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**Main Manuscript for**

Inclusive fitness forces of selection in an age-structured population

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25 **Abstract**

26 Current evolutionary theories of senescence predict that the force of selection on survival will  
27 decline from maturity to zero at the age of last reproduction, and the force of selection on  
28 reproduction will decline monotonically from birth. These predictions rest upon the assumption  
29 that individuals within a population do not interact with one another. This assumption,  
30 however, is violated in social species, where an individual's survival and/or reproduction may  
31 shape the fitness of other group members. In such species, it is inclusive fitness that natural  
32 selection optimises. Yet, it remains unclear how the forces of selection on survival and  
33 reproduction might be modified when inclusive fitness, rather than population growth rate, is  
34 considered the appropriate metric for fitness. Here, we derive inclusive fitness forces of  
35 selection for hypothetical populations of social species. We show that selection on survival is  
36 not always constant before maturity, and can remain above zero in post-reproductive age  
37 classes, contrary to conventional models of senescence. We also show how the trajectory of  
38 the force of selection on reproduction does not always decline monotonically from birth, as  
39 predicted by classical theory, but instead depends on the balance of benefits to direct fitness  
40 and costs to indirect fitness. Our theoretical framework provides the unique opportunity to  
41 expand our understanding of senescence across social species, with important implications to  
42 species with variable life histories.

43

44

45 **Main Text**

46 To date, there are no general theories for how senescence might evolve differently in groups  
47 of social species. At the demographic level, senescence is defined as the decline in organismal  
48 fitness with increasing age (1). Hamilton (2) provided a mathematical explanation for the  
49 seemingly counter-intuitive evolution of senescence: the force of natural selection weakens  
50 with age, and so detrimental alleles acting late in life can persist despite their negative effects  
51 on fitness (3 – 6). Two years prior, Hamilton (7 – 8) also introduced the concept of inclusive  
52 fitness, which has had a profound impact on our understanding of the evolution of social life  
53 histories (9 – 11). Inclusive fitness quantifies (*i*) an individual's number of offspring in the  
54 absence of social effects and (*ii*) the effects an individual has on the number of offspring  
55 produced by other individuals, weighted by relatedness (7 – 8). It has not yet been fully  
56 considered from a theoretical standpoint, however, how these effects an individual has on the  
57 fitness of others may alter the evolution of senescence.

58

59 An age-specific force of selection describes the relative effect on fitness at different age classes  
60 of a mutant allele that impacts survival or reproduction. How might the components that  
61 contribute to such age-specific forces of selection differ between a solitary and a social species?  
62 First, consider an individual of a solitary species. When this individual dies, it loses access to  
63 any future reproduction it might have achieved. If a mutant allele arises in this population that  
64 increases the risk of dying at a certain age, say  $x$ , then the force of selection that acts against  
65 the allele is proportional to the expectation of residual reproduction that the individual may  
66 have realised (2). Now, imagine instead a social species in which individuals within a group  
67 influence one another's survival and reproduction, for example, through the provision of  
68 alloparental care or through competition for limiting resources. For an individual, death means  
69 the loss of any future reproduction, just as in the solitary case. However, in social species, an

70 individual's death may also alter the survival and reproduction of other individuals (12 – 13).  
71 For instance, the death of an individual providing alloparental care may lead to a reduction in  
72 breeder productivity. Alternatively, where there is competition within groups for resources, the  
73 death of an individual may release resources that other group members may use for survival  
74 and reproduction. If individuals within a group are related, then these effects will be under kin  
75 selection. For example, an increase in mortality late in life can be adaptive if relatives stand to  
76 benefit from the death of a focal individual (14 – 20). On the other hand, mortality may be  
77 more strongly selected against if individuals can transfer beneficial resources to others (21 –  
78 23). When the death and reproduction of a focal individual not only impacts its own fitness,  
79 but also the fitness of relatives, the force of selection acting on a mutant allele at age  $x$  must  
80 also consider these complex social effects.

81  
82 To incorporate social interactions into the evolutionary theory of senescence, we develop a  
83 general model for quantifying age-specific inclusive fitness forces of selection in social  
84 species. Here, we focus on the effects of cooperative interactions between individuals and the  
85 corresponding forces of selection, but note that our model also has scope to consider other  
86 scenarios, such as cases of harm (see **Discussion**). Using an infinite island framework to  
87 describe a resident social population (16, 20, 24 – 34), we explore the fate of a mutant allele  
88 that alters *(i)* survival rate from age  $x$  to age  $x + 1$  and *(ii)* reproduction at age  $x$ . We derive  
89 inclusive fitness forces of selection acting on these mutant alleles, which indicate how the  
90 efficacy of natural selection changes with age with respect to socio-demographic parameters.  
91 After deriving general analytical results, we explore the applicability of our framework to  
92 different social settings by providing numerical solutions for two examples of social structures:  
93 *(i)* the grandmother hypothesis: post-reproductive individuals aiding juvenile survival and *(ii)*

94 cooperative breeding: juveniles aiding reproduction by adults. We conclude by discussing the  
95 implications and possible extensions for our model.

96

## 97 **Model**

98 We consider a population divided into an infinite number of patches, and model the population  
99 dynamics of a focal patch. This infinite island approach (16, 20, 24 – 34) allows kin selection  
100 to be modelled while also considering the effects of demography, which is appropriate for  
101 considering an age-structured population in which individuals have effects on one another's  
102 fitness. Each patch, which could also be conceptualised as a territory, contains discrete groups  
103 of exactly  $N$  individuals that are, for simplicity, haploid and asexual. We also assume that  
104 patches produce a large number of offspring in each breeding season so that no position on any  
105 patch is vacant at the start of each breeding season (*i.e.* a density-dependent stationary  
106 population). Offspring that establish on to a patch are designated age 1 and can survive until  
107 some maximum age,  $\omega$ , at which point they die. Time proceeds in a series of discrete breeding  
108 seasons, during which individuals have a probability of surviving to the next breeding season,  
109  $p(x)$ , and a rate of reproduction,  $b(x)$ , that may vary with age, and can be described by a  
110 population matrix model ( $A$ ). Individuals may receive contributions to their survival and  
111 reproduction from the other  $N - 1$  individuals on their patch, and may themselves contribute  
112 to the survival and reproduction of the  $N - 1$  conspecifics on the patch.

113

114 Fundamental to this model is the concept of ‘transfers’. Biologically, transfers represent the  
115 help or harm to other individual's fitness components: survival and reproduction. Transfers  
116 occur in the currency of genetic offspring equivalents, the same currency as survival and  
117 reproduction. Here, we assume that the transfers an individual makes to others is a function of  
118 the ages of both the actor and recipient (Fig. 1). We display transfers between individuals as

119  $T_{yz}^x$ : if  $y = 1$ , this represents an individual in age class  $x$ 's social effect on the reproduction of  
120 age class  $z$ , while  $y = z + 1$  would represent an individual in age class  $x$ 's social effect on the  
121 survival of age class  $z$ .

122

123 To quantify the inclusive fitness contributions of a focal individual of age  $x$ , a series of key  
124 considerations must be made. Specifically, we must (i) exclude the fraction of the class- $y$   
125 offspring of a focal class- $x$  individual that are born or survive as a consequence of the social  
126 environment (the help or harm of other individuals), and (ii) augment the total production of  
127 class- $y$  offspring from all other age classes, including other individuals in age class  $x$ , that are  
128 born or survive due to the social contributions of a focal class- $x$  individual. These latter  
129 offspring contributions are weighted by the coefficient of relatedness between an individual of  
130 age class  $x$  and the class- $y$  offspring of the recipient class (7 – 8). For example, a focal  
131 individual aged  $x$  survives with probability  $p(x)$  and has a rate of reproduction  $b(x)$ . A fraction  
132 of these rates of survival and reproduction may be due to social interactions. These fractions  
133 are excluded from the inclusive fitness of the focal individual, leaving  $\dot{p}(x)$  and  $\dot{b}(x)$ , with dot  
134 notation representing the effect of a focal individual's own genotype on its own survival or rate  
135 of reproduction, *i.e.* direct fitness. Of the  $\dot{b}(x)$  offspring produced due to the genotype of an  
136 individual aged  $x$ , a proportion  $d$  disperse, and a proportion  $1 - d$  remain at their natal patch.  
137 A fraction  $c$  of the dispersing offspring die, representing a cost of dispersal. Surviving,  
138 dispersed offspring are evenly distributed among all sites, regardless of distance, and compete  
139 (fair lottery) for sites freed by adults that die in the current breeding season. Asymmetric  
140 competition is assumed so that juveniles do not displace resident adults, and die if they do not  
141 gain a breeding position on a patch. Offspring of a focal individual aged  $x$  face a probability

142 of establishment  $g(x)$  onto their natal patch, and  $\bar{g}$  on a different, random patch in the  
 143 population.

144

145 In a population with social interactions between patch members, we can populate a matrix ( $\mathbf{W}$ )  
 146 with the inclusive fitness (genetic offspring) contributions of individuals in age class  $x$  to  
 147 individuals in age class  $y$  ( $w_{yx}$ ):

148

$$149 \quad w_{yx} = \begin{cases} \dot{p}(x) + T_{x+1,x}^x, & \text{if } y = x + 1 \\ \dot{F}(x), & \text{if } y = 1 \\ 0 \text{ OR } T_{yz}^x \text{ if } y = z + 1 \end{cases},$$

150 [1]

151 where

$$152 \quad \dot{p}(x) = p(x) - \sum_z T_{x+1,x}^z$$

153 [2]

154 and

$$155 \quad \dot{b}(x) = b(x) - \sum_z T_{1,x}^z$$

156 [3]

157 and

$$158 \quad \dot{F}(x) = \dot{b}(x) + \sum_z T_{1,z}^x [(1-d)g(x) + (1-c)d\bar{g}].$$

159 [4]

160

161 The proportions of the survival and reproduction of a focal individual aged  $x$  that are due to  
 162 the genotypes of other individuals are represented in the summation terms on the right-hand  
 163 side of [2] (survival) and [3] (reproduction) (where  $0 \leq \sum_z t_{x+1,x}^z < p(x)$ , and  $0 \leq \sum_z t_{1,x}^z <$

164  $b(x)$ ). Importantly, these proportions are distributed to other age classes, thus ensuring that no  
165 offspring is ‘double counted’ (35 – 36). A focal individual of age  $x$  may also contribute to the  
166 survival and reproduction of others, accumulating indirect fitness through the transfer of  
167 genetic offspring. Contributions to survival are captured as  $T_{y,z}^x$  (where  $y = z + 1$ , and  $y \neq$   
168 1), and reproduction as  $T_{1,z}^x$  (summed across age classes to equal  $\sum_z T_{1,z}^x$ ). The magnitude of  
169 these contributions will depend on i) the expected number of individuals in the recipient age  
170 class, ii) the fraction of the total contribution of all age classes combined to the survival or  
171 reproduction of the recipient age class individuals that is due to a focal individual aged  $x$ , and  
172 iii) the relatedness between a focal individual aged  $x$  and an individual in the recipient age  
173 class (see **Supplementary Information Appendix C**). This approach to modelling social  
174 interactions assumes that there are fractions of survival and fecundity of each age class that are  
175 due to the social environment (which could equal zero), and that these fractions are distributed  
176 to other individuals across age classes. If there are no explicit social interactions between  
177 multiple individuals on a patch, equation [2] simplifies to a population with limited dispersal  
178 and Ronce & Promislow’s (20) kin competition selection gradients can be computed. With full  
179 dispersal (no offspring stay at the patch in which they’re born) and no social interactions,  
180 equation [1] simplifies to Hamilton’s panmictic population, and his forces of selection can be  
181 computed (2).

182

### 183 **An inclusive fitness force of selection**

184 To compute forces of selection, we are ultimately concerned with a hypothetical mutation that  
185 alters survival rate or rate of reproduction at age  $x$ . The derivative of the growth rate of the  
186 mutant population,  $\lambda$ , with respect to the phenotypic effect of the mutation,  $\delta$ , gives an indicator  
187 of the force of selection acting on the mutant allele (2, 20, 37 – 38). We consider mutations of  
188 weak effects (small  $\delta$ ) and first-order effects of selection (39). Using this ‘sensitivity’ approach



189 for an age-structured population (20, 37 – 38, 40 – 42), the force of selection acting on a mutant  
190 allele can be written as:

191

$$192 \quad S = \frac{d\lambda}{d\delta_{\delta=0}} = \sum_x \sum_y \frac{f_x v_y}{\mathbf{f} \cdot \mathbf{v}} \frac{dw_{yx}}{d\delta_{\delta=0}}$$

193 [5]

194 where  $\mathbf{f}$  and  $\mathbf{v}$  are the vector of asymptotic frequencies and the vector of inclusive reproductive  
195 values for the different age classes in the resident population. The term  $f_x$  denotes the  
196 asymptotic frequency of age class  $x$ , and  $\mathbf{f}$  is the dominant right eigenvector of the demographic  
197 projection matrix ( $\mathbf{A}$ ). In this model, the term  $v_x$  represents the inclusive reproductive value of  
198 age class  $x$ , and is instead derived from an inclusive fitness matrix ( $\mathbf{W}$ ) that decomposes the  
199 demographic projection matrix into inclusive fitness contributions between age classes.  
200 Therefore,  $\mathbf{v}$  is the dominant left eigenvector of  $\mathbf{W}$ . Thus, the growth rate of the mutant  
201 population,  $\lambda$ , represents an inclusive fitness growth rate of the allele. Finally, the term  $w_{yx}$   
202 represents the class  $y$  offspring of a class  $x$  individual (genetic offspring equivalents).  
203 Therefore,  $dw_{yx}$  represents the difference in the contribution of an individual age  $x$  to  
204 individuals aged  $y$  in the mutant population compared to the resident population. Overall, the  
205 sign of  $S$  predicts the direction of selection on the mutant allele with respect to the resident  
206 population wild type allele, whilst the magnitude of  $S$  conveys information about the force of  
207 selection (2, 20).

208

### 209 **The inclusive fitness force of selection on survival**

210 A mutant allele that alters the survival rate between age  $x$  and  $x + 1$  changes inclusive fitness  
211 contributions between age class according to the following (see **Supplementary Information**  
212 **Appendix B**):

$$213 \quad dw_{yx} = \begin{cases} d\dot{p}(x), & \text{if } y = x + 1 \\ -d\dot{p}(x)[\dot{h}(x) + \dot{k}(x)\hat{r}(x)], & \text{if } y = 1 \\ 0, & \text{otherwise} \end{cases}$$

214 [6]

215 where  $\dot{h}(x)$  is the proportion of offspring after dispersal at the local patch that are the direct  
 216 and indirect contributions of a focal individual aged  $x$ ,  $\dot{k}(x)$  is the proportion of offspring that  
 217 are born due to the genotypes of other individuals on the patch, and  $\hat{r}(x)$  is the relatedness of  
 218 an individual aged  $x$  to the offspring of other patch mates (see **Supplementary Information**  
 219 **Appendix A**). As we assume mortality occurs between breeding seasons, a focal individual's  
 220 contributions to the survival and reproduction of other age classes are only affected at  $x + 1$ ,  
 221 not in the current breeding season.

222

223 Let  $S_p(x)$  be the component of the force of selection due the effect of a mutant allele on the  
 224 survival rate between age  $x$  and  $x + 1$ . Using equations [5] and [6], in a stationary population  
 225 with limited dispersal and social interactions between individuals, this can be written as:

226

$$227 \quad S_p(x) = \frac{d\dot{p}(x)}{d\delta} \frac{f_x(v_{x+1} - [\dot{h}(x) + \dot{k}(x)\hat{r}(x)]v_1)}{\mathbf{f} \cdot \mathbf{v}}$$

228 [7]

229

230 Equation [7] shows that the overall direction of the force of selection acting on a mutant allele  
 231 that affects the survival rate between age  $x$  and  $x + 1$  is a balance of two forces: the inclusive  
 232 reproductive value at age  $x + 1$  vs the reproductive value of offspring (displaced by the  
 233 survival of the focal individual) that have varying relatedness to the focal individual aged  $x$ .

234 The term  $\mathbf{f} \cdot \mathbf{v}$  acts to scale the forces of selection in terms of generation time (2, 20).

235

236 **The inclusive fitness force of selection on reproduction**

237 A mutant allele that alters reproduction at age  $x$  changes inclusive fitness contributions  
 238 between age class according to the following (see **Supplementary Information Appendix B**  
 239 **and D**):

240

$$241 \quad dw_{yx} = \begin{cases} 0, & \text{if } y = x + 1 \\ db(x)[(1-d)g(x)[(1-h(x)) - i(x) - k(x)\hat{r}(x)] + (1-c)d\bar{g}], & \text{if } y = 1 \\ 0, & \text{otherwise} \end{cases}$$

242 **[8]**

243

244 Then, let  $S_m(x)$  be the component of the force of selection due the effect of a mutant allele on  
 245 reproduction at age  $x$ . Using **[5]** and **[8]**, in a stationary population with limited dispersal and  
 246 social interactions between individuals, this can be written as:

247

$$248 \quad S_m(x) = \frac{d\dot{b}(x) f_x v_1}{d\delta \mathbf{f} \cdot \mathbf{v}} [(1-d)g(x)[(1-h(x)) - i(x) - k(x)\hat{r}(x)] + (1-c)d\bar{g}].$$

249 **[9]**

250

251 where

252

$$253 \quad i(x) = \frac{\sum_z T_{1,z}^x (1-d)}{b(x)(1-d) + (N-1)\bar{b}(1-d) + N\bar{b}(1-c)d}$$

254 **[10]**

255 is the fraction of all offspring at the local patch after dispersal that exist due to indirect effects  
 256 of the genotype of a focal individual aged  $x$ . Equation **[9]** shows that the overall force of  
 257 selection acting on a mutant allele that affects the rate of reproduction at age  $x$  is also comprised  
 258 of two components: (i) the effect of the allele on the probability of establishment of different

259 types of offspring onto the local patch and (ii) the effect of the allele on the dispersing offspring  
260 that are part of the direct fitness of the focal individual aged  $x$ . Selection for effect (ii) will  
261 always be positive; however, selection for effect (i) will depend on the relative weights each  
262 class of offspring contributes to the overall effect. In this model, an increase in direct  
263 reproduction is, all else being equal, beneficial for the direct fitness of a focal individual, but  
264 detrimental to the indirect fitness of the focal individual.

265

### 266 **Applications of the model**

267 Equations [7] and [9] provide general solutions for age-specific inclusive fitness forces of  
268 selection on individual survival and reproduction in group structured populations. To visualise  
269 the results, we consider two hypothetical populations of iteroparous individuals with social  
270 interactions (Fig 1, Fig 2). For each, we consider background demography described by age-  
271 specific vital rates,  $p(x)$  and  $b(x)$ . We parameterise mortality risk at age  $x$  using the Siler  
272 model (43):

$$273 \quad \mu(x) = \alpha_1 e^{-\beta_1 x} + \alpha_2 e^{\beta_2 x} \quad [11]$$

274  
275 The probability of survival at age  $x$ ,  $p(x)$ , is therefore equal to  $e^{-\mu(x)}$ . The probability of  
276 survival to age  $x$  ( $l(x)$ ) is then  $l(x) = \prod_1^{x-1} p(x)$ , with  $l(1) = 1$ . As we assume all patches  
277 have no breeding positions available at the start of each breeding seasons (*i.e.*, a density-  
278 dependent stationary population), we can calculate the asymptotic frequency ( $f_x$ ) of each age  
279 class as

$$280 \quad f_x = \frac{l(x)}{\sum_y l(y)}. \quad [12]$$

281  
282 We then parameterise individual rate of reproduction at age  $x$  as:

283

$$284 \quad b(x) = \begin{cases} 0, & \text{if } x < \varepsilon \\ (x - \varepsilon)e^{-\varphi(x-\varepsilon)}, & \text{if } x \geq \varepsilon \\ 0, & \text{if } x > \kappa \end{cases}$$

285 [13]

286 where  $\varepsilon$  designates the age of reproductive maturity,  $\kappa$  represents an age at which reproduction  
287 ceases, and  $\varphi$  modulates the shape of reproduction across age classes.

288

289 Fig. 1A and Fig. 2A illustrate the life cycles of the two hypothetical social populations. Fig. 1A  
290 considers a population with post-reproductive individuals providing care for juveniles, as seen  
291 in humans (44), orcas (45), and Asian elephants (46). Fig. 2A considers a population with  
292 juvenile individuals providing help to the reproduction adult breeders, as is found in many  
293 cooperatively-breeding species (47). Fig. 1B and Fig. 2B display the modelled survivorship  
294 and reproduction as a function of individual age. We then apply our methodology (see **Model**  
295 **and Appendix C**) to partition these vital rates into inclusive fitness contributions between age  
296 classes and compute a fitness matrix (**W**) with elements described in [1]. Fig. 1C and Fig. 2C  
297 show the forces of selection acting on survival and reproduction at age  $x$  in these hypothetical  
298 social populations according to equations [7] and [9].

299

300 We show that the force of selection acting on survival in social populations is not necessarily  
301 constant before maturity, as predicted by classical theory (2). The exact pattern depends on  
302 whether pre-reproductive individuals gain indirect fitness through transfers or not. When  
303 juveniles do not engage in helping behaviour, the force of selection increases in the juvenile  
304 period as relatedness to newborn offspring decreases with increasing juvenile age (Fig. 1C;  
305 Fig.1D). This decline in local relatedness facilitates a more ‘selfish’ force of selection on  
306 survival throughout the juvenile period. On the other hand, when juveniles provide help to

307 adult reproduction, the force of selection on survival generally decreased from the age at which  
308 indirect fitness was first accrued (Fig. 2C; Fig. S3), rather than the age of first reproduction. In  
309 both examples, the force of selection on survival then declines throughout adulthood as future  
310 inclusive reproductive value declines and the relatedness to newborn offspring increases. When  
311 post-reproductive adults continue to accrue indirect fitness, the force of selection on survival  
312 can remain above zero in post-reproductive age classes (Fig. 1C; Fig. S1). The magnitude of  
313 the force of selection is greater in post-reproductive age classes when juvenile dispersal is  
314 lower (and so there is higher local relatedness) and the magnitude of help provided by post-  
315 reproductive individuals is higher (Fig. S1). In general, the force of selection on survival will  
316 always have a positive component until the final age at which inclusive fitness is accrued,  
317 rather than necessarily the age of last reproduction. At this age, when future survival is no  
318 longer possible, the first term on the numerator of Equation [7] is zero, and so, if there is some  
319 level of local relatedness (*i.e.*  $\hat{r}(x) > 0$ ), selection will favour increased mortality as it will  
320 benefit the establishment of related juveniles.

321  
322 In populations with relatively long lifespans, the force of selection on reproduction was weaker  
323 than the force of selection on survival. The force of selection acting on reproduction at age  $x$   
324 generally declined from birth, as predicted by Hamilton's model (2), but not always (Fig. S4),  
325 and the decline was more rapid when the rate of dispersal was lower (Fig. S2). This more rapid  
326 decline is likely due to the greater inclusive fitness costs of increasing personal reproduction  
327 when local relatedness is higher. The force of selection on reproduction in early life is also  
328 weaker when post-reproductive adults have a more significant impact on juvenile survival. In  
329 all iterations of the model (Fig. 2C; Fig. S3), there was a slight increase in the force of selection  
330 acting on reproduction in the final age class, when the force of selection on rate of survival  
331 becomes negative.

## 332 **Discussion**

333 When considering the evolution of demographic senescence, evolutionary biologists use  
334 population growth rate,  $r$ , as the measure of fitness (48, but see 49). The magnitude of the  
335 change in population growth rate due to an age-specific change in survival and/or reproduction  
336 generally declines with age (but see (50) for other indicators of the force of selection), and this  
337 decline facilitates the evolution of senescence (2). However, for social species, it is crucial to  
338 consider explicitly the inclusive fitness of individuals as the quantity that natural selection  
339 seeks to maximise (10). Indeed, the change in inclusive fitness due to an age-specific change  
340 in individual survival and/or reproduction must consider the combined effect on all individuals  
341 that are affected by the change (24). Here, we show that, in an age-structured model for patch-  
342 structured social populations, considering the inclusive fitness effects of an allele significantly  
343 alters the form of the forces of selection acting on age-specific survival rate and rate of  
344 reproduction.

345

346 Our framework provides several key insights into the force of selection acting on survival and  
347 reproduction in social species. First, the force of selection acting on the survival rate of that  
348 age class is the product of future inclusive reproductive value (IRV), rather than conventional  
349 reproduction value (RV (48)), and the asymptotic frequency (stationary age distribution) of  
350 that age class. Since IRV remains above zero after reproduction ceases, if post-reproductive  
351 adults continue to accrue indirect fitness benefits, selection on survival of post-reproductive  
352 age-classes does not necessarily go to zero as in Hamilton's model (2). Importantly, this finding  
353 provides a formal inclusive fitness framework for the 'grandmother hypothesis' (51 – 52),  
354 supporting work that has suggested indirect fitness benefits are essential to sustained post-  
355 reproductive lifespan (23, 31). In our framework, the force of selection on survival of social  
356 species will remain non-zero until there is no future IRV. At this point, if there is some local

357 relatedness, the force of selection on increased survival will be negative. Combined with an  
358 increase in the force of selection on reproduction at a ‘final age class’, a kin-selected terminal  
359 investment strategy, in which it pays to invest heavily in reproduction at the expense of survival  
360 to maximise the establishment of kin, may be favoured (19).

361  
362 The incorporation of age-specific indirect fitness into the evolutionary theory of senescence  
363 means that selection on survival before maturity is not necessarily constant (Fig. 1C; Fig. 2C).  
364 This difference occurs because of the balance between the future IRV of the individual and the  
365 IRV of newborns displaced by increased survival. If relatedness to other individuals declines  
366 throughout the juvenile period as a focal individual ages, and the focal individuals own IRV  
367 increases as they approach maturity, the balance in Equation [7] is weighed more heavily  
368 towards the first term, and the force of selection on increased survival will increase. On the  
369 other hand, in populations where juveniles help and accrue indirect fitness, the force of  
370 selection on survival will decline from the age at which indirect fitness is first gained. This  
371 result implies that, in species with pre-reproductive help, senescence should start from the age  
372 at which inclusive fitness is first gained, rather than the age of first reproduction, as in  
373 conventional models (2, 20).

374  
375 An inclusive fitness force of selection acting on reproduction depends on the costs and benefits  
376 associated with increasing personal reproduction. In our framework, selection for increased  
377 reproduction will always have a positive component due to the increased probability of an  
378 offspring (whether philopatric or dispersive) establishing on to a patch. However, the  
379 subsequent decrease in probability of other locally produced offspring establishing on to the  
380 patch reduces the magnitude of the force of selection acting on reproduction. This result may  
381 be especially important for groups experiencing strong competition over resources (12). For



382 example, a negligible force of selection on reproduction may favour reproductive restraint by  
383 some individuals within cooperatively-breeding groups, when access to reproduction is limited  
384 and inclusive fitness costs of increasing personal reproduction would be substantial (32).

385

386 Our framework builds on previous work that has made significant ground in incorporating  
387 social effects into the evolutionary theory of senescence. Lee's (23) model showed that the  
388 force of selection acting on age-specific mortality can be modified by intergenerational  
389 transfers of resources. However, kin selection did not enter the formal model as no explicit  
390 spatial structure was considered. Here, by explicitly considering a patch structured population  
391 with dispersal, we allow for variation in relatedness and thus a larger breadth of possible kin  
392 selection effects to be considered. Ronce & Promislow (20) derived analytical solutions that  
393 provided the baseline framework for the model here, showing that the force of selection on  
394 increased survival includes a negative component driven by the displacement of offspring from  
395 establishing on the local patch. This term is similar to the negative term in [7]; however, our  
396 framework also explicitly considers the impact of survival on the establishment of other locally  
397 produced offspring. By only considering single individuals on a patch, social interactions in  
398 Ronce & Promislow's model were limited to kin competition between parent and offspring  
399 over residency on the patch. Here, by including multiple individuals on the patch, we can also  
400 incorporate social effects into the form of the force of selection on reproduction ([10]). Finally,  
401 Moorad & Nussey (53) took a quantitative genetics approach to add indirect genetic effects,  
402 explicitly considering maternal effect senescence, but modelled no explicit demography. A  
403 combination of explicit demography, as modelled here, and quantitative genetics could prove  
404 a major future step.

405

406 The framework we present here provides a base to expand our understanding of senescence  
407 across social species. For example, previous work has found mixed evidence for extended  
408 lifespan in cooperative breeders (54 – 57), and some evidence for differences in rates of  
409 senescence between cooperative and non-cooperative breeders (58). Previous theory suggests  
410 that it is longer life and overlapping generations that initially favour cooperation (26), but also  
411 that a delayed age of first reproduction as a result of queuing for reproduction might be a self-  
412 reinforcing mechanism for extended lifespan in cooperative breeders (59). However, multiple  
413 other facets of the demography of cooperative breeding systems, including the process of group  
414 formation (60), the structure of dominance hierarchies (61) and levels of reproductive skew  
415 (62) all have the potential to play a role in determining lifespan and rates of senescence. All  
416 have the potential to contribute to the shape of the age class asymptotic frequency and inclusive  
417 reproductive value distributions that, as we have shown here, underpin inclusive fitness forces  
418 of selection. Our model provides a framework to stimulate further theoretical work for how  
419 these features of cooperative breeding systems may impact the evolution of lifespan and  
420 senescence.

421  
422 Here, we focused on how cooperative interactions between members of a group can alter age-  
423 specific inclusive fitness forces of selection. However, in many groups, competitive  
424 interactions over limited resources are also rife. In our model, transfers between age classes  
425 reflect the net effect of the presence of an individual in one age class on the survival and  
426 reproduction of an individual in another age class. If the net effect is negative, then the genetic  
427 offspring transfer is also negative. For example, consider again the social system illustrated in  
428 Fig. 1. Instead of post-reproductive individuals having a positive effect of the survival of  
429 juveniles, let us instead imagine a scenario in which the presence of post-reproductive  
430 individuals is harmful to the survival of juveniles. An allele that increases the rate of survival

431 in such post-reproductive individuals will be selected against due to the inclusive fitness costs  
432 imposed from the negative effects on related juvenile individuals, potentially hastening the  
433 evolution of more rapid senescence. Finally, in our model, we only considered indirect fitness  
434 returns from social interactions. In many cooperative breeding systems, however, direct fitness  
435 returns from social interactions can be the main driver for alloparental care (47). Some form of  
436 direct fitness benefits could be incorporated into the model by delaying the age at which returns  
437 from social interactions are realised, as hypothesised by group augmentation theory (63).

438

439 In summary, recent research has focused on the potential for social interactions to drive  
440 variation in senescence across species (1, 64). The model we present here shows that when  
441 inclusive fitness consequences of increasing individual survival or reproduction are considered,  
442 age-specific forces of selection can vary markedly from previous asocial models. Our results  
443 thus support the hypothesis that sociality can shape patterns of senescence in nature. Further  
444 theoretical, empirical and comparative studies are now needed to determine the amount of  
445 variation in senescence patterns that can be explained by social modes of life.

446

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452

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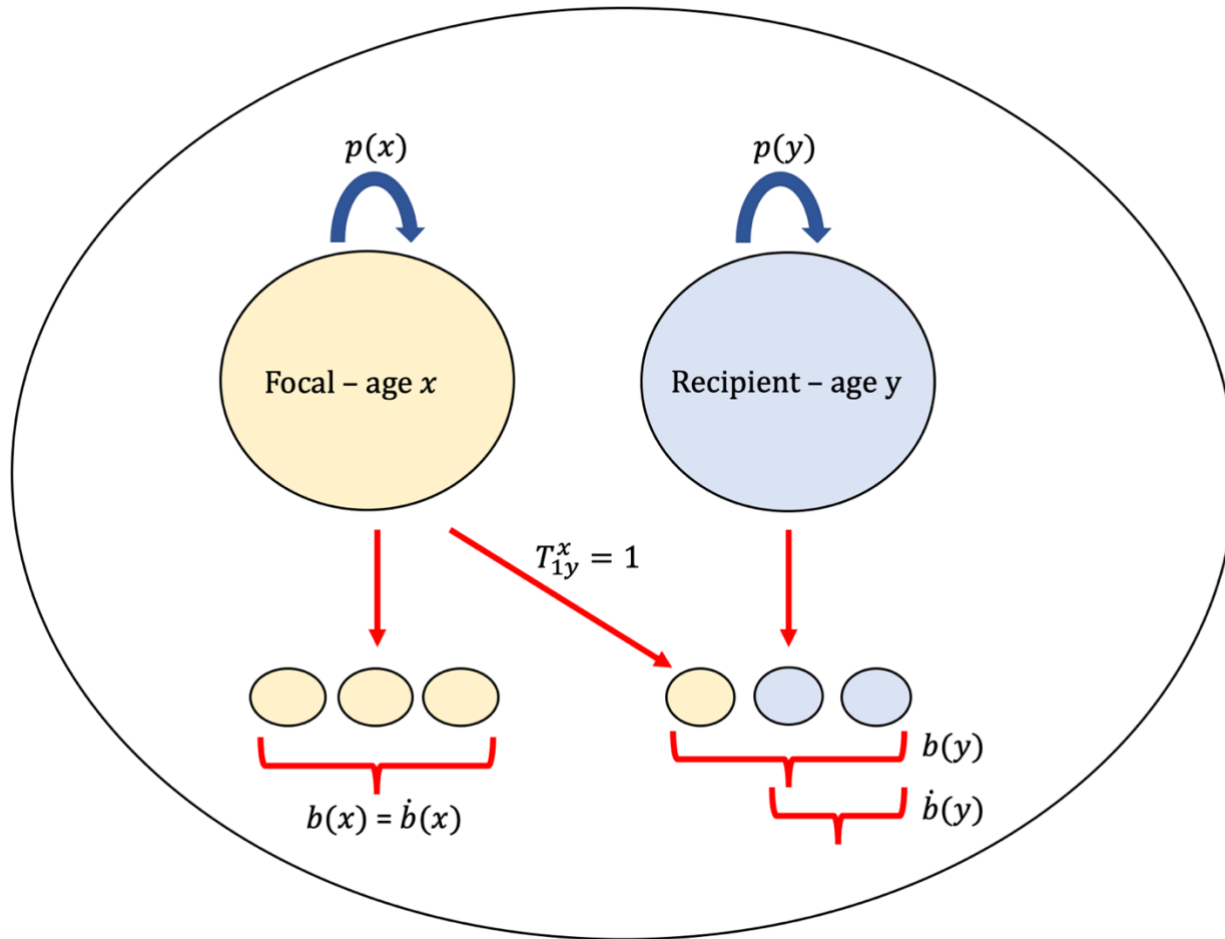
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583 **Figures**

584 **Figure 1**

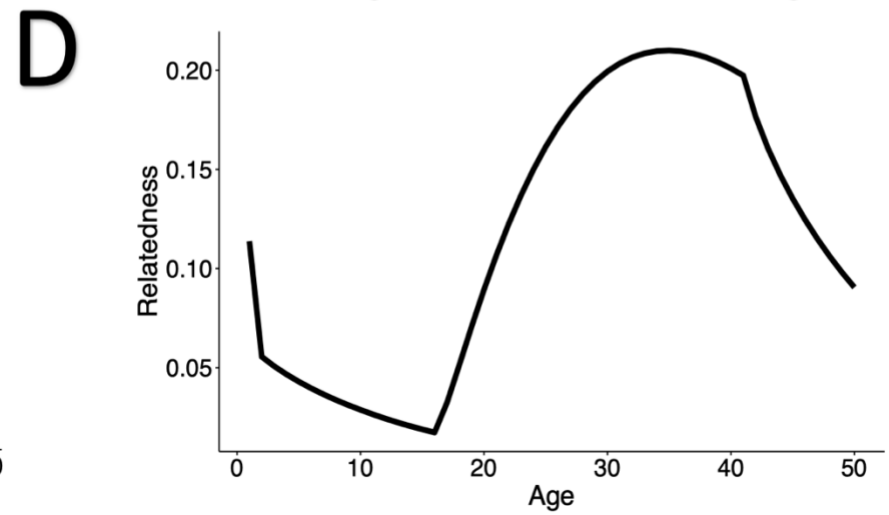
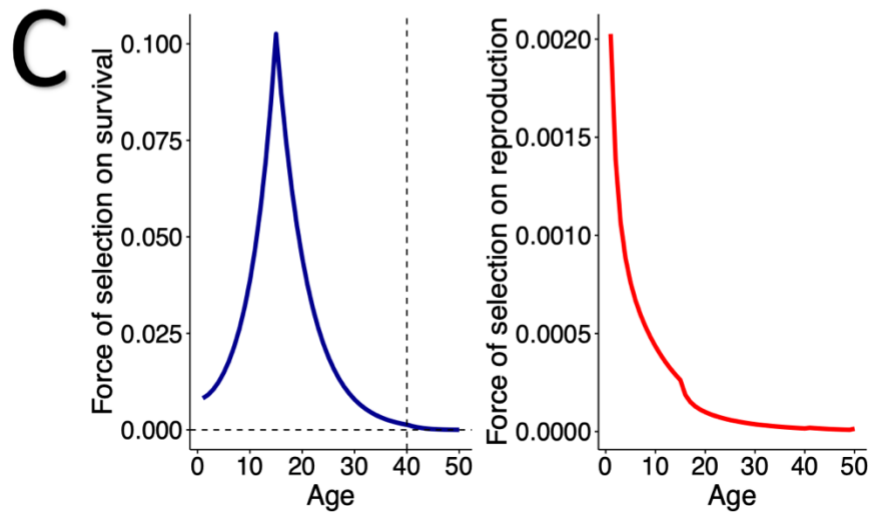
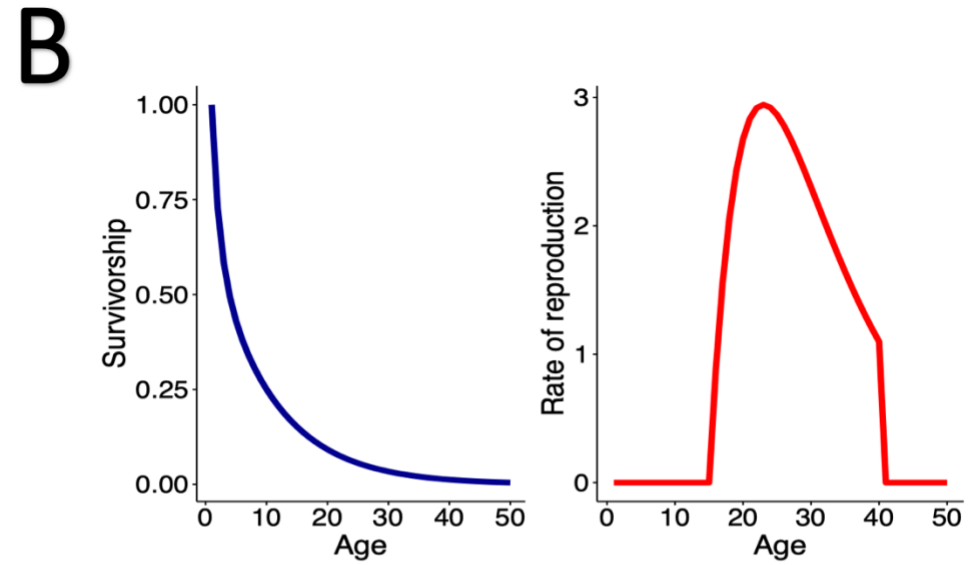
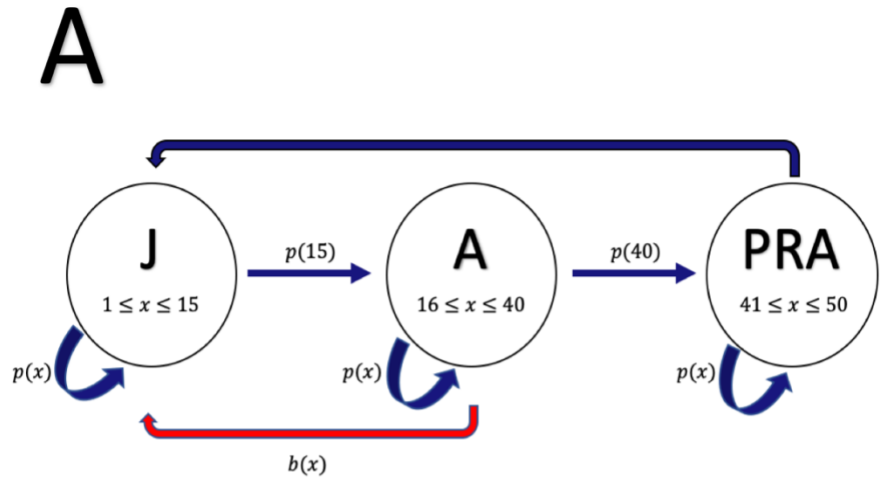
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586

599 **Figure 1. An example of a genetic offspring transfer between two individuals using inclusive fitness.** To illustrate transfers, we consider a  
600 patch with two individuals, one of age  $x$  and the other of age  $y$ . The individual aged  $x$  has  $b(x)$  offspring, survives with probability  $p(x)$ , and  
601 receives no social transfers from other individuals in the population when aged  $x$ . We imagine a social behaviour exists whereby the individual  
602 aged  $x$  contributes to the reproduction of individuals aged  $y$ . In this scenario, the individual aged  $y$  has  $b(y)$  offspring in the current breeding  
603 season, but one of these offspring is due to the transfer from the focal individual aged  $x$ . Following inclusive fitness logic, the offspring produced  
604 due to the social behaviour of the individual aged  $x$  is stripped from the inclusive fitness of the individual aged  $y$ , leaving  $\dot{b}(y)$  as their inclusive  
605 fitness contribution to age class 1. The inclusive fitness contribution of the focal individual aged  $x$  to age class 1 is  $\dot{b}(x) + T_{1y}^x \hat{r}(x)$ , where  $\hat{r}(x)$   
606 represents the relatedness of an individual aged  $x$  to the offspring it helped to produce.

607



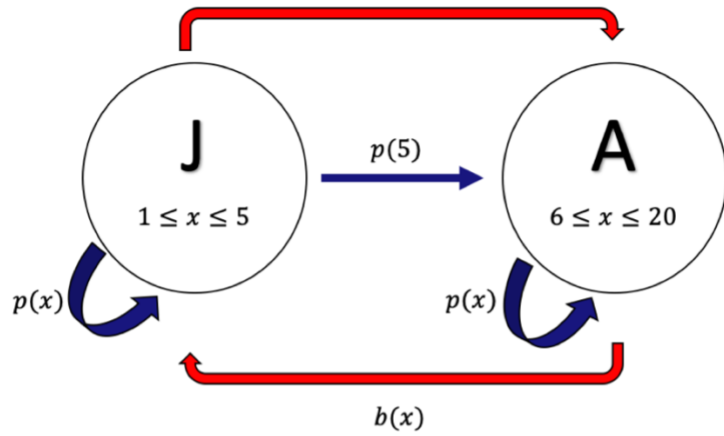
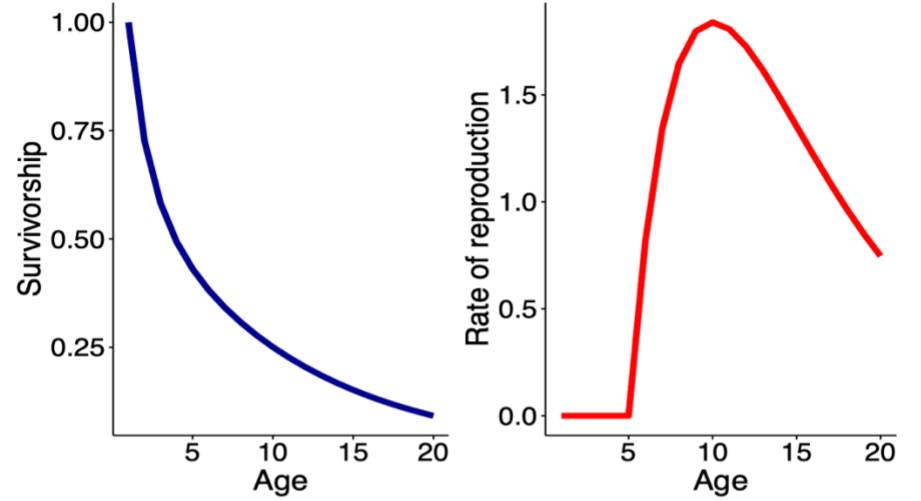
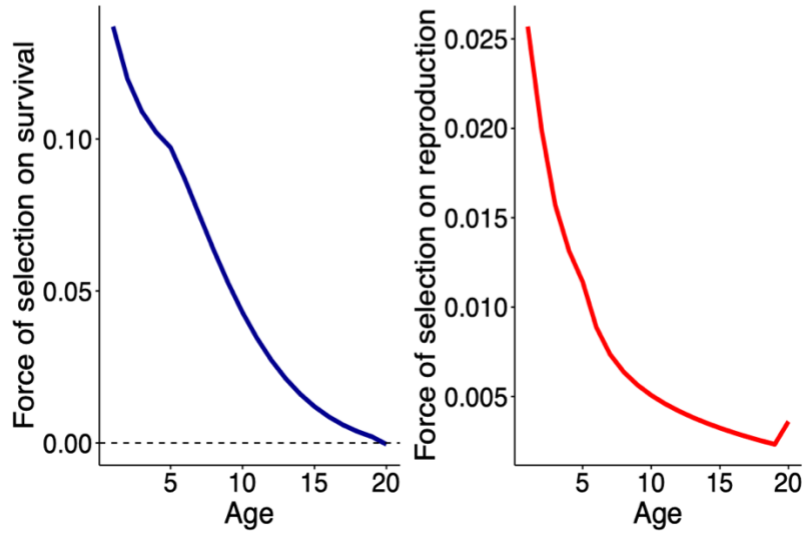
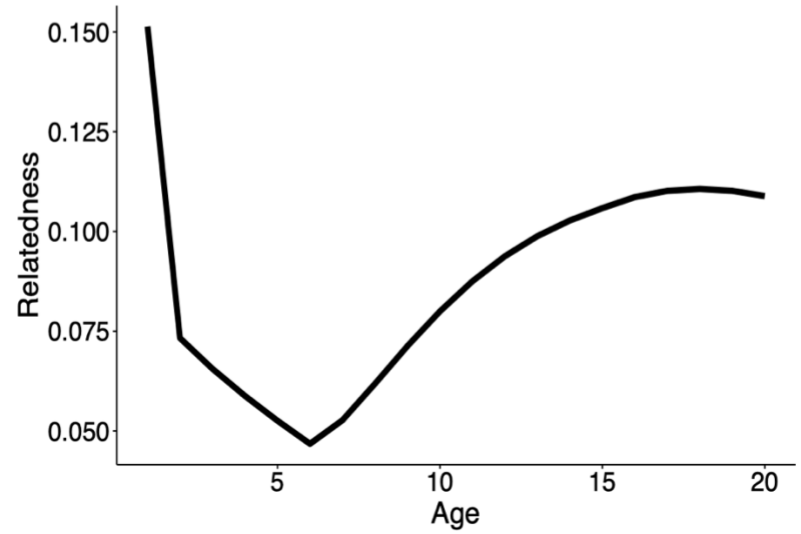
609 **Figure 2. Age specific forces of selection in a social population with post-reproductive help.** A) A hypothetical population of iteroparous  
610 individuals classified into three life cycle stages: juvenile (J), reproductive adult (A), and post-reproductive adult (PRA). The red arrow from A to  
611 J represents the reproduction of adult individuals, whereas the dark blue arrow from PRA to J represents the social contributions from post-  
612 reproductive adults to the survival of juveniles. B) The background vital rates of survivorship and reproduction of the model social population.  
613 Survival probability at age  $x$  is produced from a Siler model ([11]) with parameters:  $\alpha_1 = 0.4$ ,  $\beta_1 = 0.6$ ,  $\alpha_2 = 0.1$ ,  $\beta_2 = 0$  (See SOM for further  
614 details). Reproduction at age  $x$  is modelled according to [13] with parameters:  $\varepsilon = 15$ ,  $\varphi = 0.125$ , and  $\kappa = 40$  (SOM). C) The forces of selection  
615 acting on survival at age  $x$  increases during the juvenile period and then decreases but remains above zero in the post-reproductive period. The  
616 force of selection acting on reproduction at age  $x$  is weaker than the force of selection acting on survival and declines from birth. Other demographic  
617 parameters to produce these forces of selection were set to  $c = 0$ ,  $d = 0.5$ ,  $N = 4$  and  $\omega = 50$ . (see **Model** and SOM D) The relatedness of an  
618 individual aged  $x$  to another random individual on the patch declines throughout the juvenile (pre-reproductive) window, and then increases during  
619 adult reproduction before declining again as reproduction ceases.

621

620

622

623

**A****B****C****D**

625 **Figure 3. Age specific forces of selection in a social population with pre-reproductive help.** A) A hypothetical population of iteroparous  
626 individuals with two lifecycle stages: juvenile (J) and reproductive adult (A). The red arrow from J to A represents the social contributions from  
627 juveniles to the reproduction of adults. Note that here, help is in the currency of reproduction, rather than survival (See Fig. 2A). B) The background  
628 vital rates of survivorship and reproduction. Survival at age  $x$  is produced from a Siler model ([11]) with parameters:  $\alpha_1 = 0.4$ ,  $\beta_1 = 0.6$ ,  $\alpha_2 =$   
629  $0.1$ ,  $\beta_2 = 0$ . Rate of reproduction at age  $x$  is modelled according to [13] with parameters:  $\varepsilon = 5$ ,  $\varphi = 0.2$ , and  $\kappa = 21$ . C) The force of selection  
630 acting on survival at age  $x$  declines from birth. The force of selection acting on reproduction at age  $x$  is weaker than the force of selection on  
631 survival and also declines from birth but then increases in the final age class. Other demographic parameters to produce these forces of selection  
632 were set to  $c = 0$ ,  $d = 0.5$  and  $N = 4$  and  $\omega = 20$ . D) The relatedness of an individual aged  $x$  to another random individual on the patch declines  
633 throughout the juvenile period, and then increases during adult reproduction.