Maternal effect killing by a supergene controlling ant social organization

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Supergenes underlie striking polymorphisms in nature, yet the evolutionary mechanisms by which they arise and persist remain enigmatic. These clusters of linked loci can spread in populations because they captured coadapted alleles or by selfishly distorting the laws of Mendelian inheritance. Here, we show that the supergene haplotype associated with multiple-queen colonies in Alpine silver ants is a maternal effect killer. All eggs from heterozygous queens failed to hatch when they did not inherit this haplotype. Hence, the haplotype specific to multiple-queen colonies is a selfish genetic element that enhances its own transmission by causing developmental arrest of progeny that do not carry it. At the population level, such transmission ratio distortion favors the spread of multiple-queen colonies, to the detriment of the alternative haplotype associated with single-queen colonies. Hence, selfish gene drive by one haplotype will impact the evolutionary dynamics of alternative forms of colony social organization. This killer hidden in a social supergene shows that large nonrecombining genomic regions are prone to cause multifarious effects across levels of biological organization.

selfish genetic elements | maternal effect killer | transmission ratio distortion | supergene | queen number

Supergenes control a variety of complex polymorphic phenotypes, including sexes, distyly, ecotypes, cryptic morphs, mating morphs, and alternative forms of social organization (1–5). How did such supergenes evolve? A prevailing model is that the suppression of recombination linked alleles that are advantageous or show positive epistasis in only one of the discrete phenotypes, resulting in clusters of coadapted alleles. Natural selection then favored the spread of these clusters of coadapted alleles inherited as single nonrecombining haplotypes (6, 7). In line with this adaptive view, several supergenes contain coadapted alleles (2, 8–11).

Another nonmutually exclusive model is that supergenes spread selfishly. The suppression of recombination exacerbates intragenomic conflict, as cotransmitted alleles gain power to favor their own transmission over that of alternative alleles, at a cost to their bearers (12–14). Selfish genetic elements that distort Mendelian transmission, here defined as the expected 1:1 transmission ratio of each allele from a heterozygous parent to adult offspring, typically arise through tight linkage of a distorter and target locus, followed by further accumulation of enhancer loci (14, 15). Therefore, large nonrecombining genomic regions are likely not only to control complex phenotypes, but also to ally against other genes and cause transmission ratio distortion (12, 16–20). Such transmission ratio distortion may thus play a major role in the initial increase in frequency and later maintenance of supergenes.

Supergenes can increase their own transmission through meiotic drive, green-beard effects, or maternal effect killing (14, 21). Meiotic drive is characterized by the overrepresentation of one allele in the gametes of heterozygous individuals (16, 22, 23). Green-beard effects occur when the carrier of an allele recognizes and favors individuals that also carry this allele (24, 25). Finally, maternal effect killers are genetic elements present in the parent that cause the death of the progeny that did not inherit this element (26–29). So far, maternal effect killing by large supergenes controlling complex phenotypes has not been documented (2, 10). More generally, despite growing evidence that selfish genetic elements are common and play a major role in evolutionary innovations, their effect on supergene dynamics remains underappreciated.

A large supergene that controls colony social organization appears to selfishly distort the laws of Mendelian transmission in the Alpine silver ant, *Formica selysi* (5, 30). In colonies where a single queen reproduces, all females are homozygous for the Sm supergene haplotype. In contrast, in colonies where multiple queens contribute to offspring production, all females have one or two copies of the Sp haplotype (Fig. 1). As many as 51.3% of the mature queens heading multiple-queen colonies are Sp/Sm, and 22.7% of the Sp/Sm queens are mated with Sm males (30). Heterozygous queens are expected to produce Sm males and, when mated with Sm males, Sm/Sm females. Yet, multiple-queen colonies never produce Sm/Sm females nor Sm males. Hence, genotype frequencies in multiple-queen colonies in the field provide indirect evidence that the Sp haplotype is a transmission ratio distorter (5, 30), but so far the distortion has not been directly observed and the mechanism remains unknown. Here, we perform a series of experiments to investigate the occurrence and mechanism of drive by the social supergene.

Significance

Supergenes are clusters of linked loci producing complex alternative phenotypes. In a series of experiments, we demonstrate that a supergene controlling ant social organization distorts Mendel's laws to enhance its transmission to adult offspring. One supergene haplotype is specific to multiple-queen colonies. This haplotype kills half of the offspring from heterozygous mothers—all eggs that do not inherit the haplotype fail to hatch. Hence, the haplotype associated with multiple-queen colonies is a selfish genetic element favoring its transmission to the detriment of the alternate haplotype associated with single-queen colonies. Selfish gene drive by a large group of linked genes impacts the social organization of ant colonies, which illustrates the intricate multilevel effects of supergenes.

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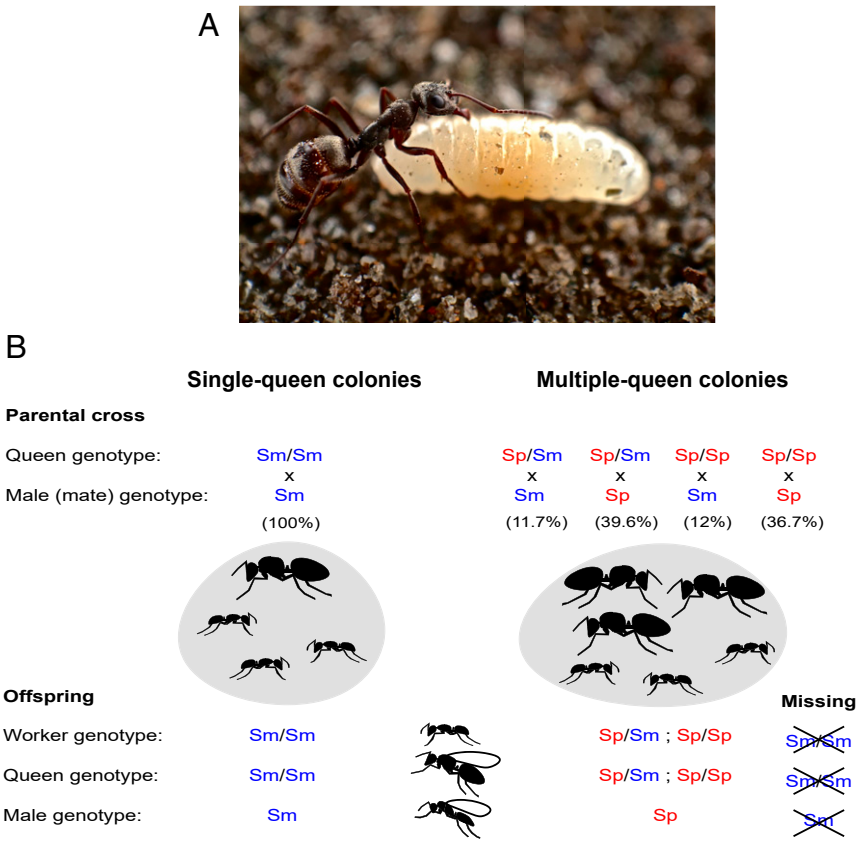

Fig. 1. Study species and genetic system underlying variation in social organization. (*A*) Worker and larva of Alpine silver ant, *F. selysi*. Image credit: Timothée Brütsch (University of Lausanne, Lausanne, Switzerland). (*B*) In this species, a supergene with two haplotypes, Sm and Sp, determines colony social organization (5, 30). Queens established in single-queen colonies (*Left*) have exclusively the supergene genotype Sm/Sm. They mated with Sm males and produce Sm/Sm workers, winged Sm/Sm females destined to become queens, and winged Sm males (males are haploid) (5, 30). By contrast, queens established in multiple-queen colonies (*Right*) always have at least one copy of the Sp haplotype, being either Sp/Sm or Sp/Sp. These queens mated with Sm males or Sp males (5, 30, 37). Percentages of each parental cross are indicated in parentheses, as inferred from previous sampling in field colonies ($n = 63$ queens from single-queen colonies and 150 queens from multiple-queen colonies) (30). Strikingly, queens established in multiple-queen colonies produce only Sp/Sm and Sp/Sp workers and winged females, along with winged Sp males (5, 30). The complete absence of Sm/Sm females and Sm males in brood produced by multiple-queen colonies in the field suggests that the Sp haplotype is a transmission ratio distorter.

Results

Experimental Approach. To document the occurrence of transmission ratio distortion, we isolated Sp/Sm queens that had mated with Sm males and genotyped their brood at the social supergene (*Materials and Methods*). To differentiate between meiotic drive and maternal effect killing, we genotyped eggs that were less than 3 d old and 3 to 6 d old, as well as young larvae. To detect potential green-beard effects from rearing workers, the brood was reared in the absence of workers, with workers from single-queen colonies or with workers from multiple-queen colonies.

Evidence for Maternal Effect Killing. There was complete transmission ratio distortion in favor of the Sp haplotype, and the pattern of brood mortality indicated that this haplotype was a maternal effect killer (Figs. 2 and 3 and *SI Appendix*, Fig. S1). Queens heterozygous at the social supergene and mated with Sm males laid Sm/Sm and Sm/Sp eggs in Mendelian proportions (Fig. 2). Strikingly, all Sm/Sm eggs failed to hatch into larvae, while Sp/Sm eggs from the same clutches hatched normally. As a result, all surviving larvae were heterozygous at the social supergene (Fig. 2).

The mortality of Sm/Sm brood depended on the presence of Sp in their mother. Indeed, Sm/Sm eggs coming from Sp-carrying queens did not hatch into larvae (mortality rate 100%). In contrast, Sm/Sm brood from queens that lacked Sp developed normally, with mortality rates similar to the ones of brood with alternative genotypes (Fig. 3). The maternally induced mortality of Sm/Sm brood laid by Sp/Sm queens occurred irrespective of whether the brood was unattended, reared by Sm/Sm workers, or reared by Sm/Sp and Sp/Sp workers (Figs. 2 and 3 and *SI Appendix*, Fig. S1).

No Evidence for Green-Beard Effects. We found no sign that the Sp haplotype caused green-beard effects. The presence and social origin of rearing workers did not alter the 1:1 Mendelian ratio in eggs and did not explain the differential mortality of embryos with alternative genotypes (Figs. 2 and 3 and *SI Appendix*, Fig. S1). Hence, workers from multiple-queen colonies, which carry at least one Sp haplotype, did not selectively eliminate brood that lacked Sp.

No Evidence for Meiotic Drive. There was no indication of meiotic drive in the eggs laid by queens heterozygous at the social supergene. The proportion of eggs carrying the Sp and Sm maternal haplotype did not depart significantly from the 1:1 Mendelian ratio, irrespective of whether the Sp/Sm queens had mated with Sm males or Sp males (Fig. 4).

Discussion

We provide strong evidence that the haplotype associated with multiple-queen colonies (Sp) in Alpine silver ants is a maternal

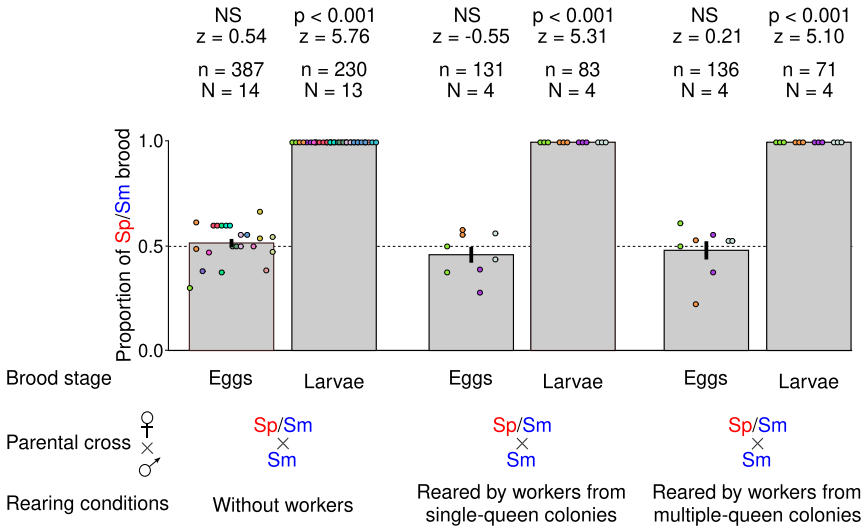

Fig. 2. Evidence that the Sp supergene haplotype is a maternal effect killer. All Sm/Sm eggs laid by heterozygous queens failed to hatch into larvae, while Sp/Sm eggs from the same clutches developed normally. This differential mortality between homozygous and heterozygous brood from heterozygous queens occurred in all rearing conditions, namely when the brood was unattended (*Left*), reared by workers from single-queen colonies (*Center*), or by workers from multiple-queen colonies (*Right*), respectively. The proportion of Sp/Sm brood was calculated across rearing units (small circles). Queen identity (shown by color) was included as a random factor in the statistical models to control for the nonindependence of brood from the same mother. The mean ± SE are shown by gray bars and vertical lines, respectively. The number of live brood items collected and genotyped (n) and the number of queens (N) are shown above the bars. *P* values above bars indicate significant departures from 1:1 Mendelian ratio, after Bonferroni correction for multiple comparisons; NS means that the deviation was not significant (GLMMs, Wald tests, z-values above bars).

effect killer. Queens heterozygous at the supergene produced eggs that inherited each haplotype in the expected 1:1 Mendelian ratio. Later in their development, all Sm/Sm eggs from heterozygous queens failed to hatch into larvae, while Sm/Sm eggs from homozygous queens hatched normally. Hence, the presence of the Sp haplotype in the mother induced developmental arrest of her brood that did not inherit this haplotype. This is a demonstration of a supergene controlling complex phenotypes that favors its own transmission through maternal effect killing.

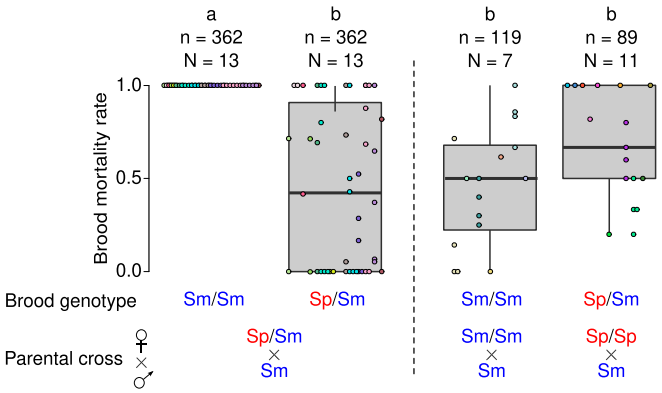

Fig. 3. Evidence for lethal maternal effect in the absence of rearing workers. Mortality rate until the larval stage for: Sm/Sm brood from Sp/Sm queens mated with Sm males (first column); Sp/Sm brood from Sp/Sm queens mated with Sm males (second column); Sm/Sm brood from Sm/Sm queens mated with Sm males (third column); Sp/Sm brood from Sp/Sp queens mated with Sm males (fourth column). In the absence of workers, all Sm/Sm brood laid by heterozygous queens died. The mortality rate of Sm/Sm brood laid by heterozygous queens (first column) was significantly higher than the one of both heterozygous brood (second and fourth columns) and Sm/Sm brood laid by homozygous queens (third column). Brood mortality rate was calculated across rearing units (small circles). Queen identity (shown by color) was included as a random factor in the statistical model. The median mortality rate is shown by horizontal lines. Boxplots depict the lower and upper quartiles and the whiskers encompass 1.5 times the interquartile range. The number of eggs at the start of the experiment (n) and the number of queens (N) are shown above the bars. The effect of brood genotype on mortality rate depended on the maternal genotype (GLMM; $\chi^2_{(3)} = 828$, $P < 0.0001$). Different letters above bars indicate significant differences in mortality (post hoc Tukey's HSD test for multiple comparisons, $P < 0.05$).

Known cases of maternal effect killing are based on modification-rescue systems, for example a maternally expressed toxin transmitted to eggs coupled with an embryo-expressed antidote (14). In *Tribolium castaneum*, maternal effect killing is associated with a large transposable element inserted into the host genome (26, 27). In *Caenorhabditis elegans*, a maternally delivered toxin is tightly linked to an antidote in an inversion polymorphism (29). Although the mechanisms causing developmental arrest can be complex and diverse (14), involving protein-coding genes or regulatory elements, cases documented so far involve tightly linked loci.

The suppression of recombination also generates other types of transmission ratio distorters. Clusters of tightly linked loci are involved in meiotic drive by the *t*-locus in *Mus musculus* (22) and by the *Segregation Distorter* gene complex in *Drosophila melanogaster* (23). The social supergene of the fire ant *Solenopsis invicta* is associated with unusual transmission ratios, likely due to the joint effects of meiotic drivers and suppressors (16). The same supergene also causes a green-beard effect (24, 25). Overall, the emerging picture is that large nonrecombining genomic regions are prone to harbor selfish genetic elements that distort Mendelian transmission ratio in many ways.

Selfish genetic elements are difficult to observe, because they either spread to fixation or are repressed by other genes (20, 31). The ones that persist are often recessive lethal or near-lethal, so that negative frequency-dependent selection prevents the fixation of the driving allele (14, 19, 22, 24). In the Alpine silver ant, viable homozygotes are commonly found for both haplotypes of the social supergene (5, 30). The social polymorphism appears stable and is widespread across *F. selysi* populations (32, 33). Moreover, the genetic polymorphism is shared across species separated by 20–40 My of independent evolution (34).

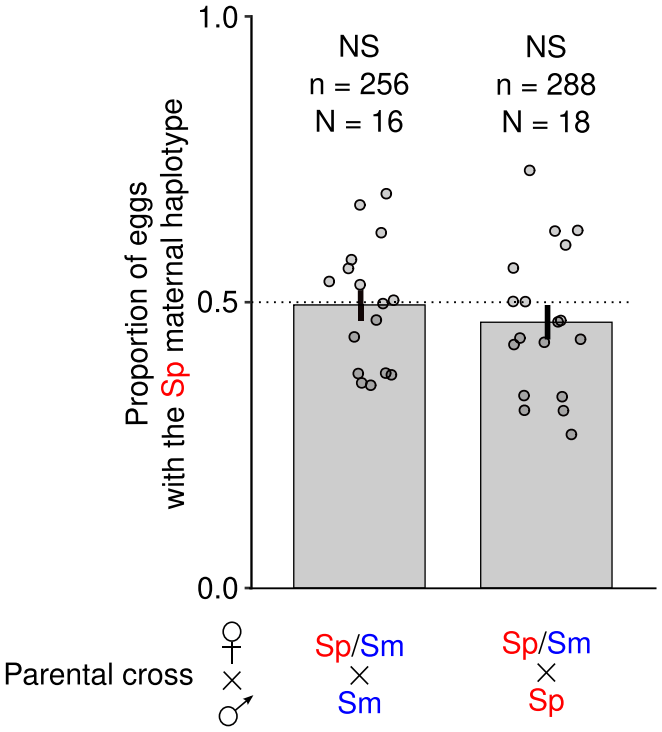

Fig. 4. No evidence for meiotic drive. Heterozygous queens transmitted the Sp and Sm haplotype to their eggs in 1:1 Mendelian ratio. The proportion of eggs that inherited the Sp maternal haplotype was calculated across eggs laid by each individual queen (small circles). The mean ± SE are shown by gray bars and vertical lines, respectively. The number of eggs genotyped (n) and the number of queens (N) are shown above the bars. Meiosis did not deviate significantly from 1:1 ratio in queens mated with Sm males (*Left*; GLM, Wald test $z = -0.04$, $P = 0.97$) or Sp males (*Right*; GLM, Wald test $z = -0.30$, $P = 0.77$).

The unusual properties of the Sp haplotype, which drives without being lethal in homozygotes, raises the question of what balances the polymorphism and prevents the driving haplotype from going to fixation? Stable polymorphic equilibria can be maintained by overdominance, by various forms of negative frequency-dependent selection such as disassortative mating, or by selection in temporally or spatially heterogenous environments (35, 36). Mating is mostly assortative in mature field colonies of *F. selysi*, although no preference was detected in mate choice experiments (30, 37). Further investigations of the behavior and fitness of males, queens, and workers with alternative genotypes will provide a more complete picture of the dynamics of this system. Overdominance, genetic load reducing the fitness of Sp/Sp females or Sp males, reduced dispersal or colonization by Sp-carrying individuals, and spatially varying selection due to habitat heterogeneity are key candidates for limiting the spread of Sp and stabilizing the polymorphism.

In conclusion, in the social supergene of Alpine silver ants, a selfish genetic element causes maternal effect killing. The suppression of recombination in a large cluster of genes created a coadapted gene complex that selfishly distorts Mendelian transmission, while promoting a form of colony social organization in which reproduction is shared by multiple queens. Supergenes controlling colony queen number have evolved independently in two highly divergent ant lineages (5). In the fire ant, the haplotype linked to multiple-queen colonies generates a green-beard effect, as workers carrying this haplotype kill queens that lack it (17, 24, 25). In the Alpine silver ant, the haplotype associated with multiple-queen colonies is a maternal effect killer, causing developmental arrest of brood that did not inherit this haplotype. Strikingly, in both systems a killer is hidden in the social supergene, and selfish drive by a nonrecombining haplotype favors the spread of multiple-queen colonies. Yet, despite drive, these supergene haplotypes did not reach fixation over extended evolutionary periods (34, 38, 39). The long-term persistence of these polymorphisms indicates that they are balanced by antagonistic selective pressures at the gene, individual, and group levels. More generally, many supergenes are likely to have multifarious effects across levels of biological organization. The suppression of recombination makes supergenes prone to harbor selfish genetic elements facilitating their spread in populations, while genetic load or other counteracting forces protect the polymorphism.

Materials and Methods

Ant Sampling, Rearing, and Genotyping. To investigate the occurrence and mechanism of transmission ratio distortion by the social supergene of *F. selysi*, we sampled mature, naturally mated queens from multiple-queen colonies (30). In complement, we sampled young virgin queens from multiple-queen colonies and mated them with males of known social origin in flight cages (37, 40). We reared queens individually in small laboratory colonies and collected their egg and larva progeny as described previously (30). The social supergene genotype of each queen, sperm in their spermatheca, eggs and larvae were determined with a polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) assay that discriminates three SNPs diagnostic for alternative haplotypes of the supergene (5, 30).

Maternal Effect Killing. To detect postsegregational lethal maternal effects, we monitored brood mortality during development when the brood was unattended (no workers), reared by workers from single-queen colonies, or reared by workers from multiple-queen colonies, respectively (see below). If the Sp haplotype is a maternal effect killer, the Sm/Sm brood laid by Sp/Sm queens mated with Sm males should die during development, while Sp/Sm brood from the same clutch should develop normally, irrespective of the presence and social origin of workers. Control brood from Sm/Sm or Sp/Sp queens mated with Sm males should develop normally.

We tested the development of unattended brood from 15 Sp/Sm queens mated with Sm males. Ten queens were mature, naturally mated queens, and five were young queens mated in flight cages. As a control, we analyzed brood from 7 Sm/Sm and 11 Sp/Sp queens mated with Sm males in flight cages. Queens were isolated for 2 d. Using a soft paintbrush cleaned with ethanol, we collected their eggs individually. We placed the eggs side by side, without contact between them, onto sterile plastic plates that served as rearing units. The eggs were incubated in a climate chamber, at a temperature of 24 °C and a relative humidity of 95%. We collected half of the surviving eggs after 4 d in the incubator, when they were 4 to 6 d old. We then monitored the remaining brood on a daily basis for 12 d and collected all hatched larvae. We genotyped the social supergene of all eggs and larvae.

Green-Beard Effects. By cross-fostering eggs, we examined whether the social origin of rearing workers influenced the survival of brood. If the Sp haplotype causes green-beard effects, workers from multiple-queen colonies, which carry at least one Sp haplotype, should selectively eliminate Sm/Sm brood, while workers from single-queen colonies should spare it. Brood bearing the Sp haplotype should develop normally, irrespective of the social origin of workers.

We cross-fostered eggs from four mature Sp/Sm queens mated with Sm males into groups of workers from single-queen colonies or multiple-queen colonies, respectively. As controls, we similarly cross-fostered eggs from five Sp/Sp queens and five Sm/Sm queens mated with Sm males in flight cages. Queens were isolated for 3 d. Their eggs were transferred to small rearing groups of 20 workers of known social origin (a single-queen or a multiple-queen field colony; five colonies per social form). Each rearing group received 17.6 ± 4.2 (mean ± SD) eggs (a ratio of eggs to workers close to 1:1 maximizes brood survival; ref. 41). Eggs from Sp/Sm queens were placed in 40 rearing groups, 20 from each social form. Eggs from Sp/Sp queens and Sm/Sm queens were placed in 20 rearing groups, 10 from each social form. For Sp/Sm queens, we collected the surviving eggs after 3 d in eight rearing groups from each social form. These eggs were 3 to 6 d old. For all other rearing groups, we monitored brood on a daily basis for 12 d and collected all hatched larvae. We genotyped the social supergene of all eggs and larvae.

Meiotic Drive. To test if the Sp haplotype distorted Mendelian segregation during meiosis, we genotyped 16 eggs per queen from 34 mature Sp/Sm queens, of which 16 had mated with Sm males and 18 with Sp males, respectively. All eggs were less than 3 d old.

Statistical Analysis. To detect transmission ratio distortion, we examined whether the supergene genotypes of brood from Sp/Sm queens departed from the expected 1:1 Mendelian ratio. We constructed two separate generalized linear mixed models (GLMMs) with binomial error, one to test for postsegregational lethal maternal effects and the other to test for green-beard effects by rearing workers. In both models, the response variable was the proportion of Sp/Sm brood per rearing unit. We used the Wald test to assess whether the intercept was significant, revealing deviation from Mendelian ratio. In each model, we included queen identity as a random factor, to control for the nonindependence of brood from the same mother. We also weighted the proportion of Sp/Sm brood by the initial number of eggs per rearing unit. In tests of postsegregational lethal maternal effects, we used the brood developmental stage (eggs or larvae) as a fixed factor. When testing for green-beard effects, we included as a fixed factor the interaction between brood stage and social origin of workers. To test for meiotic drive, we constructed a generalized linear model (GLM). The response variable was the proportion of eggs that inherited the Sp maternal haplotype for each individual queen. We included the genotype (Sm or Sp) of the male (= the queen's mate) as a fixed factor.

To test whether the effect of brood genotype on brood mortality depended on maternal genotype, we constructed three GLMMs with a binomial error distribution—one model for offspring reared without workers, one for offspring reared by workers from single-queen colonies, and one for offspring reared by workers from multiple-queen colonies. Brood mortality rate per rearing unit was the response variable. As a fixed factor, we included the interaction between brood genotype and queen genotype. Queen identity was included as a random factor. We weighted brood mortality by the initial number of eggs per rearing unit. For heterozygous queens, we assumed an equal proportion of each genotype in the eggs (no meiotic drive), and the mortality rate was constrained between zero and one.

Larvae never carried the maternal Sm haplotype, causing complete separation in the data. Complete separation affects the estimation of regression coefficients in GLMMs. Therefore, when testing for postsegregational lethal maternal effects and green-beard effects by analyzing genotype proportions and mortality rates in brood, we applied Bayesian inference for regression models, using the "bglmer" function implemented in the R package "blme." We tested for pairwise differences in brood mortality with a post hoc Tukey's honestly significant difference (HSD) test, as implemented in the R package "multcomp." For the test of meiotic drive, we used the "glm" function implemented in the R package "stats." All of the statistics were performed in R version 3.6.1 (42).

Data Availability. The dataset has been deposited in Dataset S1.

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