Caste Load and the Evolution of Reproductive Skew

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Online enhancement: simulation code.

ABSTRACT: Reproductive skew theory seeks to explain how reproduction is divided among group members in animal societies. Existing theory is framed almost entirely in terms of selection, though nonadaptive processes must also play some role in the evolution of reproductive skew. Here I propose that a genetic correlation between helper fecundity and breeder fecundity may frequently constrain the evolution of reproductive skew. This constraint is part of a wider phenomenon that I term "caste load," which is defined as the decline in mean fitness caused by caste-specific selection pressures, that is, differential selection on breeding and nonbreeding individuals. I elaborate the caste load hypothesis using quantitative and population genetic arguments and individual-based simulations. Although selection can sometimes erode genetic correlations and resolve caste load, this may be constrained when mutations have similar pleiotropic effects on breeder and helper traits. I document evidence for caste load, identify putative genomic adaptations to it, and suggest future research directions. The models highlight the value of considering adaptation within the boundaries imposed by genetic architecture and incidentally reaffirm that monogamy promotes the evolutionary transition to eusociality.

Keywords: cooperative breeding, eusociality, **G** matrix, evolutionary constraints, pleiotropy.

Introduction

Evolution can result from natural selection as well as nonadaptive processes such as mutation, drift, migration, and genetic hitchhiking. Some phenotypes also represent nonadaptive by-products of selection on correlated characters (Gould and Lewontin 1979). For some topics in evolutionary biology, ample consideration is given to both adaptive and nonadaptive causes of observed phenotypic diversity; examples include range size evolution (Kirkpatrick and Barton 1997), speciation (Gavrilets 2003), genome evolution (Lynch et al. 2006), and sexual dimorphism (Bonduriansky and Chenoweth 2009). In other fields, selection takes center stage, and nonadaptive processes are rarely considered.

Reproductive skew theory is concerned with explaining how breeding is divided among group members in animal societies (reviewed in Clutton-Brock 1998; Johnstone 2000; Magrath and Heinsohn 2000; Buston and Zink 2009; Nonacs and Hager 2011). "Cooperative breeders" are characterized by reproductive totipotency, meaning that all individuals are capable of breeding, at least in principle (Crespi and Yanega 1995). Some cooperative breeders exhibit strong reproductive skew, including meerkats (Griffin et al. 2003), mole rats (Bennett and Faulkes 2000), ambrosia beetles (Biedermann and Taborsky 2011), paper wasps (Reeve and Keller 2001), cichlids (Wong and Balshine 2011), and most cooperatively breeding birds (Raihani and Clutton-Brock 2010); while in other species such as banded mongooses (Cant 2000) and lions (Packer 2001), reproduction is shared almost equally among group members (low skew). By contrast, "eusocial" species are here defined as those with behaviorally distinct reproductive "castes" of "queens" and "workers," which are set irreversibly once development is complete (Crespi and Yanega 1995). Reproductive skew is typically high in eusocial species, though there is much variation in the proportion of the colony's offspring that is produced by workers (Wenseleers and Ratnieks 2006*a*).

Current reproductive skew theory is framed almost entirely in terms of selection (see reviews listed above). Skew is proposed to be an emergent property of selection on breeders and helpers to optimize their inclusive fitness. For example, worker reproduction in social insects is often interpreted as an attempt to gain direct fitness in spite of the costs to other colony members (Wenseleers et al. 2004); workers are hypothesized to weigh the direct benefits of reproduction against its direct costs (e.g., the risk of punishment; Wenseleers and Ratnieks 2006*b*) and its indirect costs (reduced effort in helping relatives; Mattila et al. 2012). Another common approach is to regard division of reproduction as a negotiation between dominants and subordinates. In this negotiation, dominants permit subor-

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dinates to reproduce just enough to make it worthwhile for the latter to stay and assist in group tasks that benefit the dominant, such as vigilance or alloparental care (e.g., Buston and Zink 2009). Negotiation models often focus on how skew is affected by the availability of alternative breeding opportunities, relatedness between social partners, and the power disparity between them.

Nonadaptive Explanations for Patterns of Reproductive Skew

To fully understand a phenomenon, one should endeavor to investigate all potential causal factors and to determine and explain their relative importance. Although adaptive hypotheses have been quite successful at explaining variation in reproductive skew (e.g., Wenseleers and Ratnieks 2006*a*; Cornwallis et al. 2010; Lukas and Clutton-Brock 2012), focusing on selection alone produces an incomplete picture.

A plausible nonadaptive cause of variation in reproductive skew was proposed by Van Dyken et al. (2011), who used a model to show that "cheaters" (e.g., reproductive subordinates whose reproduction harms the inclusive fitness of other group members) can persist in a social system even when cheating carries a net cost to the inclusive fitness of the cheater, provided that cheating is constantly reintroduced into the population by mutation. Because selection on social behaviors occurs partly through kin selection, which becomes progressively weaker as relatedness among social partners declines, these maladaptive cheaters may be present at considerable frequencies. Nonacs and Hager (2011) similarly argued that reproductive skew is probably a highly polygenic trait and should thus have a high mutational input. High mutation pressure could keep populations away from their optimum skew level, and variation in the degree of mutational input might explain some of the observed variation in skew.

Here I present another nonadaptive hypothesis for patterns of reproductive skew. The hypothesis relies on the fact that the response to selection can be constrained by genetic correlations among traits (Cheverud 1984; Orr 2000; Otto 2004; Blows and Hoffmann 2005). When two traits have a shared genetic basis, the evolutionary response of one trait is tied to that of the other. One can consider the same trait expressed in two different contexts as two potentially genetically correlated traits (Falconer 1952), and phenotypic plasticity can be constrained when this genetic correlation is strong (Via and Lande 1985). A well-known example is intralocus sexual conflict, in which males and females are unable to reach their sex-specific phenotypic optima because of cross-sex genetic correlations (reviewed in Arnqvist and Rowe 2005; Bonduriansky and Chenoweth 2009; van Doorn 2009). Genetic correlations between the sexes coupled with sex-specific selection create the "gender load," lowering mean fitness across the population.

Caste Load

I propose that eusocial and cooperatively breeding species experience "caste load" resulting from conflicting selective pressures and genetic covariance between the castes (I will henceforth use "caste" to distinguish breeders and helpers, even in noneusocial species where this term is not typically used). I define caste load as the decline in mean individual fitness caused by antagonistic selection pressures on breeders and helpers. For example, in eusocial insects, queens may benefit from being large, long-lived, and nonaggressive, while the optimal worker might be small, short-lived, and aggressive. In such cases, genetic correlations between the castes for body size, life span, and aggression could place a limit on the evolution of caste dimorphism in these traits and thereby impose a fitness cost (Linksvayer and Wade 2005). Similarly, in social vertebrates, aggression might be beneficial when expressed in the dominant breeder but disadvantageous for a subordinate.

Like any trait under caste-specific selection, fecundity (and hence, reproductive skew) should be affected by caste load. Primary reproductives such as dominant breeders and queens will generally be selected for higher fecundity than subordinates and workers. The optimal nonbreeder fecundity should range from zero to the same as that of a breeder, depending on the net inclusive fitness effects of worker/subordinate reproduction (e.g., Johnstone 2000; Wenseleers et al. 2004; Nonacs and Hager 2011). The evolved level of reproductive skew will depend on both selection and nonadaptive processes, including genetic constraints: when there is an "intercaste genetic correlation" for fecundity, selection may produce a different evolutionary outcome than when fecundity is genetically independent in the castes.

Previous models of reproductive skew have not considered the possibility that caste dimorphism in fecundity is constrained by a positive intercaste genetic correlation for fecundity. Although it has been measured only once to my knowledge (Holman et al. 2013), I will argue that this genetic constraint is likely to be both common and strong and that incorporating it into reproductive skew models can change their predictions. I will then elaborate the caste load hypothesis with quantitative and population genetic arguments and an individual-based simulation, and review pertinent empirical data.

Genetic Correlations Can Constrain the Evolution of Caste Dimorphism

Genetic correlations result from a combination of pleiotropy, in which a single gene affects more than one trait, and linkage disequilibrium, in which alleles affecting two or more traits are nonrandomly associated across the population. The multivariate breeder's equation can be used to predict the evolutionary response to selection (the change in mean phenotype, $\Delta\mathbf{z}$) in two or more traits (e.g., Lande 1979). The equation is usually written $\Delta\mathbf{z} = \mathbf{G}\boldsymbol{\beta}$, where $\Delta\mathbf{z}$ is a vector of changes in mean trait values, $\mathbf{G}$ is the additive genetic variance-covariance matrix, and $\boldsymbol{\beta}$ is a vector of selection gradients for the traits. In the case of two traits (z_1 and z_2), the equation can be written in matrix notation as

$$\begin{bmatrix}\Delta z_1\\ \Delta z_2\end{bmatrix} = \begin{bmatrix}G_{11} & G_{12}\\ G_{12} & G_{22}\end{bmatrix}\begin{bmatrix}\beta_1\\ \beta_2\end{bmatrix}, \tag{1}$$

where G_{11} and G_{22} are the additive genetic variances of the two traits, G_{12} is the additive genetic covariance between them, and β_1 and β_2 are selection gradients. Equation (1) can be rewritten as

$$\Delta z_1 = G_{11}\beta_1 + G_{12}\beta_2 \tag{2a}$$

and

$$\Delta z_2 = G_{22}\beta_2 + G_{12}\beta_1. \tag{2b}$$

These equations illustrate that the response to selection in one trait will be either augmented or depressed whenever selection acts on the other trait and there is a nonzero genetic covariance between them. For example, if trait 1 is positively selected ($\beta_1 > 0$) and trait 2 is negatively selected ($\beta_2 < 0$), then a positive genetic correlation ($G_{12} > 0$) will weaken or even reverse the response in the focal trait that would occur if the traits were genetically independent. Equation (2) also shows that when a genetic correlation is present ($G_{12} \neq 0$), adaptive evolution may cease despite the presence of both selection and additive genetic variation for both traits (see also Blows and Hoffmann 2005).

Now consider a cooperatively breeding or eusocial species: let z_1 and z_2 be breeder fecundity and helper fecundity, respectively. Equation (2a, 2b) shows that selection on the fecundity of one caste will influence the response to selection in the other whenever there is an intercaste genetic correlation for fecundity. The observation that some helpers reproduce is usually assumed to demonstrate that reproduction is selectively advantageous to these helpers (this is implicit whenever reproductive helpers are called "cheaters" or "selfish"). However, equation (2) shows that selection can increase helper fecundity even when helpers would be most fit if they were sterile, provided that $G_{12} > 0$ and breeder fecundity is positively selected. In other words, helper reproduction might be maintained as a maladaptive or selectively neutral by-product of selection on breeders because of a positive intercaste genetic correlation for fecundity.

But Genetic Correlations Can Evolve: Are Constraints Still Important?

Genetic correlations are not necessarily an absolute evolutionary constraint, not least because genetic correlations can themselves evolve (Steppan et al. 2002; Arnold et al. 2008). In the short term, a genetic correlation between two traits can change because of a breakdown of linkage disequilibrium between alleles that independently affect both traits. Over the longer term, alleles with pleiotropic effects on both traits may change in frequency in the population. For example, if breeder fecundity were selected for high values and helper fecundity were selected for low values, then pleiotropic alleles conferring these effects would tend to increase in frequency as a result of selection. Over still longer timescales, new pleiotropic alleles may appear by mutation or migration and then fix. New alleles potentially increase the phenotypic space available to the population and allow the evolution of ever more pronounced caste dimorphism.

The potential for genetic correlations to evolve has led to suggestions that they can be safely ignored over long periods of evolutionary time. For example, in the current context, caste-specific selection has been postulated to cause "genetic release" of the queen and worker castes in social insects, possibly mediated by gene duplication (West-Eberhard 1996; Gadagkar 1997). However, just as for single traits, nonadaptive processes such as mutation and drift can thwart the adaptive evolution of genetic correlations. I will now argue that genetic correlations that lead to caste load might sometimes persist over evolutionary time in spite of their fitness costs.

To recap, the long-term evolution of genetic correlations depends on the fixation of new mutations (Steppan et al. 2002). When selection on a mutation is weak and/or the effective population size is small, its fixation probability becomes almost independent of selection and approaches the value for a selectively neutral mutation (Kimura 1957). The allele is then said to be "nearly neutral." Even when a mutation is quite strongly beneficial, its fixation probability can be low because there is a good chance that the allele will be lost to drift while it is still rare.

Now imagine that selection favors greater caste dimorphism in fecundity, for example, because high reproductive skew increases the inclusive fitness of all group members by bringing greater efficiency. New mutations that elevate

breeder fecundity or depress helper fecundity, as well as pleiotropic mutations that simultaneously raise the fecundity of breeders and lower that of helpers, will generally be positively selected. By contrast, all other mutations will lower dimorphism and hence be negatively selected. However, unless alleles causing elevated caste dimorphism have a strong selective advantage and/or the effective population size is large, their fixation probability will essentially be the same as that of alleles that degrade caste dimorphism and in any case may be low.

These arguments imply that the frequency with which different types of mutations appear will have an important effect on the change in caste dimorphism with time. Let μ^+ be the rate of appearance of new mutations that increase caste dimorphism in fecundity and μ^- be the rate of appearance of new mutations that decrease it. If f is the average fixation probability of a mutation that increases caste dimorphism relative to one that decreases it (such that $f > 1$ when "increased caste dimorphism" alleles have a higher fixation probability because they are positively selected), then the change in mean caste dimorphism over time due to the fixation of new mutations will tend to be positive when $\mu^+ f > \mu^-$.

Therefore, when mutations increasing caste dimorphism are comparatively rare ($\mu^+ < \mu^-$), f must be large (i.e., selection on caste dimorphism must be strong and the effective population size must be large; Kimura 1957) for caste dimorphism to reach high levels. The inequality $\mu^+ f > \mu^-$, therefore, illustrates two factors that limit the evolution of genetic correlations. First, genetic correlations cannot readily respond to selection unless selection is strong relative to drift. Second, biased mutation can affect the adaptive evolution of genetic correlations (Steppan et al. 2002).

These two factors are relevant to the current problem. First, many eusocial insects and cooperatively breeding vertebrates are expected to have effective population sizes substantially lower than their total population sizes, since only a subset of individuals breed. Many eusocial insects are also haplodiploids with complementary sex determination; both traits further reduce effective population size (Zayed 2004). Therefore, alleles affecting caste dimorphism might sometimes display nearly neutral evolution ($f \approx 1$). Second, I predict that a positive mutational correlation will frequently exist between breeder fecundity and helper fecundity; that is, mutations that affect breeder fecundity tend to pleiotropically affect helper fecundity in the same direction ($\mu^+ < \mu^-$). Fecundity is a complex trait that probably depends on many loci with diverse functions, for example, in nutrition, the endocrine system, behavior, and sensitivity to social cues, so genes that affect these traits in one direction in breeders probably have the same effect in helpers more often than not (e.g., Amdam 2004; Grozinger et al. 2007). Ultimately, the strength of selection and drift and the magnitude of the mutational correlation between breeder fecundity and helper fecundity are empirical questions. However, it is premature to conclude that selection will always eliminate genetic correlations that cause maladaptation given enough time.

One should note that the arguments in this section generalize well beyond the current topic of caste load. Pleiotropic mutations might reduce fitness whenever the mutational correlation is misaligned with the adaptive landscape (Jones et al. 2007). That pleiotropy and complexity carry a selective cost is also a general principle: populations tend to be kept further from their multivariate phenotypic optimum as pleiotropy and the number of traits increase (Fisher 1930; Orr 2000). Complexity in the current context refers to having caste-specific rather than fixed trait expression, but any form of phenotypic plasticity may carry a selective load (Via and Lande 1985; Snell-Rood et al. 2010; Van Dyken and Wade 2010).

Individual-Based Simulation of Caste Load for Fecundity

To further investigate caste load in a more biologically explicit setting, I wrote an individual-based simulation. The model considers a life cycle representative of an annual eusocial hymenopteran, though its qualitative conclusions likely generalize to cooperative breeders and to other taxa. The aim of the model is to assess the impact of a nonadaptive factor (the strength of the mutational correlation between queen fecundity and worker fecundity) on the evolution of caste dimorphism in fecundity, while incorporating pleiotropy, selection, mutation, and drift.

The population is composed of social insect colonies, each of which contains one queen and the stored sperm of her p mates (modeled explicitly) and their many worker offspring (modeled implicitly). Queens, males, and workers are all haploid and have L unlinked loci that interact additively to determine fecundity (the assumption of haploidy simplifies the model by allowing dominance to be ignored and is unlikely to affect the model's qualitative conclusions). Every locus has a pleiotropic effect on worker fecundity and queen fecundity and has one of four possible alleles. I chose to consider only pleiotropic loci because they affect genetic correlations and are affected by selection on multiple traits. The model allows four possible pleiotropic alleles, which were coded 1–4 and have the following phenotypic effects: 1 = low queen fecundity and low worker fecundity; 2 = low queen fecundity and high worker fecundity; 3 = high queen fecundity and low worker fecundity; and 4 = high queen fecundity and high worker fecundity. "Low" and "high" are relative terms and

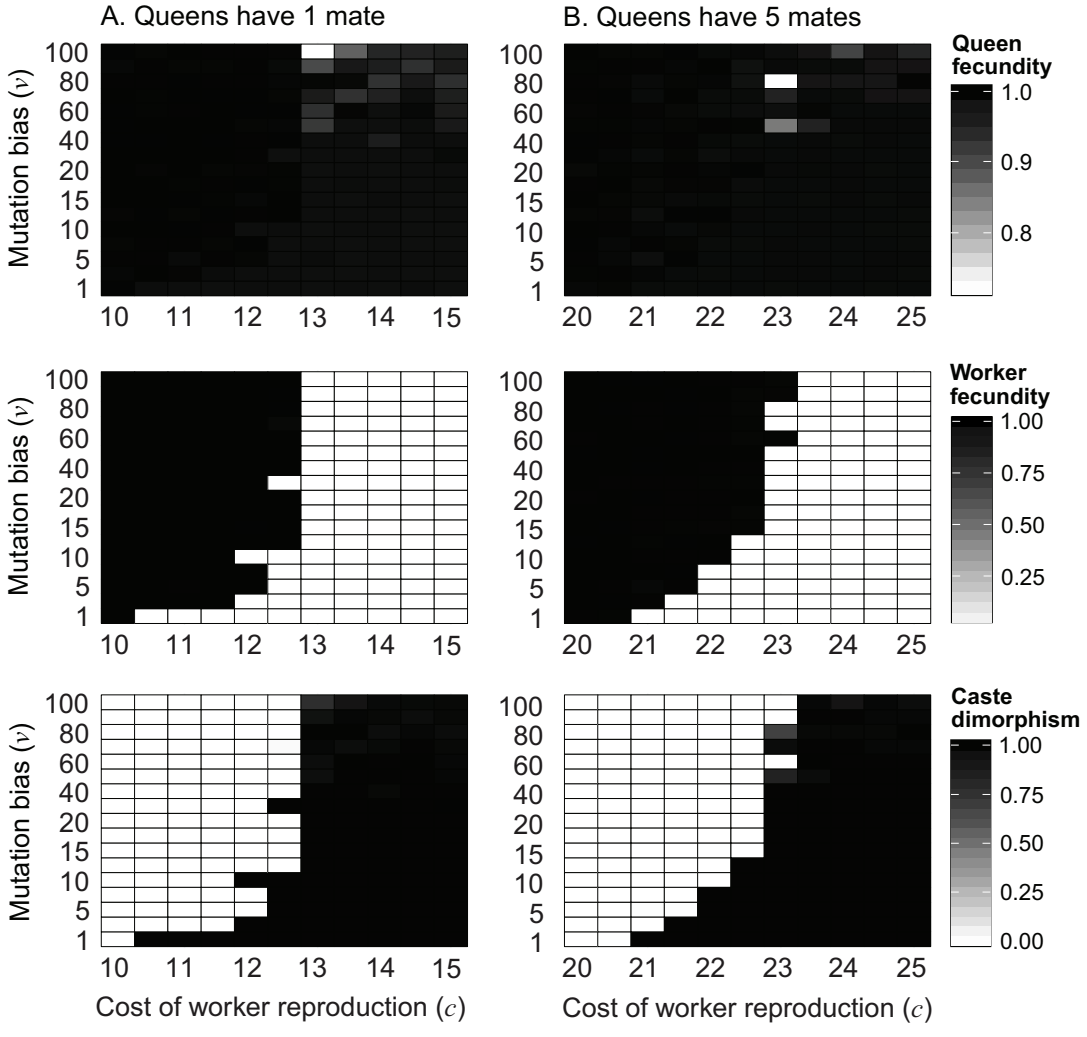

Figure 1: Both the cost of worker fecundity (a selective pressure) and the mutational correlation between queen fecundity and worker fecundity (a nonadaptive factor) affect the evolution of reproductive skew. The *X*-axes show how much worker fecundity harms the colony's competitiveness (c), while high values on the *Y*-axes indicate a strong positive mutational correlation between queen fecundity and worker fecundity (v). Simulations in column *A* assume monogamy ($p = 1$), while those in column *B* assume queens have exactly 5 mates ($p = 5$). The first two rows show the evolved mean values of queen fecundity and worker fecundity, while the bottom row shows the evolved mean caste dimorphism in fecundity (i.e., queen fecundity minus worker fecundity). Note the discontinuous scale on the *Y*-axis; rows 1–20 proceed in increments of 2.5 and rows thereafter in increments of 10. The figure assumes $L = 50$, $t = 5$, and $\mu = 0.0001$; $N = 200$ colonies.

describe how each allele affects the breeding value of the traits relative to other alleles.

From the genotypes of the queen and her workers, one can deduce their relative fecundities, which together determine the contribution of the colony to the next generation. Highly fecund workers were assumed to reduce the total contribution of their colony to the next generation; however, fecund workers were more likely to produce some of the colony's sons than were less fecund workers. I investigated the evolution of caste dimorphism under different caste-specific selection regimes, while varying the mutational correlation between queen and worker fecundity. The model varied the mutational correlation by adjusting the frequency with which 1 and 4 alleles were generated by mutation relative to 2 and 3 alleles. Full details of the simulation are given in the appendix.

Simulation Results: Caste Load Affects Reproductive Skew

Figure 1 shows that when worker fecundity is sufficiently harmful to the colony's total offspring production, caste

dimorphism in fecundity evolves, leading to high reproductive skew. There was a clear threshold value of *c* at which the inclusive fitness benefits of reduced worker fecundity exceeded its costs and reproductive skew evolved from being absent to almost complete. This existence of such a threshold is predicted by inclusive fitness theory (e.g., Hamilton 1964; Wenseleers et al. 2004). However, the position of the threshold was affected by the mutational correlation: stronger benefits of sterility were needed for it to evolve when there was a positive mutational correlation between queen fecundity and worker fecundity (note diagonal area of black in the bottom part of the lower four panels of fig. 1). Therefore, the model shows that identical selective pressures can produce markedly different evolutionary outcomes under different levels of mutational pleiotropy.

The mutational correlation had another consequence: when it was sufficiently strong, caste dimorphism frequently failed to reach maximal values (fig. 1, upper right of queen fecundity panels). In effect, queen fecundity was dragged down as a by-product of selection for lower worker fecundity. With a low mutational correlation, the "eusocial" 3 allele was able to reach close to 100% frequency at all 50 loci (except for a small "cheater load"; Van Dyken et al. 2011), resulting in near-maximal values of reproductive skew. Conversely, when the mutational correlation was strong, populations contained both 1 (i.e., low-low) and 3 (high queen–low worker) alleles, with further evolution limited by the rate at which 1 alleles mutated to 3 alleles and then successfully fixed.

In a finite population, especially a small one, genetic drift can fix negatively selected alleles. The resulting loss in population mean fitness is termed the drift load (Whitlock 2000). When the mutational correlation between breeder fecundity and helper fecundity is sufficiently strong, evolution toward the selected level of skew (e.g., fixation of the 3 allele at all loci) may be so slow that factors such as drift load have significant effects. In other words, the longer it takes for the 3 allele to arise by mutation and fix at each locus, the more opportunity there is for loci that have already fixed the 3 allele to lose it again by drift. Drift load may explain how mutational pleiotropy produced the maladaptive equilibrium levels of reproductive skew seen in the upper parts of the panels in figure 1. Another explanation is that the populations were still proceeding very slowly toward higher levels of skew when the simulation was terminated after 500,000 generations. This possibility cannot be ruled out (although caste dimorphism was almost static in the last 50,000 generations; appendix). However, on such large timescales, there is ample opportunity for the relative fitness of the alleles to change due to factors not included in the model, for example, changes in the environment or genetic changes at epistatically interacting loci. Therefore, slowing progression toward the optimal reproductive skew may be enough to prevent the population from ever reaching it, even over very long periods of time.

The effect of the mutational correlation was similar under monogamy and polyandry (fig. 1). However, comparing figure 1*A* and 1*B* reveals that polyandry hindered the evolution of high reproductive skew. When queens had 5 mates rather than 1, worker sterility evolved only when it had very strongly beneficial effects on queen fecundity (note *X*-axis scales). This is because polyandry decreases relatedness among siblings, reducing the inclusive fitness benefits to workers of augmenting their mother's reproductive output (Boomsma 2007).

Some of my simplifying assumptions might underestimate the strength of caste load. For example, the effects of the alleles were assumed to be constant in space, time, and different genetic backgrounds, but capricious selection and epistasis can increase genetic variation and result in populations that are farther from their phenotypic optima (Lenormand 2002). The 50 loci were also assumed to recombine freely: assuming linkage would hinder fixation of beneficial mutations linked to deleterious ones and promote hitchhiking by deleterious alleles. Both of these factors could make it harder for selection to break down the genetic correlation between breeder fecundity and helper fecundity. The model also has no population structure and so effectively assumes hard selection on caste dimorphism—soft selection generally impedes adaptive evolution (Agrawal 2010) and hence should increase caste load. Selection on caste dimorphism is expected to be soft whenever it derives partly from local competition among colonies/groups, which seems likely for many eusocial taxa and cooperative breeders.

Conversely, some of my assumptions might overestimate caste load. In the interests of computation time, I assumed a modest effective population size: 200 pairs and their reproductive offspring under monogamy, or 200 females, each with 5 mates plus their reproductive offspring, under polyandry. Although this effective population size is not necessarily artificially low (e.g., Ellis et al. 2006), assuming a higher population size would decrease the influence of genetic drift and potentially reduce maladaptation. However, the overall patterns in figure 1 would likely be similar for higher effective population sizes: populations should still be relatively maladapted and less likely to evolve high reproductive skew under a high mutational correlation.

In sum, the model suggests that caste load exists whenever there is a genetic correlation between breeder fitness and helper fitness, especially when selection is unable to break down this genetic correlation (e.g., because of a strong mutational correlation and strong drift relative to

selection). The strength of caste load likely varies between taxa from strong to almost nonexistent because of variation in genetic architecture and strength of selection relative to drift.

Discussion

The above models and arguments show that reproductive skew can be influenced by genetic correlations linking the respective fecundity of breeders and helpers. This effect is especially pronounced when there is also a mutational correlation, such that mutations tend to affect breeder fecundity and helper fecundity simultaneously and in the same direction. Selection can sometimes erode the genetic correlation and thereby alleviate caste load, but mutational pleiotropy may sometimes prevent this from happening, especially when selection is weak and/or when mutations that erode caste dimorphism are much more common than those that increase it. This latter condition might be true in most cases, because intuitively I expect that helper fecundity and breeder fecundity will often be similarly affected by mutations. For example, any mutations that interfere with the ability to assimilate nutrients, produce yolk, or resist infection should lower fecundity in both castes.

For caste load to influence reproductive skew, there must be an intercaste genetic correlation for fecundity. To my knowledge, intercaste genetic correlations of any kind have been estimated only once. Holman et al. (2013) found a significant positive correlation between measures of ovarian development in queens and their cross-fostered worker offspring in the ant *Lasius niger*, implying the existence of a positive intercaste genetic correlation for fecundity. *Lasius niger* workers occasionally produce males in the presence of the queen (Fjerdingstad et al. 2002), though it is unclear whether worker reproduction is positively or negatively selected. Interestingly, worker-laid eggs (both fertile eggs and nonfertile "trophic eggs") are fed to larvae (Baroni Urbani 1991), which returns their resources to the colony. The consumption of worker-laid eggs by other workers, brood, or the queen occurs in many social insects (Perry and Roitberg 2006) and may in part reflect an adaptation to caste load. Worker reproduction presumably loses some or all of its colony-level cost if the eggs are used as food. As shown in figure 1, lower costs of worker reproduction can allow queen fecundity to evolve to higher levels under restrictive genetic architecture.

Although several studies have applied quantitative genetic methods to the honeybee *Apis mellifera*, I am not aware of any studies that have explicitly measured intercaste genetic correlations in this species. However, a microarray study of brain tissue (Grozinger et al. 2007) found that of the 155 genes that were upregulated in reproductive workers relative to sterile workers, 62% were also upregulated and only 3% were downregulated in queens relative to sterile workers. Similarly, of the 66 genes that were downregulated in reproductive workers relative to sterile workers, 57% were also downregulated and only 2% were upregulated in queens versus sterile workers. This study, therefore, identified approximately $155 \times 0.62 + 66 \times 0.57 = 134$ genes that might pleiotropically affect queen fecundity and worker fecundity in the same direction.

Studies of the clonal ants *Platythyrea punctata* and *Cerapachys biroi* provide further evidence of caste load. Their colonies are composed of genetically identical females, all of which are potentially capable of clonal reproduction. *Platythyrea punctata* colonies contain only one or two highly fertile individuals that monopolize reproduction, while *C. biroi* colonies go through reproductive phases in which many individuals lay eggs, followed by brood-rearing phases in which the colony is sterile. *Platythyrea punctata* workers that become fertile in the presence of a dominant reproductive are attacked by their nestmates (Hartmann et al. 2003), as are *C. biroi* workers that reproduce during the wrong phase (Teseo et al. 2013). Because all workers are genetically identical (barring de novo mutations), their evolutionary interests are perfectly aligned, implying that "illegitimate" reproduction reduces the inclusive fitness of the workers involved. This apparently maladaptive reproduction may reflect caste load: selection may be unable to simultaneously maximize the fecundity of breeding workers and ensure complete sterility in nonbreeding workers because of genetic correlations between breeder fecundity and nonbreeder fecundity. Alternatively, the cheater load concept of Van Dyken et al. (2011) may be at work: the fertile workers may carry de novo mutations that cause them to reproduce contrary to their own fitness interests. As evidence that minor genetic changes can increase the fecundity of workers, Jarosch et al. (2011) identified a genetic region that caused honeybee workers to become fertile when knocked out using RNAi.

The current simulations revealed that selection might sometimes remove the caste load by favoring alleles that increase caste dimorphism and lower the intercaste genetic correlation. The literature on sexual conflict provides several ideas on the proximate mechanisms by which this might be accomplished (reviewed in Bonduriansky and Chenoweth 2009), and there are striking parallels here between gender load and caste load. Cross-sex genetic correlations, and hence the gender load, can be reduced by gene duplication followed by sex-specific expression of each copy (Gallach and Betrán 2011), sex-specific alternative splicing (Telonis-Scott et al. 2008), or sex-specific genomic imprinting (Day and Bonduriansky 2004; Hager et al. 2008). Similar mechanisms appear to have evolved

to accomplish caste-specific gene expression in social insects. In honeybees, there is some evidence that caste-specific selection has led to gene duplication (Honeybee Genome Sequencing Consortium 2006; Xu et al. 2010), caste-specific splicing (Aamodt 2008; Jarosch et al. 2011), and caste-specific methylation patterns (Weiner and Toth 2012). Such traits could be regarded as putative genomic adaptations to caste load.

It is worth noting that even perfect caste-specific gene expression may be unable to completely mitigate caste load. Sexual conflict researchers have pointed out that genes or transcripts with sex-specific expression are only subject to selection in ~50% of the population, which should double the load of deleterious mutations at these loci, all else being equal (Bonduriansky and Chenoweth 2009; Connallon et al. 2010). Similar concerns hold for caste-specific gene expression, such that there should be a genetic load even when gene expression is caste specific at all loci with antagonistic fitness effects on the castes.

My simulation is also pertinent to the debate over the evolution of eusociality and the utility of inclusive fitness theory. Hamilton's rule implies that, all else being equal, monogamy favors the evolution of eusociality by increasing relatedness between workers and the siblings that they help to rear (Boomsma 2007). Although diverse modeling frameworks concur with this prediction (Queller 1992; Bijma and Wade 2008; McGlothlin et al. 2010; Fromhage and Kokko 2011) and there is little evidence that eusociality ever evolved in nonmonogamous taxa (Boomsma et al. 2011), there are claims that the inclusive fitness prediction that monogamy assists the transition to eusociality is incorrect (e.g., Wilson and Hölldobler 2005; Nowak et al. 2010; Nonacs 2011). This model reaffirms that monogamy does favor the evolution of eusociality. As in Hamilton's rule, there was a critical threshold at which the benefits of sterility equaled the costs, beyond which eusociality evolved. The benefits required to pass this threshold were much lower under monogamy than under polyandry (fig. 1). By contrast, another simulation study found that polyandry sometimes allows a new mutation coding for a worker phenotype to spread more rapidly than monogamy and concluded that inclusive fitness theory might have got it wrong (Nonacs 2011). However, this result stems from that simulation's assumption that sibling helpers compete to inherit their mother's nest (see Leggett et al. 2012) and therefore may apply only to systems in which helpers can replace the breeder. Moreover, figures 1*B*, 4*B* and 6 in Nonacs (2011) apparently show that there are parameter spaces in which eusociality can invade under monogamy but not under polyandry, as well as regions in which eusociality can invade when queens have 2 mates but not 5, which concurs with inclusive fitness predictions.

In order to better understand caste load and reproductive skew, future studies could attempt to estimate the genetic correlation between breeder and helper fecundity. This could be accomplished using breeding designs (Holman at al. 2013), phenotyping wild populations with known pedigrees (Kruuk et al. 2008), or comparing populations or selected lines. The caste load hypothesis also produces the testable prediction that interpopulation variation in caste dimorphism should be positively correlated with estimates of fitness, just as sexual dimorphism may increase mean fitness by lowering the gender load (Rankin and Arnqvist 2008). Additionally, the strength of mutation relative to selection should be positively correlated with caste load whenever there is a positive mutational correlation between the castes. Since population size and relatedness among social partners affect the efficacy of selection (Van Dyken et al. 2011), these factors should predict caste load. Caste load may have especially interesting consequences in unicolonial ants, in which workers care for the offspring of queens to which they are entirely unrelated, completely removing selection on traits expressed in workers provided that workers are sterile (Helanterä et al. 2009). Workers traits, therefore, should be further from their optimum, and queen traits should be better adapted than in nonunicolonial species.

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APPENDIX

Details of the Caste Load Simulation

Initialization

The simulation was written in R, and the script is included as supplementary material.[1] Simulations were initialized by creating 200 colonies containing haploid queens, each with p haploid mates, with randomly assigned genotypes at each of their L loci. Of the four possible alleles (codes: 1 = low queen fecundity and low worker fecundity; 2 = low queen fecundity and high worker fecundity; 3 = high queen fecundity and low worker fecundity; and 4 = high queen fecundity and high worker fecundity), only the 1 and 4 alleles were present in the initial population.

[1] Code that appears in the *American Naturalist* is provided as a convenience to the readers. It has not necessarily been tested as part of the peer review.

Determining Colony Competitiveness

In each generation, colonies competed to produce the new sexual offspring that would form the new population. A given colony's competitiveness was determined by its queen's fecundity, its average worker fecundity, and a global parameter, the colony-level cost of worker fecundity (c). Colonies with a fecund queen tended to contribute more sexual offspring to the next generation, while those with relatively fecund workers were less productive provided $c > 0$.

Specifically, the simulation first calculated the fecundity of the queen and the fecundity of the workers in the colony given the queen and male genotypes. Queen fecundity was equal to the total number of 3 and 4 alleles carried by the queen divided by L, such that queen fecundity ranged between zero and one. I assumed that paternity was shared equally by the p males and that the number of workers in each colony was large, such that knowing the alleles present in the queen and her mates allows accurate estimation of the mean fecundity of the workers in the colony. The mean worker fecundity ($\bar{w}$) was calculated for each colony as

$$\bar{w} = \frac{1}{L}\sum_{i=1}^{L}\left(0.5Q_i + \frac{0.5}{p}\sum_{j=1}^{p}M_{ij}\right), \tag{A1}$$

where Q_i is the queen's breeding value for worker fecundity at locus i (where the "high worker fecundity" alleles 2 and 4 have a breeding value of one, and the "low worker fecundity" alleles 1 and 3 have a breeding value of zero), and M_{ij} is the allelic value of her jth mate at locus i (using the same scheme). The $1/L$ term ensured that worker fecundity ranged between zero and one.

Having calculated the queen and worker fecundities for each colony, the simulation then determined each colony's competitiveness, which influenced realized colony fecundities. The competitiveness of a given colony was equal to $qe^{-c\bar{w}}$, where q is the queen's fecundity and c determines how much worker fecundity reduces colony productivity ($c \geq 0$). The colonies then competed to produce the N gynes (unmated queens) and Np males that would make up the next generation. Competition was modeled by sampling colonies with replacement to determine which one produced each offspring and where the probability of being picked was equal to colony competitiveness, divided by the sum of the competitiveness of all the colonies.

Assigning Genotypes to the New Gynes and Males

I assumed that all newly produced gynes were offspring of the queen produced by sexual reproduction, as in most eusocial insects. The genotype of each gyne was determined by selecting a random allele at each locus from the mother queen (with probability 0.5) or one of her mates (with probability $0.5/p$ for each of the p mates).

As in many social Hymenoptera, both workers and queens could produce males asexually. The probability that each male from a specific colony was worker produced was assumed to be equal to $\bar{w}/(\bar{w} + q)$, where $\bar{w}$ and q are queen fecundity and mean worker fecundity, respectively. For colonies with $\bar{w} = q = 0$, this value was set to 0.5.

Because the model considers haploid individuals, queen-produced males were clones of the queen, whereas worker-produced males were clones of one of the possible worker genotypes in the colony. Moreover, I assumed that genotypes conferring high worker fecundity were more likely to contribute to the pool of worker-laid males than lower fecundity worker genotypes in the same colony. For each worker-produced male, I therefore randomly selected male genotypes from the possible worker genotypes, assuming that alleles conferring high worker fecundity (the 2 and 4 alleles) were t times more likely to be chosen than the 1 and 3 alleles. The simulation is thus somewhat similar to models of segregation distorters (selfish genetic elements that disrupt Mendelian inheritance; Lyttle 1991): alleles conferring high worker fecundity reduce colony-level fecundity whenever $c > 0$, but they have a higher probability of being present in each offspring when $t > 1$ (t stands for "transmission bias"). Therefore, the relative magnitudes of c and t determined the direction of selection on worker fecundity, though the evolved outcome also depends on the genetic architecture.

Mutation and Mating

Each allele in every newborn individual had an independent probability μ of mutating to a randomly selected allele. I assumed that the alleles 1 (low worker fecundity and low queen fecundity) and 4 (high queen fecundity and high worker fecundity) were ν times more likely to arise by mutation than the "opposite effect" 2 and 3 alleles. Setting $\nu > 1$ thus creates a positive mutational correlation between queen fecundity and worker fecundity.

The model assumes that newly produced gynes mate with exactly p randomly selected males and that multiple mating by males does not occur (as in many eusocial Hymenoptera; Boomsma et al. 2005). These queens and the stored sperm of their mates went on to lead the colonies of the next generation, replacing the parental generation.

Running the Simulation

The simulation was run for 500,000 generations, at which point the mean fecundity of queens and workers was recorded. From this, I calculated the population-wide caste

dimorphism in fecundity as the average of the caste difference in fecundity across colonies (possible range: 0–1). In all simulations, I assumed $L = 50$, $t = 5$, and $\mu = 0.0001$. Assuming fixed t (the parameter that determines the relative frequency with which alleles for high worker fecundity are found in worker-produced males) likely does not affect the results because the strength and direction of selection on worker fecundity depends on the ratio of t and c. I kept t constant and varied c, but doing the opposite would also vary the strength and direction of selection and likely produce qualitatively similar results. The relatively high mutation rate was chosen in the interests of computation time. A lower mutation rate would reduce the frequency of maladaptive genetic variation at equilibrium (i.e., it would lower the cheater load; Van Dyken et al. 2011), but inspection of individual simulation runs confirmed that genetic variance was already low (the gray areas in the upper right of fig. 1 are predominantly caused by 1 alleles being fixed at some loci and 3 alleles being fixed at others, and not by polymorphism at individual loci). Therefore, cheater load had minor influence on the results relative to selection and pleiotropy.

I did not replicate individual parameters spaces because this comes as a trade-off with the number of parameter spaces that can be examined in a given time frame. This method also allows one to gauge the repeatability of simulation runs by examining the similarity of neighboring parameter spaces (fig. 1). All simulations were checked for convergence: the mean range in caste dimorphism in the final 50,000 generations was 0.022 ± 0.0009.

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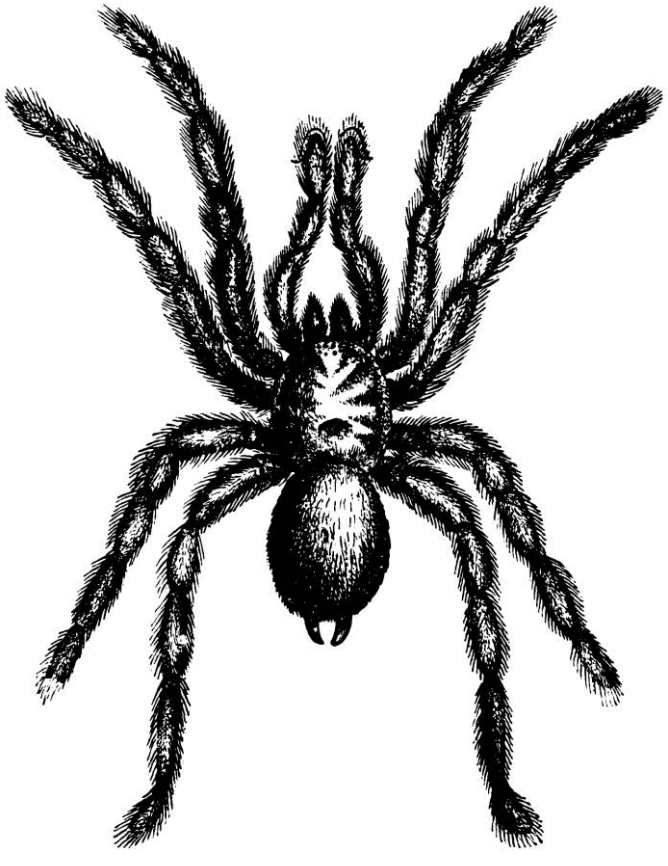

"The Tarantula Killer pursues several other species of the large ground spiders, but the Mygale Hentzii [*Aphonopelma hentzi*, shown above], or Tarantula, is his favorite. I have sometimes found under shelving rocks, and other sheltered places, dauber's nests that were doubtless several years old. In some of the cells, where the egg had proved abortive, the spiders were still there, still limber, with no signs of decomposition about them. They did not seem to be dead, but looked as if they could almost move their legs, and were perhaps not unconscious of their deplorable condition." From "The Tarantula Killers of Texas" by G. Lincecum (*American Naturalist*, 1867, 1:137–141).