biology letters

Kin selection, genomics and caste-antagonistic pleiotropy

David W. Hall, Soojin V. Yi and Michael A. D. Goodisman

Biol. Lett. 2013 **9**, 20130309, published 16 October 2013

biology letters

rsbl.royalsocietypublishing.org

Research

Cite this article: Hall DW, Yi SV, Goodisman MAD. 2013 Kin selection, genomics and caste-antagonistic pleiotropy. Biol Lett 9: 20130309.
http://dx.doi.org/10.1098/rsbl.2013.0309

Received: 6 April 2013
Accepted: 25 July 2013

Subject Areas:
behaviour, evolution

Keywords:
antagonistic selection, eusocial insect caste, molecular evolution, sexual selection, social conflict

Author for correspondence:
Michael A. D. Goodisman
e-mail: michael.goodisman@biology.gatech.edu

One contribution of 12 to the Special Feature '50 Years on: the legacy of William Donald Hamilton' organized by Joan Herbers and Neil Tsutsui.

Electronic supplementary material is available at http://dx.doi.org/10.1098/rsbl.2013.0309 or via http://rsbl.royalsocietypublishing.org.

Royal Society Publishing
Informing the science of the future

Evolutionary biology

Kin selection, genomics and caste-antagonistic pleiotropy

David W. Hall[1], Soojin V. Yi[2] and Michael A. D. Goodisman[2]

[1]Department of Genetics, University of Georgia, Athens, GA 30602, USA
[2]School of Biology, Georgia Institute of Technology, Atlanta, GA 30332, USA

Kin selection is a fundamentally important process that affects the evolution of social behaviours. The genomics revolution now provides the opportunity to test kin selection theory using genomic data. In this commentary, we discuss previous studies that explored the link between kin selection and patterns of variation within the genome. We then present a new theory aimed at understanding the evolution of genes involved in the development of social insects. Specifically, we investigate caste-antagonistic pleiotropy, which occurs when the phenotypes of distinct castes are optimized by different genotypes at a single locus. We find that caste-antagonistic pleiotropy leads to narrow regions where polymorphism can be maintained. Furthermore, multiple mating by queens reduces the region in which worker-favoured alleles fix, which suggests that multiple mating impedes worker caste evolution. We conclude by discussing ways to test these and other facets of kin selection using newly emerging genomic data.

1. William D. Hamilton and kin selection theory

William D. Hamilton revolutionized the study of sociality [1]. Arguably, Hamilton's most important work focused on the process of kin selection. Kin selection occurs when alleles for social behaviours are selected because these behaviours affect the fitness of relatives [2]. Fundamentally, the idea underlying kin selection is that an allele can be transmitted not only through personal reproduction, but also through the reproduction of kin.

Kin selection is responsible for the evolution of many of the remarkable actions displayed by social animals, such as the extreme helping behaviours displayed by social insects (figure 1) [3–4]. Kin selection also underlies the social actions of microbes, including the production of public goods [5]. Remarkably, even plants show evidence of kin-selected 'behaviours', such as competition through root growth, which may vary based on kinship [6]. Indeed, the evolution of many of the cooperative actions among entities at all levels of biological organization relied on kin selection-like processes [7]. Thus, kin selection represents a fundamentally important mechanism governing biological group formation.

2. Kin selection and molecular evolution

Kin selection theory has been primarily applied to explain the evolution of social behaviours at the phenotypic level. However, the signatures of kin selection should be seen at the genomic level as well. For example, Linksvayer & Wade [8] and Hall & Goodisman [9] determined the effects of kin selection on molecular evolution. They showed that the strength of kin selection, measured as the probability of fixation of a newly arising allele experiencing directional selection, could equal that of direct selection, but only when relatedness was high. As relatedness declined, so too did the strength of kin selection (figure 2*a*). Consequently, genes experiencing kin selection and direct selection may exhibit different rates of molecular evolution depending on the social system of the species in question.

Figure 1. The behaviours of social insects, such as (*a*) the honeybee (*Apis mellifera*), (*b*) the fire ant (*Solenopsis invicta*) and (*c*) the yellowjacket wasp (*Vespula maculifrons*) have been shaped extensively by the process of kin selection. Social insects often exhibit phenotypically distinct castes, the evolution of which may be hampered by caste-antagonistic pleiotropy, particularly in species where queens mate with multiple males such as *A. mellifera* and *V. maculifrons* (see text for details). (Online version in colour.)

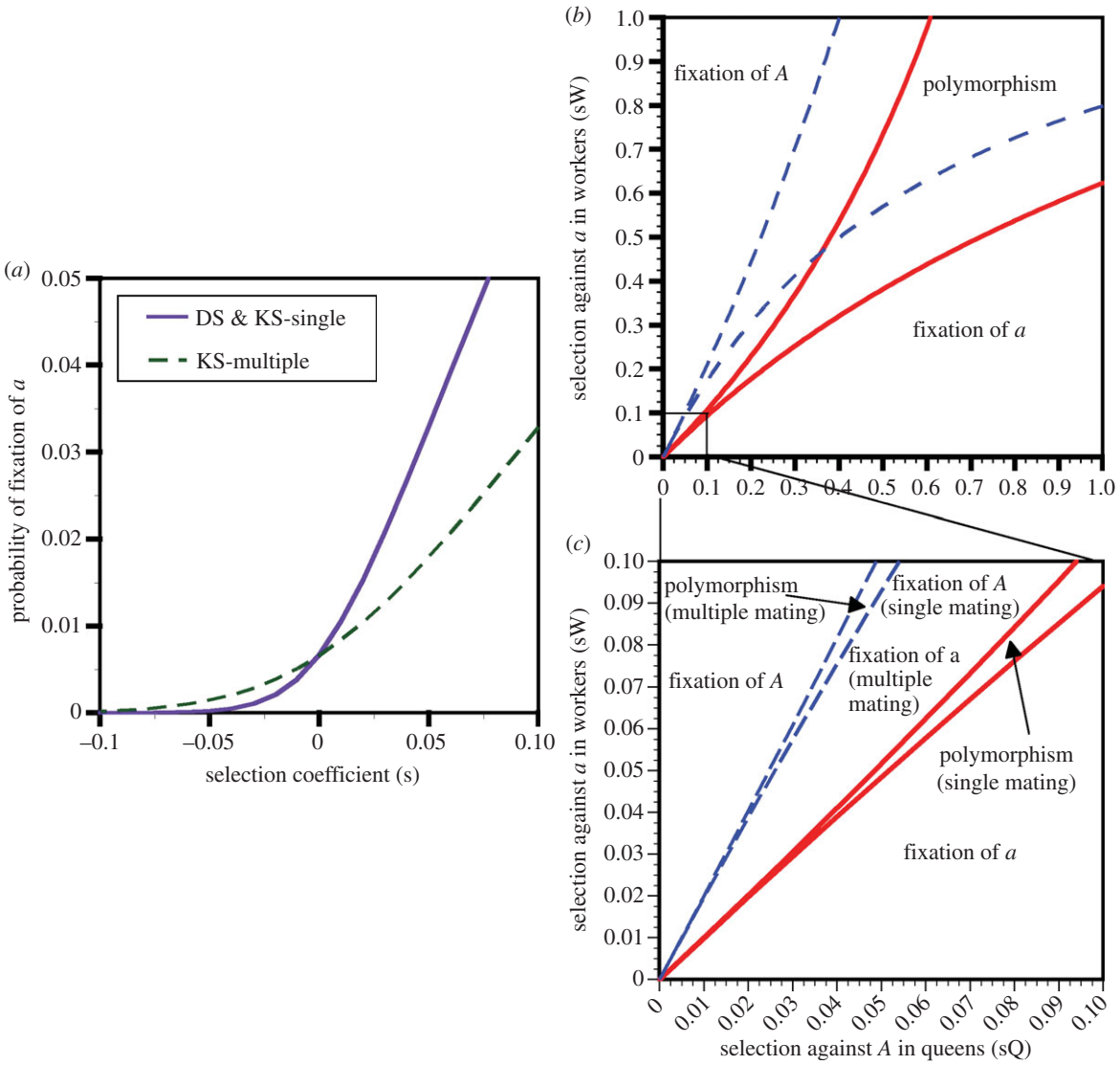

Figure 2. The outcome of selection for genes experiencing directional selection or caste-antagonistic selection. (*a*) The probability of fixation of newly arising additive alleles as a function of the strength of selection is identical under direct selection (DS) on queens or kin selection (KS) on workers when queens are singly mated (KS-single). However, when queens are multiply mated, beneficial alleles ($s > 0$) fix at lower rates and deleterious alleles ($s < 0$) fix at higher rates, when they are subjected to KS in workers (KS-multiple). (*b*,*c*) Regions in which antagonistic selection results in fixation of the queen-favoured *a* allele, the worker-favoured *A* allele or polymorphism. Solid lines and dashed lines delineate regions for singly and multiply mated queens, respectively. sQ and sW represent the strength of selection on performance against alleles *A* and *a* in queens and workers, respectively. In (*b*), all possible combinations of selection coefficients are shown, whereas panel (*c*) expands the region with more realistic, weaker selection. (Online version in colour.)

This prediction was tested using data in two social insects, the fire ant *Solenopsis invicta* and the honeybee *Apis mellifera* [9] (figure 1*a*,*b*). The queens of these two species mate different numbers of times; fire ant queens mate once, whereas honeybee queens mate multiply. Thus, the rates of evolution of queen- and worker-biased genes were predicted to be different in the honeybee but similar in the fire ant. Queen- and worker-biased genes did evolve at significantly

different rates in the honeybee and non-significantly different rates in the fire ant, consistent with expectations [9]. Thus, these studies began to link molecular and genome evolution with theoretical predictions of kin selection.

3. Caste-antagonistic pleiotropy in social genomes

Extending Hamilton's ideas to genome evolution requires the development of theory to predict how kin selection affects patterns of genetic variation. Of particular relevance for highly social species, such as social insects, is the case of antagonistic selection between castes [10], which occurs when distinct castes have different phenotypic optima for the same trait (cf. [11]). If this trait is controlled by the same gene(s) in both castes, then alleles favoured in one caste may be disfavoured in another. For example, wing muscle development may be beneficial for queen ants, which partake in mating flights, but is unlikely to be beneficial for worker ants, which do not fly. Thus, an allele that increases wing muscle development would potentially be subject to antagonistic selection across castes.

We determined the outcome of antagonistic selection arising from caste-antagonistic pleiotropy in haplodiploid social insects. Here, we present the special case where gene effects were additive at a single locus that affected caste 'performance', which in turn affected colony fitness (details in the electronic supplementary material). In this model, queens reproduced and were subject to direct selection, whereas workers were incapable of reproduction and subject exclusively to kin selection. We assumed that allele *a* was favoured in queens and allele *A* was favoured in workers. We then determined the combinations of selection coefficients in which either allele was fixed or both alleles were maintained as a polymorphism. Our interest was in understanding whether kin selection in workers was overwhelmed by direct selection acting in the opposite direction in queens, and whether caste-antagonistic pleiotropy was likely to lead to detectable genetic patterns within the genomes of social species.

We found that the region of the parameter space in which the queen-favoured allele fixed was the same size as the region in which the worker-favoured allele fixed, but only when queens mated once (figure 2*b*,*c*). By contrast, when queens mated with multiple males, the region of the parameter space in which the worker-favoured allele fixed was substantially smaller, and the region where the queen-favoured allele fixed was substantially larger (figure 2*b*,*c*). These differences arose because kin selection operating on workers was weaker than direct selection operating on queens when queens mated many times. Regardless, in both cases, the region of the parameter space allowing polymorphism was limited, especially when selection coefficients were realistically small (figure 2*c*).

4. Outlook: kin selection and social insect genomics

The revolution in social insect genomics [12–14] now allows rates of evolution and levels of polymorphism to be determined for all loci across multiple genomes. Thus, population genetic predictions arising from kin selection theory can be tested using newly emerging genomic data.

The model presented here makes three predictions. First, antagonistic selection across castes is unlikely to maintain polymorphism. Consequently, loci affecting traits in multiple castes are not expected to show high levels of polymorphism compared with other loci. Overall, this suggests that factors other than caste-antagonistic pleiotropy may be responsible for the maintenance of genetic polymorphism in social species.

Second, the model predicts that the evolution of antagonistic alleles that are favoured in workers, but disfavoured in queens, is impeded by multiple mating by queens. Such worker-beneficial alleles are strong candidates for alleles that would lead to distinct worker phenotypes. Consequently, these results suggest that phenotypic differentiation between the queen and worker castes, or within the worker caste, could have evolved more easily in species with singly mated queens, and that multiple mating hinders the evolution of caste differences.

Interestingly, currently available empirical data suggest that hymenopteran social insects that have genetically diverse colonies (e.g. are headed by multiply mated queens) have more phenotypically diverse workers [15]. If phenotypic differentiation in workers is due to selection in workers, then available data are inconsistent with our theoretical expectations, suggesting that caste-antagonistic pleiotropy is not a pervasive force. However, caste-antagonistic pleiotropy may have been important in the early evolution of sociality, when castes were first evolving and gene expression patterns in proto-queens and workers were similar. Subsequently, caste-antagonistic pleiotropy could have been resolved through the evolution of differential expression of genes between castes [16–19], allowing worker phenotypic differences to result from genes under selection (e.g. expressed) in workers only.

Third, our model predicts that adaptive evolution of genes that function in both queens and workers is more likely to be due to fixation of alleles that give queen-favoured phenotypes in species with multiply mated queens. By contrast, adaptive alleles are expected to be just as likely to give worker-favoured as queen-favoured phenotypes in species with singly mated queens. If adaptive evolution in social species is driven primarily by selection on performance of workers, then genes that function in both queens and workers should show higher rates of adaptive evolution in species with singly mated, rather than multiply mated, queens. Conversely, if adaptive evolution is driven by selection on queen performance, the opposite pattern is predicted. Thus, the patterns of molecular evolution may give insight into whether selection acts primarily on worker or queen performance.

In conclusion, models for interpreting genomic data have great potential for testing kin selection theory by determining how social evolution affects molecular evolution (e.g. [20–27]). Moreover, these investigations provide further inspiration for the development of new theory aimed at generating predictions regarding how genes should evolve under direct and kin selection. With several large-scale sequencing projects in progress, the genomes of social animals offer a natural playground for data and theory to come together in testing kin selection theory and providing insight into the evolution of social behaviours.

References

1. Hamilton WD. 1996 *Narrow roads of gene land: evolution of social behavior*. New York, NY: W. H. Freeman & Co.
2. West SA, Griffin AS, Gardner A. 2007 Social semantics: altruism, cooperation, mutualism, strong reciprocity and group selection. *J. Evol. Biol.* **20**, 415–432. (doi:10.1111/j.1420-9101.2006.01258.x)
3. Queller DC, Strassmann JE. 1998 Kin selection and social insects. *Bioscience* **48**, 165–175. (doi:10.2307/1313262)
4. Abbot P *et al*. 2011 Inclusive fitness theory and eusociality. *Nature* **471**, E1–E4. (doi:10.1038/nature09831)
5. West SA, Griffin AS, Gardner A, Diggle SP. 2006 Social evolution theory for microorganisms. *Nat. Rev. Microbiol.* **4**, 597–607. (doi:10.1038/nrmicro1461)
6. Dudley SA, File AL. 2007 Kin recognition in an annual plant. *Biol. Lett.* **3**, 435–438. (doi:10.1098/rsbl.2007.0232)
7. Sachs JL, Mueller UG, Wilcox TP, Bull JJ. 2004 The evolution of cooperation. *Q. Rev. Biol.* **79**, 135–160. (doi:10.1086/383541)
8. Linksvayer TA, Wade MJ. 2009 Genes with social effects are expected to harbor more sequence variation within and between species. *Evolution* **63**, 1685–1696. (doi:10.1111/j.1558-5646.2009.00670.x)
9. Hall DW, Goodisman MAD. 2012 The effects of kin selection on rates of molecular evolution in social insects. *Evolution* **66**, 2080–2093. (doi:10.1111/j.1558-5646.2012.01602.x)
10. Kovacs JL, Hoffman EA, Marriner SM, Goodisman MAD. 2010 Detecting selection on morphological traits in social insect castes: the case of the social wasp *Vespula maculifrons*. *Biol. J. Linnean Soc.* **101**, 93–102. (doi:10.1111/j.1095-8312.2010.01495.x)
11. Bonduriansky R, Chenoweth SF. 2009 Intralocus sexual conflict. *Trends Ecol. Evol.* **24**, 280–288. (doi:10.1016/j.tree.2008.12.005)
12. Robinson GE, Fernald RD, Clayton DF. 2008 Genes and social behavior. *Science* **322**, 896–900. (doi:10.1126/science.1159277)
13. Robinson GE *et al*. 2011 Creating a buzz about insect genomes. *Science* **331**, 1386. (doi:10.1126/science.331.6023.1386)
14. Foster KR. 2011 The sociobiology of molecular systems. *Nat. Rev. Genet.* **12**, 193–203. (doi:10.1038/nrg2903)
15. Fjerdingstad EJ, Crozier RH. 2006 The evolution of worker caste diversity in social insects. *Am. Nat.* **167**, 390–400. (doi:10.1086/499545)
16. Mank JE, Wedell N, Hosken DJ. 2013 Polyandry and sex-specific gene expression. *Phil. Trans. R. Soc. B* **368**, 20120047. (doi:10.1098/rstb.2012.0047)
17. Toth AL *et al*. 2007 Wasp gene expression supports an evolutionary link between maternal behavior and eusociality. *Science* **318**, 441–444. (doi:10.1126/science.1146647)
18. Goodisman MAD, Kovacs JL, Hunt BG. 2008 Functional genetics and genomics in ants (Hymenoptera: Formicidae): the interplay of genes and social life. *Myrmecol. News* **11**, 107–117.
19. Smith CR, Toth AL, Suarez AV, Robinson GE. 2008 Genetic and genomic analyses of the division of labour in insect societies. *Nat. Rev. Genet.* **9**, 735–748. (doi:10.1038/nrg2429)
20. Toth AL, Robinson GE. 2010 Evo-devo and the evolution of social behavior: brain gene expression analyses in social insects. In *Cold Spring Harbor Symposia on Quantitative Biology*, Vol. LXXIV, pp. 1–8. New York, NY: Cold Spring Harbor Laboratory Press.
21. Van Dyken JD, Wade MJ. 2012 Detecting the molecular signature of social conflict: theory and a test with bacterial quorum sensing genes. *Am. Nat.* **179**, 436–450. (doi:10.1086/664609)
22. Sucgang R *et al*. 2011 Comparative genomics of the social amoebae *Dictyostelium discoideum* and *Dictyostelium purpureum*. *Genome Biol.* **12**, Artn R20. (doi:10.1186/gb-2011-12-2-r20)
23. Fischman BJ, Woodard SH, Robinson GE. 2011 Molecular evolutionary analyses of insect societies. *Proc. Natl Acad. Sci. USA* **108**, 10 847–10 854. (doi:10.1073/pnas.1100301108)
24. Hunt BG, Wyder S, Elango N, Werren JH, Zdobnov EM, Yi SV, Goodisman MAD. 2010 Sociality Is linked to rates of protein evolution in a highly social insect. *Mol. Biol. Evol.* **27**, 497–500. (doi:10.1093/molbev/msp225)
25. Hunt BG, Ometto L, Wurm Y, Shoemaker D, Yi SV, Keller L, Goodisman MAD. 2011 Relaxed selection is a precursor to the evolution of phenotypic plasticity. *Proc. Natl Acad. Sci. USA* **108**, 15 936–15 941. (doi:10.1073/pnas.1104825108)
26. Bromham L, Leys R. 2005 Sociality and the rate of molecular evolution. *Mol. Biol. Evol.* **22**, 1393–1402. (doi:10.1093/molbev/msi133)
27. Viljakainen L, Evans JD, Hasselmann M, Rueppell O, Tingek S, Pamilo P. 2009 Rapid evolution of immune proteins in social insects. *Mol. Biol. Evol.* **26**, 1791–1801. (doi:10.1093/molbev/msp086)

Online supplementary materials - doi: 10.1098/rsbl.2013.0309

Kin selection, genomics, and caste-antagonistic pleiotropy

David W. Hall[1], Soojin V. Yi[2], and Michael A. D. Goodisman[2]

[1] Department of Genetics, University of Georgia. Athens, GA 30602. [2] School of Biology, Georgia Institute of Technology, Atlanta, GA 30332.

Model and analysis of intralocus caste conflict

Our model is similar in general construction to that of Hall and Goodisman [1]. We model a population of hymenopteran (i.e., haplodiploid) social insects, such that females (queens and workers) are diploid and males are haploid. We assume a colony consists of a single queen and her daughter workers. All colonies in a population produce reproductives synchronously and generations do not overlap. Mating is random and all queens in a population mate the same number of times, either once under single mating, or many times under multiple mating.

We assume that antagonistic selection arises because variation at a single locus affects a trait that has different optima in queens and workers. Expressing a trait value that is non-optimal results in a reduction in performance. Variation in the trait, and thus performance, is determined by an individual's genotype at a single locus, which segregates for two alleles, *A* and *a*. The effects of genetic variation on queen and worker performance is antagonistic. Specifically, the *A* allele results in higher performance in workers and lower performance in queens, and vice-versa for the *a* allele. Variation at this locus affects a phenotype that has a different optimum in the two castes, such as flight muscle production, mandible size, or ovary activity. Thus the optimal value of the phenotype that maximizes performance in queens (active ovaries, for example) reduces the performance of workers, and vice versa. Queens that are homozygous *aa* have the optimal queen trait value, and maximum performance (= 1). Likewise, workers that are homozygous *AA* have the optimal worker trait value, and maximum performance (= 1). All other caste genotypes have suboptimal performance.

Caste performance affects colony 'fitness', which is equivalent to number of reproductives produced by the colony. If a queen has a genotype that reduces her performance by half, then her colony produces half as many reproductives. Likewise, the production of reproductives by a colony is reduced by half if the average performance of workers is reduced by half. The number of reproductives produced by the colony is the product of queen performance and the mean performance of all the workers in the colony. This framework is essentially identical to that used in models of sexually antagonistic loci in which particular genotypes experience different fitness in males and females (e.g., [2]). For simplicity, we assume additive effects in both castes, such that the performance of the heterozygote falls exactly in between the two homozygotes. The performances of the genotypes are:

Genotype	Queen performance	Worker performance
AA	$1 - s_Q$	1
Aa	$1 - s_Q/2$	$1 - s_W/2$
aa	1	$1 - s_W$

where s_Q and s_W are the strengths of selection on performance acting against the *A* and *a* alleles in queens and workers, respectively.

The outcome of caste-antagonistic pleiotropy depends on the actual range of selection coefficients in queens and workers. However, little is known about the fitness effects of new mutations on performance of queens and workers in social insects. In addition, we assume that colonies produce equal sex ratios of new queens and males. Thus we do not consider cases of sex ratio bias that may occur in some social species [3].

With these assumptions, we obtain the following recursions in genotype frequencies in queens and males when queens are singly mated:

$$Tf'_{AA} = \left(f_{AA}(1-s_Q)p_m + f_{Aa}\left(1-\frac{s_Q}{2}\right)\frac{p_m}{2}\left(1-\frac{s_W}{4}\right)\right);$$

$$Tf'_{Aa} = f_{AA}(1-s_Q)q_m\left(1-\frac{s_W}{2}\right) + f_{Aa}\left(1-\frac{s_Q}{2}\right)\left(\frac{p_m}{2}\left(1-\frac{s_W}{4}\right)+\frac{q_m}{2}\left(1-\frac{3s_W}{4}\right)\right) + f_{aa}p_m\left(1-\frac{s_W}{2}\right);$$

$$Tf'_{aa} = f_{aa}q_m(1-s_W) + f_{Aa}\left(1-\frac{s_Q}{2}\right)\frac{q_m}{2}\left(1-\frac{3s_W}{4}\right);$$

$$Tp'_m = f_{AA}(1-s_Q)\left(p_m + q_m\left(1-\frac{s_W}{2}\right)\right) + \frac{f_{Aa}}{2}\left(1-\frac{s_Q}{2}\right)\left(p_m\left(1-\frac{s_W}{4}\right)+q_m\left(1-\frac{3s_W}{4}\right)\right);$$

$$Tq'_m = f_{aa}(1-s_W)\left(p_m\left(1-\frac{s_W}{2}\right)+q_m(1-s_W)\right) + \frac{f_{Aa}}{2}\left(1-\frac{s_Q}{2}\right)\left(p_m\left(1-\frac{s_W}{4}\right)+q_m\left(1-\frac{3s_W}{4}\right)\right);$$

where

$$T = f_{AA}(1-s_Q)\left(p_m + q_m\left(1-\frac{s_W}{2}\right)\right) + f_{Aa}\left(1-\frac{s_Q}{2}\right)\left(p_m\left(1-\frac{s_W}{4}\right)+q_m\left(1-\frac{3s_W}{4}\right)\right) + f_{aa}(1-s_W)\left(p_m\left(1-\frac{s_W}{2}\right)+q_m(1-s_W)\right).$$

For multiply mated queens, the recursions are:

$$Tf'_{AA} = f_{AA}(1-s_Q)p_m\left(p_m + q_m\left(1-\frac{s_W}{2}\right)\right) + f_{Aa}\left(1-\frac{s_Q}{2}\right)\frac{p_m}{4}\left(p_m + \left(1-\frac{s_W}{2}\right)+q_m(1-s_W)\right);$$

$$Tf'_{Aa} = f_{AA}(1-s_Q)q_m\left(p_m + q_m\left(1-\frac{s_W}{2}\right)\right) + \frac{f_{Aa}}{4}\left(1-\frac{s_Q}{2}\right)\left(p_m + \left(1-\frac{s_W}{2}\right)+q_m(1-s_W)\right) + f_{aa}p_m\left(p_m\left(1-\frac{s_W}{2}\right)+q_m(1-s_W)\right);$$

$$Tf'_{aa} = f_{aa}q_m\left(p_m\left(1-\frac{s_W}{2}\right)+q_m(1-s_W)\right) + f_{Aa}\left(1-\frac{s_Q}{2}\right)\frac{q_m}{4}\left(p_m + \left(1-\frac{s_W}{2}\right)+q_m(1-s_W)\right);$$

$$Tp'_m = f_{AA}(1-s_Q)\left(p_m + q_m\left(1-\frac{s_W}{2}\right)\right) + \frac{f_{Aa}}{4}\left(1-\frac{s_Q}{2}\right)\left(p_m + \left(1-\frac{s_W}{2}\right)+q_m(1-s_W)\right);$$

$$Tq'_m = f_{aa}(1-s_W)\left(p_m\left(1-\frac{s_W}{2}\right)+q_m(1-s_W)\right) + \frac{f_{Aa}}{4}\left(1-\frac{s_Q}{2}\right)\left(p_m + \left(1-\frac{s_W}{2}\right)+q_m(1-s_W)\right);$$

where

$$T = f_{AA}(1-s_Q)\left(p_m + q_m\left(1-\frac{s_W}{2}\right)\right) + f_{Aa}\left(1-\frac{s_Q}{2}\right)\left(p_m + \left(1-\frac{s_W}{2}\right)+q_m(1-s_W)\right) + f_{aa}(1-s_W)\left(p_m\left(1-\frac{s_W}{2}\right)+q_m(1-s_W)\right).$$

In all recursions, f_{AA}, f_{Aa} and f_{aa} are the frequencies of queens of genotypes *AA*, *Aa* and *aa* respectively, and p_m and q_m are the frequencies of *A* and *a* males respectively.

To determine the global behavior of the system, we examined the conditions for the initial increase of each allele when rare. Our goal here was to determine the combinations of selection coefficients in queens and workers that resulted in one of four possible outcomes. (1) the *A* allele increases when rare and the *a* allele is lost when rare. In this case the *A* allele is expected to go to fixation. (2) The *a* allele increases when rare and the *A* allele is lost when rare. In this case the *a* allele is expected to go to fixation. (3) Both alleles increase when rare, in which case a protected polymorphism will result [4]. (4) Both alleles are lost when rare, implying that neither allele can invade a population fixed for the other (we never observed this outcome).

The condition for initial increase of an allele is determined by first calculating the Jacobian matrix at each fixation. The leading eigenvalue of the Jacobian matrix determines the stability of the fixation. We used Mathematica (version 9.0.1, Wolfram Research 2013) to calculate the Jacobian and its leading eigenvalue at each fixation. In all cases the leading eigenvalue was real and positive, implying instability when it is greater than 1, and stability when it is less than 1 [5]. The eigenvalues are:

Model		Leading eigenvalue
Singly-mated queen	*a* allele fixed	$\frac{(2-s_Q)(4-3s_W)+\sqrt{(2-s_Q)(4-3s_W)(72-38s_W-s_Q(4-3s_W))}}{32(1-s_w)}$
	A allele fixed	$\frac{(2-s_Q)(4-s_W)+\sqrt{(2-s_Q)(4-s_W)\left(72-34s_W-s_Q(68-33s_W)\right)}}{32(1-s_w)}$
Multiply-mated queen	*a* allele fixed	$\frac{(2-s_Q)(4-3s_W)+\sqrt{(2-s_Q)(4-3s_W)(72-70s_W-s_Q(4-3s_W))}}{32(1-s_w)}$
	A allele fixed	$\frac{(2-s_Q)(4-s_W)+\sqrt{(2-s_Q)(4-s_W)\left(72-2s_W-s_Q(68-s_W)\right)}}{32(1-s_w)}$

The analysis of the magnitude of the eigenvalues relative to 1 elucidated the regions in which the three possible outcomes shown in figure 2b,c: fixation of the worker favored *A* allele, fixation of the queen favored *a* allele, and protected polymorphism.

The *A* allele fixes when it is not too deleterious in queens and is highly beneficial in workers. The *a* allele fixes when it is not too deleterious in workers and highly beneficial in queens. Polymorphism results when selection is very strong in both queens and workers, such that the disfavored homozygote in each is very unfit. Finally, when queens are multiply mated, selection through worker performance is less efficacious because worker relatedness to reproductives is lower, and the region of the parameter space leading to fixation of the worker-favored *A* allele is smaller.

References

1. Hall DW, Goodisman MAD. 2012 The effects of kin selection on rates of molecular evolution in social insects. *Evolution* **66**, 2080-2093.

2. Connallon T, Clark AG. 2013 Antagonistic versus nonantagonistic models of balancing selection: Characterizing the relative timescales and hitchhiking effects of partial selective sweeps. *Evolution* **67**, 908-917.

3. West S. 2009 *Sex allocation*. Princeton, NJ, Princeton University Press.

4. Prout T. 1968 Sufficient conditions for multiple niche polymorphism. *Am. Nat.* **102**, 493-496.

5. Otto SP, day T. 2007 *A biologist's guide to mathematical modeling in ecology and evolution*. Princeton, NJ, Princeton University Press.