\mathrm{f})} = \frac{(1/2)(1+F)/2 + (1/2)k_{\mathrm{m}}G_{\mathrm{fm}}}{(1/2)(1+F)/2 + (1/2)k_{\mathrm{m}}G_{\mathrm{fm}}} = 1$$

for all models including LRC and LMC. The average value of $\boldsymbol{R}^{(\mathrm{f})}$ is denoted $\overline{\boldsymbol{R}}^{(\mathrm{f})}$ and is calculated

$$\begin{aligned}\frac{H_{\mathrm{ff}} + H_{\mathrm{fm}}}{(1+F)/2 + k_{\mathrm{m}}G_{\mathrm{fm}}} &= \frac{1}{N}\boldsymbol{R}^{(\mathrm{f})} + \frac{N-1}{N}\frac{(1/2)k_{\mathrm{f}}^2 G_{\mathrm{ff}} + (1/2)k_{\mathrm{m}}k_{\mathrm{f}}^2 G_{\mathrm{fm}}}{(1/2)(1+F)/2 + (1/2)k_{\mathrm{m}}G_{\mathrm{fm}}} \\ &= \begin{cases} 2(3N-1)/N(3N+1) & \text{LRC} \\ 1/N & \text{LMC} \end{cases}.\end{aligned}$$

The coefficient $\boldsymbol{R}^{(\mathrm{f+m})}$ is the relatedness of a mother (actor) to her own mated pair (recipient), measured from the perspective of the female offspring produced by the mated pair itself. We find

$$\begin{aligned}\boldsymbol{R}^{(\mathrm{f+m})} &= \frac{1}{2}(1) + \frac{1}{2}\frac{1 + k_{\mathrm{m}}G_{\mathrm{fm}}}{(1+F)/2 + k_{\mathrm{m}}G_{\mathrm{fm}}} = \begin{cases} 3/2 & \text{LRC} \\ (3N-1)/2N & \text{LMC} \end{cases}, \\ \overline{\boldsymbol{R}}^{(\mathrm{f+m})} &= \frac{(1/4)H_{\mathrm{ff}} + (1/2)H_{\mathrm{fm}} + (1/4)H_{\mathrm{mm}}}{(1/2)(1+F)/2 + (1/2)k_{\mathrm{m}}G_{\mathrm{fm}}},\end{aligned}$$

which is $H_{\mathrm{ff}} + H_{\mathrm{mm}} = 6/(3N+1)$ under LRC.

When we allow imprinting, we must normalize our boldfaced relatedness coefficients using

$$\begin{cases} F = k_{\mathrm{m}}G_{\mathrm{fm}} & \text{if } Z = \mathrm{m} \\ (1+F)/2 & \text{if } Z = \mathrm{f} \end{cases},$$

the CC between the Z-line gene in the offspring ("individual" with control) and the mother (actor).

A technical note before proceeding: any time there is no inbreeding, that is, $F = 0$, we can clearly see that the paternally derived allele that resides in a female offspring has no interest in any of the male offspring born on the same patch. It follows that a paternally derived allele in models such as LRC will favor zero investment in sons. We do not need to calculate relatedness coefficients to see that this claim holds, and so we proceed under the assumption that either $F \neq 0$ or (when $F = 0$) $Z \neq \mathrm{m}$.

Applying our definition of relatedness, we have

$$\begin{aligned}\boldsymbol{R}_Z^{(\mathrm{f})} &= 1 \text{ LRC, LMC, } Z = \mathrm{m}, \mathrm{f}, \\ \overline{\boldsymbol{R}}_Z^{(f)} &= \begin{cases} H_{\mathrm{fm}}/F & Z = \mathrm{m} \\ H_{\mathrm{ff}}/[(1+F)/2] & Z = \mathrm{f} \end{cases}, \\ \boldsymbol{R}_Z^{(\mathrm{f+m})} &= \begin{cases} [(1/2)F + (1/2)(1)]/F & Z = \mathrm{m} \\ [(1/2)(1+F)/2 + (1/2)F]/[(1+F)/2] & Z = \mathrm{f} \end{cases}, \\ \overline{\boldsymbol{R}}_Z^{(\mathrm{f+m})} &= \begin{cases} [(1/2)H_{\mathrm{fm}} + (1/2)H_{\mathrm{mm}}]/F & Z = \mathrm{m} \\ [H_{\mathrm{ff}} + H_{\mathrm{fm}}]/(1+F) & Z = \mathrm{f} \end{cases}.\end{aligned}$$

or, more specifically,

$$\overline{\boldsymbol{R}}_Z^{(\mathrm{f})} = \begin{cases} 2(3N-1)/N(3N+1) & \text{LRC, } Z = \mathrm{f} \\ 1/N & \text{LMC, } Z = \mathrm{m, f} \end{cases},$$

$$\boldsymbol{R}_Z^{(\mathrm{f+m})} = \begin{cases} 1/2 & \text{LRC, } Z = \mathrm{f} \\ 2N-1 & \text{LMC, } Z = \mathrm{m} \\ N/(2N-1) & \text{LMC, } Z = \mathrm{f} \end{cases},$$

$$\overline{\boldsymbol{R}}_{\mathrm{f}}^{(\mathrm{f+m})} = \frac{3N-1}{N(3N+1)} \text{ LRC.}$$

Polyandry

With polyandry, the only recursive equation that changes is

$$H_{\mathrm{mm},n+1} = \frac{1}{N}\left(\frac{1}{M} + \frac{M-1}{M}k_{\mathrm{m}}^2 H_{\mathrm{ff},n}\right) + \frac{N-1}{N}k_{\mathrm{m}}^2 k_{\mathrm{f}} H_{\mathrm{ff},n}.$$

Under LRC, we find

$$H_{\mathrm{ff}} = \frac{2MN+N-1}{MN(3N+1)},$$

$$H_{\mathrm{mm}} = \frac{1}{MN},$$

$$H_{\mathrm{fm}} = 0,$$

and under LMC,

$$F = \frac{1}{4N-3},$$

$$H_{\mathrm{mm}} = \frac{1}{MN}.$$

Relatedness coefficients used in maternal control "no imprinting" models include

$$R^{(\mathrm{f})} = 1 \text{ LRC, LMC,}$$

$$R^{(\mathrm{f+m})} = \begin{cases} 1/2 & \text{LRC} \\ N/(2N-1) & \text{LMC} \end{cases},$$

$$\overline{R}^{(f)} = \frac{1}{N} \text{ LMC,}$$

$$\overline{R}^{(\mathrm{f+m})} = \frac{2MN+N-1}{MN(3N+1)} \text{ LRC.}$$

With imprinting, we use

$$R_Z^{(\mathrm{f})} = 1,$$

$$\overline{R}_Z^{(\mathrm{f})} = \frac{1}{N} \text{ LMC, } Z = \mathrm{m}, \mathrm{f},$$

$$R_Z^{(\mathrm{f+m})} = \begin{cases} 1/2 & \text{LRC} \\ (2N^2 - N + 1)/N(2N-1) & \text{LMC, } Z = \mathrm{m} \\ (N+1)/2N & \text{LMC, } Z = \mathrm{f} \end{cases},$$

$$\overline{R}_Z^{(\mathrm{f+m})} = \begin{cases} (MN + N - 1)/2MN^2 & \text{LRC, } Z = \mathrm{m} \\ [MN(5N-1) + (N+1)^2]/2MN^2(3N+1) & \text{LRC, } Z = \mathrm{f} \end{cases}.$$

For offspring control,

$$\boldsymbol{R}^{(\mathrm{f})} = 1 \text{ LMC, LRC,}$$

$$\overline{\boldsymbol{R}}^{(\mathrm{f})} = \frac{1}{N} \text{ LMC,}$$

$$\boldsymbol{R}^{(\mathrm{f+m})} = \frac{1}{2}(1) + \frac{1}{2}\frac{1/M + [(M-1)/M]k_{\mathrm{m}}^2 G_{\mathrm{mm}} + k_{\mathrm{m}} G_{\mathrm{fm}}}{(1+F)/2 + k_{\mathrm{m}} G_{\mathrm{fm}}}$$

$$= \begin{cases} (M+2)/2M & \text{LRC} \\ [M(2N^2 + 3N - 1) + (4N-1)(N-1)]/4MN^2 & \text{LMC} \end{cases},$$

$$\overline{\boldsymbol{R}}^{(\mathrm{f+m})} = \frac{2(M+2)}{M(3N+1)} \text{ LRC.}$$

With imprinting,

$$\boldsymbol{R}_Z^{(\mathrm{f})} = 1 \text{ LMC, LRC, } Z = \mathrm{m}, \mathrm{f},$$

$$\overline{\boldsymbol{R}}_Z^{(\mathrm{f})} = \frac{1}{N} \text{ LMC,}$$

$$\boldsymbol{R}_Z^{(\mathrm{f+m})} = \begin{cases} \{(1/2)F + (1/2)[1/M + k_{\mathrm{m}}^2 G_{\mathrm{mm}}(M-1)/M]\}/F & Z = \mathrm{m} \ (F \neq 0) \\ (1+3F)/2(1+F) & Z = \mathrm{f} \end{cases},$$

or, more simply,

$$\boldsymbol{R}_Z^{(\mathrm{f+m})} = \begin{cases} 1/2 & \text{LRC, } Z = \mathrm{f} \\ [M(3N-1) + (4N-1)(N-1)]/2MN & \text{LMC, } Z = \mathrm{m} \\ N/(2N-1) & \text{LMC, } Z = \mathrm{f} \end{cases}.$$

$$\overline{R}_Z^{(\mathrm{f+m})} = \frac{(2M+1)N - 1}{MN(3N+1)} \text{ LRC, } Z = \mathrm{f}.$$

Polygyny

For the model of polygyny, we see that the only change to the recursive equations is

$$H_{\mathrm{mm},n+1} \equiv \frac{1}{N} + \frac{N-1}{N} k_{\mathrm{f}}^2.$$

Under LMC, $k_{\mathrm{f}} = 0$, and so $H_{\mathrm{mm}} \equiv 1/N$, the same value it takes in the case of female monogamy. It follows that none of the CCs in a polygynous system differ from those calculated under female monogamy. We report relatedness coefficients only for LRC models.

Under LRC, we find

$$H_{\mathrm{ff}} = \frac{N+1}{3N+1},$$

$$H_{\mathrm{mm}} = 1,$$

$$H_{\mathrm{fm}} = 0.$$

In maternal control models, $R^{(\mathrm{f})} = 1$, $R^{(\mathrm{f+m})} = 1/2$, and $\overline{R}^{(\mathrm{f+m})} = H_{\mathrm{ff}}$ if there is no imprinting. When we allow imprinting, $R_Z^{(\mathrm{f})} = 1$, $R_Z^{(\mathrm{f+m})} = 1/2$, and

$$\overline{R}_Z^{(\mathrm{f+m})} = \begin{cases} 1/2 & Z = \mathrm{m} \\ (N+3)/2(3N+1) & Z = \mathrm{f} \end{cases}.$$

In offspring control models without imprinting, we use $\boldsymbol{R}^{(\mathrm{f})} = 1$, $\boldsymbol{R}^{(\mathrm{f+m})} = 3/2$, and $\overline{\boldsymbol{R}}^{(\mathrm{f+m})} = 2(2N+1)/(3N+1)$.

The paternally inherited allele in this model has no interest in the production of male offspring. A paternally imprinted allele, then, always favors decreased investment in sons. To determine the equilibrium from the perspective of a maternally imprinted allele, we use $\boldsymbol{R}_{\mathrm{f}}^{(\mathrm{f})} = 1$, $\boldsymbol{R}_{\mathrm{f}}^{(\mathrm{f+m})} = 1/2$, and $\overline{\boldsymbol{R}}_{\mathrm{f}}^{(\mathrm{f+m})} = H_{\mathrm{ff}}$.

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Appendix E from G. Wild and S. A. West, "Genomic Imprinting and Sex Allocation"

(Am. Nat., vol. 173, no. 1, p. E1)

Evolutionary Dynamics and Conflict Resolution

The sex ratio conflict between two imprinted alleles is resolved through the action of selection. Recall that the sign of $\Delta W_Z(z_m, z_f)$ determines whether z_Z is increasing (if $\Delta W_Z > 0$) or decreasing (if $\Delta W_Z < 0$). If we assume that z_m and z_f change independently, then, together, the pair ΔW_m and ΔW_f determines the direction in which the population moves through z_m, z_f trait space (the unit square). Following Greenwood-Lee et al. (2001) we have

$$\begin{bmatrix} dz_m/dt \\ dz_f/dt \end{bmatrix} \propto \begin{bmatrix} \Delta W_m(z_m, z_f) \\ \Delta W_f(z_m, z_f) \end{bmatrix}, \tag{E1}$$

where t measures time on a scale much longer than one generation (i.e., t denotes "evolutionary time"). If $(z_m(t), z_f(t))$ is a solution to expression (E1), then we will consider (z_m^*, z_f^*) to be a stable pair whenever $(z_m(t), z_f(t)) \to (z_m^*, z_f^*)$ as $t \to \infty$.

The set of points (z_m, z_f) that satisfies $\Delta W_Z = 0$ is called a selection nullcline because the set itself corresponds to a collection of population "states" at equilibrium with respect to the evolution of z_Z. In our models, we always find that (*a*) selection balances z_Z on its nullcline, when such a nullcline exists; (*b*) nullclines ΔW_m and ΔW_f never intersect; and (*c*) nullclines ΔW_m and ΔW_f run parallel to and never occur on opposite sides of the antidiagonal line $(z_m + z_f = 1)$.

In reverse order, *c* tells us that maternally and paternally inherited alleles both prefer to bias the realized sex ratio in same direction, *b* tells us that the action of selection never leads to a "compromise" state in the interior of the trait space, and *a* in combination with *b* and *c* tells us that the selection promotes the perspective of the allele that prefers the more moderately biased realized sex ratio (e.g., fig. E1). It is important to note that the implications of *a*, *b*, and *c* depend on the assumption of the realized sex ratio, $z = (z_m + z_f)/2$, and so the conclusions we draw may not hold in general.

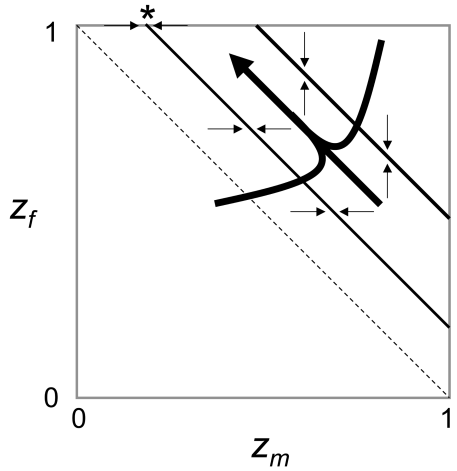

Figure E1: Sample evolutionary trajectories in z_m, z_f trait space. We find two selection nullclines (*solid lines*) that run parallel to the antidiagonal (*dashed line*). In our models, nullclines never occur on opposite sides of the antidiagonal. In this figure, they both occur above the antidiagonal, indicating that both imprinted alleles favor a

male-biased sex ratio. Selection always balances the population-average phenotype on the appropriate nullcline (*small arrows*). The net effect of this balancing selection (*large arrow*) is an evolutionary force that pushes the population to the boundary of trait space, where it ultimately settles on the less extreme nullcline, that is, the nullcline that lies closer to the antidiagonal (*asterisk*).