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

Short title: A Dynamic Framework for Optimal Birth Spacing

M. G. Thomas[1¶*], D. P. Shanley[2¶*], A. I. Houston[3], J. M. McNamara[4], R. Mace[1&], T. B. L. Kirkwood[2&]

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

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

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

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

[¶] These authors contributed equally to this work.

[&] These authors also contributed equally to this work.

Corresponding author:

Matthew G. Thomas

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

Tel: +44 (0)20 7679 8633

Fax: +44 (0)20 7679 8632

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

Abstract

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

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

Introduction

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

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

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

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

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

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

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

Models of optimal reproductive scheduling

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

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

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

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

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

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

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

Materials and Methods

The Model

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

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

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

The finite set of states is described by mother's age *x* and family structure ***C***, discussed below.

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

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

$Q_u(x, \boldsymbol{C}, \boldsymbol{C}')$ calculates the survival probabilities of each of the children in family structure ***C***, which becomes family structure ***C'*** the following year, given their mother's age, *x*, and her birth decision, *u*. This accounts for all combinations of child survival, including where all the children die, as well as the effects of sibling competition and juvenile help.

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

State Variables

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

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

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

State Transitions

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

Mortality

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

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

where:

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

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

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

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

Sibling Competition

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

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

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

where:

$$\mu_{\text{childintrinsic}}(y) = a_1 e^{-y b_1}$$

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

Juvenile Help

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

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

The Dynamic Programming Equation

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

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

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

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

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

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

where:

i) The census time is prior to the reproductive decision, therefore 15 year olds have only just matured.

ii) The probability of a 14 year old surviving to become mature in the next year is not affected by her mother's survival. However, the maturing child's survival can depend on the presence of siblings, including babies born under birth decision *u* given the mother's age and current family structure, $\boldsymbol{C}$.

iii) Mature males are assumed to have the same reproductive value as females and an even sex ratio is assumed.

iv) The minimum IBI is two years but in the event of a newborn not surviving to the next time interval, the focal female can reproduce again.

The growth rate of a population following the optimal strategy is given by the ratio $\lambda_{t+1} = f_{t+1}(\boldsymbol{s}_0)/f_t(\boldsymbol{s}_0)$ for a reference state, $\boldsymbol{s_0}$ (McNamara, 1991). The iteration process was judged to have converged on an optimal strategy when $\lambda_{t+1} \cong \lambda_t$, to seven decimal places.

The Simulated Population

The optimal IBI is determined as a function of all possible states. Stochasticity is inherent in the model as there can be a number of states in the next time interval with a calculated probability depending on the probability of survival of children and mother. The population is simulated by 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;

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

Sibling competition and juvenile help

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

Juvenile help, on the other hand, has a small effect on birth spacing, which only becomes apparent after the age of 30 (Fig. 5 and Supplementary Fig. S3). The highest level of help decreases the IBI only by a maximum of 0.15 years (in the Taiwan population with ‘medium’ sibling competition). Supplementary Table S5 shows the extent to which sibling competition and juvenile help can extend or contract birth intervals. In order to understand the effect that our assumption of weaker levels of help compared to competition, we varied the strength of juvenile help. Even when help has the same, but opposite, weighting as sibling competition, IBIs are not strongly affected except when help is ‘high’ intensity but competition is ‘low’ or absent (Supplementary Fig. S4).

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

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

Menopause can be favoured under extreme age-dependent maternal mortality

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

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

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

Discussion

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

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

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

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

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

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

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

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530

Figures

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

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

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

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

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

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