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Evolution of maternal care in diploid and haplodiploid populations

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Abstract

Maternal care has been suggested to evolve more readily in haplodiploid populations. Because maternal care appears to have been a prerequisite for the evolution of eusociality, this effect potentially explains the apparent preponderance of haplodiploidy among eusocial taxa. Here, I use a kin selection approach to model the evolution of maternal care in diploid and haplodiploid populations. In contrast to previous suggestions, I find that haplodiploidy may inhibit as well as promote the evolution of maternal care. Moreover, I find that the haplodiploidy effect vanishes in outbred populations if gene effects average rather than add together. I confirm these analytical results using numerical simulation of an explicit population genetics model. This analysis casts doubt upon the idea that haplodiploidy has promoted the evolution of maternal care and, consequently, the evolution of eusociality.

Introduction

Hamilton (1964, 1972) suggested that the inflated genetic relatedness of sisters in haplodiploid populations has predisposed such taxa to eusociality. This 'haplodiploidy hypothesis' has gradually fallen out of favour, for both empirical and theoretical reasons (reviewed by Gardner *et al.*, 2012). However, some authors have suggested that haplodiploidy may favour the evolution of eusociality for reasons other than its impact on coefficients of relatedness. For example, haplodiploidy may reduce the stochastic loss of overdominant helping genes (Reeve, 1993; Reeve & Shellman-Reeve, 1997) and may facilitate synergistic interactions between helping genes in models of strong selection (Fromhage & Kokko, 2011).

A less direct impact of haplodiploidy on the evolution of eusociality has been suggested by Wade (2001). His idea is that haplodiploidy promotes the evolution of maternal care, which has long been understood to be a prerequisite for eusociality (e.g. Wheeler, 1928). Specifi cally, Wade (2001) analysed a model of selection acting upon a maternal care gene that incurs a direct cost for its carriers and bestows an indirect benefit upon the offspring of its carriers. He considered that the direct cost of the maternal care gene may accrue to both sexes equally or to males only, and he assumed a strictly additive model of gene effects. Wade (2001) concluded that the condition for natural selection to favour such a gene is less stringent under haplodiploidy than under diploidy, and this idea appears to be widely accepted (Wade & Shuster, 2002; Wolf *et al.*, 2002; Thorne & Traniello, 2003; Linksvayer & Wade, 2005; Crozier, 2008; Cruickshank & Wade, 2008).

However, Wade's (2001) analysis neglected the possibility that the direct cost of maternal care accrues only to females. Because maternal care is expressed only by females, one might expect them to bear its cost. The consequences of female-specific costs may be particularly important, owing to selection among females having a relatively greater impact on evolutionary change in haplodiploid populations (Price, 1970). Moreover, Wade's (2001) analysis did not consider the full impact of inbreeding on genotype frequencies, some of which he approximated by their Hardy–Weinberg proportions (e.g. Wade, 2001, p454). Finally, Wade (2001) assumed that gene effects add together, such that the magnitude of a gene's contribution to the phenotype is independent of the number of genes contributing to the phenotype. Alternatively, gene effects may average together, such that the magnitude of a gene's contribution to the phenotype decreases as the number of genes contributing to the phenotype increases (Frank, 2003). This distinction could have important consequences for evolution in haplodiploid populations, in which a female's genetic complement is twice that of a male's.

Here, I use a kin selection approach (Hamilton, 1964; Taylor & Frank, 1996) to model the evolution of

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maternal care in diploid and haplodiploid populations. I extend Wade's (2001) analysis in three ways: (i) to consider the scenario in which the direct cost of maternal care accrues to females only; (ii) to consider the perturbing influence of inbreeding on genotype frequencies; and (iii) to allow for the possibility that gene effects average rather than add together. I derive analytical conditions for natural selection to favour the evolution of maternal care, and I confirm these results using numerical simulation of an explicit population genetics model. More generally, my primary aim is not to challenge Wade's (2001) results, but rather to investigate their robustness to changes in modelling assumptions, to better assess the likelihood that haplodiploidy has promoted maternal care and, consequently, the evolution of eusociality.

Model and results

Kin selection model

I assume an infinite diploid or haplodiploid population of females and males. I consider two traits, 'maternal care' and 'viability', that are controlled by the same genes. Specifically, the genes associated with enhanced maternal care, when the focal individual is an adult female, are also associated with reduced viability, in one or both of the sexes. The analysis applies more generally to any maternal effect genes that incur direct pleiotropic costs for their carriers, but I refer to these specific traits for concreteness and clarity. Thus, the fitness of a female is $w_f(G,G')$, where G is her genetic value for the compound trait and G' is her mother's genetic value, and where $C_f = -\partial w_f/\partial G$ is the viability cost for females and $B_f = \partial w_f/\partial G'$ is maternal care benefit for females. The fitness of a male is $w_m(G,G')$, where G is his genetic value and G' is his mother's genetic value, and where $C_m = -\partial w_m/\partial G$ is the viability cost for males and $B_m = \partial w_m/\partial G'$ is the maternal care benefit for males. Picking a gene at random from the individual, I denote its genic value by g, and picking a gene at random from the individual's mother, I denote its genic value by g'. I assume that an individual's genetic value is determined by his or her genic value(s), either by adding or by averaging. If the genetic value is determined by adding, then $G = g_1 + g_2$ for a diploid individual, where g_1 and g_2 are the individual's two genic values, and if the genetic value is determined by averaging, then $G = (g_1 + g_2)/2$ for a diploid individual. The genetic value of a haploid individual is always $G = g$, irrespective of adding or averaging. A more general treatment of the distinction between adding and averaging of gene effects is given in the Appendix.

Adding gene effects

Wade (2001) assumed that gene effects add together to give the phenotype. That is, if an individual carries one copy of the focal gene and one copy of a null allele (i.e. a diploid heterozygote) or one copy of the focal gene and zero copies of the null allele (i.e. a haploid individual), then the impact on the phenotype is x. If the individual carries two copies of the focal gene and zero copies of the null allele (i.e. a diploid homozygote), then the impact on the phenotype is $2x$. Following this assumption, I find that natural selection favours maternal care under diploidy when

$$-C_f(1+f) + B_f\frac{1+3f}{2} - C_m(1+f) + B_m\frac{1+3f}{2} > 0 \quad (1)$$

where $f = dg_2/dg_1$ is the coefficient of inbreeding (see Appendix for derivation). Assuming no sex difference in the viability cost ($C_f = C_m = C$) or the maternal care benefit ($B_f = B_m = B$), inequality (1) rearranges as $C/B < (1 + 3f)/(2 + 2f)$. That is, in the absence of inbreeding ($f = 0$), maternal care is favoured provided the cost is less than one half of the benefit ($C/B < ½$). A higher cost is tolerated if there is inbreeding, and in the extreme of full inbreeding ($f = 1$), maternal care is favoured provided that the cost is less than the benefit ($C/B < 1$). If only individuals of one sex experience the viability cost (i.e. $C_f = 0$ and $C_m = C$, or $C_f = C$ and $C_m = 0$) and there is no sex difference in the maternal care benefit ($B_f = B_m = B$), then the condition becomes $C/B < (1 + 3f)/(1 + f)$, such that a cost that is as great as the benefit is tolerated in the absence of inbreeding ($C/B < 1$, when $f = 0$) and a cost that is twice the benefit is tolerated in the extreme of full inbreeding ($C/B < 2$, when $f = 1$). These analytical results are summarized in Table 1 and illustrated in panels (a–c) of Fig. 1. Numerical simulation of an explicit population genetics model confirms these analytical results (see Appendix for details). The simulation data are also presented in panels (a–c) of Fig. 1.

Under haplodiploidy, the condition for natural selection to favour maternal care genes is

$$-2C_f(1+f) + B_f(1+3f) - C_m + B_m(1+f) > 0 \quad (2)$$

(see Appendix for derivation). Assuming no sex difference in the viability cost ($C_f = C_m = C$) or the maternal

Table 1 Potential for maternal care (A, such that the condition for natural selection to favour maternal care is $C/B < A$) as a function of ploidy, sex-specific effects and the coefficient of inbreeding (f).

	Potential for maternal care		
Genetic system	Direct cost accrues to both sexes	Direct cost accrues to males only	Direct cost accrues to females only
Diploidy			
Gene effects add	$\frac{1+3f}{2+2f}$	$\frac{1+3f}{1+f}$	$\frac{1+3f}{1+f}$
Gene effects average	$\frac{1+3f}{2+2f}$	$\frac{1+3f}{1+f}$	$\frac{1+3f}{1+f}$
Haplodiploidy			
Gene effects add	$\frac{2+4f}{3+2f}$	$2 + 4f$	$\frac{1+2f}{1+f}$
Gene effects average	$\frac{1+2f}{2+f}$	$1 + 2f$	$\frac{1+2f}{1+f}$

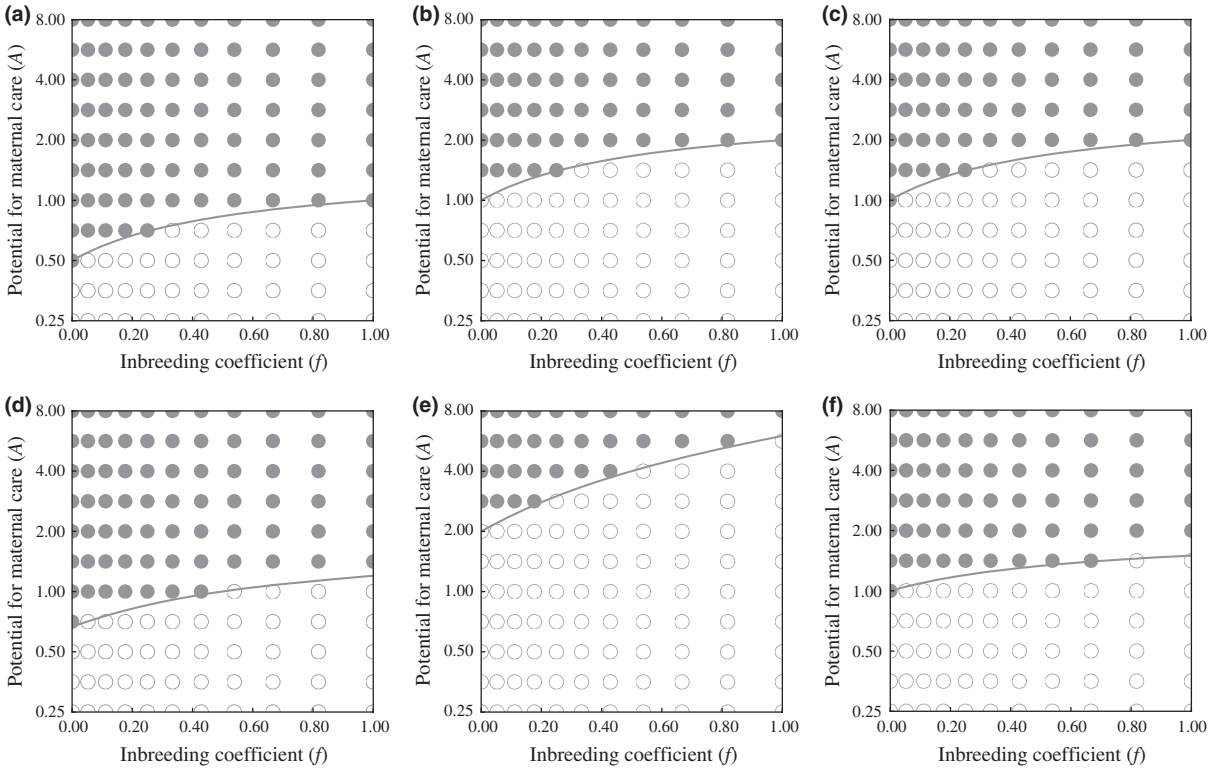

Fig. 1 Potential for maternal care under the adding model of gene effects. Analytical solutions for the potential for maternal care (A, such that the condition for natural selection to favour maternal care is $C/B < A$): for diploidy, with costs experienced by both sexes equally [panel (a), $B_f = B_m = B$, $C_f = C_m = C$], males only [panel (b), $B_f = B_m = B$, $C_f = 0$, $C_m = C$] or females only [panel (c), $B_f = B_m = B$, $C_f = C$, $C_m = 0$]; and for haplodiploidy, with costs experienced by both sexes equally [panel (d), $B_f = B_m = B$, $C_f = C_m = C$], males only [panel (e), $B_f = B_m = B$, $C_f = 0$, $C_m = C$] or females only [panel (f), $B_f = B_m = B$, $C_f = C$, $C_m = 0$]. Overlaid are numerical simulation results, indicating invasion (white disc) or noninvasion (grey disc) of a maternal care allele from rarity.

care benefit ($B_f = B_m = B$), inequality (2) rearranges as $C/B < (2 + 4f)/(3 + 2f)$. That is, in the absence of inbreeding ($f = 0$), maternal care is favoured provided the cost is less than two-thirds of the benefit ($C/B < 2/3$). An even higher cost is tolerated if there is inbreeding, and in the extreme of full inbreeding ($f = 1$), this cost may exceed the benefit by a factor of six to five ($C/B < 6/5$). If only males experience the viability cost (i.e. $C_f = 0$ and $C_m = C$) and there is no sex difference in the benefit of maternal care ($B_f = B_m = B$), then the condition becomes $C/B < 2 + 4f$, such that a cost that is twice the benefit is tolerated in the absence of inbreeding ($C/B < 2$, when $f = 0$) and a cost that is six times the benefit is tolerated in the extreme of full inbreeding ($C/B < 6$, when $f = 1$). If only females experience the viability cost (i.e. $C_f = C$ and $C_m = 0$) and there is no sex difference in the maternal care benefit ($B_f = B_m = B$), then the condition becomes $C/B < (1 + 2f)/(1 + f)$, such that a cost that is as great as the benefit is tolerated in the absence of inbreeding ($C/B < 1$, when $f = 0$) and a cost that exceeds the benefit by a factor of three to two is tolerated in the extreme of full inbreeding ($C/B < 3/2$, when $f = 1$). These results are summarized in Table 1 and illustrated in panels (d–f) of Fig. 1, along with supporting numerical simulation data (see Appendix for details).

The conditions for maternal care to be favoured by natural selection are of the form $C/B < A$, where A defines the 'potential for maternal care' (cf Gardner, 2010). Assuming that gene effects add together, the potential for maternal care under haplodiploidy is always greater than that under diploidy when the cost of maternal care accrues to both females and males [contrast Fig. 1 panels (a) and (d)] or to males only [contrast Fig. 1 panels (b) and (e)]. However, the potential for maternal care under haplodiploidy is less than (or, for $f = 0$, equal to) that under diploidy when the cost of maternal care accrues to females only [contrast Fig. 1 panels (c) and (f)].

Averaging gene effects

In the previous section, I followed Wade (2001) in assuming that gene effects add together to give the individual's phenotype. Now, I consider that gene effects average together. That is, if an individual carries one copy

of the focal gene and one copy of a null allele (i.e. a diploid heterozygote), then the impact on the phenotype is $x/2$. And if the individual carries two copies of the focal gene and zero copies of the null allele (i.e. a diploid homozygote) or one copy of the focal gene and zero copies of the null allele (i.e. a haploid individual), then the impact on the phenotype is x. Following this assumption, I find that natural selection favours maternal care genes under diploidy when

$$-C_{\mathrm{f}}\frac{1+f}{2}+B_{\mathrm{f}}\frac{1+3f}{4}-C_{\mathrm{m}}\frac{1+f}{2}+B_{\mathrm{m}}\frac{1+3f}{4}>0 \qquad (3)$$

(see Appendix for derivation). Note that this condition is exactly equivalent to that for diploidy under the adding model of genetic values – that is, inequality (1) – and hence, all of the results for diploids are the same irrespective of whether gene effects add or average. This is because the distinction between adding vs. averaging simply rescales the effect of every gene in a diploid population by a constant amount. These results are summarized in Table 1, and illustrated in panels (a–c) of Fig. 2, along with supporting numerical simulation data (see Appendix for details).

Under haplodiploidy, the condition for natural selection to favour maternal care genes in the averaging model is

$$-C_{\mathrm{f}}(1+f)+B_{\mathrm{f}}\frac{1+3f}{2}-C_{\mathrm{m}}+B_{\mathrm{m}}\frac{1+f}{2}>0 \qquad (4)$$

(see Appendix for derivation). This is different from that derived for haplodiploidy under the adding model of genetic values – that is, inequality (2) – and hence, the results for haplodiploids need not be the same under adding or averaging of gene effects. This is because the distinction between adding and averaging rescales the fitness effects of genes in diploid individuals but not in haploid individuals and hence mediates the relative strengths of selection in each sex.

Assuming no sex difference in the viability cost ($C_{\mathrm{f}} = C_{\mathrm{m}} = C$) or the maternal care benefit ($B_{\mathrm{f}} = B_{\mathrm{m}} = B$), inequality (4) rearranges as $C/B < (1 + 2f)/(2 + f)$. That is, in the absence of inbreeding ($f = 0$), maternal care is favoured provided the cost is less than one half of the benefit ($C/B < 1/2$). An even higher cost is tolerated if there is inbreeding, and in the extreme of full inbreeding ($f = 1$), maternal care is favoured provided that the cost is

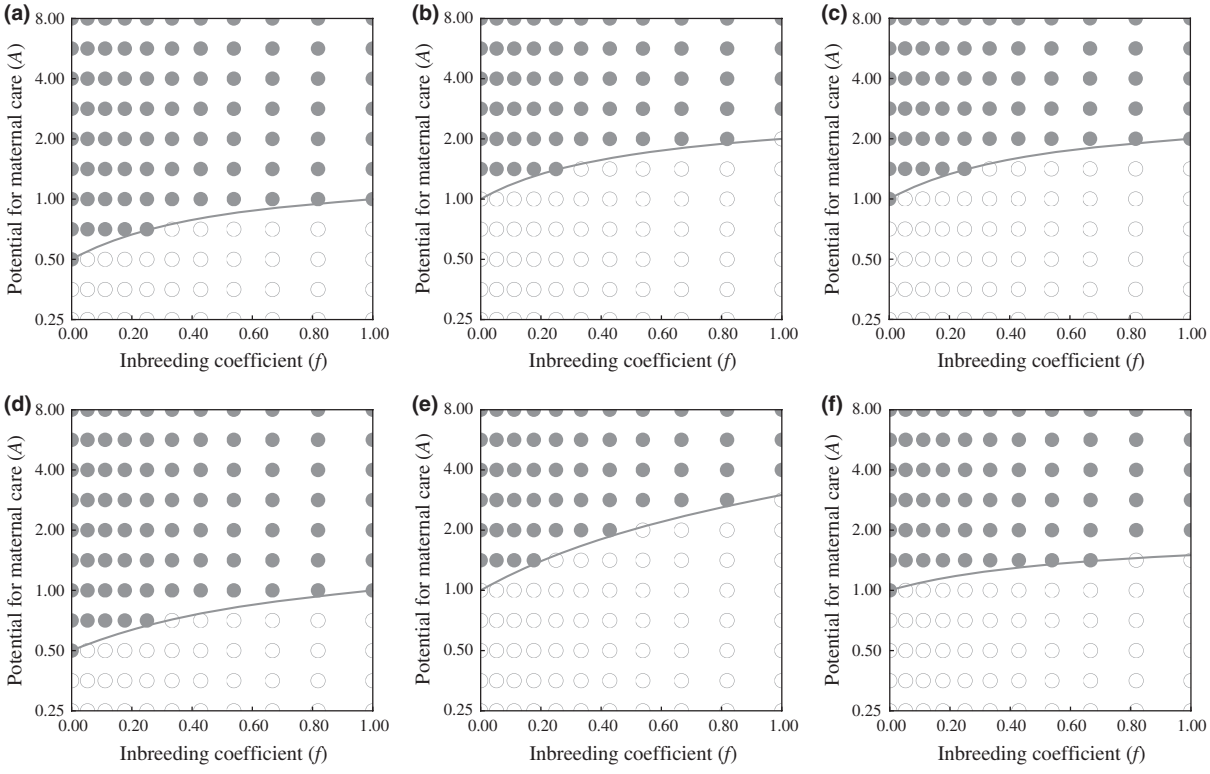

Fig. 2 The potential for maternal care under the averaging model of gene effects. Analytical solutions for the potential for maternal care (A, such that the condition for natural selection to favour maternal care is $C/B < A$): for diploidy, with costs experienced by both sexes equally [panel (a), $B_{\mathrm{f}} = B_{\mathrm{m}} = B$, $C_{\mathrm{f}} = C_{\mathrm{m}} = C$], males only [panel (b), $B_{\mathrm{f}} = B_{\mathrm{m}} = B$, $C_{\mathrm{f}} = 0$, $C_{\mathrm{m}} = C$] or females only [panel (c), $B_{\mathrm{f}} = B_{\mathrm{m}} = B$, $C_{\mathrm{f}} = C$, $C_{\mathrm{m}} = 0$]; and for haplodiploidy, with costs experienced by both sexes equally [panel (d), $B_{\mathrm{f}} = B_{\mathrm{m}} = B$, $C_{\mathrm{f}} = C_{\mathrm{m}} = C$], males only [panel (e), $B_{\mathrm{f}} = B_{\mathrm{m}} = B$, $C_{\mathrm{f}} = 0$, $C_{\mathrm{m}} = C$] or females only [panel (f), $B_{\mathrm{f}} = B_{\mathrm{m}} = B$, $C_{\mathrm{f}} = C$, $C_{\mathrm{m}} = 0$]. Overlaid are numerical simulation results, indicating invasion (white disc) or noninvasion (grey disc) of a maternal care allele from rarity.

less than the benefit ($C/B < 1$). If only males experience the viability cost (i.e. $C_f = 0$ and $C_m = C$) and there is no sex difference in the maternal care benefit ($B_f = B_m = B$), then the condition becomes $C/B < 1 + 2f$, such that a cost that is equal to the benefit is tolerated in the absence of inbreeding ($C/B < 1$, when $f = 0$) and a cost that is three times the benefit is tolerated in the extreme of full inbreeding ($C/B < 3$, when $f = 1$). If only females experience the viability cost (i.e. $C_f = C$ and $C_m = 0$) and there is no sex difference in the maternal care benefit ($B_f = B_m = B$), then the condition becomes $C/B < (1 + 2f)/(1 + f)$, such that a cost that is equal to the benefit is tolerated in the absence of inbreeding ($C/B < 1$, when $f = 0$) and a cost that exceeds the benefit by a factor of three to two is tolerated in the extreme of full inbreeding ($C/B < 3/2$, when $f = 1$). These results are summarized in Table 1 and illustrated in panels (d–f) of Fig. 2, along with supporting numerical simulation data (see Appendix for details).

Upon the assumption that gene effects average together, the potential for maternal care under haplodiploidy is less than (or, for $f = 0$ or 1, equal to) that under diploidy when the cost of maternal care accrues to both females and males [contrast Fig. 2 panels (a) and (d)]. The potential for maternal care under haplodiploidy is greater than (or, for $f = 0$, equal to) that under diploidy when the cost of maternal care accrues to males only [contrast Fig. 2 panels (b) and (e)]. And the potential for maternal care under haplodiploidy is less than (or, for $f = 0$, equal to) that under diploidy when the cost of maternal care accrues to females only [contrast Fig. 2 panels (c) and (f)].

Discussion

Wade (2001) suggested that haplodiploidy promotes the evolution of maternal care, potentially explaining the apparent preponderance of haplodiploidy among eusocial taxa. On the basis of Wade's (2001) analysis, this idea has subsequently been repeated elsewhere in the literature (Wade & Shuster, 2002; Wolf *et al.*, 2002; Thorne & Traniello, 2003; Linksvayer & Wade, 2005; Crozier, 2008; Cruickshank & Wade, 2008). However, Wade's (2001) analysis made particular assumptions about which sex bears the cost of maternal care and how gene effects combine to determine phenotype and fitness. I have extended Wade's (2001) analysis to consider the possibility that the direct cost of maternal care accrues only to females, rather than to both sexes or to males only. I have also considered that gene effects may average, rather than add, together. Using kin selection methodology (Hamilton, 1964; Taylor & Frank, 1996), I have derived analytical conditions for when natural selection favours the evolution of maternal care, and I have confirmed these analytical results using numerical simulation of an explicit population genetics model. I have found that, contrary to the previous suggestions, haplodiploidy may inhibit as well as promote the evolution of maternal care and, in many cases, is expected to have no impact at all.

Whereas Wade (2001) considered that the direct cost of maternal care accrues either to both sexes equally or to males only, I have allowed for the possibility that the direct cost of maternal care accrues to females only. Under diploidy, the total reproductive values of both sexes are equal (Fisher, 1930), such that the condition for maternal care to be favoured is the same irrespective of which sex pays the direct cost [e.g. compare Fig. 1 panels (b) and (c) and compare Fig. 2 panels (b) and (c)]. In contrast, under haplodiploidy, the total reproductive value of females is greater than the total reproductive value of males, such that selection among females has a greater impact on gene frequency change than does selection among males (Price, 1970; Taylor, 1990, 1996; Grafen, 2006). This means that the effective cost of maternal care is greater if it accrues to females rather than to males [e.g. contrast Fig. 1 panels (e) and (f) and contrast Fig. 2 panels (e) and (f)]. Consequently, if the direct cost accrues to females only, then maternal care is relatively promoted by diploidy and is relatively inhibited by haplodiploidy [e.g. contrast Fig. 1 panels (c) and (f) and contrast Fig. 2 panels (c) and (f)].

Wade (2001) also suggested that inbreeding enhances the haplodiploidy effect, because it increases the potential for maternal care under haplodiploidy but has no impact on the potential for maternal care under diploidy. In contrast, I have found that inbreeding increases the potential for maternal care under both diploid and haplodiploid modes of inheritance. This disagreement owes to Wade (2001) having not fully captured the effects of inbreeding in his equations of gene frequency change. In particular, Wade (2001, p454) approximated the population frequency of a heterozygous genotype according to its Hardy–Weinberg proportion ($H = 2pq$, and hence $H/4 = pq/2$, in Wade's notation), even in the context of inbreeding, which is well known to disturb genotype frequencies from their Hardy–Weinberg proportions. Consequently, although the results of my adding model of gene effects agree quantitatively with the corresponding results of Wade's (2001) analysis for the special case of outbreeding ($f = 0$), they disagree in the context of inbreeding ($f > 0$). However, the impact of inbreeding may be inconsequential with regard to the issue of whether any maternal-care-promoting effects of haplodiploidy have paved the way for eusociality, because eusociality most likely evolved in the context of outbred populations (Boomsma, 2007, 2009; West, 2009).

Focusing on outbred populations ($f = 0$) and adopting Wade's (2001) assumption that gene effects add together, I have found that haplodiploidy can mediate the evolution of maternal care, sometimes promoting and sometimes having an inhibitory effect. However, if gene effects average together in determining the individual's phenotype, then the haplodiploidy effect

vanishes altogether. This is because, while the response to selection is proportional to reproductive value and hence is twice as strong in females as in males under haplodiploidy, the phenotypic effect of a gene is twice as strong in a haploid male than in a diploid female, and hence, there is no net effect of ploidy. All else being equal, adding appears to be more realistic if phenotypes depend upon the absolute amount of a gene's product, whereas averaging appears to be more realistic if phenotypes depend upon the concentration of the gene's product. Factors such as dosage compensation (Ohno, 1969; Mank, 2009) may complicate this picture; however, the importance of dosage compensation among haplodiploids is unclear. Frank (2003) provides more discussion of the distinction between adding vs. averaging of gene effects, with a focus on variation in number of loci rather than variation in ploidy. This issue can only be resolved empirically, and the present analysis suggests that understanding gene effects in haplodiploids represents an important avenue for future empirical exploration.

In conclusion, this analysis has cast doubt upon the suggestion that haplodiploidy has promoted the evolution of maternal care and, consequently, the evolution of eusociality. Under the assumption that gene effects add together, I have shown that haplodiploidy may either promote or inhibit the evolution of maternal care. Under the assumption that gene effects average together, I have shown that the haplodiploidy effect may vanish altogether. More generally, I have performed this reanalysis using the standard tools of kin selection theory (Hamilton, 1964; Taylor & Frank, 1996), deriving Hamilton's (1963, 1964, 1970) rules that describe the action of selection in terms of three basic measures of value: costs/benefits, consanguinity (relatedness) and reproductive value (Frank, 1998). This clarifies that kin selection theory is well equipped to deal with the complexities of indirect genetic effects (Gardner *et al.*, 2011) and is able to reproduce the results of (often impenetrable) population genetics analyses.

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Appendix

Adding vs. averaging of gene effects

To clarify the distinction between the adding vs. averaging models of gene effects, it is helpful to expand beyond the basic scenario in which the phenotype of interest is determined by a single locus in a haploid or diploid individual, to consider a scenario in which the phenotype is determined by l loci in an individual with ploidy m. Thus, an individual's genetic value for the phenotype is given by $G = \sum_{i,j} g_{ij}$ under the adding model and by $G = \sum_{i,j} g_{ij}/n$ under the averaging model, where $i \in \{1, 2,\ldots, l\}$; $j \in \{1, 2,\ldots, m\}$; g_{ij} is the genic value of the gene at the ith locus in the individual's jth haploid genome; and $n = l \times m$ is the total number of gene positions contributing to the phenotype. This difference in scaling may be evolutionarily important if n varies between different individuals, that is, owing to different number of loci contributing to the phenotype (l; Frank, 2003) and/or different ploidy (m; this article).

Although the adding model of gene action is most familiar in the evolutionary literature, it implies that phenotypic trait values can be increased without bound simply by adding extra gene positions, which cannot be generally true owing to physical and mathematical limits. For example, the sex ratio (proportion males) cannot be increased above unity, no matter how many gene positions contribute to this phenotype. Hence, for many phenotypic traits, averaging of gene effects appears to be more natural.

Kin selection analysis

In the main text, I define female and male fitness as w_f and w_m, respectively. Writing the average female fitness and average male fitness as $\bar{w}_f$ and $\bar{w}_m$, respectively, the relative fitness of a female is $W_f = w_f/\bar{w}_f$ and the relative fitness of a male is $W_m = w_m/\bar{w}_m$. I focus on the scenario where the maternal care genes are vanishingly rare, and accordingly, I assume that $\bar{w}_f = \bar{w}_m$. Natural selection favours an increase in genetic value if $c_f\ dW_f/dg_f + c_m\ dW_m/dg_m > 0$ (Taylor, 1996; Taylor & Frank, 1996; Frank, 1997, 1998; Taylor *et al.*, 2007), where c_f and c_m are the class reproductive values of females and males, respectively (Fisher, 1930; Taylor, 1996; Grafen, 2006) and where I have added subscripts f or m to denote whether the gene is present in a female or male, respectively.

There are both direct and indirect impacts of a female's genic value on her fitness. The direct effect is due to her genic value g_f being associated with her genetic value G_f, which determines her viability, which impacts upon her fitness W_f. The indirect effect is due to her genic value g_f being associated with her mother's genetic value G'_f, which determines her mother's investment into maternal care, which impacts upon her fitness W_f. Hence, $dW_f/dg_f = [(\partial W_f/\partial G_f) \times (dG_f/dg_f)] + [(\partial W_f/G'_f) \times (dG'_f/dg_f)]$. Note that $\partial W_f/\partial G'_f = B_f/\bar{w}_f$ and $\partial W_f/\partial G_f = -C_f/\bar{w}_f$; dG_f/dg_f is equal to $2p_f$ under the adding model and p_f under the averaging model, where p_f is the consanguinity of a female to herself (Bulmer, 1994) and dG'_f/dg_f is equal to $2p_d$ under the adding model and p_d under the averaging model, where p_d is the consanguinity of mother and daughter. Similarly, the association between a male's genes and his fitness is $dW_m/dg_m = [(\partial W_m/\partial G_m) \times (dG_m/dg_m)] + [(\partial W_m/G'_m) \times (dG'_m/dg_m)]$, where $\partial W_m/\partial G'_m = B_m/\bar{w}_m$ and $\partial W_m/\partial G_m = -C_m/\bar{w}_m$; dG_m/dg_m is equal to $2p_m$ under the adding model for diploidy and p_m under the averaging model for diploidy and both adding and averaging models for haplodiploidy, where p_m is the consanguinity of a male to himself and dG'_m/dg_m is equal to $2p_s$ under the adding model and p_s under the averaging model, where p_s is the consanguinity of mother and son.

Note that $c_f = c_m = ½$ under diploidy and $c_f = 2/3$ and $c_m = 1/3$ under haplodiploidy (Price, 1970; Taylor, 1996); $p_f = (1 + f)/2$, where $f = dg_1/dg_2 = dg_2/dg_1$ is the coefficient of inbreeding; $p_d = (1/2)p_f + (1/2)f = (1 + 3f)/4$; $p_m = (1 + f)/2$ for diploid males and $p_m = 1$ for haploid males; and $p_s = (1 + 3f)/4$ for diploid males and $p_s = p_f = (1 + f)/2$ for haploid males. Making these substitutions into the condition $c_f\ dW_f/dg_f + c_m\ dW_m/dg_m > 0$ obtains expressions (1)–(4) of the main text.

Population genetics analysis

Diploidy

I assume an infinite diploid population with genetic variation at a single biallelic locus with two alleles: a maternal effect allele A and a null allele a. A proportion ϕ_x of females carry $x \in \{0, 1, 2\}$ copies of the A allele, and a proportion μ_u of males carry $u \in \{0, 1, 2\}$ copies of the A allele. I assume an equal number

of breeders of each sex. Mating pairs are constructed as follows: aa females are paired to aa males, to form $\min(\phi_0,\mu_0)$ aa/aa pairs and $\max(\phi_0,\mu_0) - \min(\phi_0,\mu_0)$ unpaired aa individuals of the rarer sex; Aa females and Aa males, and AA females and AA males, are paired in the same way; all unpaired individuals are paired at random; a proportion $1-\alpha$ of pairs are broken up at random, and these unpaired individuals are repaired with each other at random; the female and male in each pair then mate with each other. Thus, the frequency of x-female/u-male mating pairs is $\gamma_{xu} = \alpha \min(\phi_x,\mu_u) + (1-\alpha)\phi_x\mu_u$ for $x = u$ and $\gamma_{xu} = \alpha (\phi_x - \min(\phi_x,\mu_x))(\mu_u - \min(\phi_u,\mu_u))/(\sum_{z\in\{0,1,2\}}(\mu_z - \min(\phi_z,\mu_z))) + (1-\alpha)\phi_x\mu_u$ for $x \neq u$. In the special case of the genotype frequencies being the same in the two sexes, this gives $\gamma_{xu} = \phi_x(\alpha + (1-\alpha)\mu_u)$ for $x = u$ and $\gamma_{xu} = (1-\alpha)\phi_x\mu_u$ for $x \neq u$. Mated pairs each produce a large number K of offspring, with an even sex ratio. Each offspring's genotype is generated in the usual way, by choosing a random gene from their mother and a random gene from their father. Thus, the fraction of females who carry x copies of the A allele and whose mother carried y copies of the A allele is $\phi_{xy} = \pi_{xy0}\gamma_{y0} + \pi_{xy1}\gamma_{y1} + \pi_{xy2}\gamma_{y2}$, where: $\pi_{xyz} = \sum_{X\in\{0,1\}} \mathscr{B}(X;y/2)\mathscr{B}(x-X;z/2)$ is the probability that a daughter born to a genotype-y mother and a genotype-z father is of genotype-x; and $\mathscr{B}(q;r) = r$ if $q = 1$, $1-r$ if $q = 0$, and 0 otherwise. Similarly, the fraction of males who carry u copies of the A allele and whose mother carried v copies of the A allele is $\mu_{uv} = \rho_{uv0}\gamma_{v0} + \rho_{uv1}\gamma_{v1} + \rho_{uv2}\gamma_{v2}$, where $\rho_{uvz} = \sum_{U\in\{0,1\}}\mathscr{B}(U;v/2)\ \mathscr{B}(u-U;z/2)$ is the probability that a son born to a genotype-v mother and a genotype-z father is of genotype-u. Offspring then undergo viability selection, with a proportion $s_{xy} = \beta + B_f\ y\varepsilon_f - C_f\ x\varepsilon_f$ of genotype-x females with genotype-y mothers surviving to adulthood, where β is baseline viability, B_f is the indirect benefit of the maternal effect gene as experienced by females, C_f is the direct viability cost of the maternal effect gene as experienced by females, $\varepsilon_f = 1$ under the adding model of gene effects, and $\varepsilon_f = \frac{1}{2}$ under the averaging model of gene effects. Similarly, a proportion $t_{uv} = \beta + B_m\ v\varepsilon_f - C_m\ u\varepsilon_m$ of genotype-u males with genotype-v mothers survive to adulthood, where B_m is the indirect benefit of the maternal effect gene as experienced by males, C_m is the direct viability cost of the maternal effect gene as experienced by males, $\varepsilon_m = 1$ under the adding model of gene effects, and $\varepsilon_m = \frac{1}{2}$ under the averaging model of gene effects. The average viability of all females in the population is $S = \sum_{x\in\{0,1,2\},y\in\{0,\ 1,2\}} s_{xy}\phi_{xy}$, and the average viability of all males in the population is $T = \sum_{u\in\{0,1,2\},v\in\{0,1,2\}}\ t_{uv}\mu_{uv}$. Finally, an equal number of adult females and adult males are chosen at random to become breeders, returning the population to the beginning of the lifecycle. The genotype frequencies among the breeders are $\phi'_x = \sum_{y\in\{0,1,2\}} s_{xy}\phi_{xy}/S$ for females and $\mu'_u = \sum_{v\in\{0,1,2\}} t_{uv}\mu_{uv}/T$ for males.

Haplodiploidy

I assume a model identical to that for diploidy, above, except for the following details: the male genotypes are restricted to $u \in \{0,1\}$; AA females preferentially pair with A males, aa females preferentially pair with a males, and Aa females exhibit no preference; the probability that a daughter born to a genotype-y mother and a genotype-z father is of genotype-x is $\pi_{xyz} = \sum_{X\in_{\{0,1\}}}\mathscr{B}(X;y/2)\mathscr{B}(x-X;z)$ the probability that a son born to a genotype-v mother and a genotype-z father is of genotype-u is $\rho_{uvz} = \mathscr{B}(u;v/2)$; and $\varepsilon_m = 1$ under both the adding and averaging models of gene effects.

Inbreeding

I define a random variable κ according to the presence ($\kappa = 1$) or absence ($\kappa = 0$) of allele A in a randomly chosen gamete produced by a randomly chosen breeding female, and I define a random variable λ according to the presence ($\lambda = 1$) or absence ($\lambda = 0$) of allele A in a randomly chosen gamete produced by her mate. The coefficient of inbreeding is defined as $f = \mathrm{cov}(\kappa,\lambda)/\mathrm{cov}(\kappa,\kappa)$. Note that $\mathrm{cov}(\kappa,\lambda) = \mathrm{E}(\kappa\lambda) - \mathrm{E}(\kappa)\mathrm{E}(\lambda) = (\frac{1}{4}\ \gamma_{11} + \frac{1}{2}\ \gamma_{12} + \frac{1}{2}\ \gamma_{21} + \gamma_{22}) - (\frac{1}{2}\ \gamma_{10} + \frac{1}{2}\ \gamma_{11} + \frac{1}{2}\ \gamma_{12} + \gamma_{20} + \gamma_{21} + \gamma_{22})\ (\frac{1}{2}\ \gamma_{01} + \frac{1}{2}\ \gamma_{11} + \frac{1}{2}\ \gamma_{21} + \gamma_{02} + \gamma_{12} + \gamma_{22})$ and $\mathrm{cov}(\kappa,\kappa) = \mathrm{E}(\kappa^2) - \mathrm{E}(\kappa)^2 = (\frac{1}{2}\ \gamma_{10} + \frac{1}{2}\ \gamma_{11} + \frac{1}{2}\ \gamma_{12} + \gamma_{20} + \gamma_{21} + \gamma_{22})(1 - \frac{1}{2}\ \gamma_{10} - \frac{1}{2}\ \gamma_{11} - \frac{1}{2}\ \gamma_{12} - \gamma_{20} - \gamma_{21} - \gamma_{22})$, under diploidy and $\mathrm{cov}(\kappa,\lambda) = \mathrm{E}(\kappa\lambda) - \mathrm{E}(\kappa)\mathrm{E}(\lambda) = (\frac{1}{2}\ \gamma_{11} + \gamma_{21}) - (\frac{1}{2}\ \gamma_{10} + \frac{1}{2}\ \gamma_{11} + \gamma_{20} + \gamma_{21})(\gamma_{01} + \gamma_{11} + \gamma_{21})$ and $\mathrm{cov}(\kappa,\kappa) = \mathrm{E}(\kappa^2) - \mathrm{E}(\kappa)^2 = (\frac{1}{2}\ \gamma_{10} + \frac{1}{2}\ \gamma_{11} + \gamma_{20} + \gamma_{21})(1 - \frac{1}{2}\ \gamma_{10} - \frac{1}{2}\ \gamma_{11} - \gamma_{20} - \gamma_{21})$, under haplodiploidy.

Numerical analysis

I perform numerical simulations, using the above recursions for ϕ_x and μ_u. The initialization values for the genotype frequencies are $\phi_0 = \mu_0 = 0.999$, $\phi_1 = \mu_1 = 0.001$ and $\phi_2 = \mu_2 = 0.000$ for diploidy and $\phi_0 = 0.999$, $\phi_1 = 0.001$, $\phi_2 = 0.000$, $\mu_0 = 0.9995$ and $\mu_1 = 0.0005$ for haplodiploidy. I perform iterations for 100 generations with the maternal effect gene 'switched off' (i.e. setting $B_f = B_m = C_f = C_m = 0$). At the end of the 100th generation, I compute the frequency of the maternal effect gene (i.e. $(\frac{1}{2})(\frac{1}{2}\phi_1 + \phi_2) + (\frac{1}{2})(\frac{1}{2}\mu_1 + \mu_2)$ for diploidy and $(2/3)(\frac{1}{2}\phi_1 + \phi_2) + (1/3)\mu_1$ for haplodiploidy), and I compute the coefficient of inbreeding, f. I then 'switch on' the maternal effect gene (i.e. setting B_f, B_m, C_f and C_m to their desired values) and perform iterations for a further 900 generations. The A allele is considered to have 'invaded' if its frequency at the end of the 900 generations of selection is greater than its frequency at the beginning of those 900 generations.

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