

1 **A Dynamic Framework for the Study of Optimal Birth Intervals Reveals the Importance of**
2 **Sibling Competition and Mortality Risks**

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4 **Short title:** A Dynamic Framework for Optimal Birth Spacing

5 M. G. Thomas^{1¶*}, D. P. Shanley^{2¶*}, A. I. Houston³, J. M. McNamara⁴, R. Mace^{1&}, T. B. L. Kirkwood^{2&}

6 ¹ – Human Evolutionary Ecology Group, Department of Anthropology, University College London,
7 WC1H 0BW, UK

8 ² – Institute for Ageing and Health, Newcastle University, Newcastle upon Tyne NE4 5PL, UK

9 ³ – School of Biological Sciences, Woodland Road, Bristol, BS8 1UG, UK

10 ⁴ – School of Mathematics, University Walk, Bristol, BS8 1TW, UK

11 ¶ These authors contributed equally to this work.

12 & These authors also contributed equally to this work.

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14 **Corresponding author:**

15 Matthew G. Thomas

16 Department of Anthropology, University College London, 14 Taviton Street, London, WC1H 0BW, UK

17 Tel: +44 (0)20 7679 8633

18 Fax: +44 (0)20 7679 8632

19 Email: m.thomas.10@ucl.ac.uk

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22 **Abstract**

23 Human reproductive patterns have been well studied but the mechanisms by which physiology,
24 ecology and existing kin interact to affect the life history need quantification. Here, we create a
25 model to investigate how age-specific interbirth intervals adapt to environmental and intrinsic
26 mortality, and how birth patterns can be shaped by competition and help between siblings. The
27 model provides a flexible framework for studying the processes underlying human reproductive
28 scheduling. We developed a state-based optimality model to determine age-dependent and family-
29 dependent sets of reproductive strategies, including the state of the mother and her offspring. We
30 parameterised the model with realistic mortality curves derived from five human populations.
31 Overall, optimal birth intervals increase until the age of 30 after which they remain relatively
32 constant until the end of the reproductive lifespan. Offspring helping each other does not have much
33 effect on birth intervals. Increasing infant and senescent mortality in different populations decreases
34 interbirth intervals. We show that sibling competition and infant mortality interact to lengthen
35 interbirth intervals. In lower-mortality populations, intense sibling competition pushes births further
36 apart. Varying the adult risk of mortality alone has no effect on birth intervals between populations;
37 competition between offspring drives the differences in birth intervals only when infant mortality is
38 low. These results are relevant to understanding the demographic transition, because our model
39 predicts that sibling competition becomes an important determinant of optimal interbirth intervals
40 only when mortality is low, as in post-transition societies. We do not predict that these effects alone
41 can select for menopause.

42 **Keywords:** interbirth intervals; humans; state-dependent optimality modelling; life history evolution;
43 sibling competition.

44

45 **Introduction**

46 On attaining sexual maturity, humans have substantial reproductive potential and populations are
47 capable of rapid expansion. This feature of the human life history may have contributed to the
48 successful migration and colonisation that has been a characteristic of our species. Present day
49 populations exhibiting natural fertility have a typical interbirth interval (IBI) in the range of 3-5 years
50 (Sear & Mace, 2008). Shorter first birth intervals are associated with increased lifetime reproductive
51 success (Nenko et al., 2013). Moreover, IBIs increase with age until reproduction is physiologically no
52 longer possible after the age of menopause, although the age at last reproduction tends to occur
53 well before this (Sievert, 2006). Explanations of the (proximate) mechanisms underlying these
54 patterns have so far met with mixed success.

55 Here, we construct a flexible framework in which factors relating to individual human reproductive
56 success are analysed from an evolutionary perspective. Our model explores how reproductive
57 schedules adapt to mortality risks (both intrinsic and environmental) and kin effects, potentially
58 explaining the variation in human life history across the world.

59 At birth, human children are particularly altricial compared to other great apes and require intensive
60 and protracted maternal investment. While mothers are breastfeeding, fertility is usually suppressed
61 (but see Short et al. 1991). This can act as a natural contraceptive, protecting both the mother and
62 existing children from too close birth spacing (Ellison et al., 1993). Nevertheless, human infants are
63 weaned early compared to other great apes. This increases the fertility of the mother and may
64 require alloparents (usually kin) to help in providing for the child (Bogin, 1997; Hawkes et al., 1997).
65 Although young children are capable of foraging to some degree, they remain nutritionally
66 dependent on others for many years (Kaplan, 1996). The age of puberty depends on rates of growth
67 and development, which in turn depend on the levels of nutrition received during infancy and
68 childhood.

69 Human females suffer an unusually high hazard during childbirth, which increases with age (Grimes,
70 1994; Abitbol, 1996). At older maternal ages, there is a general age-related increase in IBIs and
71 offspring are weaned later, as in many other primates (Caro et al., 1995). Younger offspring are
72 particularly vulnerable if their mother dies (Willführ & Gagnon, 2013).

73 There is inevitable competition between siblings for maternal attention and resources. Newborns
74 are likely to divert maternal attention from existing children and the youngest child must usually be
75 weaned before the mother is again fertile. Young children with many young siblings may therefore
76 be exposed to higher mortality risks than if they are the sole recipient of the mother's provisioning.
77 In order to combat these risks, the World Health Organization recommends a minimum birth interval
78 of two years (WHO, 2006).

79 Older children can share some of the burden of care for the young with their mother, helping to
80 reduce the mortality risk of their younger siblings. This has been observed in some farming
81 populations such as the Mandinka in Gambia, the Maya in Mexico, and the Chewa in Malawi, as well
82 as in a 17th Century Québécoise population (Sear et al., 2002; Beise, 2005; Sear, 2008; Kramer, 2010).

83 The timing of births can have important consequences for reproductive success. The risks of adverse
84 outcomes due to short IBIs are well documented. However, there is evidence to suggest that
85 extended spacing between births (longer than 50 months) is also linked to events such as preterm
86 birth and low birth weight (Conde-Agudelo et al., 2006). This may be due to phenotypic correlations
87 whereby a female may already be experiencing low fertility or poor nutritional status. Thus,
88 understanding how mechanisms such as sibling competition can affect birth spacing might be
89 important for understanding patterns of infant mortality.

90 **Models of optimal reproductive scheduling**

91 In foraging populations, women have no option but to carry infants, which poses a considerable
92 energetic burden. The !Kung San -- a foraging people of the Kalahari desert -- are largely dependent

93 on mongongo nuts as a food source (Howell, 1979). They frequently move foraging site and must
94 carry both food and young offspring. Mathematical models quantifying the load of food that can be
95 efficiently carried, along with the demands of young offspring who must be provisioned and also
96 carried, predicted an optimal IBI of approximately 4 years, which is typical of this population (Blurton
97 Jones & Sibly, 1978; Blurton Jones, 1986; Anderies, 1996). However, other related groups of San
98 people who have a different local ecology, which does not expose them to the same reproductive
99 constraint imposed by the need to carry food, have similar IBIs of 4 years (Hill & Hurtado, 1996).

100 A more complex model considered the influence of a female's age and stochasticity in her foraging
101 success on the survival of her children and her optimal reproductive strategy (Anderies, 1996),
102 where older females were assumed to forage less efficiently. A female's probability of survival
103 depended on her age and, if she gave birth, on the risk of mortality in childbirth. Through maximising
104 lifetime reproductive success, an IBI of 4 years was a robust response to all realistic conditions, and
105 showed only a small increase in the optimal interval with age of the mother. The predicted optimal
106 IBIs matched observations of !Kung reproductive decisions (Anderies, 1996).

107 Mace (1998) used the same framework to show how reproductive decisions are sensitive to
108 inherited wealth, when parental resources are required for the next generation to marry and
109 reproduce; the more parental resources are needed, the smaller the optimal family size. When this
110 was the case, higher mortality risk in the environment caused increased fertility through
111 'replacement' births even though the overall family size of surviving offspring was not much altered.

112 Here, we apply the well-developed technique of state-dependent optimality modelling (Houston &
113 McNamara, 1999) to investigate reproductive decisions in human life history. We develop a general
114 but comprehensive dynamic model that offers the flexibility to examine optimal age-related
115 reproductive strategies across a variety of contexts relevant to human physiology, ecology and social
116 organisation. A dynamic modelling framework can add greater realism to models of reproductive

117 behaviour. This allows maternal decisions to be evaluated in terms of their long-term fitness
118 consequences; crucially, decisions depend on the mother's state.

119 The aim is to identify the key determinants of the age-related increase in IBI given exposure to
120 mortality hazards from the mother's socioecological environment. Rather than generating
121 quantitative predictions for observed birth intervals, this model is intended to understand the
122 factors driving human life history variation. This is not explicitly a model of menopause, as it does
123 not include a third generation with which to explore grandmother effects. However in one set of
124 experiments we extend the possible reproductive span to the end of life in order to investigate
125 whether maternal mortality hazards and offspring effects can select for reproductive cessation.

126 **Materials and Methods**

127 **The Model**

128 The purpose of the model is to determine the optimal IBIs over the course of an individual female
129 life cycle. A woman can produce a child once every two or more years. However, there are
130 considerable risks associated with reproduction both for the mother and her existing family. First,
131 the mother is exposed to the risks associated with childbirth, which increase with age (Grimes, 1994;
132 Blanc et al., 2013). Second, offspring spaced too closely encounter competition for maternal
133 provisions; for example, the youngest child must be weaned before the next is born.

134 For each existing child in the model, a newborn sibling diverts attention from the mother that would
135 otherwise be directed towards them. A newborn child can therefore bring an associated reduction in
136 survival for all siblings. Finally, even in the absence of a newborn child, existing siblings have a
137 detrimental effect on one another. The model examines the interaction of these parameters in
138 determining an optimal schedule of births for a female.

139 In order to determine the optimal birth decisions, the model can be characterised as a discrete-time
140 Markov Decision Process (MDP) and solved by stochastic dynamic programming. The MDP contains
141 the following elements:

142 The finite set of states is described by mother's age x and family structure \mathbf{C} , discussed below.

143 \mathcal{U} is the set of actions {reproduce, do not reproduce}.

144 $P_u(x)$ is the mother's probability of surviving, given her age, x , and her birth decision, u .

145 $Q_u(x, \mathbf{C}, \mathbf{C}')$ calculates the survival probabilities of each of the children in family structure \mathbf{C} , which
146 becomes family structure \mathbf{C}' the following year, given their mother's age, x , and her birth decision, u .

147 This accounts for all combinations of child survival, including where all the children die, as well as the
148 effects of sibling competition and juvenile help.

149 $R_u(x, \mathbf{C}, \mathbf{C}')$ is one half of the expected number of offspring that mature next year, given the
150 mother's age, x , her birth decision u and the effects of sibling competition or help on the maturing
151 child's survival as family structure \mathbf{C} transitions to \mathbf{C}' (Houston & McNamara, 1999). \mathbf{C}' is the family
152 structure corresponding to \mathbf{C} with children ageing one year and newborns being present (or not)
153 according to birth decision u and the mortality risks for the mother and her children. This element is
154 half the total expected offspring since the model tracks only females.

155 **State Variables**

156 Females in the model make an annual decision (u) whether to give birth or not, depending on their
157 age and the structure of their existing family. The state variables are the mother's age and the age
158 and number of children in her existing family. A female is assumed to mature at 15 years. The model
159 tracks her birth decisions from sexual maturity until the age of 50. Twinning is excluded from the
160 model so she can only give birth to a single child and the minimum birth spacing is set at two years,
161 to allow a reasonable period of lactational amenorrhea while remaining computationally tractable.
162 Given these constraints, a mother can have 987 possible family compositions. (A family of children
163 aged between 1 and 14 can be represented as a 14-bit binary string where the presence of a child is

164 marked with a 1. For example, a family with a two-year-old and a 14-year old would be
165 01000000000001. Neighbouring binary digits cannot both be 1; hence, there are 987 possible
166 combinations.) Since the sexually mature lifespan is 35 years, the model will optimise birth decisions
167 over $35 \times 987 = 34,545$ states. The state space is (x, \mathbf{C}) where:

- 168 1. x is the set of maternal ages between 15 years and 50 years.
- 169 2. \mathbf{C} is the family structure (i.e. mother's offspring): a set of child ages between 1 year and 14
170 years for up to 7 children, including no offspring. There will always be a minimum spacing of
171 2 years between children.

172 **State Transitions**

173 The model considers all possible combinations of family in each year that can result in the case of
174 none, any or all children surviving. One of the strengths of state-dependent optimality modelling as a
175 methodology is its ability to account for a range of future states. The probability of each permutation
176 is calculated from mortality data that, in turn, depend on the structure of the family, the mother's
177 age and whether or not she gives birth.

178 **Mortality**

179 The mother's mortality rate is comprised of age-specific senescent and maternal components, and
180 an age-independent extrinsic term (equation (1)); child mortality is a decreasing exponential
181 function of age (equation (2)) (Siler, 1979). In order to situate the model in a real-world context, we
182 parameterise the mortality model using cross-cultural data (see Supplementary Table S1 and Fig.
183 1a).

$$\mu_{\text{adult}}(x) = \mu_{\text{extrinsic}} + \mu_{\text{senescent}}(x) + \mu_{\text{maternal}}(x) \quad (1)$$

184 where:

$$185 \mu_{\text{extrinsic}} = a_2$$

$$186 \mu_{\text{senescent}}(x) = a_3 e^{xb_3}$$

187
$$\mu_{\text{maternal}}(x) = \begin{cases} \alpha_{\text{birth}}x^2 - x\beta_{\text{birth}} + \gamma_{\text{birth}} \\ \alpha_{\text{birth}}e^{(x-x_{\text{maturity}})\beta_{\text{birth}}} + (\mu_{\text{extrinsic}} - \alpha_{\text{birth}}) \end{cases}$$

188 Here, x is the mother's age; a_2 , a_3 and b_3 are population-specific mortality parameters; α_{birth} , β_{birth}
 189 and γ_{birth} are maternal mortality parameters. The two maternal mortality functions are discussed
 190 below.

191 The sources of mortality are considered to be independent and can therefore simply be added
 192 together to obtain total mortality. The annual probability of survival is $\exp\{-\mu_{\text{adult}}(x)\}$.

193 According to how these parameters have been estimated in the published literature, the hunter-
 194 gatherer populations (Ache and Tsimane) have the lowest infant mortality rates for newborns but
 195 eventually have the highest infant, extrinsic and senescent mortality rates. Hunter-gatherers and the
 196 Taiwanese pastoralists have the greatest increases in senescent mortality while modern Swedes
 197 have the lowest; the Gambia data provide an intermediate case. We also parameterised the model
 198 with artificially low and high mortality curves to ensure our results are not confounded by these
 199 counterintuitive published mortality parameters (results not shown).

200 Infant mortality is characterised by two age-related curves, describing mortality in the presence and
 201 absence of the mother. If the mother dies, her child is exposed to a ten-fold increase in mortality risk
 202 (Shanley et al., 2007). Children under the age of two will die if their mothers die.

203 Maternal mortality is either a J-shaped or exponential function (equation 1). Parameters for the J-
 204 shaped function were calculated from data presented in Blanc et al. (2013) fitted to a second-degree
 205 polynomial; the exponential function was fitted to data in Grimes (1994). See Supplementary Table
 206 S2 for parameter values and Fig. 1b for a visual representation of maternal mortality.

207 The model is run under different assumptions concerning the relative importance of the inter-
 208 relationships of children with each other and their mothers, firstly with each factor in isolation and
 209 then in combination. As the state variables are the mother's and her children's ages and, as the

210 decision of whether to give birth is annual, these ages are simply incremented by one year. A 14 year
 211 old child matures and becomes independent of the mother the following year. Mortality introduces
 212 a stochastic element into the model, as there is a finite probability that the mother and any one (or
 213 even all) the children may not survive to the following year. For example, for a 30 year old woman
 214 with a 3 year old child who gives birth, there are 8 different states that need to be considered in the
 215 following year (see Supplementary Table S3 for an example calculation).

216 **Sibling Competition**

217 Siblings compete for maternal resources and thus have detrimental effects on each other's survival
 218 (Hill & Hurtado, 1996; Rutstein, 2005; Bøhler & Bergström, 2008). To model this, we calculate a
 219 weighting factor for each child that increases or decreases her mortality risk, depending on the ages
 220 of her siblings. In the absence of quantitative models of human sibling competition in the literature,
 221 we assume a linear, additive effect for four levels of competition: none, low, medium and high (Fig.
 222 2a).

223 A high weighting results in a large effect on mortality; conversely a low weighting results in a
 224 negligible effect on mortality. For a child aged y with siblings in family structure \mathbf{C} , the total mortality
 225 rate for the child, $\mu_{\text{child}}(y)$, is given by her intrinsic mortality, $\mu_{\text{childintrinsic}}(y)$, modified by the sum
 226 of these weightings:

$$\mu_{\text{child}}(y) = \mu_{\text{childintrinsic}}(y) \times \left(1 + \sum_{\mathbf{C}} \text{weightings}\right) \quad (2)$$

where:

$$\mu_{\text{childintrinsic}}(y) = a_1 e^{-yb_1}$$

227

228 Here, a_1 , b_1 are population-specific mortality parameters. The sum of weights due to family
 229 structure, \mathbf{C} , exclude the weight of the focal child age y . The child's annual probability of survival is
 230 $\exp\{-\mu_{\text{child}}(y)\}$.

231 **Juvenile Help**

232 In some models we assume children over the age of 10 can have beneficial effects in the family by
233 decreasing their siblings' risk of dying. As for sibling competition, quantitative models of age-based
234 levels of help are absent from the literature. Thus, we model help as a linear, additive effect which
235 decreases the detrimental effect of the weighting described above for four different intensities of
236 help: none, low, medium and high (Fig. 2b).

237 Juvenile help, as modelled here, has a weaker effect than sibling competition. In order to investigate
238 the extent to which this assumption affects our results, we also conducted a sensitivity analysis
239 where we varied the weightings of help relative to competition.

240 **The Dynamic Programming Equation**

241 For each birth decision (action u) taken by an adult female of a certain age (x) with family structure
242 (set of children) \mathbf{C} , we calculate the number of offspring in the following year from:

- 243 1. The adult female's probability of surviving to the next year.
- 244 2. For each possible family structure next year, the probability the mother is in the new state
245 (age and family structure), given her survival and the survival of her offspring.
- 246 3. For each possible family structure next year, the probability that a new child is born and
247 survives.

248 The decision of whether or not to give birth is taken in view of the risk of childbirth and the burden
249 of having a dependent child the following year, if it survives. Children that are 15 years old are
250 considered independent of the mother and, assuming female demographic dominance, only adult
251 females are included in the calculations (Charlesworth, 1994).

252 Given the mother's age and present family structure, the optimal birth strategy is determined by the
253 fitness of the strategy, i.e. maximising the maximum eigenvalue of the projection matrix (Houston &
254 McNamara, 1999). We define $f_t(x, \mathbf{C})$ as the expected number of descendants left t years in the

255 future by a female in state (x, \mathbf{C}) . Initially $f_0(x, \mathbf{C}) = 1$ for all ages x and family structures \mathbf{C} except
 256 $f_0(x_{\text{dead}}, \emptyset) = 0$ (i.e. there are no fitness benefits to dying without children). From f_0 , we can
 257 calculate f_1, f_2 , etc. from the dynamic programming equation:

$$f_{t+1}(x, \mathbf{C}) := \max_u \sum_{\mathbf{C}'} \{ [P_u(x) Q_u(x, \mathbf{C}, \mathbf{C}') f_t(x+1, \mathbf{C}')] + [(1 - P_u(x)) Q_u(x_{\text{dead}}, \mathbf{C}, \mathbf{C}') f_t(x_{\text{dead}}, \mathbf{C}')] + [R_u(x, \mathbf{C}, \mathbf{C}') f_t(15, \emptyset)] \} \quad (3)$$

258 where:

- 259 i) The census time is prior to the reproductive decision, therefore 15 year olds have only just
 260 matured.
- 261 ii) The probability of a 14 year old surviving to become mature in the next year is not affected
 262 by her mother's survival. However, the maturing child's survival can depend on the
 263 presence of siblings, including babies born under birth decision u given the mother's age
 264 and current family structure, \mathbf{C} .
- 265 iii) Mature males are assumed to have the same reproductive value as females and an even
 266 sex ratio is assumed.
- 267 iv) The minimum IBI is two years but in the event of a newborn not surviving to the next time
 268 interval, the focal female can reproduce again.

269 The growth rate of a population following the optimal strategy is given by the ratio $\lambda_{t+1} =$
 270 $f_{t+1}(\mathbf{s}_0)/f_t(\mathbf{s}_0)$ for a reference state, \mathbf{s}_0 (McNamara, 1991). The iteration process was judged to
 271 have converged on an optimal strategy when $\lambda_{t+1} \cong \lambda_t$, to seven decimal places.

272 **The Simulated Population**

273 The optimal IBI is determined as a function of all possible states. Stochasticity is inherent in the
 274 model as there can be a number of states in the next time interval with a calculated probability
 275 depending on the probability of survival of children and mother. The population is simulated by
 276 modelling population growth forward in time using the state-dependent optimal strategy. The

277 annual population growth rate at the stable age distribution has the same value as the relative
278 fitness determined in the dynamic optimisation procedure outlined above.

279 In the results that follow, the population is described in terms of the average IBIs. There are a
280 number of different ways to define IBI, such as an average of all birth spacings at a given age, or the
281 interval between a newborn and the next child. For example, a 35 year old female with 3 children of
282 5, 9 and 12 years old who gives birth has an average birth spacing of 4 years or alternatively a birth
283 interval at 35 of 5 years. An additional problem in defining IBI is how to include children who have
284 died. For example in the previous example the 35 year old female may have given birth in the
285 previous year, in which case the IBI at 34 was 4 years, but the baby died. In the work that follows,
286 the IBI relates to the spacing between a newborn baby and the next youngest child, unless stated
287 otherwise.

288 **Probabilistic Age at First Birth**

289 In order to involve fewer degrees of freedom, the model fixes the age of first birth at 15 years and
290 does not impose menopause. Although this paper is concerned with reproductive schedules
291 throughout the lifespan rather than the initial decision to reproduce, we ran a set of experiments
292 where age at first birth was probabilistic. Females were still assumed to mature at age 15 but gave
293 birth for the first time with a probability calculated from the function $y = 0.25 + 0.15x$, where x
294 is the age between 15 and 20. Thus, newly mature females have a probability = 0.25 of giving birth at
295 age 15, linearly increasing such that first birth is guaranteed by age 20.

296 The code is freely available; see Supplementary Information for download instructions.

297 **Results**

298 IBIs increase from first reproduction until age 30 in the Ache, Sweden and Taiwan populations (Fig.
299 3; red, green and blue lines, respectively), after which they remain relatively constant until the end
300 of the reproductive span at age 50. Birth intervals in the Tsimane and Gambian populations (Fig. 3;

301 purple and yellow lines) decrease slightly from the age of 20 and again remain constant until aged
302 50. Fig. 3 shows these effects for the cases where there is no risk of dying in childbirth, averaged
303 across all sibling effects (competition and juvenile help). The average IBI hovers in the range 2.05-
304 2.72 years across populations.

305 **Sibling competition and juvenile help**

306 Length of the optimal IBI is sensitive to how severely children compete for maternal resources as
307 well as to mortality risks in the population (Fig. 4). In the Taiwanese population, for example,
308 increasing the intensity of sibling competition from 'none' to 'high' causes the median IBI to increase
309 by 1.24 years. When there are higher levels of environmental mortality, such as in the Gambian and
310 Tsimane populations, birth intervals are less affected by the level of sibling competition. In 'easier'
311 environments, such as Sweden, birth intervals increase with the intensity of sibling competition.

312 Juvenile help, on the other hand, has a small effect on birth spacing, which only becomes apparent
313 after the age of 30 (Fig. 5 and Supplementary Fig. S3). The highest level of help decreases the IBI only
314 by a maximum of 0.15 years (in the Taiwan population with 'medium' sibling competition).

315 Supplementary Table S5 shows the extent to which sibling competition and juvenile help can extend
316 or contract birth intervals. In order to understand the effect that our assumption of weaker levels of
317 help compared to competition, we varied the strength of juvenile help. Even when help has the
318 same, but opposite, weighting as sibling competition, IBIs are not strongly affected except when help
319 is 'high' intensity but competition is 'low' or absent (Supplementary Fig. S4).

320 In order to tease apart the independent effects of infant and senescent mortality, we ran the model
321 holding each of these two factors constant in turn. When children were not exposed to any mortality
322 hazards -- but the rate of senescent and extrinsic mortality could vary across all populations -- birth
323 intervals remained at the minimum of 2 years, regardless of the levels of sibling competition or
324 juvenile help (results not shown but follow the same pattern as the red lines in Fig. 4). This is
325 unsurprising, since sibling effects cannot occur when there is no infant mortality.

326 Increasing the intensity of sibling competition lengthens the birth intervals when infant mortality
327 occurred but the rate of adult mortality was held constant across populations (results not shown but
328 are the same as in Fig. 4). Thus, infant mortality, in the presence of sibling competition, appears to
329 drive increases in IBIs.

330 **Menopause can be favoured under extreme age-dependent maternal mortality**

331 To explore the circumstances that might select for menopause, we increased the potential
332 reproductive span to a maximum age of 90. An age-related risk of dying during childbirth has a
333 negligible effect on birth spacing when the mortality function is J-shaped; females continued to
334 reproduce until death (Supplementary Fig. S2, panel A). Reproductive cessation only becomes
335 adaptive under extreme levels of maternal mortality risk that increase exponentially with age
336 (Supplementary Fig. S2, panel B). It should be noted that effects in old age, such as menopause,
337 would be more realistic had the model included grandmaternal effects on child survival (which this
338 model does not attempt to do; see Discussion).

339 **Probabilistic age at first birth does not affect birth decisions later in life**

340 When age at first birth was probabilistic rather than fixed at 15, the female experienced an initial
341 spike in birth intervals where they increased to a maximum of 3.95 years (Supplementary Fig. S5)
342 before dropping, at age 22, to the minimum of 2 years. After this point, optimal birth intervals follow
343 the same pattern as shown in Fig. 4.

344 **Discussion**

345 The model uses a comprehensive description of the mother and her family structure to obtain the
346 optimal birth strategy that maximises the number of offspring who survive to sexual maturity, a key
347 component of fitness. Alongside this, the model takes into account the stochastic year to year
348 changes that can occur in the family across a set of realistic mortality hazards derived from five
349 human populations. Optimal reproductive decisions are based on the complex interaction of family

350 members and the environment. Although not explicitly included, the strength of these interactions is
351 likely to be determined by resource availability.

352 The dynamic, state-dependent framework presented here shows how mortality hazards and sibling
353 competition interact to produce a range of life history strategies. IBIs increase with age in three of
354 the five simulated populations until the age of 30, after which birth spacing remains constant (Fig. 3).
355 In low mortality environments (e.g. modern Sweden), increasing the intensity of sibling competition
356 results in longer IBIs compared to high mortality environments (e.g. Tsimane; Fig. 4). Even at young
357 ages, mothers reproduce below their maximum potential level of reproductive output in order to
358 enhance the survival prospects of existing children (Figs. 3 and 4). Siblings providing help to each
359 other did little to reduce optimal IBIs (Fig. 5, Supplementary Table S5 and Fig. S4). These effects
360 alone do not induce menopause; it is only in the presence of extreme and exponentially increasing
361 age-related risks of dying during childbirth that reproductive cessation becomes adaptive
362 (Supplementary Fig. S2).

363 Our results predict many aspects of observed life history patterns. Among Ache hunter-gatherers,
364 the initial birth interval for women giving birth at age 15 was ~2.5 years; the median IBI of Ache
365 women is 3 years and remains relatively constant throughout her life (Hill & Hurtado, 1996). Under
366 high levels of sibling competition, the Ache IBI in our model reached a maximum of 2.96 years (Fig. 4,
367 top-left panel).

368 In the absence of published empirical data, we modelled sibling competition and juvenile help as
369 linearly increasing or decreasing (respectively) the mortality risks of other children in the family. The
370 effect of a particular child depended on her age and affected all siblings equally. A more realistic
371 implementation of this might include the ages of siblings in the effects. Newborns could have a more
372 deleterious effect on young siblings rather than older ones who are capable of provisioning
373 themselves, although this will depend of ecology; among the Ache, for example, children older than
374 10 years who were raised with more competing juvenile siblings suffered higher mortality (Hill &

375 Hurtado, 1996). In order to understand kin effects independently of environment-specific mortality,
376 the patterns of kin effects were assumed to be the same across all five modelled populations. Future
377 work could also tailor the levels of help and competition to the mortality rates in different ecologies.
378 We expect that altering the dynamics of kin effects in these ways would lead to greater divergence
379 in reproductive schedules between populations but less variation within a population. Less intense
380 sibling competition brought about by 'easier' environments might lead to shorter birth intervals, all
381 else equal. Introducing other allocarers, such as grandparents (see below), into the model could also
382 alleviate the effects of sibling competition.

383 We assumed that sibling competition occurs over maternal resources that are directly invested in
384 one offspring at the expense of others, with effects that diminish with age, although sibling
385 competition for parental resources can continue into adulthood (Mace, 2013). Social institutions
386 such as arranged marriages can also affect sibling competition depending on birth order, the
387 presence of same-sex siblings and local demography. In South Asia, for example, the presence of
388 older sisters can increase a girl's education by allowing her to remain in school rather than marry
389 (Vogl, 2013).

390 Our modelling framework also assumes that all offspring have equal quality. However, the
391 reproductive value – and sex – of the youngest child can affect a mother's IBIs. For example,
392 firstborn boys of high reproductive value often receive additional care with an associated delay to
393 the next child (Mace & Sear, 1997). Other primates, and indeed other mammals, also have a delayed
394 interval following the birth of a male offspring (Bercovitch & Berard, 1993; Birgersson, 1998).
395 Children in Tanzania were more likely to be weaned later when they were later-born or heavier at
396 birth, while socioeconomic status also played a role: high-status females and low-status males
397 received less parental investment in the form of breastfeeding (Wander & Mattison, 2013).

398 Maternal quality could be modelled by introducing a probability of birth depending on her fertility.
399 Fertility is variable in terms of ecological conditions and physiological status of women, as shown by

400 the seasonality of birth, response to food supply, and the effect of lactational amenorrhoea (Ellison
401 et al., 1993; Kaplan, 1996). Juveniles will inevitably vary according to the quality of care they have
402 received with a corresponding effect on growth and age of reproductive maturity (McNamara &
403 Houston, 1996).

404 In addition to the sex, reproductive value, and birth order of offspring, birth spacing decisions
405 respond to other circumstances, such as the constraint that carrying food imposes on the number of
406 young offspring (Blurton Jones & Sibly, 1978; Blurton Jones, 1986; Anderies, 1996). In farming and
407 herding populations, the heritable resources needed in adult life to go on to reproduce also
408 constrain reproductive schedules (Mace, 1998). The current version of the model only tracks female
409 offspring. It would be interesting to include males in order to test hypotheses about birth order and
410 the sex ratios of offspring (following, e.g. Trivers & Willard, 1973 and Leimar, 1996).

411 The model tracks individual females and their children under the age of 15 years, from which IBIs are
412 calculated. Once a female offspring is beyond the age of 15, it is no longer possible to know if her
413 mother is alive or how old she is. In some model scenarios, this leads to a sudden increase in
414 reproduction in late life, because the risk of death no longer has any cost once children are 15
415 (Supplementary Fig. S1). In reality, grandmothers can continue to enhance the fitness of older
416 offspring (Sear & Mace, 2008), so such late life peaks in fertility are an artefact of the model
417 structure being limited to two generations. Clearly, to examine more closely the effect of
418 grandmaternal care, children must be followed beyond independence and the influence of the
419 maternal grandmother can then be modelled explicitly. In a similar fashion, we expect that inclusion
420 of a grandmaternal generation would allow menopause to evolve in the model without assuming
421 extreme maternal mortality rates.

422 In summary, we have developed a comprehensive, dynamic framework for the study of optimal IBIs
423 and explored how sibling behaviour affects maternal reproductive success in different ecologies. At
424 high levels of infant mortality, sibling effects become less important, presumably as the risk of

425 mortality exceeds the risks associated with competition should each child survive (Fig. 4). This
426 suggests a stronger role for sibling competition only when mortality is low, as has been noted in
427 some modern populations (Lawson & Mace, 2009).

428 As mentioned above, extensions of this framework could take into account a third, grandmothing
429 generation in order to test hypotheses about the evolution of menopause. As it stands, our model
430 treats the nuclear family as an 'island', unaffected by the lives and strategies of others. Future work
431 might investigate the effects of other family members (e.g. grandparents, spouses, in-laws,
432 stepparents) on optimal reproductive scheduling..

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- 530

531 **Figures**

532 **Figure 1:** (a) Age-specific mortality risk in each of the five modelled populations: Ache (red), Gambia
533 (yellow), Sweden (green), Taiwan (blue), Tsimane (purple) from birth until a maximum lifespan of 90
534 (although note that reproductive spans last until age 50). (b) Maternal mortality hazards during the
535 reproductive span (ages 15-50): Solid lines show fitted probability functions for a J-shaped mortality
536 function derived from Blanc et al. (2013) with three levels: low (blue); medium (green); high (red).
537 Dashed lines show an exponential maternal mortality function derived from Grimes (1994) with
538 three levels: low (brown), medium (green) and high (red). See Supplementary Tables S1 and S2 for
539 parameters and references.

540 **Figure 2:** Age-specific sibling effects. Higher weights have stronger effects on sibling survival. (a)
541 Sibling competition (b) Sibling help. Intensities of sibling effects: none (red), low (yellow), medium
542 (blue) and high (purple).

543 **Figure 3:** Age-specific interbirth intervals (IBIs) for each of the five modelled populations: Ache (red),
544 Gambia (yellow), Sweden (green), Taiwan (blue), Tsimane (purple). Infant, adult and extrinsic
545 mortality vary according to the population parameters (see Table S1). Here, maternal mortality was
546 set to 'none', meaning the focal female did not face any increase in mortality due to giving birth.
547 Data points are the mean IBI values across the range of sibling competition and juvenile help
548 parameters. Values presented here do not include IBIs after the death of children, so IBIs are
549 independent of any replacement effect.

550 **Figure 4:** The effects of sibling competition on interbirth interval for the five modelled populations.
551 Here, each population experiences mortality according to the parameters in Table S1. Each panel
552 shows IBI for the four intensities of sibling competition: none (red); low (yellow); medium (blue);
553 high (purple).

554 **Figure 5:** Juvenile help does not have a strong effect on interbirth intervals. The curves are optimal
555 birth intervals for Sweden, for each of the four intensities of sibling competition: none (red); low

556 (yellow); medium (blue); high (purple). Panels show, from left to right, increasing levels of sibling
557 help (see Fig. 2b). Supplementary Fig. S3 shows the effects of juvenile help across all modelled
558 populations and Supplementary Fig. S4 illustrates a sensitivity analysis on our juvenile help
559 assumptions.