

1 Monogamy promotes worker sterility in insect societies

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9 tion of inclusive-fitness theory that monogamy promotes altruistic sib-rearing in eusocial insects.

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18 **Abstract**

19 Inclusive-fitness theory highlights monogamy as a key driver of altruistic sib-rearing. Accordingly,
20 monogamy should promote the evolution of worker sterility in social insects when sterile workers
21 make for better helpers. However, a recent population-genetics analysis (Olejarz *et al.* 2015) found
22 no clear effect of monogamy on worker sterility. Here, we revisit this analysis. First, we relax
23 genetic assumptions, considering not only alleles of extreme effect—encoding either no sterility or
24 complete sterility—but also alleles with intermediate worker-sterility effects. Second, we broaden
25 the stability analysis—which focused on the invasibility of populations where either all work-
26 ers are fully-sterile or all workers are fully-reproductive—to identify where intermediate pure or
27 mixed evolutionarily-stable states may occur. Finally, we consider additional, demographically-
28 explicit ecological scenarios relevant to worker non-reproduction. This extended analysis demon-
29 strates that an exact population-genetics approach strongly supports the prediction of inclusive-
30 fitness theory that monogamy promotes sib-directed altruism in social insects.

31 **Introduction**

32 Altruism among animals is epitomised by the workers of insect societies, who sacrifice their per-
33 sonal reproductive success to promote their siblings' welfare. This remarkable self-abnegation—
34 seemingly at odds with the “survival of the fittest”—is traditionally explained by kin selection: a
35 gene causing workers to share provisions or defend the communal nest can spread if the workers'
36 sacrifice increases the survival of their siblings, who are likely to carry copies of the same gene.
37 Higher genetic relatedness between the altruist and her beneficiaries would therefore—all else
38 being equal—promote selection for altruism (Hamilton 1964). Accordingly, monogamy is often
39 highlighted as a key promoter of sibling altruism, since maternal promiscuity decreases related-
40 ness between siblings, diminishing the inclusive-fitness benefits of sib-rearing (Hamilton 1972;
41 Charlesworth 1978; Charnov 1978; Boomsma 2007, 2009, 2013; Gardner *et al.* 2012; Davies *et al.*
42 2016). A wealth of empirical evidence supports this view, revealing a strong association between

43 monogamy and sib-directed altruism in arthropods (Hughes *et al.* 2008), birds (Cornwallis *et al.*
44 2010), and mammals (Lukas & Clutton-Brock 2012).

45 A conspicuous example of sib-directed altruism in the social Hymenoptera (wasps, bees, and
46 ants) is worker sterility. In many hymenopteran species, female workers lay unfertilised eggs in
47 their natal colony, which develop into males on account of their haplodiploid mode of sex deter-
48 mination. But in some species, workers have partly or entirely stopped making sons to focus their
49 efforts on helping instead. A standard account of inclusive-fitness theory would predict that—as
50 with other forms of sibling altruism—monogamy should promote helpful worker sterility.

51 However, this prediction has recently been challenged by Olejarz *et al.*'s (2015) mathematical
52 analysis of worker sterility in haplodiploid insect colonies, which uses an intricate population-
53 genetics model to derive exact conditions for the invasion and stability of a worker-sterility allele.
54 Surprisingly, this analysis could not identify a consistent effect of monogamy on the evolution of
55 non-reproductive workers. In this *Research Advance*, we revisit this analysis, exploring alternative
56 assumptions concerning the genetics, evolution, and ecology of worker sterility. We find that
57 a more-comprehensive investigation of Olejarz *et al.*'s (2015) exact population-genetics approach
58 strongly supports the view that monogamy promotes helpful worker sterility in insect societies
59 and corroborates inclusive-fitness theory more generally.

60 **Unconstrained allelic effects: monogamy promotes worker steril-** 61 **ity**

62 Olejarz *et al.* (2015) investigated the spread of an allele that renders workers carrying the allele—
63 who would otherwise produce sons through arrhenotokous parthenogenesis, substituting them
64 for the queen's sons—completely sterile. As the proportion z of sterile workers in a colony in-
65 creases, the proportion p_z of males produced by the queen rather than by workers also increases,
66 while overall colony productivity r_z may increase or decrease. Following these assumptions, they
67 found that—in a seeming challenge to inclusive-fitness theory—worker sterility sometimes in-
68 vades under single mating ($n = 1$) only, sometimes under double mating ($n = 2$) only, sometimes

69 under both single and double mating, and sometimes under neither, suggesting no clear effect of
70 monogamy on the invasion of sterility (Olejarz *et al.* 2015).

71 To explore the generality of this unexpected finding, we take up a suggestion by Olejarz *et*
72 *al.* (2015, p. 13) and extend their analysis to consider alleles with intermediate effects on worker
73 sterility (as was done for a similar model by Olejarz *et al.* 2016). Intermediate-effect alleles may
74 exhibit incomplete penetrance (such that each carrier has an intermediate probability of being ster-
75 ile), or may encode intermediate phenotypes (such that each carrier divides her resources between
76 colony tasks and personal reproduction); these scenarios are mathematically equivalent, but for
77 simplicity, we focus on the former. This suggested extension seems particularly apt, as the incom-
78 plete penetrance of sterility has been shown to be important for the evolution of reduced worker
79 reproduction both in theory and in empirical practice (Charlesworth 1978; Ratnieks *et al.* 2006;
80 Wenseleers & Ratnieks 2006b; Ronai *et al.* 2016); indeed, some form of incomplete penetrance is
81 required to preserve the fecundity of queens carrying the sterility allele. Accordingly, we have de-
82 rived exact conditions for the invasion of a recessive or dominant sterility allele with arbitrary pen-
83 etrance (see Methods). When we require mutant worker-sterility alleles to show full penetrance,
84 our analysis exactly recovers Olejarz *et al.*'s (2015) results (Fig. 1a). However, when we allow mu-
85 tant worker-sterility alleles to show incomplete penetrance, we find that—strikingly—monogamy
86 always promotes the invasion of helpful worker sterility (Fig. 1b). (Note that monogamy may
87 inhibit worker sterility when sterility is harmful; see Methods.)

88 Why does allowing intermediate effects make such a categorical difference? The population
89 genetics of invasion is the key. For example, a recessive sterility allele, when rare, is almost always
90 expressed in colonies founded by a heterozygous female who has mated with one mutant male
91 and $n - 1$ wild-type males. Other colony types occur, but are either comparatively rare (because
92 they require more copies of the mutant allele among mating partners), or exhibit exactly the same
93 phenotype as wild-type colonies (because sterility is expressed only when both parents pass the
94 recessive mutant allele to their daughters). Therefore, sterility can only invade if these “mutant”
95 colonies—in which a proportion $z = \frac{v}{2n}$ of workers are sterile, where v is the allele's penetrance—
96 succeed in spreading the sterility allele. If we only permit alleles with full penetrance ($v = 1$) to

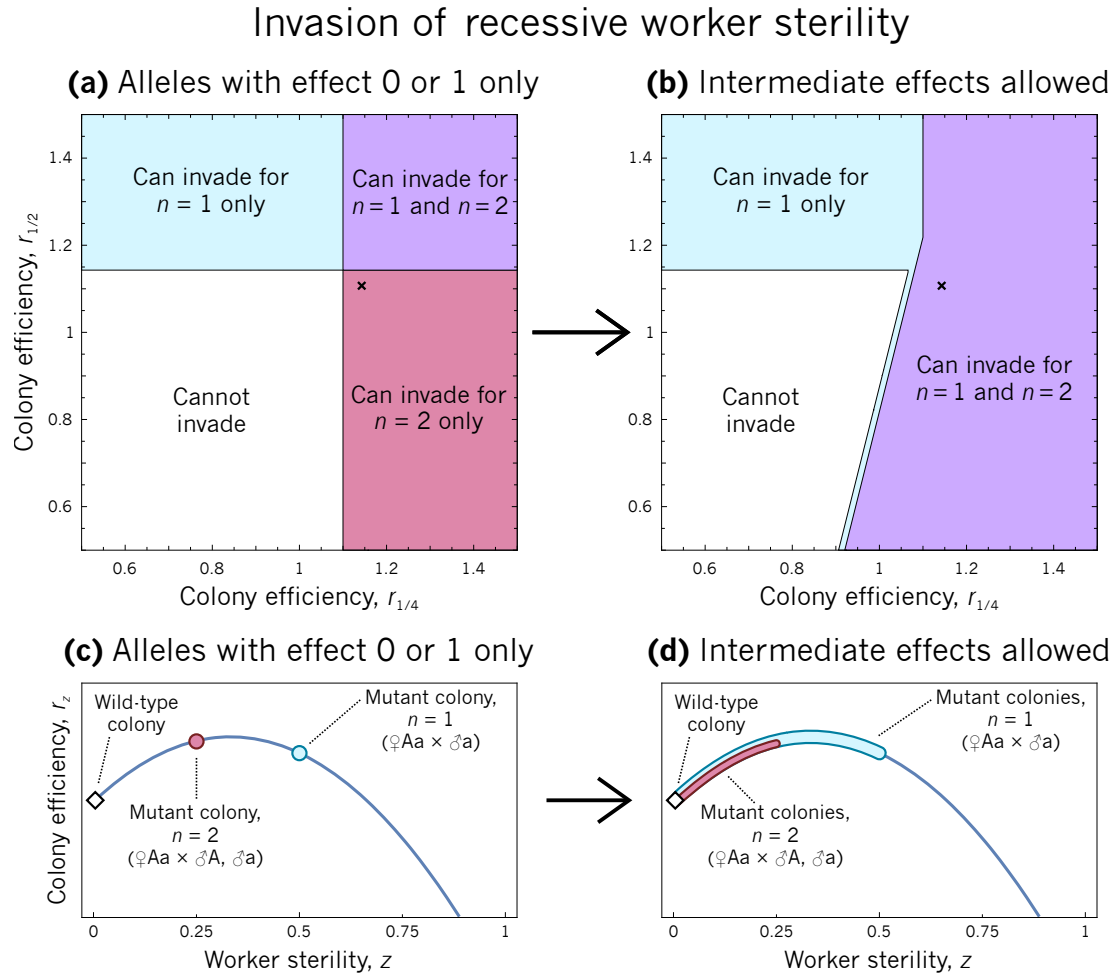


Figure 1: The invasion of worker sterility under recessive genetics, exploring the regions of parameter space where sterility can invade under single mating only, double mating only, both, or neither. **(a)** If we assume that only full-sterility alleles can arise, double mating sometimes promotes the invasion of sterility over single mating. But **(b)** if we assume that alleles encoding intermediate worker sterility may arise, double mating never promotes the invasion of sterility over single mating, depending on the colony efficiency values $r_0 = 1$, $r_{1/4}$, and $r_{1/2}$. This is because **(c)** for a rare allele encoding full sterility, mutant colonies have the phenotype $z = 1/2$ under single mating and $z = 1/4$ under double mating. Therefore, sterility may invade more easily under double mating if colony efficiency is relatively peaked near $z = 1/4$. But **(d)** for a rare allele encoding intermediate sterility, mutant colonies may express any phenotype $0 < z \leq 1/2$ under single mating and $0 < z \leq 1/4$ under double mating, depending on the allele's effect, and so mutant phenotypes are less constrained by the population's mating number. In order to facilitate comparison with Fig. 3A of Olejarz *et al.* (2015), we assume $p_z = 0.2 + 0.8z$, and for r_z we use the unique quadratic curve passing through the points specified by $r_0 = 1$, $r_{1/4}$, and $r_{1/2}$.

97 arise, this allelic constraint may overpower the altruism-promoting effect of higher relatedness:
98 for example, double mating ($n = 2$) may facilitate sterility's invasion over single mating ($n = 1$)
99 if colony efficiency is relatively high when $z = 1/4$ and relatively low when $z = 1/2$ (Fig. 1c). In
100 contrast, if we permit alleles with incomplete penetrance ($0 < v \leq 1$) to arise, mutant colonies
101 may exhibit any one of a range of phenotypes, depending on v (namely, $0 < z \leq 1/2$ for single
102 mating, and $0 < z \leq 1/4$ for double mating), and monogamy always promotes the invasion of
103 helpful worker sterility over promiscuity, by both maximizing sibling relatedness and allowing a
104 wider range of phenotypes to be explored (Fig. 1d; see Methods for the corresponding analysis
105 assuming dominant sterility).

106 **Beyond invasion: monogamy promotes worker sterility**

107 These results explain why promiscuity sometimes promotes the invasion of helpful sterility over
108 monogamy under specific genetic constraints. But to only consider whether sterility invades may
109 be misleading, for two reasons. First, that a sterility allele spreads from rarity says little about its
110 equilibrium frequency, which may be a more-relevant measure of monogamy's impact on worker
111 altruism than mere invasion. Indeed, although promiscuity sometimes promotes sterility's inva-
112 sion *per se* under constrained penetrance, we find that monogamy typically promotes equilibrium
113 sterility under the same conditions (Fig. 2).

114 Second, if we do allow intermediate-effect alleles, then considering only a single invasion is in-
115 adequate, because long-term evolution is likely to involve multiple successive invasions (*cf.* Ham-
116 merstein 1996). How can we predict the outcome without knowing in advance which alleles may
117 arise, and when? The solution is that, over the long term, populations exposed to sufficient genetic
118 variation will converge on an evolutionarily-stable strategy (ESS; Maynard Smith & Price 1973)—a
119 level of sterility that cannot be invaded by an allele encoding any other level of sterility. To identify
120 a candidate ESS for sterility, we further extend Olejarz *et al.*'s (2015) population-genetics analysis to
121 derive an exact condition for the invasion of an allele encoding a small increase to average sterility,

122 Z:

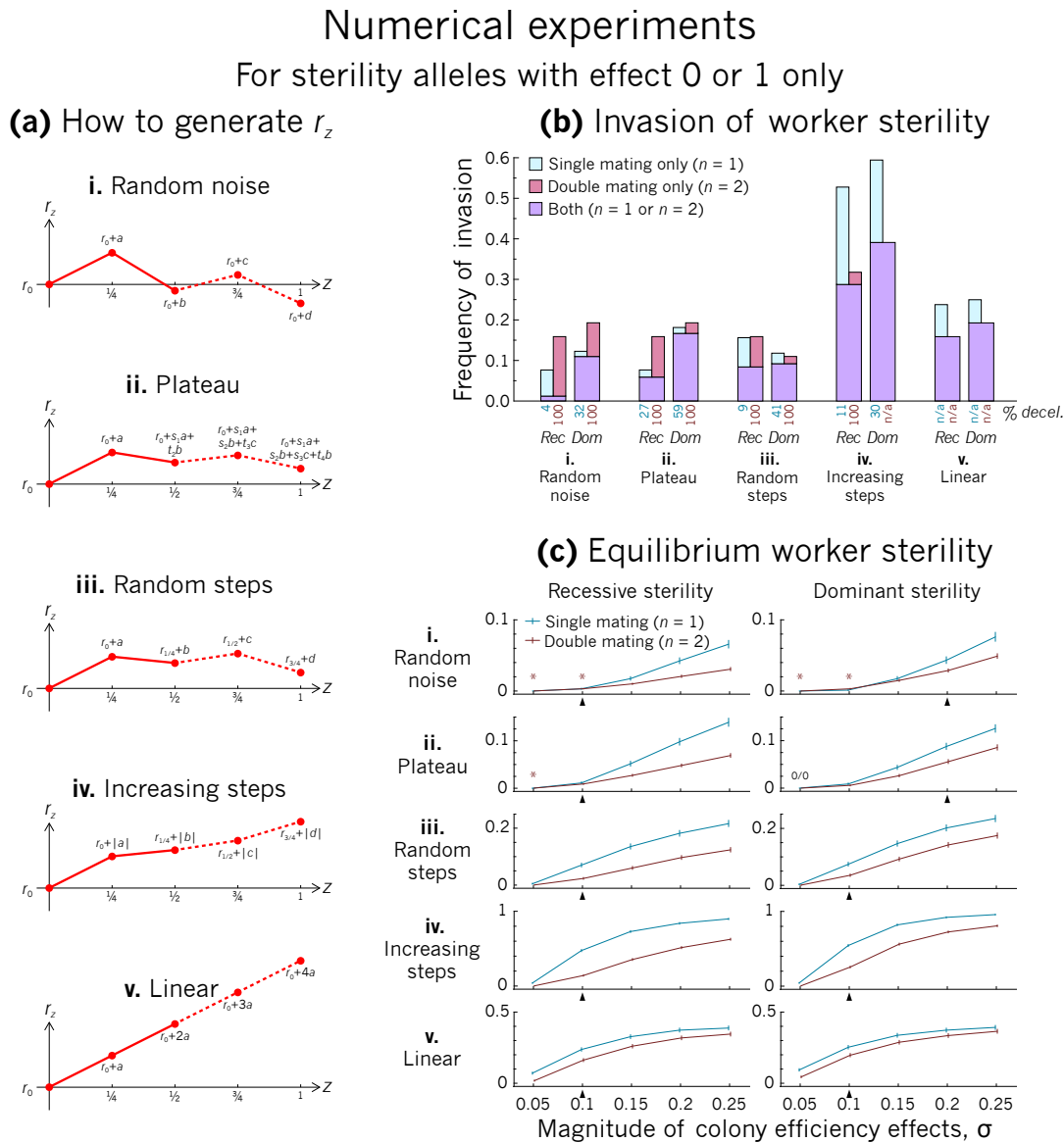


Figure 2: Here, we compare the evolution of worker sterility under single versus double mating by revisiting the numerical experiments of Olejarz *et al.* (2015). **(a)** There are many possible ways to construct the colony efficiency function r_z based on picking random numbers from a normal distribution. Five alternatives are shown here, including the two procedures used by Olejarz *et al.* (“Random noise”, their Procedure 1, and “Plateau”, their Procedure 2). For testing whether sterility invades, only two points are needed (solid lines), but this can be extended to four points (dashed lines) for measuring sterility at equilibrium. **(b)** We record the frequency of invasion of a full-sterility allele under single ($n = 1$) versus double mating ($n = 2$), running 10 million experiments for each scenario. Percentages beneath the bar chart show that an initially-decelerating r_z is required for sterility to invade under double mating only (see Methods). **(c)** We record the average worker sterility at equilibrium over 5000 experiments for each scenario. Except when r_z is constructed using the “random noise” or “plateau” procedure and the magnitude of efficiency effects is small (asterisks), single mating tends to promote average worker sterility at equilibrium over double mating (the 0/0 denotes no worker sterility under either single or double mating). This can happen even if sterility is more likely to invade under double mating (for example, compare results of procedures i-iii in panel **(b)** versus panel **(c)**). Arrowheads beneath the x-axis show where parameters coincide with those used in panel **(b)**. The “magnitude of colony efficiency effects” is the standard deviation of normally-distributed variates used for constructing r_z . For panels **(b)** and **(c)**, we assume $p_z = 0.2 + 0.8z$. See Methods for details.

Evolutionarily-stable level of sterility

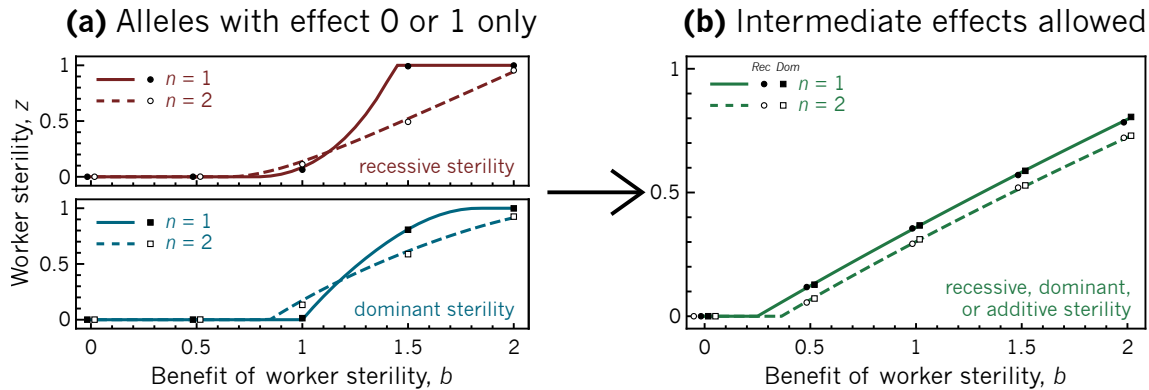


Figure 3: The evolutionarily-stable level of sterility under single versus double mating, for **(a)** constrained allelic variation, with recessive (top) versus dominant (bottom) sterility and **(b)** unconstrained allelic variation, regardless of whether sterility is recessive, dominant, or additive. **(a)** When allelic variation is constrained, double mating (dashed lines) can sometimes promote sterility over single mating (solid lines). But **(b)** when allelic variation is unconstrained, single mating always promotes sterility. Overlaid markers show results of a stochastic individual-based model (see Methods), matching well with the predicted evolutionarily-stable levels of worker sterility. To illustrate a scenario where constraints on heritable variation may lead to promiscuity promoting worker sterility over monogamy, we use the colony efficiency function $r_z = 1 + bz - z^2$, with a “benefit of worker sterility” term bz and a “decelerating” term $-z^2$. For the proportion of male eggs laid by the queen, we again use $p_z = 0.2 + 0.8z$.

$$-\frac{1}{1-z}(1-p_z)(3n-2) + \frac{r'_z}{r_z}(4+3n(1+p_z)) - p'_z(2-n) > 0, \quad (1)$$

123 where r'_z and p'_z are the slopes of the r_z and p_z functions at z , respectively. Remarkably, this
 124 exact condition holds for both recessive and dominant genetics. Using this condition and a global
 125 stability analysis, we find that the ESS for helpful sterility is always highest under single mating—
 126 that is, over long-term evolution, monogamy always promotes helpful worker sterility (Fig. 3; see
 127 Methods).

128 Intuition for this exact population-genetics result may be obtained by recasting condition 1 in
 129 terms of inclusive fitness (Hamilton 1964). Accordingly, natural selection favours an increase to
 130 average sterility, z , when

$$\underbrace{-\frac{1-p_z}{1-z}R_{\text{son}}}_{\text{sacrifice effect}} + \underbrace{\frac{r'_z}{r_z}(R_{\text{sis}} + p_z R_{\text{bro}} + (1-p_z)R_{\text{neph}})}_{\text{efficiency effect}} + \underbrace{p'_z R_{\text{bro}} + \left(\frac{1-p_z}{1-z} - p'_z\right)R_{\text{neph}}}_{\text{male production effect}} > 0, \quad (2)$$

131 where $R_{\text{son}} = \frac{1}{2}$, $R_{\text{neph}} = \frac{2+n}{8n}$, $R_{\text{sis}} = (1+p_z)\frac{2+n}{8n}$, and $R_{\text{bro}} = \frac{1}{4}$ are the life-for-life related-
 132 ness of a worker to her son, her nephew (a random worker's son), her reproductive sister, and
 133 her brother, respectively (Hamilton 1972). Note that promiscuity decreases worker relatedness to
 134 sisters and nephews, but not to sons or brothers. The left-hand side of condition 2 can be inter-
 135 preted as the inclusive-fitness effect experienced by a focal worker who stops laying male eggs.
 136 The "sacrifice effect" captures the direct cost of her sterility, in that she forfeits her relative share
 137 $\frac{1-p_z}{1-z}$ of all worker-laid males. The "efficiency effect" captures her impact on colony efficiency,
 138 which increases by a relative amount $\frac{r'_z}{r_z}$, augmenting the production of her sisters and of colony-
 139 produced males, a proportion p_z of whom are her brothers, and a proportion $1-p_z$ of whom are
 140 her nephews. And the "male production effect" captures her impact on the proportion of male
 141 eggs produced by the queen versus workers: her relative gain of brothers is p'_z , while her relative
 142 gain or loss of nephews exactly balances her forfeited sons and gained brothers.

143 Condition 2 clarifies the impact of monogamy upon helpful worker sterility: by increasing a
 144 worker's relatedness to her nephews and sisters, monogamy increases her inclusive-fitness benefit
 145 of promoting colony efficiency, and by increasing a worker's relatedness to her nephews, it in-
 146 creases her inclusive-fitness benefit of augmenting her fellow workers' production of sons. Hence,
 147 overall, monogamy promotes helpful worker sterility. Condition 2 also clarifies how Olejarz *et*
 148 *al.*'s (2015) model differs from Boomsma's (2007, 2009, 2013) model for the evolution of eusociality:
 149 in Boomsma's model, females trade away offspring for siblings as dispersers evolve into a non-
 150 totipotent worker caste, while in Olejarz *et al.*'s model, an existing non-totipotent worker caste
 151 trades away sons for brothers and nephews. Conditions 1 and 2 are exactly equivalent, are valid
 152 for recessive, dominant, or additive genetics, and can be obtained using standard kin-selection
 153 methodology (see Methods).

154 **Alternative ecological scenarios: monogamy promotes worker steril-** 155 **ity**

156 Finally, we consider some alternative scenarios for the evolution of worker non-reproduction, us-
157 ing a demographically-explicit model of queen-worker competition over egg-laying. Whether we
158 investigate sex-blind egg replacement by workers, soldier sterility in claustral inbreeders, or the
159 evolution of eusociality via female non-dispersal, we find that monogamy always promotes help-
160 ful worker sterility (Fig. 4). This conclusion also holds if we alternatively consider a diploid mode
161 of inheritance (see Methods).

162 **Conclusion: monogamy promotes worker sterility**

163 In seeming contrast to the predictions of inclusive-fitness theory, Olejarz *et al.*'s (2015) exact population-
164 genetics analysis could not identify a consistent effect of monogamy on the evolution of worker
165 sterility. This surprising result, if robust, would have not only overturned a considerable theoretic-
166 al consensus, but would also have left a number of empirically-described patterns bereft of a pre-
167 dictive, explanatory framework. Happily, we have shown that by relaxing constraints on genetic
168 variation (Fig. 1), considering the consequences of invasion rather than just its occurrence (Fig. 2),
169 describing long-term evolutionarily-stable states (Fig. 3), and exploring a wide range of ecological
170 scenarios (Fig. 4), a clear sterility-promoting effect of monogamy consistently emerges. Moreover,
171 we have shown that the long-term evolutionary outcome is readily described, conceptualised, and
172 explained by standard inclusive-fitness theory. In sum, a more comprehensive analysis based on
173 Olejarz *et al.*'s (2015) exact population-genetics approach supports inclusive-fitness theory and its
174 prediction that monogamy promotes the evolution of worker sterility.

175 **Acknowledgements**

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Alternative ecological scenarios

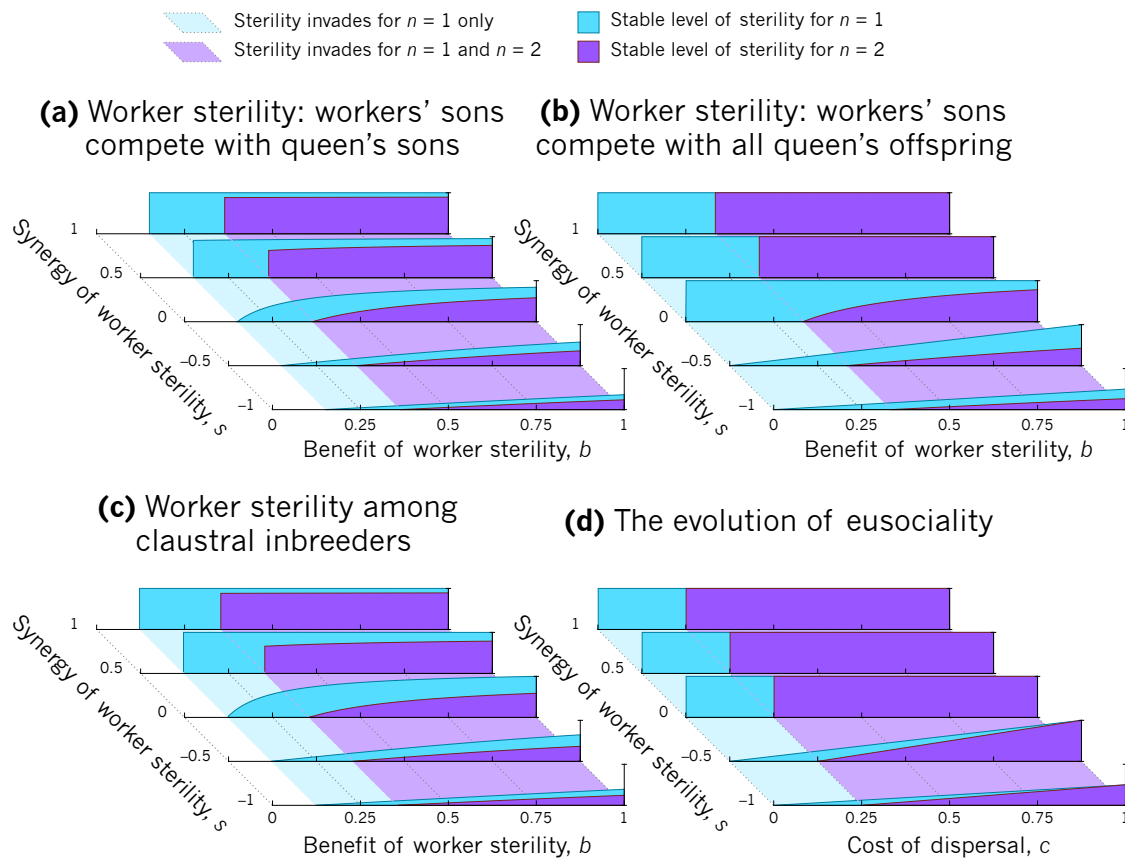


Figure 4: The evolution of worker sterility under alternative ecological scenarios. Here, we determine the stable level of worker sterility under four demographically-explicit models of worker sterility; see Methods for full details. (a) One possible assumption is that worker-laid males only compete with the queen's sons (*cf.* Olejarz *et al.* 2015). In this case, monogamy promotes worker sterility over promiscuity. (b) It is also possible to assume that worker-laid males compete with the queen's offspring of both sexes, and not just with the queen's sons. In this case, monogamy promotes worker sterility over promiscuity. (c) In the gall-forming thrips, the foundress produces an initial brood of female and male soldiers, who may produce part of the next brood by inbreeding amongst themselves (Chapman *et al.* 2002). Female soldiers can sacrifice part of their reproductive potential to invest more in defending their nestmates. In this case, monogamy promotes worker sterility over promiscuity. (d) A possible model for the evolution of eusociality involves dispersing, fully-reproductive females evolving into sterile workers, who stay in the nest to help, producing no offspring (Boomsma 2007, 2009, 2013). In this case, monogamy promotes worker sterility over promiscuity. We show results for $k = 4$ in (a) and $k = 2$ in (b) and (c) (see Methods for details).

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224 **Methods**

225 **Helpful versus harmful worker sterility and policing**

226 Throughout the main text, our focus is on helpful worker sterility, where giving up some or all
227 of her reproductive potential allows a worker to provide more help within her colony, as this
228 biological assumption underpins most work on altruistic sib-rearing in social insects. However,
229 the model of Olejarz *et al.* (2015), despite making strong genetic assumptions, makes few ecological
230 assumptions about worker sterility, which means it may also describe harmful worker sterility. If
231 worker sterility is harmful—namely, if worker sterility reduces colony efficiency and/or reduces
232 other workers' personal fitness—monogamy may inhibit worker sterility, depending on the overall
233 impact of sterility on a worker's inclusive fitness.

234 In this model, harmful worker sterility may occur via two routes—one operating through
235 colony efficiency, r_z , and one operating through the queen's production of males, p_z . The first
236 case occurs when an increase in average worker sterility decreases colony efficiency—for exam-
237 ple, if the sterility allele has a pleiotropic effect on worker condition which results in less-efficient
238 work. In such a case, monogamy will inhibit the evolution of worker sterility relative to promis-
239 cuity, since promiscuity decreases relatedness between relatives, thereby lessening the harmful
240 impact of sterility upon a worker's inclusive fitness via colony efficiency.

241 The second case occurs when an increase in a focal worker's sterility harms the reproduc-
242 tive success of other workers. In the main text, we assume that when a worker becomes sterile,
243 her forfeited sons are replaced partly by the queen's sons and partly by her sisters' sons, such that
244 by forfeiting sons she gains both nephews and brothers. But if, due to the shape of the p_z function,
245 the queen gains a larger proportion of sons than the worker forfeits (that is, when $p'_z > \frac{1-p_z}{1-z}$), this
246 "outsized gain" by the queen must be balanced by *decreased* male production by other workers,
247 such that, by becoming sterile, the focal worker loses nephews overall. If the focal worker loses
248 nephews by becoming sterile (*i.e.*, when $\frac{1-p_z}{1-z} - p'_z < 0$; see condition 2), then promiscuity, by de-
249 creasing the worker's relatedness to nephews, may promote this harmful form of worker sterility
250 over monogamy, unless this relative cost of sterility is countered by a colony efficiency benefit of

251 sterility, which would be largest in magnitude under monogamy.

252 This second form of harmful worker sterility is connected with worker policing—that is, when
 253 workers invest resources in preventing other workers from laying eggs (Ratnieks 1988; Ratnieks
 254 & Visscher 1989). If worker sterility is harmful, then a worker gives up part of her personal fitness
 255 in order to decrease the reproduction of her fellow workers; this is analogous to costly worker
 256 policing. Standard inclusive-fitness theory (Ratnieks 1988; Ratnieks & Visscher 1989; Ratnieks
 257 *et al.* 2006) and empirical evidence (Wenseleers & Ratnieks 2006a, 2006b) have emphasised that
 258 promiscuity promotes worker policing, so the result that this harmful form of worker sterility
 259 may be promoted by promiscuity is not at all surprising.

260 For non-incremental increases in sterility, the condition for harmful sterility becomes $\frac{p_v - p_u}{v - u} >$
 261 $\frac{1 - p_u}{1 - u}$, where u is the level of worker sterility in the monomorphic population before the mutant
 262 allele is introduced, and v is the level of worker sterility encoded by the mutant allele.

263 **Explicit population-genetics analysis**

264 In Appendix A, we extend the methods of Olejarz *et al.* (2015) to consider the invasion of an allele
 265 with an arbitrary effect on worker sterility; the results of this analysis are presented here. We
 266 find that a recessive allele encoding worker sterility v can invade a population monomorphic for
 267 sterility u when

$$\frac{r \frac{(2n-1)u+v}{2n}}{r_u} > \frac{2(2n(1-u) + u - v)(2 + n(1 + p_u))}{\left(\begin{array}{l} n(8 + 4n(1-u) - 3u - 5v) + 2(u-v) \\ + (2+n)(2n(1-u) + u - v)p_u \\ - 2n(2 - u - v - n(1-u))p \frac{(2n-1)u+v}{2n} \end{array} \right)}. \quad (3)$$

268 Similarly, we find that a dominant allele encoding worker sterility v can invade a population
 269 monomorphic for sterility u when

$$\frac{r_{\frac{u+v}{2}}}{r_u} \left(\begin{array}{l} 1 + \left(\frac{(1-u)p_{\frac{u+v}{2}}}{2-u-v} + \frac{(1-v) \left((2-n)(u-v) + n(2-u-v)p_{\frac{(n-1)u+v}{n}} \right)}{2(n(1-u)+u-v)(2-u-v)} \right) \frac{r_{\frac{(n-1)u+v}{n}}}{r_u} \\ + \frac{n(1-v)(1-p_{\frac{(n-1)u+v}{n}}) r_{\frac{(n-1)u+v}{n}}}{n(1-u)+u-v} \frac{r_{\frac{(n-1)u+v}{n}}}{r_{\frac{u+v}{2}}} \end{array} \right) > 2. \quad (4)$$

270 Note that conditions 3 and 4 give both the invasion and stability of a given level of sterility:
 271 that is, if a sterility allele with effect v can invade a population monomorphic for sterility u , then
 272 this is the same as saying that a population monomorphic for sterility u is not stable to invasion
 273 by a sterility allele with effect v . For example, substituting $n = 1$, $u = 0$, $v = 1$ into condition 3
 274 yields the condition for the invasion of a recessive sterility allele under single mating from Olejarz
 275 *et al.* (2015; their condition 1), while substituting $n = 1$, $u = 1$, $v = 0$ into condition 4 yields the
 276 condition for the stability of a recessive sterility allele under single mating from Olejarz *et al.* (2015;
 277 their condition 3).

278 In order to find when natural selection will favour a small increase in sterility δz , we make the
 279 substitution $v = u + \delta z$ into conditions 3 and 4 above. Then, by linearizing r_z and p_z around the
 280 point $z = u$, we can recast these conditions in terms of the value and slope of r_z and p_z at this
 281 point. More specifically, for a recessive sterility allele, substituting $v = u + \delta z$ into condition 3
 282 yields

$$\frac{r_{u+\frac{\delta z}{2n}}}{r_u} > \frac{2(2n(1-u) - \delta z)(2 + n(1 + p_u))}{\left(\begin{array}{l} 4n(2+n)(1-u) - (2+5n)\delta z \\ + (2+n)(2n(1-u) - \delta z)p_u \\ - 2n(2-n - (2-n)u - \delta z)p_{u+\frac{\delta z}{2n}} \end{array} \right)}.$$

283 Linearizing r_z and p_z around $z = u$, we replace $r_{u+\frac{\delta z}{2n}}$ with $r + \frac{\delta z}{2n}r'$, where $r = r_u$ and $r' = \frac{dr}{dz}|_{z=u}$.

284 Similarly, we replace $p_{u+\frac{\delta z}{2n}}$ with $p + \frac{\delta z}{2n}p'$, where $p = p_u$ and $p' = \frac{dp}{dz}|_{z=u}$. This yields

$$\frac{r + \frac{\delta z}{2n} r'}{r} > \frac{2(2n(1-u) - \delta z)(2 + n(1+p))}{\left(\begin{array}{l} 4n(2+n)(1-u) - (2+5n)\delta z \\ + (2+n)(2n(1-u) - \delta z)p \\ - 2n(2-n - (2-n)u - \delta z)(p + \frac{\delta z}{2n} p') \end{array} \right)}.$$

285 Eliminating the fractions on both sides, discarding terms of order δz^2 or higher, substituting z for
 286 u and simplifying yields

$$-\frac{1}{1-z}(1-p_z)(3n-2) + \frac{r'_z}{r_z}(4+3n(1+p_z)) - p'_z(2-n) > 0,$$

287 which is condition 1 of the main text.

288 Similarly, for a dominant sterility allele, substituting $v = u + \delta z$ into condition 4 yields

$$\frac{r_{u+\frac{\delta z}{2}}}{r_u} \left(\begin{array}{l} 1 + \left(\frac{(1-u)p_{u+\frac{\delta z}{2}}}{2-2u-\delta z} + \frac{(1-u-\delta z)(n(2-2u-\delta z)p_{u+\frac{\delta z}{2}} - (2-n)\delta z)}{2(n(1-u)-\delta z)(2-2u-\delta z)} \right) \frac{r_{u+\frac{\delta z}{2}}}{r_u} \\ + \frac{n(1-u-\delta z)(1-p_{u+\frac{\delta z}{2}})}{n(1-u)-\delta z} \frac{r_{u+\frac{\delta z}{2}}}{r_{u+\frac{\delta z}{2}}} \end{array} \right) > 2.$$

289 By linearizing r_z and p_z around $z = u$ as above, we obtain

$$\frac{r + \frac{\delta z}{2} r'}{r} \left(\begin{array}{l} 1 + \left(\frac{(1-u)(p + \frac{\delta z}{2} p')}{2-2u-\delta z} + \frac{(1-u-\delta z)(n(2-2u-\delta z)(p + \frac{\delta z}{2} p') - (2-n)\delta z)}{2(n(1-u)-\delta z)(2-2u-\delta z)} \right) \frac{r + \frac{\delta z}{2} r'}{r} \\ + \frac{n(1-u-\delta z)(1-p - \frac{\delta z}{2} p')}{n(1-u)-\delta z} \frac{r + \frac{\delta z}{2} r'}{r + \frac{\delta z}{2} r'} \end{array} \right) > 2.$$

290 Expanding all terms, discarding terms of order δz^2 or higher, substituting z for u and simplifying
 291 yields

$$-\frac{1}{1-z}(1-p_z)(3n-2) + \frac{r'_z}{r_z}(4+3n(1+p_z)) - p'_z(2-n) > 0,$$

292 which, again, is condition 1 of the main text.

293 **Invasion of a dominant sterility allele**

294 In the main text, we discuss why promiscuity can sometimes favour the invasion of a recessive
295 worker sterility allele. The invasion of a dominant worker sterility allele is similar, but in this
296 case there are two “mutant” mating types which determine whether sterility can invade: a het-
297 erozygous mutant female mating with n wild-type males, and a wild-type female mating with
298 one mutant male and $n - 1$ wild-type males. These mating types produce colonies with a pro-
299 portion $z = \frac{v}{2}$ and $z = \frac{v}{n}$ of sterile workers, respectively. Hence, for a sterility allele with full
300 penetrance, under single mating ($n = 1$), it is the relative success of colonies with 50% and 100%
301 sterile workers which determines whether a sterility allele with full penetrance can invade, while
302 under double mating ($n = 2$), only the relative success of colonies with 50% sterile workers deter-
303 mines whether a sterility allele with full penetrance can invade. Therefore, if the relative success of
304 colonies with 100% sterile workers is low, this could be enough to disfavour the invasion of a fully-
305 penetrant worker sterility allele into a wild-type population under single but not double mating.
306 Nonetheless, for the scenario investigated by Olejarz *et al.* (2015, their Fig. 8), we find that single
307 mating always promotes the invasion of dominant sterility over double mating (Fig. 5). Moreover,
308 allowing for helpful-sterility alleles showing the full range of degrees of penetrance and domi-
309 nance/recessivity, there is no scenario under which *some* sterility alleles can invade under double
310 mating, and yet *no* sterility allele can invade under single mating.

311 **Numerical experiments**

312 Olejarz *et al.* (2015) performed numerical experiments to see whether sterility was more likely to
313 invade under single mating or double mating. To do so, they constructed randomly-generated
314 r_z functions according to one of two procedures. Here, we add to these procedures, bringing
315 the number of possible methods for constructing the r_z function to five (Fig. 2a). Each involves
316 drawing four random variates—here, notated as a , b , c , and d —from a normal distribution with

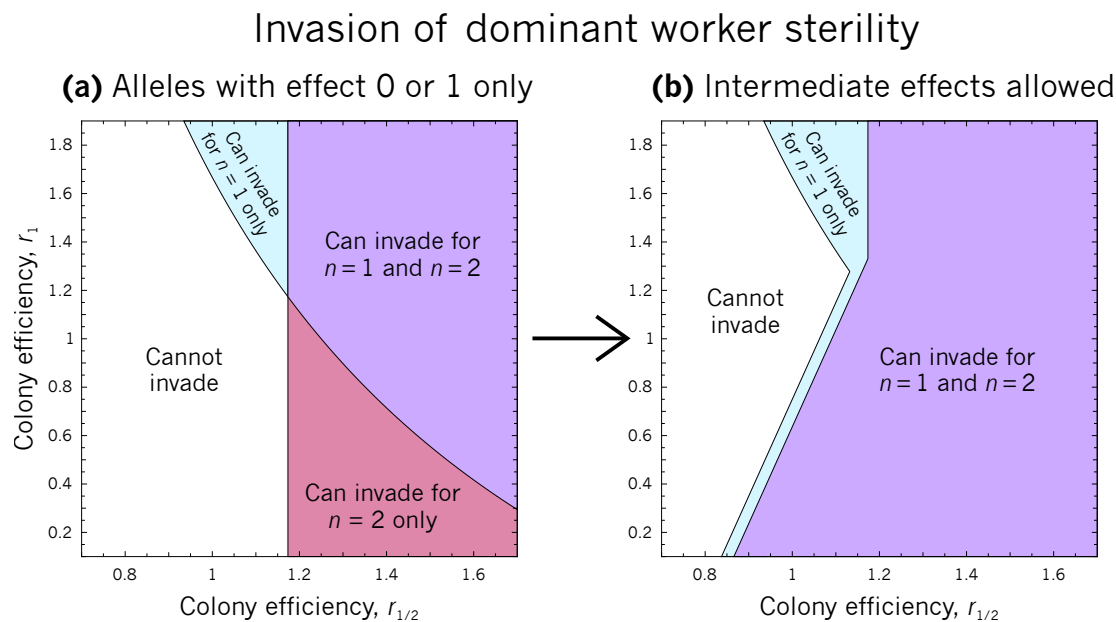


Figure 5: The invasion of worker sterility under dominant genetics, exploring the regions of parameter space where sterility can invade under single mating only, double mating only, both, or neither. **(a)** If we assume that only full-sterility alleles can arise, double mating sometimes promotes the invasion of sterility over single mating. But **(b)** if we assume that alleles encoding intermediate worker sterility can arise, double mating never promotes the invasion of sterility over single mating. In order to facilitate comparison with Figure 8 of Olejarz *et al.* (2015), we assume $p_z = 0.2 + 0.8z$, and for r_z we use the unique quadratic curve passing through the points specified by $r_0 = 1, r_{1/2}$, and r_1 .

317 mean 0 and standard deviation σ . In all cases, we assume $r_0 = 1$, and use the random variates to
318 generate $r_{1/4}$, $r_{1/2}$, $r_{3/4}$, and r_1 , which suffice to numerically integrate the evolutionary dynamics
319 of worker sterility using the system of ODEs described by Olejarz *et al.* (2015). We restrict our
320 attention here to the invasion of an allele encoding full sterility in its carriers, under either recessive
321 or dominant genetics.

322 The first procedure, “random noise”, is equivalent to Procedure 1 in Olejarz *et al.* (2015). Here,
323 we set $r_{1/4} = r_0 + a$, $r_{1/2} = r_0 + b$, $r_{3/4} = r_0 + c$, and $r_1 = r_0 + d$. Note that the four values are
324 completely uncorrelated with each other; sequential values of r_z are independent from previous
325 values, which is why we have named this procedure “random noise”. This procedure might gener-
326 ate plausible r_z functions for a population where every colony-level increase in worker sterility
327 were to completely erase the effect of any previous increase in worker sterility, replacing it with a
328 new, random effect.

329 The second procedure, “plateau”, is equivalent to Procedure 2 in Olejarz *et al.* (2015). Here, the
330 values $r_{1/4}$, $r_{1/2}$, $r_{3/4}$, and r_1 are drawn from a correlated multivariate normal distribution. This
331 can be simulated by transforming four uncorrelated normal variates; one way of doing this is by
332 using the matrix

$$\begin{bmatrix} 1 & \rho & \rho & \rho \\ \rho & 1 & \rho & \rho \\ \rho & \rho & 1 & \rho \\ \rho & \rho & \rho & 1 \end{bmatrix},$$

333 where ρ is the desired correlation between each variate. By multiplying the vector of uncorre-
334 lated variates by the Cholesky decomposition of this matrix, one obtains four correlated variates

$$\begin{aligned} a' &= a \\ b' &= a\rho + b\sqrt{1-\rho^2} \\ c' &= a\rho + b\frac{\rho\sqrt{1-\rho}}{\sqrt{1+\rho}} + c\sqrt{3-2\rho - \frac{2}{1+\rho}} \end{aligned}$$

$$d' = a\rho + b\frac{\rho\sqrt{1-\rho}}{\sqrt{1+\rho}} + c\frac{\rho\sqrt{3-2\rho-\frac{2}{1+\rho}}}{1+2\rho} + d\sqrt{\frac{1+\rho(2-3\rho)}{1+2\rho}}.$$

335 Now, we set $r_{1/4} = r_0 + a'$, $r_{1/2} = r_0 + b'$, $r_{3/4} = r_0 + c'$, and $r_1 = r_0 + d'$. Note that, because
 336 the variables are correlated, the first “step” (from r_0 to $r_{1/4}$) tends to be larger in magnitude than
 337 subsequent “steps” (*i.e.*, from $r_{1/4}$ to $r_{1/2}$, $r_{1/2}$ to $r_{3/4}$, or $r_{3/4}$ to r_1), which is why we have named
 338 this procedure “plateau”. This procedure might generate plausible r_z functions for a population in
 339 which worker sterility brings diminishing returns to colony productivity, where these diminishing
 340 returns happen to set in near $z = 1/4$.

341 Note that both the “random noise” and “plateau” procedures tend to produce r_z functions that
 342 disadvantage single mating relative to double mating. For the “random noise” procedure, this
 343 is because although the procedure is just as likely to produce a peak at $z = 1/2$ (which would
 344 favour single mating) as at $z = 1/4$ (which would favour double mating), workers at $z = 1/2$ are
 345 typically “trading away” more male production than workers at $z = 1/4$ (since $p_{1/2} \geq p_{1/4}$), yet,
 346 on average, they are receiving the same expected increase in productivity; hence, single mating
 347 is relatively disfavoured. And since the “plateau” procedure tends to produce colony efficiency
 348 functions with diminishing returns on worker sterility for colonies with $z > 1/4$, it is much more
 349 likely to produce an r_z function with a relative peak at $z = 1/4$ rather than a relative peak at $z = 1/2$.

350 The third procedure, “random steps”, sets each point in r_z to the value of the previous point
 351 plus a random perturbation: $r_{1/4} = r_0 + a$, $r_{1/2} = r_{1/4} + b$, $r_{3/4} = r_{1/2} + c$, and $r_1 = r_{3/4} + d$.
 352 This procedure might generate plausible r_z functions if each increase in worker sterility had a
 353 random increasing or decreasing effect on colony productivity. The fourth procedure, “increasing
 354 steps”, is similar, except steps are constrained to be positive: $r_{1/4} = r_0 + |a|$, $r_{1/2} = r_{1/4} + |b|$,
 355 $r_{3/4} = r_{1/2} + |c|$, and $r_1 = r_{3/4} + |d|$. This procedure might generate plausible r_z functions
 356 if each increase in worker sterility added a random increase to colony productivity. The fifth
 357 procedure, “linear”, uses a single normal variate to establish a constant step size for r_z : $r_{1/4} =$
 358 $r_0 + a$, $r_{1/2} = r_{1/4} + a$, $r_{3/4} = r_{1/2} + a$, and $r_1 = r_{3/4} + a$. This procedure might generate plausible
 359 r_z functions if each increase in worker sterility had a consistent increasing or decreasing effect on

360 colony productivity. For each of these new procedures, later points in r_z depend on earlier points,
361 but there is no tendency for “steps” between points in r_z to change in average magnitude.

362 In Fig. 2, we test each of these 5 procedures to see whether single or double mating promotes
363 the invasion (Fig. 2b) or equilibrium level of sterility (Fig. 2c) more, for recessive versus dominant
364 sterility. The form of p_z we use ($p_z = k + (1 - k)z$, with $k = 0.2$), chosen for comparison with
365 the numerical experiments of Olejarz *et al.* (2015, their Table 1), prevents worker sterility from
366 resulting in a net loss of nephews (see *Helpful versus harmful worker sterility and policing*, above).
367 Beneath the bar charts in Fig. 2d, we show the percentage of experiments for which the exclusive
368 invasion of sterility under either single or double mating occurred with an initially-decelerating r_z
369 (*i.e.*, where $r_{1/2} - r_{1/4} < r_{1/4} - r_0$). Note that, for these values of p_z , double mating only promotes
370 the invasion of sterility over single mating when r_z is initially-decelerating. In Fig. 2c, error bars
371 show bootstrapped 95% confidence intervals for average worker sterility.

372 ESS analysis

373 By setting the left-hand side of condition (2) to zero, it is possible to find a convergence-stable
374 point (Davies *et al.* 2016) for worker sterility. At these points, natural selection will not favour the
375 invasion of an allele encoding either a small increase or a small decrease to worker sterility (*i.e.*,
376 convergence-stable points are stable to small perturbations); moreover, for a population playing
377 a strategy that is close to a convergence-stable point, natural selection will favour the invasion
378 of strategies between the population strategy and the convergence-stable point (*i.e.*, convergence-
379 stable states are reachable from nearby states). However, a convergence-stable point is only an
380 evolutionarily-stable strategy (ESS) if *no* alternative allele can invade at this point. Therefore, in
381 order to find a true ESS, we treat convergence-stable points as “candidate ESSs”, then use condi-
382 tions 3 and 4 to determine whether any alternative allele can invade a population monomorphic
383 for the candidate ESS under the appropriate regime of dominance or recessivity. If no alternative
384 allele can invade, the candidate ESS is a true ESS. In Figure 3, true ESSs are shown.

385 Note that it is possible for an ESS to *not* be convergence-stable, and this method will not identify
386 such states. However, we are only interested in ESSs that are reachable, *i.e.*, both convergence-

387 stable and evolutionarily-stable. Such strategies are called “continuously-stable strategies” (CSSs;
388 Eshel 1983).

389 Demographically-explicit ecological scenarios

390 In Appendix B, we develop a general kin-selection model for the evolution of worker sterility. This
391 analysis can be used to investigate a variety of ecological scenarios. Here, we present four such
392 scenarios for the evolution of worker sterility.

393 Scenario A. Workers’ sons replace queen’s sons

394 In this scenario, we assume that non-sterile workers replace the queen’s sons with their own sons,
395 as in the model of Olejarz *et al.* (2015). Following these assumptions, we find that natural selection
396 will favour an increase to worker sterility, z , when

$$\underbrace{-\frac{1-p_z}{1-z}R_{\text{son}}}_{\text{sacrifice effect}} + \underbrace{\frac{r'_z}{r_z}(R_{\text{sis}} + p_z R_{\text{bro}} + (1-p_z)R_{\text{neph}})}_{\text{efficiency effect}} + \underbrace{p'_z R_{\text{bro}} + \left(\frac{1-p_z}{1-z} - p'_z\right)R_{\text{neph}}}_{\text{male production effect}} > 0, \quad (5)$$

397 where $R_{\text{son}} = \frac{1}{2}$, $R_{\text{neph}} = \frac{2+n}{8n}$, $R_{\text{sis}} = (1+p_z)\frac{2+n}{8n}$, and $R_{\text{bro}} = \frac{1}{4}$. As explained in the main
398 text, the left-hand side of condition 5 can be interpreted as the inclusive-fitness effect experienced
399 by a worker who stops laying male eggs. The “sacrifice effect” captures the direct cost of her
400 sterility, in that she forfeits her relative share $\frac{1-p_z}{1-z}$ of all worker-laid males. The “efficiency effect”
401 captures her impact on colony efficiency, which increases by a relative amount $\frac{r'_z}{r_z}$, augmenting the
402 production of her sisters and of colony-produced males, a proportion p_z of whom are her brothers,
403 and a proportion $1-p_z$ of whom are her nephews. And the “male production effect” captures her
404 impact on the proportion of male eggs produced by the queen versus workers: her relative gain
405 of brothers is p'_z , while her relative gain or loss of nephews exactly balances her forfeited sons and
406 her gained brothers.

407 Similarly, natural selection favours an increase to the queen’s sex allocation, x (her proportion

408 of resources allocated to daughters), when

$$\frac{1}{x} - \frac{1}{1-x} > 0. \quad (6)$$

409 That is, natural selection favours an increased investment into daughters when $x < 1/2$, and a
 410 decreased investment into daughters when $x > 1/2$, such that an even sex ratio is favoured overall,
 411 regardless of worker sterility.

412 B. Workers' sons compete with all queen's offspring

413 It is also possible to assume that, rather than only displacing the queen's sons, workers' sons
 414 compete with the queen's sons and daughters equally. This scenario may apply if workers do
 415 not discern between fertilised and unfertilised eggs when they replace the queen's eggs with their
 416 own; alternatively, it may apply if rather than replacing the queen's eggs, the workers simply lay
 417 their eggs in the communal nest, and all queen-produced and worker-produced offspring have the
 418 same expected survival. Following these assumptions, we find that natural selection will favour
 419 an increase to worker sterility, z , when

$$\underbrace{-\frac{1-p_z}{1-z}R_{\text{son}}}_{\text{sacrifice effect}} + \underbrace{\frac{r'_z}{r_z} \left(xp_z R_{\text{sis}} + (1-x)p_z R_{\text{bro}} + (1-p_z)R_{\text{neph}} \right)}_{\text{efficiency effect}} + \underbrace{xp'_z R_{\text{sis}} + (1-x)p'_z R_{\text{bro}} + \left(\frac{1-p_z}{1-z} - p'_z \right) R_{\text{neph}}}_{\text{juvenile production effect}} > 0 \quad (7)$$

420 where p_z is the proportion of all juveniles on the patch that are produced by the queen, $R_{\text{son}} = \frac{1}{2}$,
 421 $R_{\text{neph}} = \frac{2+n}{8n}$, $R_{\text{sis}} = \frac{1+(1-2x)p_z}{xp_z} \frac{2+n}{8n}$, and $R_{\text{bro}} = \frac{1}{4}$. In this model, queen sex allocation alters
 422 the relative reproductive value of a female compared to that of a male, $\frac{1+(1-2x)p_z}{xp_z}$ (the product of
 423 the relative reproductive value of all females compared to that of all males, $\frac{1+(1-2x)p_z}{1-xp_z}$, and the
 424 number of females relative to the number of males, $\frac{1-xp_z}{xp_z}$), which comes into the expression for

425 R_{sis} . Similarly to condition 5, the left-hand side of condition 7 can be interpreted as the inclusive-
426 fitness effect experienced by a worker who stops laying male eggs. Here, the “sacrifice effect”
427 captures the direct cost of her sterility, in that she forfeits her relative share $\frac{1-p_z}{1-z}$ of all worker-
428 laid males. The “efficiency effect” captures her impact on colony efficiency, which increases by
429 a relative amount $\frac{r'_z}{r_z}$, a proportion $x p_z$ of which goes toward sisters, $(1-x)p_z$ toward brothers,
430 and $1-p_z$ toward nephews. And the “juvenile production effect” captures her impact on the
431 proportion of eggs produced by the queen versus workers: her relative gain of sisters is $x p'_z$, and
432 her relative gain of brothers is $(1-x)p'_z$, and so her relative gain of nephews exactly balances her
433 lost sons, less her gained brothers and sisters.

434 In this scenario, queen sex allocation is not independent of worker sterility. We find that natural
435 selection favours an increase to the queen’s investment in daughters, x , when

$$\frac{1+p_z}{2x} - \frac{p_z}{1-x} > 0; \quad (8)$$

436 hence, when all colony offspring are queen-laid ($p_z = 1$), the queen favours an even sex ratio
437 ($x = 1/2$), but as the proportion of colony offspring laid by workers increases, the queen favours
438 an increasingly female-biased sex ratio. Specifically, the equilibrium sex ratio is $x^* = \frac{1+p_z}{1+3p_z}$.

439 **Scenario C. Worker sterility among claustral inbreeders**

440 Here, we assume that the queen produces a first brood of female and male soldiers, who mate
441 amongst themselves; the second brood of female and male dispersers is partly produced by the
442 queen and partly produced by the soldiers, as in the gall-forming social thrips (Chapman 2002).
443 For simplicity, we assume here that queens and soldiers produce an even sex ratio for the second
444 brood, but allowing sex ratio evolution does not change the results qualitatively (not shown).
445 Following these assumptions, we find that natural selection favours an increase to the sterility of
446 female soldiers, z , when

Alternative ecological scenarios (diploidy)

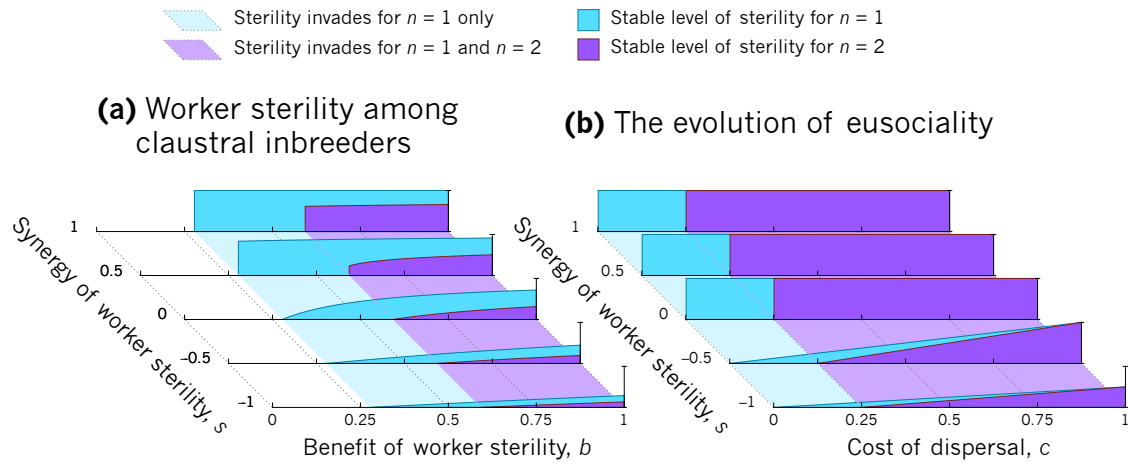


Figure 6: The evolution of worker sterility under alternative ecological scenarios, for diploidy. Here, we determine the stable level of worker sterility under two demographically-explicit models of worker sterility; see Methods for full details. **(a)** For claustral inbreeders under diploidy, monogamy promotes worker sterility over promiscuity; we show results for $k = 4$ here. **(b)** For the evolution of eusociality via non-dispersing female workers under diploidy, monogamy promotes worker sterility over promiscuity.

$$\begin{aligned}
 & \underbrace{-\frac{1-p_z}{1-z}(R_{\text{dau}} + R_{\text{son}})}_{\text{sacrifice effect}} + \underbrace{\frac{r'_z}{r_z} \left(p_z(R_{\text{sis}} + R_{\text{bro}}) + (1-p_z)(R_{\text{niece}} + R_{\text{neph}}) \right)}_{\text{efficiency effect}} \\
 & + \underbrace{p'_z(R_{\text{sis}} + R_{\text{bro}}) + \left(\frac{1-p_z}{1-z} - p'_z \right) (R_{\text{niece}} + R_{\text{neph}})}_{\text{juvenile production effect}} > 0 \quad (9)
 \end{aligned}$$

447 where, under haplodiploidy, $R_{\text{dau}} = \frac{5+p_z}{6}$, $R_{\text{son}} = \frac{3+p_z}{6}$, $R_{\text{niece}} = \frac{3+6n+p_z}{12n}$, $R_{\text{neph}} = \frac{3+2n+p_z}{12n}$,
 448 $R_{\text{sis}} = \frac{3+2n+p_z}{6n}$, and $R_{\text{bro}} = \frac{1}{3}$. Because this scenario does not require arrhenotokous partheno-
 449 genesis of males, it also applies to diploid populations. Under diploidy, $R_{\text{dau}} = R_{\text{son}} = \frac{11+p_z}{16}$
 450 and $R_{\text{niece}} = R_{\text{neph}} = R_{\text{sis}} = R_{\text{bro}} = \frac{1+n}{4n}$ (Fig. 6a). Similarly to condition 7, the left-hand side of
 451 condition 9 can be interpreted as the inclusive-fitness effect experienced by a worker who stops
 452 laying male eggs; but in condition 9, the female worker's "sacrifice effect" involves giving up

453 both daughters and sons; the “efficiency effect” involves an increase in both niece and nephew
454 production as well as sister and brother production; and the “juvenile production effect” involves
455 the focal worker gaining both sisters and brothers, while her gain or loss of nieces and nephews
456 balances her forfeited offspring and her gained siblings.

457 **Scenario D. The evolution of eusociality**

458 Here, we assume that the queen produces and provisions a first brood of females, and then pro-
459 duces a second batch of female and male eggs. Each first-brood female can either disperse—leave
460 the nest, mate, and produce female and male offspring on her own—or work—stay in the nest
461 and help to raise the queen’s second-brood offspring without producing any offspring of her own.
462 We assume that each worker can raise b siblings, on average, in her natal nest, and that each dis-
463 perser can raise $b(1 - c)$ offspring, on average, in her newly-founded nest, where c represents the
464 cost of dispersal; and, additionally, that workers may synergistically or antagonistically interact
465 according to the parameter s , such that if the total number of female workers is Kz , then in total
466 workers can raise $Kzb(1 + sz)$ of the queen’s second-brood offspring. This model is similar to the
467 one considered by Boomsma (2007, 2009, 2013) for the evolution of eusociality. Following these
468 assumptions, we find that natural selection will favour an increase to worker sterility, z , when

$$\underbrace{-b(1 - c)(R_{\text{dau}} + R_{\text{son}})}_{\text{sacrifice effect}} + \underbrace{b(1 + 2sz)(R_{\text{sis}} + R_{\text{bro}})}_{\text{efficiency effect}} > 0, \quad (10)$$

469 where $R_{\text{dau}} = R_{\text{son}} = \frac{1}{2}$, $R_{\text{sis}} = \frac{2+n}{4n}$, and $R_{\text{bro}} = \frac{1}{4}$. As with scenario C, this scenario also applies
470 to diploid populations; under diploidy, $R_{\text{dau}} = R_{\text{son}} = \frac{1}{2}$ and $R_{\text{sis}} = R_{\text{bro}} = \frac{1+n}{4n}$ (Fig. 6b). When
471 $z = 0$, this condition reduces to

$$c > \frac{n - 1}{2n}$$

472 under both haplodiploidy and diploidy; that is, under strict monogamy ($n = 1$), any marginal
473 benefit of rearing siblings over offspring (for example, any non-zero cost of dispersal, mating, or

474 nest founding) suffices to favour the invasion of sterile workers, regardless of the level of worker
475 synergy, s ; but with any level of multiple mating ($n > 1$), a threshold dispersal cost of at least
476 $\frac{n-1}{2n}$ is required for natural selection to favour the invasion of sterile workers (Fig. 4d; Fig. 6b). In
477 other words, only marginal efficiency gains are needed for worker sterility to invade under strict
478 monogamy (Boomsma 2007, 2009, 2013).

479 **Explicit forms for r_z and p_z**

480 Scenarios A, B, and C above are independent of the particular r_z and p_z functions used. However,
481 for preparing Figs. 4 and 6, we used the explicit forms

$$r_z = 1 + bz + sz^2 \text{ and}$$
$$p_z = \frac{1}{1 + k(1 - z)}.$$

482 The r_z function above has three components: a baseline efficiency of 1; bz , representing a linear
483 fitness benefit for each sterile worker; and sz^2 , representing an “interaction effect” of worker steril-
484 ity. We use the parameter s to examine scenarios where multiple sterile workers results in either
485 synergy ($s > 0$) or diminishing returns ($s < 0$) to colony productivity.

486 The p_z function given above corresponds to a model in which the queen and $k(1 - z)$ repro-
487 ductive workers each take an equal share of offspring production. Alternatively, k can capture not
488 only the total number of workers but also their ability to control offspring production relative to
489 the queen; for example, halving k could represent either a halving in the number of workers or
490 a halving of their relative ability to control offspring production, keeping the number of workers
491 constant.

492 A function of this form can also model more complicated demographic processes: for example,
493 if we assume that there are N workers, each of whom replaces a random egg with their own at rate
494 W , while the queen can replace a workers’ egg with her own at rate Q , then the form above gives
495 the proportion of eggs produced by the queen at equilibrium when $k = \frac{NW}{Q}$. In models where

496 worker-laid and queen-laid individuals compete equally, regardless of their sex, production of
497 eggs and replacement of eggs will often be equivalent processes: that is, the form given above
498 for p_z also holds if workers, rather than replacing the queen's eggs, simply lay their own eggs in
499 the communal nest without replacement. In that case, the r_z function would capture the overall
500 production and survival of eggs.

501 **Stable level of sterility**

502 For Fig. 4, we determine the convergence-stable point (Davies *et al.* 2016) for sterility by numer-
503 ically integrating the selection gradients for sterility and sex allocation (left-hand sides of condi-
504 tions 5-10). First, we set the sex ratio to $x = \bar{x} = 1/2$ and allow it to evolve in the absence of worker
505 sterility ($Z = z = \bar{z} = 0$) until it reaches its equilibrium value. Then, we allow both the sex ratio
506 and sterility to coevolve, until equilibrium is reached for both traits.

507 **Stochastic individual-based model**

508 To verify the results of our kin selection analysis (Fig. 3), we implemented a stochastic individual-
509 based model in C++. Here, each individual comprises a locus encoding their breeding value for
510 worker sterility, Z . The locus comprises one or two genes, depending on whether the individual is
511 haploid or diploid, and each gene is represented by a real number $\gamma \in [0, 1]$. Breeding values are
512 determined by averaging genic values: hence, a haploid individual with genotype γ has breeding
513 value $Z = \gamma$, while a diploid individual with genotype γ_1, γ_2 has breeding value $Z = (\gamma_1 + \gamma_2)/2$.

514 At the beginning of each generation, M mated females each produce K female workers on
515 their home patch. Each worker has a probability Z of being sterile. The patch average sterility
516 z determines the colony productivity r_z and the proportion of males produced by the queen p_z .
517 The next generation of breeders is then produced: first, a patch is randomly selected from the
518 population with probability proportional to its colony efficiency, r_z , and a female is produced by
519 the queen on that patch; then, another n patches are randomly selected with replacement, with
520 probability proportional to their colony efficiency, and each of these n patches produces a male
521 (from the queen with probability p_z , or from a random reproductive worker on that patch with

522 probability $1 - p_z$); the female mates with these n males, and this process is performed M times,
 523 at which point all the M mated females replace the foundresses of existing patches. All other
 524 individuals on each patch die, returning the population to the beginning of the life cycle.

525 Simulations start with a monomorphic population in which all $\gamma = 0$, and hence $Z = 0$ for each
 526 individual. A gene in a newly-produced individual has a 1% probability of mutating, in which
 527 case its genic value changes from γ to $\gamma' = \max(0, \min(\gamma + \delta, 1))$, where δ is drawn from a normal
 528 distribution with mean 0 and standard deviation 0.01. We validated this stochastic individual-
 529 based model by using it to verify the analytical conditions of Olejarz *et al.* (2015; not shown).

530 Appendix A: Explicit population-genetics analysis

531 Here, we analyse the invasion of a sterility allele into a wild-type population. The population
 532 is initially monomorphic for an allele A encoding sterility with penetrance $0 \leq u \leq 1$, and a rare
 533 mutant allele a is introduced which encodes sterility with penetrance $0 \leq v \leq 1$. Throughout, we
 534 closely follow the approach of Olejarz *et al.* (2015), whose analysis is equivalent to ours with the
 535 assumptions that u and v are restricted to either 0 or 1.

536 We denote colony types by the genotype of the queen and the genotypes of her mating partners.
 537 Hence, $X_{AA,m}$ is the frequency of colonies with an AA queen, m mutant (a) males, and $n - m$
 538 wild-type (A) males; similarly for $X_{Aa,m}$ and $X_{aa,m}$. At any given time step, we also keep track
 539 of the number of reproductive females of each genotype— x_{AA} , x_{Aa} , and x_{aa} —and the number
 540 of reproductive males of each genotype— y_A and y_a . Matings between reproductives lead to the
 541 establishment of new colonies; hence, the evolutionary dynamics of colony types are captured by:

$$\begin{aligned}
 \dot{X}_{AA,m} &= x_{AA} \binom{n}{m} y_A^{n-m} y_a^m - \phi X_{AA,m} \\
 \dot{X}_{Aa,m} &= x_{Aa} \binom{n}{m} y_A^{n-m} y_a^m - \phi X_{Aa,m} \\
 \dot{X}_{aa,m} &= x_{aa} \binom{n}{m} y_A^{n-m} y_a^m - \phi X_{aa,m}.
 \end{aligned} \tag{11}$$

542 That is, the rate of establishment of new AA , m colonies is proportional to the frequency of repro-
 543 ductive AA females, multiplied by their probability of mating with exactly $n - m$ wild-type males
 544 and m mutant males; similarly for Aa , m and aa , m colonies.

545 The death rate of existing colonies, ϕ , is defined as

$$\phi = (x_{AA} + x_{Aa} + x_{aa})(y_A + y_a)^n \quad (12)$$

546 in order to enforce a density constraint, namely:

$$\sum_{m=0}^n (X_{AA,m} + X_{Aa,m} + X_{aa,m}) = 1. \quad (13)$$

547 **Reproductives if the mutant allele is dominant**

548 When the mutant allele is dominant, the production of each type of reproductive female (x_{AA} , x_{Aa} ,
 549 x_{aa}) and male (y_A , y_a) is:

$$x_{AA} = \sum_{m=0}^n \left\{ \frac{n-m}{n} r \frac{(n-m)u+mv}{n} X_{AA,m} + \frac{n-m}{2n} r \frac{(n-m)u+(n+m)v}{2n} X_{Aa,m} \right\}$$

$$x_{Aa} = \sum_{m=0}^n \left\{ \frac{m}{n} r \frac{(n-m)u+mv}{n} X_{AA,m} + \frac{1}{2} r \frac{(n-m)u+(n+m)v}{2n} X_{Aa,m} + \frac{n-m}{n} r v X_{aa,m} \right\}$$

$$x_{aa} = \sum_{m=0}^n \left\{ \frac{m}{2n} r \frac{(n-m)u+(n+m)v}{2n} X_{Aa,m} + \frac{m}{n} r v X_{aa,m} \right\}$$

$$y_A = \sum_{m=0}^n \left\{ \begin{aligned} & \left(p \frac{(n-m)u+mv}{n} + \frac{n-m}{n} \frac{(1-u) + \frac{1}{2} \frac{m}{n} (1-v)}{(1-u) + \frac{m}{n} (1-v)} \left(1 - p \frac{(n-m)u+mv}{n} \right) \right) r \frac{(n-m)u+mv}{n} X_{AA,m} \\ & + \left(\frac{1}{2} p \frac{(n-m)u+(n+m)v}{2n} + \frac{n-m}{2n} \frac{(1-u) + \frac{1}{2} (1-v)}{(1-u) + \frac{m}{2n} (1-v)} \left(1 - p \frac{(n-m)u+(n+m)v}{2n} \right) \right) r \frac{(n-m)u+(n+m)v}{2n} X_{Aa,m} \\ & + \left(\frac{1}{2} \frac{n-m}{n} \right) (1 - p v) r v X_{aa,m} \end{aligned} \right\}$$

$$y_a = \sum_{m=0}^n \left\{ \begin{aligned} & \left(\frac{\frac{1}{2} \frac{m}{n} (1-v)}{\frac{n-m}{n} (1-u) + \frac{m}{n} (1-v)} \left(1 - p_{\frac{(n-m)u+mv}{n}} \right) \right) r_{\frac{(n-m)u+mv}{n}} X_{AA,m} \\ & + \left(\frac{1}{2} p_{\frac{(n-m)u+(n+m)v}{2n}} + \frac{\frac{1}{4} (1-v) + \frac{m}{2n} (1-v)}{\frac{n-m}{2n} (1-u) + \frac{1}{2} (1-v) + \frac{m}{2n} (1-v)} \left(1 - p_{\frac{(n-m)u+(n+m)v}{2n}} \right) \right) r_{\frac{(n-m)u+(n+m)v}{2n}} X_{Aa,m} \\ & + \left(p_v + \left(\frac{1}{2} \frac{n-m}{n} + \frac{m}{n} \right) (1 - p_v) \right) r_v X_{aa,m} \end{aligned} \right\}. \quad (14)$$

550 These equations can be understood as follows. First, note that in an AA, m colony, a fraction
 551 $z = \frac{n-m}{n} u + \frac{m}{n} v = \frac{(n-m)u+mv}{n}$ of workers will be sterile (AA workers with probability u , and Aa
 552 workers with probability v); in an Aa, m colony, a fraction $z = \frac{n-m}{2n} u + \frac{1}{2} v + \frac{m}{2n} v = \frac{(n-m)u+(n+m)v}{2n}$
 553 of workers will be sterile (AA workers with probability u , and Aa and aa workers with probability
 554 v); and in an aa, m colony, a fraction $z = \frac{n-m}{n} v + \frac{m}{n} v = v$ of workers will be sterile (Aa and aa
 555 workers with probability v). That is why these values of z as subscripts to the r_z and p_z functions
 556 are always associated, above, with their associated colony frequencies, $X_{AA,m}$, $X_{Aa,m}$, and $X_{aa,m}$,
 557 respectively.

558 For female reproductives, each separate term within the curly braces above combines three
 559 elements; we will take the first term in curly braces in the x_{AA} line,

$$\frac{n-m}{n} r_{\frac{nu+m(v-u)}{n}} X_{AA,m},$$

560 as an example. The three elements are the frequency of a given colony type (*i.e.*, $X_{AA,m}$); the
 561 productivity of that colony type, as a function of the fraction of sterile workers within colonies of
 562 that type (*i.e.*, $r_{\frac{nu+m(v-u)}{n}}$); and the fraction of females and/or males produced by that colony type
 563 with the corresponding genotype (*i.e.*, a fraction $\frac{n-m}{n}$ of females produced in AA, m colonies have
 564 genotype AA , which is why they add to the quantity x_{AA}). Each term within equation 14 can be
 565 broken down in this way.

566 Accordingly, the production of female reproductives can be understood as follows: AA, m
 567 colonies produce $\frac{n-m}{n}$ AA females and $\frac{m}{n}$ Aa females; Aa, m colonies produce $\frac{n-m}{2n}$ AA females, $\frac{1}{2}$

568 Aa females, and $\frac{m}{2n}$ aa females; and aa , m colonies produce $\frac{n-m}{n}$ Aa females and $\frac{m}{n}$ aa females.

569 Male production is more complicated, since both queens and workers produce males, but the
570 principle is the same. We will take the first term in curly braces in the y_A line,

$$\left(p \frac{(n-m)u+mv}{n} + \frac{\frac{n-m}{n}(1-u) + \frac{1}{2} \frac{m}{n}(1-v)}{\frac{n-m}{n}(1-u) + \frac{m}{n}(1-v)} \left(1 - p \frac{(n-m)u+mv}{n} \right) \right) r \frac{(n-m)u+mv}{n} X_{AA,m},$$

571 as an example. Here, the overall productivity of AA, m colonies (*i.e.*, $r \frac{(n-m)u+mv}{n} X_{AA,m}$) goes to-
572 ward the production of both the queen's sons and workers' sons. In particular, the queen is AA ,
573 so all her sons have genotype A , and the queen produces a fraction $p \frac{(n-m)u+mv}{n}$ of males in the
574 colony. Simultaneously, the workers—whose sons comprise a fraction $1 - p \frac{(n-m)u+mv}{n}$ of colony
575 male production—are $\frac{n-m}{n}$ AA and $\frac{m}{n}$ Aa ; in the former group, workers are reproductive with
576 probability $1 - u$, while in the latter group, workers are reproductive with probability $1 - v$; and
577 all the sons of the first group will be A , while only half of the sons of the second group will be
578 A . Hence, overall, a fraction $\left(\frac{\frac{n-m}{n}(1-u) + \frac{1}{2} \frac{m}{n}(1-v)}{\frac{n-m}{n}(1-u) + \frac{m}{n}(1-v)} \right) \left(1 - p \frac{(n-m)u+mv}{n} \right)$ of males produced in AA, m
579 colonies are A males produced by workers. Note that the expressions for y_A and y_a can be further
580 simplified, but we have left them in the form above to maximise clarity.

581 Accordingly, the production of male reproductives can be understood as follows. In AA, m
582 colonies, the queen's sons are all A ; all of the sons of AA workers and half of the sons of Aa
583 workers are A , while the other half of the sons of Aa workers are a . In Aa, m colonies, the queen's
584 sons are half A and half a ; all of the sons of AA workers and half of the sons of Aa workers are A ,
585 while the other half of the sons of Aa workers and all of the sons of aa workers are a . Finally, in
586 aa, m colonies, the queen's sons are all a ; half of the sons of Aa workers are A , while the other half
587 of the sons of Aa workers and all the sons of aa workers are a .

588 **Reproductives if the mutant allele is recessive**

589 Along similar principles, when the mutant allele is recessive, the production of each type of repro-
590 ductive female and male is:

$$\begin{aligned}
 x_{AA} &= \sum_{m=0}^n \left\{ \frac{n-m}{n} r_u X_{AA,m} + \frac{n-m}{2n} r_{\frac{(2n-m)u+mv}{2n}} X_{Aa,m} \right\} \\
 x_{Aa} &= \sum_{m=0}^n \left\{ \frac{m}{n} r_u X_{AA,m} + \frac{1}{2} r_{\frac{(2n-m)u+mv}{2n}} X_{Aa,m} + \frac{n-m}{n} r_{\frac{(n-m)u+mv}{n}} X_{aa,m} \right\} \\
 x_{aa} &= \sum_{m=0}^n \left\{ \frac{m}{2n} r_{\frac{(2n-m)u+mv}{2n}} X_{Aa,m} + \frac{m}{n} r_{\frac{(n-m)u+mv}{n}} X_{aa,m} \right\} \\
 y_A &= \sum_{m=0}^n \left\{ \begin{aligned} &\left(p_u + \left(\frac{n-m}{n} + \frac{1}{2} \frac{m}{n} \right) (1-p_u) \right) r_u X_{AA,m} \\ &+ \left(\frac{1}{2} p_{\frac{(2n-m)u+mv}{2n}} + \frac{\frac{n-m}{2n}(1-u) + \frac{1}{4}(1-u)}{\frac{n-m}{2n}(1-u) + \frac{1}{2}(1-u) + \frac{m}{2n}(1-v)} \left(1 - p_{\frac{(2n-m)u+mv}{2n}} \right) \right) r_{\frac{(2n-m)u+mv}{2n}} X_{Aa,m} \\ &+ \left(\frac{\frac{1}{2} \frac{n-m}{n}(1-u)}{\frac{n-m}{n}(1-u) + \frac{m}{n}(1-v)} \right) (1 - p_{\frac{(n-m)u+mv}{n}}) r_{\frac{(n-m)u+mv}{n}} X_{aa,m} \end{aligned} \right\} \\
 y_a &= \sum_{m=0}^n \left\{ \begin{aligned} &\left(\frac{1}{2} \frac{m}{n} \right) (1-p_u) r_u X_{AA,m} \\ &+ \left(\frac{1}{2} p_{\frac{(2n-m)u+mv}{2n}} + \frac{\frac{1}{4}(1-u) + \frac{m}{2n}(1-v)}{\frac{n-m}{2n}(1-u) + \frac{1}{2}(1-u) + \frac{m}{2n}(1-v)} \left(1 - p_{\frac{(2n-m)u+mv}{2n}} \right) \right) r_{\frac{(2n-m)u+mv}{2n}} X_{Aa,m} \\ &+ \left(p_{\frac{(n-m)u+mv}{n}} + \left(\frac{\frac{1}{2} \frac{n-m}{n}(1-u) + \frac{m}{n}(1-v)}{\frac{n-m}{n}(1-u) + \frac{m}{n}(1-v)} \right) (1 - p_{\frac{(n-m)u+mv}{n}}) \right) r_{\frac{(n-m)u+mv}{n}} X_{aa,m} \end{aligned} \right\}. \tag{15}
 \end{aligned}$$

591 These equations can be understood similarly to equation 14; in fact, they are identical, except
 592 for two general changes. First, the subscripts to r_z and p_z are different, because the mutant allele is
 593 recessive instead of dominant, which results in different proportions of sterile workers in colonies
 594 of each type: in an AA, m colony, a fraction $z = \frac{n-m}{n}u + \frac{m}{n}u = u$ of workers will be sterile; in an
 595 Aa, m colony, a fraction $z = \frac{n-m}{2n}u + \frac{1}{2}u + \frac{m}{2n}v = \frac{(2n-m)u+mv}{2n}$ of workers will be sterile; and in an
 596 aa, m colony, a fraction $z = \frac{n-m}{n}u + \frac{m}{n}v = \frac{(n-m)u+mv}{n}$ of workers will be sterile. Second, because

597 of these differing proportions of sterile workers, the production of sons by workers is different, so
 598 the coefficients of $1 - p_z$ in the fourth and fifth lines are different.

599 **Condition for invasion of a dominant mutant sterility allele**

600 Continuing to follow the approach of Olejarz *et al.* (2015): for a dominant mutant sterility allele,
 601 whether the allele increases in frequency from rarity is governed by the behaviour of $AA, 0$, $AA, 1$,
 602 and $Aa, 0$ colonies. Colony types with more copies of the mutant allele are rarer, and so will have
 603 a negligible effect on invasion. Therefore, from equation 11, we need only consider:

$$\begin{aligned}\dot{X}_{AA,0} &= x_{AA}y_A^n - \phi X_{AA,0} \\ \dot{X}_{AA,1} &= nx_{AA}y_A^{n-1}y_a - \phi X_{AA,1} \\ \dot{X}_{Aa,0} &= x_{Aa}y_A^n - \phi X_{Aa,0}.\end{aligned}\tag{16}$$

604 We start with a wild-type population ($X_{AA,0} = 1$) and introduce a small perturbation of magni-
 605 tude $\epsilon \ll 1$. Considering the density constraint (equation 13), and only keeping terms up to order
 606 ϵ , this gives

$$\begin{aligned}X_{AA,0} &= 1 - \epsilon(\delta_{AA,1}^{(1)} + \delta_{Aa,0}^{(1)}) - \mathcal{O}(\epsilon^2) \\ X_{AA,1} &= \epsilon\delta_{AA,1}^{(1)} + \mathcal{O}(\epsilon^2) \\ X_{Aa,0} &= \epsilon\delta_{Aa,0}^{(1)} + \mathcal{O}(\epsilon^2),\end{aligned}\tag{17}$$

607 which implies that

$$\begin{aligned}\dot{X}_{AA,1} &= \epsilon\dot{\delta}_{AA,1}^{(1)} + \mathcal{O}(\epsilon^2) \\ \dot{X}_{Aa,0} &= \epsilon\dot{\delta}_{Aa,0}^{(1)} + \mathcal{O}(\epsilon^2).\end{aligned}\tag{18}$$

608 Substituting 17 into 14, and keeping terms only up to order ϵ , gives

$$\begin{aligned}
 x_{AA} &= r_u + \epsilon \left(-r_u(\delta_{AA,1}^{(1)} + \delta_{Aa,0}^{(1)}) + \frac{n-1}{n} r_{\frac{(n-1)u+v}{n}} \delta_{AA,1}^{(1)} + \frac{1}{2} r_{\frac{u+v}{2}} \delta_{Aa,0}^{(1)} \right) + \mathcal{O}(\epsilon^2) \\
 x_{Aa} &= \epsilon \left(\frac{1}{n} r_{\frac{(n-1)u+v}{n}} \delta_{AA,1}^{(1)} + \frac{1}{2} r_{\frac{u+v}{2}} \delta_{Aa,0}^{(1)} \right) + \mathcal{O}(\epsilon^2) \\
 x_{aa} &= 0 + \mathcal{O}(\epsilon^2) \\
 y_A &= r_u + \epsilon \left(\begin{aligned} &-r_u(\delta_{AA,1}^{(1)} + \delta_{Aa,0}^{(1)}) + \frac{2n(1-u)+2u-1-v+(1-v)p}{2(n(1-u)-(v-u))} r_{\frac{(n-1)u+v}{n}} \delta_{AA,1}^{(1)} \\ &+ \frac{3-2u-v-(1-u)p}{2(2-u-v)} r_{\frac{u+v}{2}} \delta_{Aa,0}^{(1)} \end{aligned} \right) + \mathcal{O}(\epsilon^2) \\
 y_a &= \epsilon \left(\frac{(1-v)(1-p)}{2(n(1-u)-(v-u))} r_{\frac{(n-1)u+v}{n}} \delta_{AA,1}^{(1)} + \frac{1-v+(1-u)p}{2(2-u-v)} r_{\frac{u+v}{2}} \delta_{Aa,0}^{(1)} \right) + \mathcal{O}(\epsilon^2) \quad (19)
 \end{aligned}$$

609 Finally, substituting 12, 18, and 19 into 16 and discarding powers of ϵ^2 or higher gives

$$\begin{aligned}
 \epsilon \dot{\delta}_{AA,1} &= \epsilon r_u^n \left(-r_u \delta_{AA,1}^{(1)} + n \left(\frac{(1-v)(1-p)}{2(n(1-u)-(v-u))} r_{\frac{(n-1)u+v}{n}} \delta_{AA,1}^{(1)} + \frac{1-v+(1-u)p}{2(2-u-v)} r_{\frac{u+v}{2}} \delta_{Aa,0}^{(1)} \right) \right) \\
 \epsilon \dot{\delta}_{Aa,0} &= \epsilon r_u^n \left(\frac{1}{n} r_{\frac{(n-1)u+v}{n}} \delta_{AA,1}^{(1)} - r_u \delta_{Aa,0}^{(1)} + \frac{1}{2} r_{\frac{u+v}{2}} \delta_{Aa,0}^{(1)} \right).
 \end{aligned}$$

610 This can be rewritten in matrix form as

$$\begin{bmatrix} \dot{\delta}_{AA,1}^{(1)} \\ \dot{\delta}_{Aa,0}^{(1)} \end{bmatrix} = \begin{bmatrix} r_u^n \left(-r_u + n \frac{(1-v)(1-p)}{2(n(1-u)-(v-u))} r_{\frac{(n-1)u+v}{n}} \right) & r_u^n n \frac{1-v+(1-u)p}{2(2-u-v)} r_{\frac{u+v}{2}} \\ r_u^n \frac{1}{n} r_{\frac{(n-1)u+v}{n}} & r_u^n \left(-r_u + \frac{1}{2} r_{\frac{u+v}{2}} \right) \end{bmatrix} \begin{bmatrix} \delta_{AA,1}^{(1)} \\ \delta_{Aa,0}^{(1)} \end{bmatrix}.$$

611 If the dominant eigenvalue of the above matrix is greater than zero, then a dominant sterility
 612 allele with penetrance v can invade a population monomorphic for sterility with penetrance u .

613 This condition, after simplification, is

$$\frac{r \frac{u+v}{2}}{r_u} \left(1 + \left(\frac{(1-u)p \frac{u+v}{2}}{2-u-v} + \frac{(1-v) \left((2-n)(u-v) + n(2-u-v)p \frac{(n-1)u+v}{n} \right)}{2(n(1-u)+u-v)(2-u-v)} \right) \frac{r \frac{(n-1)u+v}{n}}{r_u} \right) + \frac{n(1-v)(1-p \frac{(n-1)u+v}{n}) r \frac{(n-1)u+v}{n}}{n(1-u)+u-v} \frac{r \frac{u+v}{2}}{r_u} > 2. \quad (20)$$

614 Condition for invasion of a recessive mutant sterility allele

615 For a recessive mutant sterility allele, whether the allele increases in frequency from rarity is gov-
 616 erned by the behaviour of $AA,0$, $AA,1$, $Aa,0$, $AA,2$, $Aa,1$, and $aa,0$ colonies. Colony types with
 617 more copies of the mutant allele are rarer, and so will have a negligible effect on invasion. There-
 618 fore, from equation 11, we need only consider:

$$\begin{aligned} \dot{X}_{AA,0} &= x_{AA}y_A^n - \phi X_{AA,0} \\ \dot{X}_{AA,1} &= nx_{AA}y_A^{n-1}y_a - \phi X_{AA,1} \\ \dot{X}_{Aa,0} &= x_{Aa}y_A^n - \phi X_{Aa,0} \\ \dot{X}_{AA,2} &= \frac{n(n-1)}{2}x_{AA}y_A^{n-2}y_a^2 - \phi X_{AA,2} \\ \dot{X}_{Aa,1} &= nx_{Aa}y_A^{n-1}y_a - \phi X_{Aa,1} \\ \dot{X}_{aa,0} &= x_{aa}y_A^n - \phi X_{aa,0}. \end{aligned} \quad (21)$$

619 We start with a wild-type population ($X_{AA,0} = 1$) and introduce a small perturbation of mag-
 620 nitude $\epsilon \ll 1$. Considering the density constraint (equation 13), and only keeping terms up to
 621 order ϵ^2 (since terms of order ϵ alone are not sufficient to determine whether the recessive allele
 622 invades), this gives

$$X_{AA,0} = 1 - \epsilon \delta_{AA,0}^{(1)} - \epsilon^2 \delta_{AA,0}^{(2)} - \mathcal{O}(\epsilon^3)$$

$$\begin{aligned}
 &= 1 - \epsilon(\delta_{AA,1}^{(1)} + \delta_{Aa,0}^{(1)}) - \epsilon^2(\delta_{AA,1}^{(2)} + \delta_{Aa,0}^{(2)} + \delta_{AA,2}^{(2)} + \delta_{Aa,1}^{(2)} + \delta_{aa,0}^{(2)}) - \mathcal{O}(\epsilon^3) \\
 X_{AA,1} &= \epsilon\delta_{AA,1}^{(1)} + \epsilon^2\delta_{AA,1}^{(2)} + \mathcal{O}(\epsilon^3) \\
 X_{Aa,0} &= \epsilon\delta_{Aa,0}^{(1)} + \epsilon^2\delta_{Aa,0}^{(2)} + \mathcal{O}(\epsilon^3) \\
 X_{AA,2} &= \epsilon^2\delta_{AA,2}^{(2)} + \mathcal{O}(\epsilon^3) \\
 X_{Aa,1} &= \epsilon^2\delta_{Aa,1}^{(2)} + \mathcal{O}(\epsilon^3) \\
 X_{aa,0} &= \epsilon^2\delta_{aa,0}^{(2)} + \mathcal{O}(\epsilon^3), \tag{22}
 \end{aligned}$$

623 which implies that

$$\begin{aligned}
 \dot{X}_{AA,0} &= -\epsilon\dot{\delta}_{AA,0}^{(1)} - \epsilon^2\dot{\delta}_{AA,0}^{(2)} - \mathcal{O}(\epsilon^3) \\
 &= -\epsilon(\dot{\delta}_{AA,1}^{(1)} + \dot{\delta}_{Aa,0}^{(1)}) - \epsilon^2(\dot{\delta}_{AA,1}^{(2)} + \dot{\delta}_{Aa,0}^{(2)} + \dot{\delta}_{AA,2}^{(2)} + \dot{\delta}_{Aa,1}^{(2)} + \dot{\delta}_{aa,0}^{(2)}) - \mathcal{O}(\epsilon^3) \\
 \dot{X}_{AA,1} &= \epsilon\dot{\delta}_{AA,1}^{(1)} + \epsilon^2\dot{\delta}_{AA,1}^{(2)} + \mathcal{O}(\epsilon^3) \\
 \dot{X}_{Aa,0} &= \epsilon\dot{\delta}_{Aa,0}^{(1)} + \epsilon^2\dot{\delta}_{Aa,0}^{(2)} + \mathcal{O}(\epsilon^3) \\
 \dot{X}_{AA,2} &= \epsilon^2\dot{\delta}_{AA,2}^{(2)} + \mathcal{O}(\epsilon^3) \\
 \dot{X}_{Aa,1} &= \epsilon^2\dot{\delta}_{Aa,1}^{(2)} + \mathcal{O}(\epsilon^3) \\
 \dot{X}_{aa,0} &= \epsilon^2\dot{\delta}_{aa,0}^{(2)} + \mathcal{O}(\epsilon^3). \tag{23}
 \end{aligned}$$

624 Substituting equation 22 into equation 15, and keeping terms only up to order ϵ^2 , gives

$$\begin{aligned}
 x_{AA} &= r_u + \epsilon \left(-\frac{1}{n}r_u\delta_{AA,1}^{(1)} - \frac{1}{2}r_u\delta_{Aa,0}^{(1)} \right) + \epsilon^2 \left(\begin{array}{l} -\frac{1}{n}r_u\delta_{AA,1}^{(2)} - \frac{1}{2}r_u\delta_{Aa,0}^{(2)} - \frac{2}{n}r_u\delta_{AA,2}^{(2)} \\ + \left(\frac{n-1}{2n}r_{\frac{(2n-1)u+v}{2n}} - r_u \right) \delta_{Aa,1}^{(2)} - r_u\delta_{aa,0}^{(2)} \end{array} \right) + \mathcal{O}(\epsilon^3) \\
 x_{Aa} &= \epsilon \left(\frac{1}{n}r_u\delta_{AA,1}^{(1)} + \frac{1}{2}r_u\delta_{Aa,0}^{(1)} \right) + \epsilon^2 \left(\frac{r_u}{n}\delta_{AA,1}^{(2)} + \frac{1}{2}r_u\delta_{Aa,0}^{(2)} + \frac{2}{n}r_u\delta_{AA,2}^{(2)} + \frac{1}{2}r_{\frac{(2n-1)u+v}{2n}} + r_u\delta_{aa,0}^{(2)} \right) + \mathcal{O}(\epsilon^3) \\
 x_{aa} &= \epsilon^2 \left(\frac{1}{2n}r_{\frac{(2n-1)u+v}{2n}}\delta_{Aa,1}^{(2)} \right) + \mathcal{O}(\epsilon^3)
 \end{aligned}$$

$$\begin{aligned}
 y_A &= r_u + \epsilon \left(-\frac{1-p_u}{2n} r_u \delta_{AA,1}^{(1)} - \frac{1+p_u}{4} r_u \delta_{Aa,0}^{(1)} \right) \\
 &\quad + \epsilon^2 \left(-\frac{2-p_u}{2n} r_u \delta_{AA,1}^{(2)} - \frac{1+p_u}{4} r_u \delta_{Aa,0}^{(2)} - \frac{1-p_u}{n} r_u \delta_{AA,2}^{(2)} \right. \\
 &\quad \left. + \left(\frac{(3n-2)(1-u) + (2-n(1-u)-u-v)p}{2n(1-u)-(v-u)} \frac{(2n-1)u+v}{2n} r_{\frac{(2n-1)u+v}{2n}} - r_u \right) \delta_{Aa,1}^{(2)} - \frac{1+p_u}{2} r_u \delta_{aa,0}^{(2)} \right) + \mathcal{O}(\epsilon^3) \\
 y_a &= \epsilon \left(\frac{1-p_u}{2n} r_u \delta_{AA,1}^{(1)} + \frac{1+p_u}{4} r_u \delta_{Aa,0}^{(1)} \right) \\
 &\quad + \epsilon^2 \left(\frac{1-p_u}{2n} r_u \delta_{AA,1}^{(2)} + \frac{1+p_u}{4} r_u \delta_{Aa,0}^{(2)} + \frac{1-p_u}{n} r_u \delta_{AA,2}^{(2)} \right. \\
 &\quad \left. + \frac{2+n(1-u)-2v-(2-n(1-u)-u-v)p}{2(2n(1-u)-(v-u))} \frac{(2n-1)u+v}{2n} r_{\frac{(2n-1)u+v}{2n}} \delta_{Aa,1}^{(2)} + \frac{1+p_u}{2} r_u \delta_{aa,0}^{(2)} \right) + \mathcal{O}(\epsilon^3). \quad (24)
 \end{aligned}$$

625 Substituting equations 12, 23, and 24 into equation 21 and discarding powers of ϵ^2 or higher
 626 gives, in matrix form,

$$\begin{bmatrix} \delta_{AA,1}^{(1)} \\ \delta_{Aa,0}^{(1)} \end{bmatrix} = r_u^{n+1} \begin{bmatrix} -\frac{1+p_u}{2} & \frac{n(1+p_u)}{4} \\ \frac{1}{n} & -\frac{1}{2} \end{bmatrix} \begin{bmatrix} \delta_{AA,1}^{(1)} \\ \delta_{Aa,0}^{(1)} \end{bmatrix}.$$

627 The dominant eigenvalue is 0, and its corresponding eigenvector is $\begin{bmatrix} n \\ 2 \end{bmatrix}$, which gives

$$\begin{aligned}
 \delta_{AA,1}^{(1)} &= \frac{n}{n+2} \delta_{AA,0}^{(1)} \\
 \delta_{Aa,0}^{(1)} &= \frac{2}{n+2} \delta_{AA,0}^{(1)}. \quad (25)
 \end{aligned}$$

628 (In other words, this tells us how to “distribute” the first-order perturbation to $X_{AA,0}$ over the
 629 first-order perturbations to $X_{AA,1}$ and $X_{Aa,0}$.)

630 Substituting equations 12, 23, 24, and 25 into equation 21, and keeping terms up to order ϵ^2 ,
 631 gives

$$\begin{aligned}
-\delta_{AA,0}^{(2)} &= \frac{2-n-np_u}{4n} r_u^{n+1} \left(-2\delta_{AA,1}^{(2)} + n\delta_{Aa,0}^{(2)} \right) + \frac{(-2+np_u)}{n} r_u^{n+1} \delta_{AA,2}^{(2)} \\
&\quad + r_u^n \left(r_u + \frac{\begin{pmatrix} v-u-n(2+n^2(1-u)-u-v+2n(2-u-v)) \\ +n^2(2-n(1-u)-u-v) p \frac{(2n-1)u+v}{2n} \end{pmatrix}}{2n(2n(1-u)-(v-u))} r \frac{(2n-1)u+v}{2n} \right) \delta_{Aa,1}^{(2)} \\
&\quad - \frac{1}{2} n(1+p_u) r_u^{n+1} \delta_{aa,0}^{(2)} + \frac{n(3+n)r_u^{n+1}}{2(2+n)^2} \left(\delta_{AA,0}^{(1)} \right)^2 \\
\delta_{AA,1}^{(2)} &= \frac{1}{4} (1+p_u) r_u^{n+1} \left(-2\delta_{AA,1}^{(2)} + n\delta_{Aa,0}^{(2)} \right) - (-1+p_u) r_u^{n+1} \delta_{AA,2}^{(2)} \\
&\quad + \frac{\left(n \left(n(-1+u) + 2(-1+v) + (2+n(-1+u) - u - v) p \frac{(2n-1)u+v}{2n} \right) r_u^n r \frac{(2n-1)u+v}{2n} \right)}{2(2n(-1+u) - u + v)} \delta_{Aa,1}^{(2)} \\
&\quad + \frac{1}{2} n(1+p_u) r_u^{n+1} \delta_{aa,0}^{(2)} - \frac{(n(1+n)r_u^{n+1})}{(2+n)^2} \left(\delta_{AA,0}^{(1)} \right)^2 \\
\delta_{Aa,0}^{(2)} &= -\frac{r_u^{n+1}}{2n} \left(-2\delta_{AA,1}^{(2)} + n\delta_{Aa,0}^{(2)} \right) + \frac{2r_u^{n+1}}{n} \delta_{AA,2}^{(2)} \\
&\quad + \frac{1}{2} r_u^n r \frac{(2n-1)u+v}{2n} \delta_{Aa,1}^{(2)} + r_u^{n+1} \delta_{aa,0}^{(2)} - \frac{2nr_u^{n+1}}{(2+n)^2} \left(\delta_{AA,0}^{(1)} \right)^2 \\
\delta_{AA,2}^{(2)} &= -r_u^{n+1} \delta_{AA,2}^{(2)} + \frac{(n-1)nr_u^{n+1}}{2(2+n)^2} \left(\delta_{AA,0}^{(1)} \right)^2 \\
\delta_{Aa,1}^{(2)} &= -r_u^{n+1} \delta_{Aa,1}^{(2)} + \frac{2nr_u^{n+1}}{(2+n)^2} \left(\delta_{AA,0}^{(1)} \right)^2 \\
\delta_{aa,0}^{(2)} &= -r_u^{n+1} \delta_{aa,0}^{(2)} + \frac{r_u^n r \frac{(2n-1)u+v}{2n}}{2n} \delta_{Aa,1}^{(2)}. \tag{26}
\end{aligned}$$

632 Now, each of these equations must be solved.

633 The equation for $\delta_{AA,2}^{(2)}$ can be directly integrated, yielding:

$$\delta_{AA,2}^{(2)} = \frac{n(n-1)}{2(n+2)^2} \left(\delta_{AA,0}^{(1)} \right)^2 \left(1 - \exp(-r_u^{n+1}t) \right). \quad (27)$$

634 The same can be done for $\delta_{Aa,1}^{(2)}$, yielding:

$$\delta_{Aa,1}^{(2)} = \frac{2n}{(n+2)^2} \left(\delta_{AA,0}^{(1)} \right)^2 \left(1 - \exp(-r_u^{n+1}t) \right). \quad (28)$$

635 Equation 28 can be used to solve for $\delta_{aa,0}^{(2)}$, yielding:

$$\delta_{aa,0}^{(2)} = \frac{r^{\frac{(2n+1)u+v}{2n}}}{(2+n)^2 r_u} \left(\delta_{AA,0}^{(1)} \right)^2 \left(1 - (1 + r_u^{n+1}t) \exp(-r_u^{n+1}t) \right). \quad (29)$$

636 The equations for $\delta_{AA,1}^{(2)}$ and $\delta_{Aa,0}^{(2)}$ can be manipulated to yield

$$\begin{aligned} \frac{d}{dt} (-2\delta_{AA,1}^{(2)} + n\delta_{Aa,0}^{(2)}) &= -\frac{(2+p_u)r_u^{n+1}}{2} (-2\delta_{AA,1}^{(2)} + n\delta_{Aa,0}^{(2)}) + 2p_u r_u^{n+1} \delta_{AA,2}^{(2)} \\ &\quad - \frac{n \left(4 - u - 3v - 2(2 - n(1-u) - u - v) p^{\frac{(2n-1)u+v}{2n}} \right)}{2(2n(1-u) - (v-u))} r_u^n r^{\frac{(2n-1)u+v}{2n}} \delta_{Aa,1}^{(2)} \\ &\quad - np_u r_u^{n+1} \delta_{aa,0}^{(2)} + \frac{2n}{(2+n)^2} r_u^{n+1} \left(\delta_{AA,0}^{(1)} \right)^2, \end{aligned}$$

637 which can be integrated to give

$$\begin{aligned}
 -2\delta_{AA,1}^{(2)} + n\delta_{AA,0}^{(2)} = & \\
 & \left(\frac{2n(2+(n-1)p_u)}{(2+n)^2(2+p_u)} - \frac{2np_u r \frac{(2n-1)u+v}{2n}}{(2+n)^2(2+p_u)r_u} \right) \left(\delta_{AA,0}^{(1)} \right)^2 \\
 & - \frac{2n^2 \left(4-u-3v-2(2-n(1-u)-u-v)p \frac{(2n-1)u+v}{2n} \right) r \frac{(2n-1)u+v}{2n}}{(2+n)^2(2n(1-u)-(v-u))(2+p_u)r_u} \left(\delta_{AA,0}^{(1)} \right)^2 \\
 & + \left(\frac{2n \left(r_u \left(n-1-tr_u^n r \frac{(2n-1)u+v}{2n} \right) - r \frac{(2n-1)u+v}{2n} \right)}{(2+n)^2 r_u} \right) \left(\delta_{AA,0}^{(1)} \right)^2 \exp(-r_u^{n+1}t) \\
 & - \frac{2n \left((3n-2)(v-u)+2n(2-n(1-u)-u-v)p \frac{(2n-1)u+v}{2n} \right) r \frac{(2n-1)u+v}{2n}}{(2+n)^2(2n(1-u)-(v-u))p_u r_u} \left(\delta_{AA,0}^{(1)} \right)^2 \exp(-r_u^{n+1}t) \\
 & + \left(\frac{4n(n-2)}{(2+n)^2(2+p_u)} \right) \left(\delta_{AA,0}^{(1)} \right)^2 \exp\left(-\frac{2+p_u}{2}r_u^{n+1}t\right) \\
 & + \frac{4n \left((3n-2)(v-u)+2n(2-n(1-u)-u-v)p \frac{(2n-1)u+v}{2n} \right) r \frac{(2n-1)u+v}{2n}}{(2+n)^2(2n(1-u)-(v-u))p_u(2+p_u)r_u} \left(\delta_{AA,0}^{(1)} \right)^2 \exp\left(-\frac{2+p_u}{2}r_u^{n+1}t\right).
 \end{aligned} \tag{30}$$

638 We solve for $\delta_{AA,0}^{(2)}$ by substituting equations 27–30 into equation 26. In doing so, we permit t to
 639 become relatively large, such that all the time-dependent terms in equations 27–30 approach zero.
 640 Accordingly, the sign of $\delta_{AA,0}^{(2)}$ tells us that the mutant sterility allele will invade if:

$$\lim_{t \rightarrow \infty} \delta_{AA,0}^{(2)} > 0$$

641 That is, after substitution and simplification, a recessive sterility allele with penetrance v will in-

642 vade a population monomorphic for sterility with penetrance u if

$$\frac{r \frac{(2n-1)u+v}{2n}}{r_u} > \frac{2(2n(1-u) + u - v)(2 + n(1 + p_u))}{\left(\begin{array}{l} n(8 + 4n(1-u) - 3u - 5v) + 2(u-v) \\ + (2+n)(2n(1-u) + u - v)p_u \\ - 2n(2-u-v - n(1-u))p \frac{(2n-1)u+v}{2n} \end{array} \right)}. \quad (31)$$

643 Appendix B: Kin-selection analysis

644 Here, we develop a general model of the evolution of wholly or partly non-reproductive workers
 645 using standard kin selection methodology (Taylor & Frank 1996, Frank 1998). In this model, a
 646 mated queen founds a colony by producing an initial brood of females and/or males. Depend-
 647 ing on the model scenario, first-brood females may either mate with first-brood males—from their
 648 own or from a different colony—or remain unmated. Then, according to the level of worker steril-
 649 ity z , a focal first-brood female (*i.e.*, a worker) invests a proportion of her resources into helping
 650 to raise the colony's next brood—which consists partly of queen-produced offspring (queen-laid
 651 females, notated f , and queen-laid males, notated m) and partly of worker-produced offspring
 652 (worker-laid females, notated ϕ , and worker-laid males, notated μ)—and a proportion of her re-
 653 sources into producing her own offspring. Individuals of the second brood disperse and mate,
 654 with each female mating with n males, and mated females then found new patches, restarting the
 655 cycle.

656 In this model, we denote a focal worker's sterility by Z , the average sterility on a focal patch by
 657 z , and the average sterility in the population by \bar{z} . A focal queen's sex ratio strategy for her second
 658 brood is denoted by x , and the average sex ratio strategy among all queens in the population is
 659 denoted by \bar{x} . The production of queen-laid second-brood females on a focal patch is $f = f(z, x)$;
 660 the production of queen-laid second-brood males on a focal patch is $m = m(z, x)$; the production
 661 of worker-laid females by a focal worker is $\phi = \phi(Z, z, x)$; and the production of worker-laid males
 662 by a focal worker is $\mu = \mu(Z, z, x)$. We denote by $\bar{f} = f(\bar{z}, \bar{x})$, $\bar{m} = m(\bar{z}, \bar{x})$, $\bar{\phi} = \phi(\bar{z}, \bar{z}, \bar{x})$, and
 663 $\bar{\mu} = \mu(\bar{z}, \bar{z}, \bar{x})$ the population-average production of each of these four classes, respectively, and by

664 $\bar{f} = f/\bar{f}$, $\bar{m} = m/\bar{m}$, $\bar{\phi} = \phi/\bar{\phi}$, and $\bar{\mu} = \mu/\bar{\mu}$ the relative production of each of these four classes.

665 For a gene increasing worker sterility to spread, its carriers, on average, should leave more
 666 descendants than other members of the population. Accordingly, natural selection will favour an
 667 increase in worker sterility, z , when

$$\frac{\partial \bar{f}}{\partial z} R_{\text{sis}} + \frac{\partial \bar{m}}{\partial z} R_{\text{bro}} + \frac{\partial \bar{\phi}}{\partial z} R_{\text{dau}} + \frac{\partial \bar{\phi}}{\partial z} R_{\text{niece}} + \frac{\partial \bar{\mu}}{\partial z} R_{\text{son}} + \frac{\partial \bar{\mu}}{\partial z} R_{\text{neph}} > 0. \quad (32)$$

668 Above, R_{sis} , R_{bro} , R_{dau} , R_{niece} , R_{son} , and R_{neph} are the (life-for-life) relatedness between a fo-
 669 cal female worker and her sister, brother, daughter, niece, son, and nephew, respectively, and all
 670 derivatives are evaluated at $Z = z = \bar{z}$.

671 Each term on the left-hand side of condition 32 captures how a small increase in worker steril-
 672 ity impacts upon the fitness of different individuals in the population, weighted by the life-for-life
 673 relatedness between those individuals and a focal worker, which combines both (i) the reproduc-
 674 tive value of those individuals (*i.e.*, their capacity for projecting genes into future generations) and
 675 (ii) the extent to which those individuals themselves carry the gene increasing worker sterility. Al-
 676 ternatively, each term can be read as an inclusive-fitness effect experienced by a focal worker who
 677 gives up reproduction to become sterile. These interpretations are mathematically equivalent, but
 678 we focus on the inclusive-fitness interpretation here, as it is conceptually simpler.

679 Similarly, natural selection will favour an increase in the queen's sex allocation strategy (her
 680 investment in daughters), x , when

$$\frac{\partial \bar{f}}{\partial x} R_{\text{dau|Q}} + \frac{\partial \bar{m}}{\partial x} R_{\text{son|Q}} + \frac{\partial \bar{\phi}}{\partial x} R_{\text{gdau|Q}} + \frac{\partial \bar{\mu}}{\partial x} R_{\text{gson|Q}} > 0. \quad (33)$$

681 Above, $R_{\text{dau|Q}}$ is the relatedness between a focal queen and her daughter, $R_{\text{son|Q}}$ is the relat-
 682 edness between a focal queen and her son, $R_{\text{gdau|Q}}$ is the relatedness between a focal female and
 683 her granddaughter (her daughter's daughter), $R_{\text{gson|Q}}$ is the relatedness between a focal female
 684 and her grandson (her daughter's son), and all derivatives are evaluated at $x = \bar{x}$. Each term
 685 on the left-hand side of condition 33 captures how a small increase in the queen's investment in
 686 daughters, as opposed to sons, impacts upon the fitness of different individuals in the population;

687 alternatively, each term can be read as an inclusive-fitness effect experienced by a focal queen who
688 gives up one of her sons to raise an extra daughter.

689 For scenario A, the production of queen-laid females, queen-laid males, worker-laid females,
690 and worker-laid males is $f = xr_z$, $m = (1 - x)r_z p_z$, $\phi = 0$, and $\mu = (1 - x)r_z(1 - p_z)\frac{1-Z}{1-z}$,
691 respectively. For scenario B, we use $f = xr_z p_z$, $m = (1 - x)r_z p_z$, $\phi = 0$, and $\mu = r_z(1 - p_z)\frac{1-Z}{1-z}$. For
692 scenario C, we use $f = xr_z p_z$, $m = (1 - x)r_z p_z$, $\phi = yr_z(1 - p_z)\frac{1-Z}{1-z}$, and $\mu = (1 - y)r_z(1 - p_z)\frac{1-Z}{1-z}$.
693 And for scenario D, we use $f = x(z + sz^2)$, $m = (1 - x)(z + sz^2)$, $\phi = y(1 - Z)(1 - c)$, and $\mu =$
694 $(1 - y)(1 - Z)(1 - c)$. Substituting these definitions into conditions 32 and 33 recovers conditions
695 5-10 above.

696 Relatedness calculations

697 The life-for-life relatedness of individual A to individual B is $R_{AB} = \frac{F_{AB} c_B}{F_{AA} c_A}$, where F_{AB} is the con-
698 sanguinity of individual A and individual B, F_{AA} is the consanguinity of individual A to herself,
699 c_B is the class reproductive value of individual B, and c_A is the class reproductive value of individ-
700 ual A (Bulmer 1994). Note that since individual A is always the same individual within a given
701 condition above, we can instead use $R_{AB} = F_{AB}c_B$ or any multiple thereof without affecting the
702 resulting conditions.

703 Accordingly, consanguinities needed for the conditions above can be found in Table 1. The con-
704 sanguinities for a female worker under claustral inbreeding are obtained by first calculating the
705 coefficient of inbreeding for a foundress in this mating system (the probability that her two genes
706 at a given locus are identical by descent). Suppose that a juvenile is foundress-laid with probabil-
707 ity Q , and soldier-laid with probability $1 - Q$. If foundress-laid, her coefficient of consanguinity
708 is zero, because patch founders are unrelated. If worker-laid, then her paternally-inherited gene
709 comes from her grandmother, and her maternally-inherited gene comes, with equal probability,
710 either from her grandfather—who is unrelated to her grandmother—or from her grandmother; in
711 the latter case, her two genes are either copies of the “same” gene in her grandmother, in which
712 case they are identical by descent with probability 1, or are copies of “different” genes from her
713 grandmother, in which case they are identical by descent with probability G , where G is the ju-

For outbreeders

Relationship	Notation	Haplodiploidy	Diploidy
female to daughter	F_{dau}	$\frac{1}{4}$	$\frac{1}{4}$
female to son	F_{son}	$\frac{1}{2}$	$\frac{1}{4}$
female to sister	F_{sis}	$\frac{2+n}{8n}$	$\frac{1+n}{8n}$
female to brother	F_{bro}	$\frac{1}{4}$	$\frac{1+n}{8n}$
female to niece	F_{niece}	$\frac{2+n}{16n}$	$\frac{1+n}{16n}$
female to nephew	F_{neph}	$\frac{2+n}{8n}$	$\frac{1+n}{16n}$
female to daughter's daughter	F_{gdau}	$\frac{1}{8}$	$\frac{1}{8}$
female to daughter's son	F_{gson}	$\frac{1}{4}$	$\frac{1}{8}$

For claustral inbreeders

Relationship	Notation	Haplodiploidy	Diploidy
female worker to daughter	$F_{\text{dau c}}$	$\frac{5+Q}{4(3+Q)}$	$\frac{11+Q}{8(3+Q)}$
female worker to son	$F_{\text{son c}}$	$\frac{1}{2}$	$\frac{11+Q}{8(3+Q)}$
female worker to sister	$F_{\text{sis c}}$	$\frac{3+2n+Q}{4n(3+Q)}$	$\frac{1+n}{2n(3+Q)}$
female worker to brother	$F_{\text{bro c}}$	$\frac{1}{3+Q}$	$\frac{1+n}{2n(3+Q)}$
female worker to niece	$F_{\text{niece c}}$	$\frac{3+6n+Q}{8n(3+Q)}$	$\frac{1+n}{2n(3+Q)}$

Table 1: Consanguinities used in inclusive-fitness models.

714 venile's grandmother's coefficient of inbreeding. That is, overall, the probability that these two
 715 genes are identical by descent is $F = (1 - Q)\frac{1}{2} \left(\frac{1+G}{2} \right)$, and at equilibrium, $G = F$, which gives
 716 $F = \frac{1-P}{3+P}$. A similar argument gives the same result under diploidy.

717 Class reproductive values

718 To determine the class reproductive value of each of the four juvenile classes (queen-laid females,
 719 class f ; queen-laid males, class m ; worker-laid females, class φ ; and worker-laid males, class μ), we
 720 first solve for the total reproductive value of all females, $c_F = c_f + c_\varphi$, and the total reproductive
 721 value of all males, $c_M = c_m + c_\mu$. Defining $Q = \frac{\bar{f}}{\bar{f} + \bar{\phi}}$ as the probability that a random female is
 722 queen-laid, and $P = \frac{\bar{m}}{\bar{m} + \bar{\mu}}$ as the probability that a random male is queen-laid, note that a random
 723 female inherits half of her genes from a female in the previous census if she is queen-laid, and three
 724 quarters of her genes from a female in the previous census if she is worker-laid; and a random
 725 male inherits all his genes from a female in the previous census if he is queen-laid, and half of his
 726 genes from a female in the previous census if he is worker-laid. Hence, the recurrence relation
 727 $c_F = \left(\frac{Q}{2} + \frac{3(1-Q)}{4} \right) c_F + \left(P + \frac{1-P}{2} \right) c_M$, with the constraint that $c_M = 1 - c_F$, can be solved to give
 728 $c_F = \frac{2(1+P)}{3+2P+Q}$ and $c_M = \frac{1+Q}{3+2P+Q}$. Since an individual's mating success is not affected by whether
 729 they are queen- or worker-laid, we have $c_f = Qc_F$, $c_\varphi = (1 - Q)c_F$, $c_m = Pc_M$, and $c_\mu = (1 - P)c_M$,
 730 which, overall, gives

$$\begin{aligned} c_f &= \frac{2(1+P)Q}{3+2P+Q} \\ c_m &= \frac{P(1+Q)}{3+2P+Q} \\ c_\varphi &= \frac{2(1+P)(1-Q)}{3+2P+Q} \\ c_\mu &= \frac{(1-P)(1-Q)}{3+2P+Q}. \end{aligned}$$

731 When all second-brood juveniles are queen-laid ($P = Q = 1$), this yields the expected result
 732 that $c_f = 2/3$, $c_m = 1/3$, $c_\varphi = 0$, and $c_\mu = 0$; when all second-brood juveniles are worker-laid

733 ($P = Q = 0$), this yields the expected result that $c_f = 0$, $c_m = 0$, $c_\varphi = 2/3$, and $c_\mu = 1/3$ (Price 1970,
734 Taylor 1996).

735 It is illustrative to examine a special case. When all second-brood females are queen-laid ($Q =$
736 1), this reduces to

$$\begin{aligned}c_f &= \frac{1 + P}{2 + P} \\c_m &= \frac{P}{2 + P} \\c_\varphi &= 0 \\c_\mu &= \frac{1 - P}{2 + P}.\end{aligned}$$

737 In this case, when $P = 1$, we have the expected result that the total value of juvenile females is
738 $2/3$ and the total value of juvenile males is $1/3$, because of the usual asymmetries of haplodiploidy.
739 But when $P = 0$, the total value of juvenile females is $1/2$ and the total value of juvenile males is
740 $1/2$. This is because new juvenile females get half their genes from their mother and half from their
741 father, while new juvenile males are parthenogenetically produced by worker females, and so ul-
742 timately get half their genes from their mother's mother and half their genes from their mother's
743 father. In this way, juvenile females and males have an equal share in producing the next genera-
744 tion of juveniles (*cf.* Boomsma & Grafen 1991).