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SELECTION ON MENOPAUSE IN TWO PREMODERN HUMAN POPULATIONS: NO EVIDENCE FOR THE MOTHER HYPOTHESIS

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Evolutionary theory suggests that natural selection should synchronize senescence of reproductive and somatic systems. In some species, females show dramatic discordance in senescence rates in these systems, leading to a clear menopause coupled with prolonged postreproductive life span. The Mother Hypothesis proposes that menopause evolved to avoid higher reproductive-mediated mortality risk in late-life and ensure the survival of existing offspring. Despite substantial theoretical interest, the critical predictions of this hypothesis have never been fully tested in populations with natural fertility and mortality. Here, we provide an extensive test, investigating both short- and long-term consequences of mother loss for offspring, using multigenerational demographic datasets of premodern Finns and Canadians. We found no support for the Mother Hypothesis. First, although the risk of maternal death from childbirth increased from middle age, the risk only reached 1–2% at age 50 and was predicted to range between 2% and 8% by 70. Second, offspring were adversely affected by maternal loss only in their first two years (i.e., preweaning), having reduced survival probability in early childhood as well as ultimate life span and fitness. Dependent offspring were not affected by maternal death following weaning, either in the short- or long-term. We suggest that although mothers are required to ensure offspring survival preweaning in humans, maternal loss thereafter can be compensated by other family members. Our results indicate that maternal effects on dependent offspring are unlikely to explain the maintenance of menopause or prolonged postreproductive life span in women.

KEY WORDS: **Aging, death from childbirth, Grandmother hypothesis, longevity, maternal effects, senescence.**

The life-history of women is striking for at least two reasons. First, compared to our extant primate relatives, humans have a long period of dependence and yet short birth intervals, leading to unusually high numbers of offspring being cared for simultaneously (Hawkes and Paine 2006). Second, compared with mammals generally (Packer et al. 1998), women worldwide show an unusually clear menopause, with all women losing their reproductive potential by their 50s (Pavelka and Fedigan 1991), but having the potential to survive for decades thereafter (Hawkes et al. 1998). Menopause is not only a medical and social issue (Pavelka and Fedigan 1991; Lahdenperä et al. 2004a), but represents an evolutionary conundrum because classical evolutionary theory suggests that natural selection should lead to an individual's reproductive capacity ending in unison with the capacity for somatic maintenance (Cole 1954; Williams 1957; Hamilton 1966). Although in social species, with extended parental care and/or auxiliary care from other family members, it is now known that discrepancies between investment in reproductive and somatic maintenance can evolve (Lee 2003; Bourke 2007), the question of why this has evolved in female humans in the form of menopause remains.

Menopause is not a consequence of modern improvements to nutrition, hygiene or medicine, for it is well known from both prehistoric times and modern-day hunter–gatherers (Hawkes et al. 1998; Gurven and Kaplan 2007; Cant et al. 2009). Broadly, four hypotheses have been proposed to explain the evolution of menopause. For example, it has been suggested to result from: benefits of ceasing reproduction early to invest in the reproductive attempts of offspring (Grandmother Hypothesis; Hawkes et al. 1998); strong positive selection on male life span (Male Longevity Hypothesis; Kaplan et al. 2000; Marlowe 2000; Tuljapurkar et al. 2007); and/or costs of co-breeding with daughters-in-law (Reproductive Conflict Hypothesis; Cant and Johnstone 2008; Cant et al. 2009). However, the potential importance of these hypotheses has been overshadowed by the popularity of another hypothesis, the Mother Hypothesis (Williams 1957; Packer et al. 1998; Moss de Oliveira et al. 1999; Peccei 2001). This latter hypothesis proposes that because humans have unusually high numbers of dependent offspring present simultaneously and because offspring fitness might be expected to be tightly linked with the amount of care they receive, mothers who shut-down their reproductive system earlier and concentrated on rearing their already existing offspring would have been selected over those which produced offspring throughout life. This is because where costs of reproduction increase with age, those adopting the latter strategy and dying as a consequence risked either losing large numbers of their offspring or reducing the quality and future success of those offspring (Williams 1957; Lancaster and Lancaster 1983; Peccei 1995, 2001). Thus, primarily two factors acting in unison are suggested to be sufficient to select for the evolution of menopause late in life (Pavard et al. 2008). These are prohibitive age-specific increases in risks of death from childbirth and consequential detrimental effects on all dependent offspring. Despite the popularity of the aforementioned hypothesis, the evidence for each of these is weak.

First, at some point, the costs of reproduction, principally manifest through death from childbirth, should increase markedly with increasing maternal age and/or parity, leading to selection against further reproduction. There is supporting evidence that maternal mortality from childbirth can rise with increasing maternal age, but the risk still remains small, ranging from approximately 1–2.5% after age 40 in historical as well as contemporary populations with generally high maternal mortality rates (Yerushalmy et al. 1940; Högberg and Broström 1985; Fortney et al. 1988; Knodel 1988; Loudon 1992; Mbizvo et al. 1993; Donoso and Villarroel 2003; Temmerman et al. 2004; Ujah et al. 2005). Although such studies suggest mortality risk to be insufficient to select for menopause, they should be viewed with caution because they seldom use longitudinal data meaning that differences in maternal quality cannot be controlled (van de Pol and Verhulst 2006; Nussey et al. 2008). In addition, they have often used women with access to health care, which will likely confound estimates of mortality risk from childbirth (Fortney et al. 1988; Mbizvo et al. 1993; Donoso and Villarroel 2003; Temmerman et al. 2004).

Second, and more critically, maternal death from childbirth should not only have a significant negative impact on the survival probability of the new child, but also on all those previous offspring that are still dependent on their mother for sustenance (suggested to be up to 15 years; Peccei 1995, 2001; Kaplan and Lancaster 2003; Pavard et al. 2008). This being the case, the Mother Hypothesis not only predicts the evolution of menopause, but also a postreproductive life span sufficient to ensure the survival of the last offspring to adulthood (Shanley et al. 2007). Previous studies show support for the importance of the mother on child survival preweaning (Andersson et al. 1996; Sear et al. 2000; Reher and Gonzáles-Quiñones 2003; Masmas et al. 2004; Zaba et al. 2005, reviewed in Sear and Mace 2008). However, the nature of the interaction between maternal death and offspring age pre- and postweaning on all offspring fitness parameters, including offspring survival to maturity (but see Pavard et al. 2005), ultimate life span (but see Campbell and Lee 2009; Willführ 2009) and future reproductive potential, have not been explored. Additionally, when such factors have been considered previously, little or no attempt has been made to control adequately for confounding effects of the presence of other carers that might ameliorate the costs of maternal loss for offspring (Sear and Mace 2008). Quantifying the effect of mother loss at multiple ages and on multiple life-history traits, while controlling for confounding influences of other family members, is essential for a full test of the Mother Hypothesis (Williams 1957).

Thus, despite the Mother Hypothesis being proposed more than 50 years ago and commonly being cited as the explanation for the evolution of menopause (see above), there are a number of significant deficiencies in previous studies. (1) Few studies have used human populations experiencing natural levels of fertility and mortality (Rogers 1993; Shanley and Kirkwood 2001, 2007; Pavard et al. 2008). (2) Only four studies have studied the effects of mother loss on child survival to adulthood while considering confounding influences (reviewed in Sear and Mace 2008), despite the assumption of the Mother Hypothesis being that mothers are required throughout offspring development (Williams 1957; Kaplan and Lancaster 2003). (3) Only three studies have considered the effect of mother loss at various ages during offspring development on offspring survival through the rest of childhood (Pavard et al. 2005) or adulthood (Campbell and Lee 2009; Willführ 2009), and none has considered the reproductive consequences. This is despite the fact that early conditions are known to have life-long consequences (Metcalfe and Monaghan 2001; Lummaa and Clutton-Brock 2002) and that Williams (1957) highlighted that the benefits of menopause evolution should manifest as overall fitness advantages incorporating short- and long-term consequences of mother loss for both survival and reproduction. Therefore, rigorous tests of the Mother hypothesis are currently lacking.

In this study, we provide the first comprehensive test of the main two predictions of the Mother Hypothesis outlined above using multigenerational individual-based data from two countries: Finland ($n = 1965$ mothers, 10,260 offspring) and Canada ($n = 1642$ mothers, 14,923 offspring). The two populations experienced natural fertility and mortality (Finland: Soininen 1974; Canada: Gauvreau 1993; Gauvreau et al. 2007) but differed in religion, culture, and population growth; hence improving the capacity for generalizations. We have shown previously in both populations that women gained significant fitness from surviving beyond menopause and helping to improve the reproductive success of their offspring (Lahdenperä et al. 2004b). These results are consistent with the Grandmother Hypothesis, which has been proposed to account for prolonged life span in women but does not necessarily provide an adaptive explanation for menopause per se (Hawkes et al. 1998). Here, we focus on the predictions of the Mother Hypothesis for the evolution of menopause. First, we determine the risk of dying from childbirth and investigate how this changes with maternal age and parity. Second, we investigate the immediate and delayed effect of maternal loss on offspring survival between birth and adulthood (age 15 years), as well as the effect of maternal loss at varying ages during development (up to age 15 years) on their life span and reproductive capacity in adulthood. Our analyses control for an unprecedented number of possible confounding effects, including: birth cohort, sex, and order; maternal socioeconomic status and previous birth interval; the presence of other carers (father, grandparents); and repeated measures of the same mother.

Methods

STUDY POPULATIONS

Finns

Data for preindustrial Finnish people were collected from the parish records of the Lutheran Church, obliged by law to maintain accurate registers of all births, movements, marriages and deaths in the country since the seventeenth century (Luther 1993). Our data contain survival and reproductive details from four geographically distinct communities, including cause of death. The communities included three parishes from coastal (Kustavi, Rymättylä, and Hiittinen) and one parish from inland (Ikaalinen) areas. In general, the main source of livelihood was farming, supplemented by fishing in coastal areas leading to greater predictability of food at the coast (Lummaa et al. 1998). Overall for Finland, we obtained data for 10,260 children born between 1741 and 1908 to 1965 mothers and 1834 fathers (born 1709–1876). The study period ended before industrialism and before more liberal economics, birth-control methods, and higher standards of living influenced fertility and survival in Finland (Soininen 1974). The occupation (e.g., tenant farmer, fisherman, landowner, servant) of each father was recorded at the time the children were born, allowing us to rank child socioeconomic status (rich, average, poor) (Lahdenperä et al. 2004b). Overall, the standard of living was low; climatic conditions in Finland can be extremely challenging and both famines and diseases were common (Turpeinen 1973; Pitkänen 1993). Surviving offspring usually moved away from home from age 15 to work but commonly returned (Moring 2003). The average age at first marriage was 25 (range 15–54) and 26 (14–61) for women and men, respectively, and 88% of offspring married if they survived to maturity (defined here as 15 years of age, the youngest known reproducer in our population). The mating system was monogamous, and both divorce and extra-marital affairs were outlawed (Sundin 1992).

Canadians

The Canadian data were obtained using the BALSAC population register based at the University of Quebec, Chicoutimi (BALSAC Project 2009). The register contains demographic and genealogical information collected from baptism, marriage and death certificates from all individuals in the Saguenay region of Quebec during the nineteenth and twentieth centuries (Bouchard et al. 1995; Bouchard 2008). The opening of the Saguenay region to sustained settlement began in the early 1830s, with founders coming mainly from the adjacent region of Charlevoix, south of the Saguenay (Gauvreau et al. 1991). The population was Catholic and until the beginning of the twentieth century, mainly

agricultural (Bouchard 1996). Overall for Canada, we obtained data for 14,923 children born from 1891 to 1929 to 1642 mothers and 1627 fathers (born 1853–1885). Climatic conditions in this area were extreme and mothers experienced natural fertility and mortality, for benefits of health care were not apparent in the region until after the 1930s (Pouyez and Lavoie 1983; Gauvreau 1993; Gauvreau et al. 2007). The data also contain information of the occupation (e.g., farmer, blacksmith, merchant, journeyman) of the father in each family, allowing us to rank child socioeconomic status as for the Finns. The average age at first marriage was 22 (14–67) and 26 years (16–65) for women and men, respectively, and 91% of offspring married if they survived to maturity (age 15 years). The population was monogamous, illegitimate births were very rare (<1%), and all children were baptized (Bouchard 1996).

STATISTICAL ANALYSES

Survival costs of reproduction for mothers

Increasing reproductive costs with age could be manifest in two ways. First, high investment in reproduction early in life could reduce female life span, leading to an increased mortality risk in later ages among heavily investing females. Previous analysis of our Finnish dataset has failed to detect a cost of reproductive effort in terms of increased mortality risk following high overall fecundity (Helle et al. 2004), as has been typical even for humans living in conditions of natural fertility (Helle et al. 2005; Hurt et al. 2006; Le Bourg 2007). For example, Hurt et al. (2006) conducted a meta-analysis of ∼30 articles to investigate the relationship between the number of children born and mortality in women living beyond 40 years, and failed to show a consistent effect of parity on longevity. Similarly, Helle et al. (2005) show that up to 61% of 31 studies either shows a positive link between parity and longevity or no link at all. Thus, we do not consider maternal mortality to be a consequence of overall parity, although we do control for potential effects of parity (equivalent to offspring birth order) in the analysis of the age-specific probability of dying from a given childbirth (see below).

Second, increasing reproductive costs with age could be manifest as an increased probability of dying from a given childbirth. Our analysis of the age-specific risk of dying from childbirth was conducted using both populations (Finland: $n = 7354$ births by 1895 mothers; Canada: $n = 13{,}220$ births by 1463 mothers). A mother was defined as having died from childbirth if she died (excluding deaths due to accidents) within six weeks following parturition (Knodel 1988). The effect of maternal age on the probability of dying from childbirth was investigated using a generalized linear mixed effects model (GLMM) in which the data were fitted to a binomial error structure with logit link function. Linear and quadratic functions of maternal age and previous number of births were fitted as the main fixed terms of interest, while offspring sex, socioeconomic status, and birth year were fitted as potential confounding terms. Parity (offspring birth order) was fitted to control for differences in maternal investment (Faurie et al. 2009) and/or quality (van de Pol and Verhulst 2006). Maternal age and birth order were fitted as continuous variables, although deliveries for maternal ages under 18 years and over 47 years and birth orders over 10 were pooled due to small-sample sizes. We accounted for nonindependence of survival probabilities of children from the same mother by including a family-specific repeated effect in the model where necessary (mother in both countries).

Consequences of maternal death for offspring

We examined the effects of mother loss on children in three ways. First we examined the effects of mother loss on the short-term survival prospects of offspring during childhood (0–15 years). Short-term survival prospects were defined as the probability that an offspring would survive both the current and following calendar year following maternal death. Second, we examined whether mother loss had intermediate-term effects on child survival prospects during dependence and late childhood (up to age 15). Intermediate-term survival prospects were defined as the probability that an offspring would survive the rest of their childhood (to age 15 years), given they had already survived their first two calendar years following maternal death. The annual survival of 7412 Finnish and 9141 Canadian offspring was known from birth to adulthood (age 15, the youngest known reproducer in the population). Finally, we examined the long-term effects of losing a mother at various ages during dependence (0–2, 3–6, 7–15) on a child's ultimate life span and lifetime breeding success (i.e., number of offspring produced in a lifetime) (see Results for sample sizes).

Short-term effects of mother loss. The effect of mother loss on short-term offspring survival (i.e., within the same or following calendar year) was investigated in two ways. First, we used discrete time survival analyses (also known as event history analysis), which allows a sensitive analysis of the effects of time-dependent variables, such as the presence of relatives (see below), on the child's probability of dying over discrete time intervals, while accounting for repeated sampling of different offspring born to the same mother (hence experiencing a common environment, Singer and Willett 2003). Hereafter termed the time-interval analysis, this analysis allowed us to estimate the offspring risks of dying in each year from birth to age of 15 years in the presence versus absence of a living mother (i.e., whether the mother had died during the last two calendar years). For each year from birth to 15 years (16 time intervals for each child), the survival of each child was coded as survived versus died during the observation year (1/0) or missing (when the death had already occurred). Following restrictions (see below), this analysis was carried out on 77,011 datapoints in Finland

(n = 7410 children, 1481 mothers) and 107,521 datapoints in Canada (n = 9141 children, 1143 mothers).

Although, in the above analysis, an assessment of the interaction between maternal presence and child age on child survival would provide an indication of whether children are more likely to die at a given age following maternal loss in the last two calendar years, it does not provide a quantitative estimate of the child age at which maternal loss becomes unimportant for child survival. Second, therefore, to clarify whether mother loss is more detrimental to children at certain ages, we investigated the effects of mother loss on child mortality during specific child age intervals. The age categories were: preweaning (0–2 years), early childhood (3–6 years), and later childhood (7–15 years). In these age-category analyses, we also used discrete time survival analyses to determine the effect of mother loss during each time period on the survival prospects of the children during the same time period.

In both time-interval and age-category type analyses, all offspring that died before their mother and all offspring–mother couples who died within one week of each other were excluded to remove coincidental deaths due to external factors. Mother presence, child age, and the interaction between the two were fitted as the primary fixed effects of interest. In addition, however, the analyses controlled for the following confounding variables, although not all variables were available for both Canada and Finland: the presence of other relatives (father, grandparents); socioeconomic status; previous birth-interval length (only in Finland); and offspring birth year, sex and birth order. Results are presented primarily from the models using time-interval analyses, but results of the age-category analyses are also provided to clarify the child age at which maternal loss ceases to have a significant impact on child survival.

Intermediate-term effects of mother loss. Using the same two analytical approaches outlined above (time-dependent and age-category), we next investigated whether maternal death had delayed effects on child survival prospects during dependence (up to age 15). The primary difference here was that child survival prospects were considered at least two calendar years following maternal death. Therefore, in the time-dependent style analysis of the intermediate-term effects of maternal loss for child survival prospects, the mother's presence was coded as being alive or having died at least two years before each observation year (13 observations of mother presence for each child from age 3 to 15 years). In the age-category analysis, the effects of maternal death during one age-category were assessed on child survival during the following age-categories. Restrictions were as described above under short-term effects. Overall, the time-dependent analysis was conducted on 60,673 datapoints in Finland and 149,927 in Canada (7412 mothers and 1482 children in Finland and 1143 mothers and 9141 children in Canada). Again, the results are presented primarily from the time-interval analyses, with results of the age-category analyses being used to clarify contrasts in the age at which maternal effects ceased to be significant. Confounding terms were considered as for the short-term analyses outlined above.

Long-term fitness consequences of mother loss. Finally, we determined the long-term fitness consequences of mother loss during varying ages of dependence (0–2, 3–6, 7–15). In this case, we used only linear mixed effects models (see below). Fitness consequences were considered in terms of offspring life span after age 15 and total lifetime breeding success in both countries. It is essential to note that all children included in these analyses lost their mother prior to the age of 15 and hence potential grandmaternal effects are excluded. The analyses of the effects of child age at mother loss on both the child's life span after age 15 and lifetime breeding success were conducted using linear mixed effects models fitted to a normal error structure. Offspring age category at mother death was fitted as the main fixed effect. In the life span analyses: birth cohort, sex, living area (only in Finland), and socioeconomic status were fitted as potential confounding fixed effects, whereas maternal identity was fitted as a random term (n = 315 in Finland, n = 313 in Canada). The lifetime breeding success analyses considered only those offspring that were married and for which full lifetime fecundity was known with precision (n = 158 in Finland, n = 223 in Canada). Using only married individuals is justified because almost all who survived to adulthood were likely to marry at some point of their life (about 88% in Finland and 91% in Canada) and the results remained unchanged if one includes all individuals surviving to adulthood irrespective of their future marital status. In these analyses, offspring age category (0–2, 3–6, 7–15) at mother death was fitted as the main fixed effect; birth year, sex, living area (only in Finland), socioeconomic status, age at marriage and offspring life span were fitted as potential confounding fixed effects, whereas maternal identity was fitted as a random term.

All statistical analyses were conducted using SAS (SAS Institute Inc., release 9.1, 2002–2003). Confounding fixed terms were retained in models only if they improved explanatory power, determined using AIC criteria. Interactions were only tested if they were specifically predicted by the hypothesis. In all models, statistical probabilities are two-tailed, variances were not significantly dissimilar and residuals from all models were normally distributed.

Results

SURVIVAL COSTS OF REPRODUCTION FOR MOTHERS

The average number of children delivered by a woman during her lifetime was 6.6 in Finland (range 1–17) and 7.5 in Canada (range 1–20). Death from childbirth was rare. In Finland, only 0.5% of

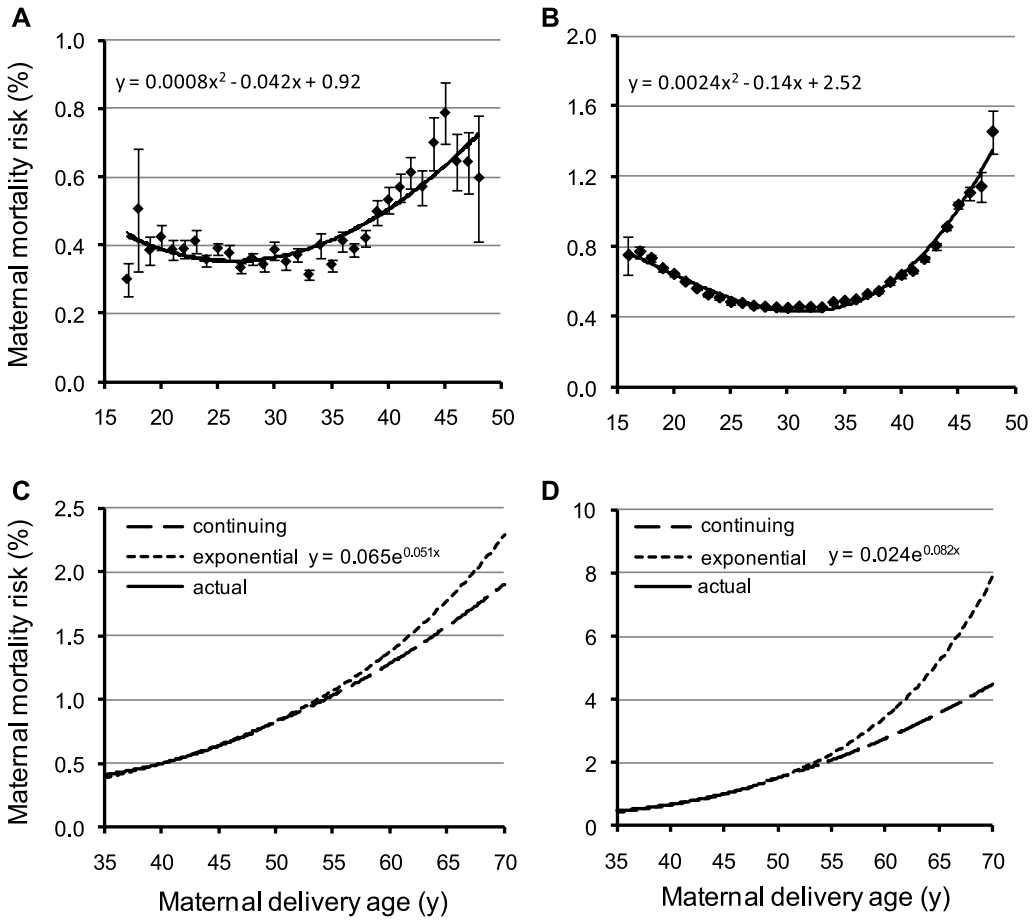

Figure 1. **Actual age-specific risk of dying from childbirth in preindustrial Finland and Canada during child-bearing years (16–50 years) (A, B) and projected risks postmenopause (50–70 years) (C, D). The actual risk of women dying from childbirth was higher among young and old mothers in (A) Finland (mother age effect $\beta = -0.79 \pm 0.17$, $\chi^2_1 = 4.85$, $P = 0.028$; mother age^2 effect, $\beta = 0.12 \pm 0.0026$, $\chi^2_1 = 23.40$, $P < 0.001$: $n = 7339$); and (B) Canada (mother age effect $\beta = -0.17 \pm 0.046$, $\chi^2_1 = 14.38$, $P < 0.001$; mother age^2 effect, $\beta = 0.0099 \pm 0.0045$, $\chi^2_1 = 4.81$, $P = 0.028$: $n = 13{,}220$). (A) and (B) show predicted means (±SE) from GLMMs (see Methods). The projected risks of mothers dying from childbirth in Finland (C) and Canada (D). The long-dotted lines (continuing function) show the projected risks of dying if the risks observed during child-bearing years are continued beyond menopause. The short-dotted lines show the risks of dying if the risk increases exponentially from the mean age at last reproduction (i.e., 38 years). Note the varying scales in A-D.**

all 7354 births ended in maternal mortality, equating to 2% of the 1895 mothers dying as a result of childbirth. Similarly, in Canada, 0.6% of 13,220 births ended in mothers dying, equating to 5% of the 1463 mothers dying from giving birth.

The probability of dying in childbirth showed a curvilinear relationship with maternal age in both Finland and Canada. In Finland, mothers became less likely to die from childbirth if they delayed reproduction until their 20s but became increasingly more likely to die in childbirth from their mid 30s (mother age effect $\beta = -0.79 \pm 0.17$, $\chi^2_1 = 4.85$, $P = 0.028$; mother age^2 effect, $\beta = 0.12 \pm 0.0026$, $\chi^2_1 = 23.40$, $P < 0.001$) (Fig. 1A). Patterns were similar in Canada, with those giving birth before their mid-20s and after their mid-30s being more likely to die as a result of childbirth (mother age effect $\beta = -0.17 \pm 0.046$, $\chi^2_1 = 14.38$, $P < 0.001$; mother age^2 effect, $\beta = 0.0099 \pm 0.0045$, $\chi^2_1 = 4.81$, $P = 0.028$) (Fig. 1B). Nevertheless, the magnitude of the effects of increasing maternal age on the risk of dying from childbirth, although statistically significant, was likely to be of limited importance biologically. For example, maternal mortality risk from childbirth increased from a low of 0.3% (Finland) and 0.45% (Canada) for women in their mid 20s to a high of 0.8% (Finland) and 1.5% (Canada) for those in their late 40s (Figs. 1A,B).

One could argue, however, that natural selection has produced women that reproduce for the last time before their probability of dying from childbirth becomes prohibitively high. Although one can never measure the risk of dying from childbirth in ages following last reproduction, it is likely that the risk would follow a function somewhere in between a continuing and exponentially increasing fitted from the mean age at last reproduction

Table 1. Discrete time survival model of the short-term effects of mother loss on offspring risk of death from 0 to 15 years in preindustrial Finland (Total $n = 77,011$ (7410 children and 1481 mothers). Estimates (positive reflect increasing mortality risk) are provided for variables and two-level factors. Terms retained and rejected in the final model (determined using AIC) are shown above and below the constant, respectively. Mother's identity was fitted as a random term. (For full details of grandmother/grandfather effects, see Lahdenperä et al. 2004b, 2007, respectively).

Term	Estimate ± SE	Statistic (χ^2)	df	*P* value
Mother's death	1.21 ± 0.15	27.74	1	<0.0001
Child age	−0.51 ± 0.022	206.12	1	<0.0001
Child age^2	0.022 ± 0.0015	105.29	1	<0.0001
Living area (coast > mainland)	−0.26 ± 0.047	28.66	1	<0.0001
Birth-order	0.055 ± 0.013	16.70	1	<0.0001
Previous birth interval	>the shorter	15.84	2	0.0004
Grandmother's death	>if dead	10.91	2	0.0043
Grandmother's death × child age		11.02	2	0.0040
Mother's death × child age	−0.088 ± 0.037	9.19	1	0.0024
Sex (boys > girls)	0.11 ± 0.041	6.71	1	0.0094
Socioeconomic status	>among poorest people	5.21	1	0.074
Birth year	−0.0018 ± 0.0010	3.14	1	0.076
Constant	1.91 ± 1.83			
Father's death	0.010 ± 0.081	0.02	1	0.90
Grandfather's death	>if dead	0.58	2	0.75

(age ~38 years). Extrapolations of mortality risk to age 70 years using a continuing function shows that it would be predicted to increase up to a maximum of 2% in Finland (Fig. 1C) and 4.5% in Canada (Fig. 1D). Extrapolations to the same age using exponentially increasing functions from the mean age at last reproduction show that maternal mortality risk would be predicted to increase to 2.4% in Finland (Fig. 1C) and 8% in Canada (Fig. 1D). Thus, our data suggest that the maternal mortality risk from giving birth would have remained low even up to the age of 70, which corresponds to both the mean age at death for women that survived to age 50 in premodern Finland and Canada (i.e., menopause; Lahdenperä et al. 2004b) as well as the projected age at last reproduction for women had menopause not evolved (Faddy et al. 1992).

CONSEQUENCES OF MATERNAL DEATH FOR OFFSPRING

Around 65% of all children delivered, survived to adulthood at age 15 years (Finland—62%; Canada—67%). Offspring survival to age 15 was influenced by a number of potentially confounding terms in both Finland (Table 1) and Canada (Table 2). For example, probability of offspring dying during their first 15 years of life was influenced by socioeconomic status, birth year and parish (Finland only); was greater for boys than for girls; and increased with increasing birth order. Offspring also had reduced mortality in the presence of their grandmother in both Finland and Canada (Lahdenperä et al. 2004b) and fathers were also beneficial for children in Canada (Table 2). Finally, in both countries, child mortality risk increased soon after birth before decreasing thereafter (see also Hamilton 1966).

By age 15, 18% (Finland) and 15% (Canada) of children had lost their mother. After controlling for the above confounding terms, we found that in both countries, the loss of the mother significantly increased the probability of offspring death by the end of the following calendar year, although this was modified by the age at which offspring lost their mother (Tables 1 and 2, Figs. 2A,C). Several pieces of evidence suggest that only maternal loss in the first two years of life (i.e., preweaning) had a significant negative effect on offspring survival probability. First, results from the short-term age-category analyses revealed that only maternal death during the first two years of life influenced the probability of a child surviving the current or subsequent calendar year (Table 3). Maternal death when offspring were aged 3–6 or 7–14 years had no influence on offspring survival prospects in the current or subsequent calendar year (Table 3). Second, results of the intermediate-term time-interval analyses revealed that if offspring lost their mother in their first two years of life, but survived both the current and subsequent year, in neither country did maternal loss influence the survival prospects of offspring during the rest of their childhood (Table 3, Figs. 2B,D). Finally, maternal loss in a previous age category did not affect child survival probability in the subsequent age category (Table 3). This again highlights that offspring surviving maternal loss early in childhood did not have impaired survival later in childhood.

Maternal loss in the first two years might also have long-term consequences for survivors, at least in Finland. After controlling

Table 2. Discrete time survival model of the short-term effects of mother loss on offspring risk of death from 0 to 15 years in preindustrial Canada (Total $n = 107,521$ (9141 children and 1143 mothers). Estimates (positive reflect increasing mortality risk) are provided for variables and two-level factors. Terms retained and rejected in the final model (determined using AIC) are shown above and below the constant, respectively. Mother's identity was fitted as a random term.

Term	Estimate ± SE	Statistic (χ^2)	df	*P* value
Mother's death	1.46 ± 0.20	32.61	1	<0.0001
Child age	-0.71 ± 0.019	200.89	1	<0.0001
Child age^2	0.032 ± 0.0015	116.01	1	<0.0001
Socioeconomic status	<among average people	15.15	2	0.0005
Birth-order	0.060 ± 0.015	13.99	1	0.0002
Birth year	-0.022 ± 0.0071	8.98	1	0.0027
Mother's death × child age	-0.14 ± 0.071	8.69	1	0.0032
Sex (boys > girls)	0.11 ± 0.042	6.51	1	0.011
Maternal grandmother's death	0.12 ± 0.048	4.89	1	0.027
Father's death	0.45 ± 0.18	4.20	1	0.040
Maternal grandfather's death	0.11 ± 0.054	3.84	1	0.050
Constant	39.95 ± 11.53			
Paternal grandmother's death	-0.062 ± 0.052	1.45	1	0.23
Paternal grandfather's death	-0.014 ± 0.055	0.06	1	0.81

for significant effects of socioeconomic status, birth year, and individual sex, we found that in Finland, life span in adulthood was impaired by early maternal loss (Fig. 3A). This result was restricted to those that lost their mother in the first two years of life, with such individuals having a 15% reduced life span after age 15 than those who lost their mother later in childhood (between 3–6 or 7–15 years). Similarly, after controlling for significant effects of birth year, offspring age at marriage, and life span, we also found that offspring who lost their mother before weaning in Finland had lower lifetime fecundity than offspring who lost their mother postweaning (Fig. 3B).

However, neither result was apparent in Canada (Figs. 3A,B). An additional model including both countries in the same analyses (controlling for significant effect of birth year; $F_{1,415} = 43.76$, $P < 0.001$) suggests that the countries differ in the effects of mother loss preweaning for ultimate life span of surviving offspring (country × age of maternal death interaction: $F_{2,686} = 3.24$, $P = 0.040$, $n = 705$). Similarly, further analysis with both countries in the same model (controlling for significant effects of birth year; $F_{1,437} = 10.21$, $P = 0.0015$ and life span; $F_{1,520} = 79.07$, $P < 0.0001$) also reveals that the countries differ in the effects of mother loss preweaning for the lifetime fecundity of surviving offspring (country × age of maternal death interaction: 0–2 vs. 3–15 $F_{1,510} = 3.95$, $P = 0.048$, $n = 528$ and 3–6 vs. 7–15 $F_{1,482} = 0.00$, $P = 0.99$, $n = 490$). The reasons for these country differences are unclear.

It is essential to note that all offspring in these analyses lost their mother before adulthood so none of the children had a helping grandmother, which we have already shown to be important for offspring reproductive success in this population (Lahdenperä et al. 2004b). Nor are our results likely to have arisen due to differential resource access because our analyses control for differences in social class. Thus the take-home message is that loss of a mother within the first two years of life can have significant negative consequences for early child survival as well as ultimate life span and lifetime fecundity, but loss of a mother later in childhood (age 3–15 years) does not have any detectable impacts on short-, intermediate-, or long-term survival or reproductive success.

Discussion

The Mother Hypothesis predicts that female menopause evolves where reproduction late life is associated with prohibitively high chances of maternal death and where maternal death has significant negative fitness consequences for the survival of all dependent offspring (Williams 1957). We found little support for either prediction, using large longitudinal demographic datasets on two separate premodern populations with contrasting religions, fertility rates, and rates of population growth. Although the risk of dying from childbirth increases later in life in both populations, the risk is low, such that women only have a 1–2% chance of dying from childbirth by age 50 and a 2–8% chance by age 70. In addition, although we found that maternal death from childbirth can have substantial negative consequences for the current child (see also Andersson et al. 1996; Sear et al. 2000; Reher and Gonzáles-Quiñones 2003; Masmas et al. 2004; Zaba et al. 2005; Sear and Mace 2008), we found no evidence to suggest that it has negative consequences for existing older offspring. This is evidenced by the findings that maternal death after offspring are age 3 has little effect on their survival in childhood, their

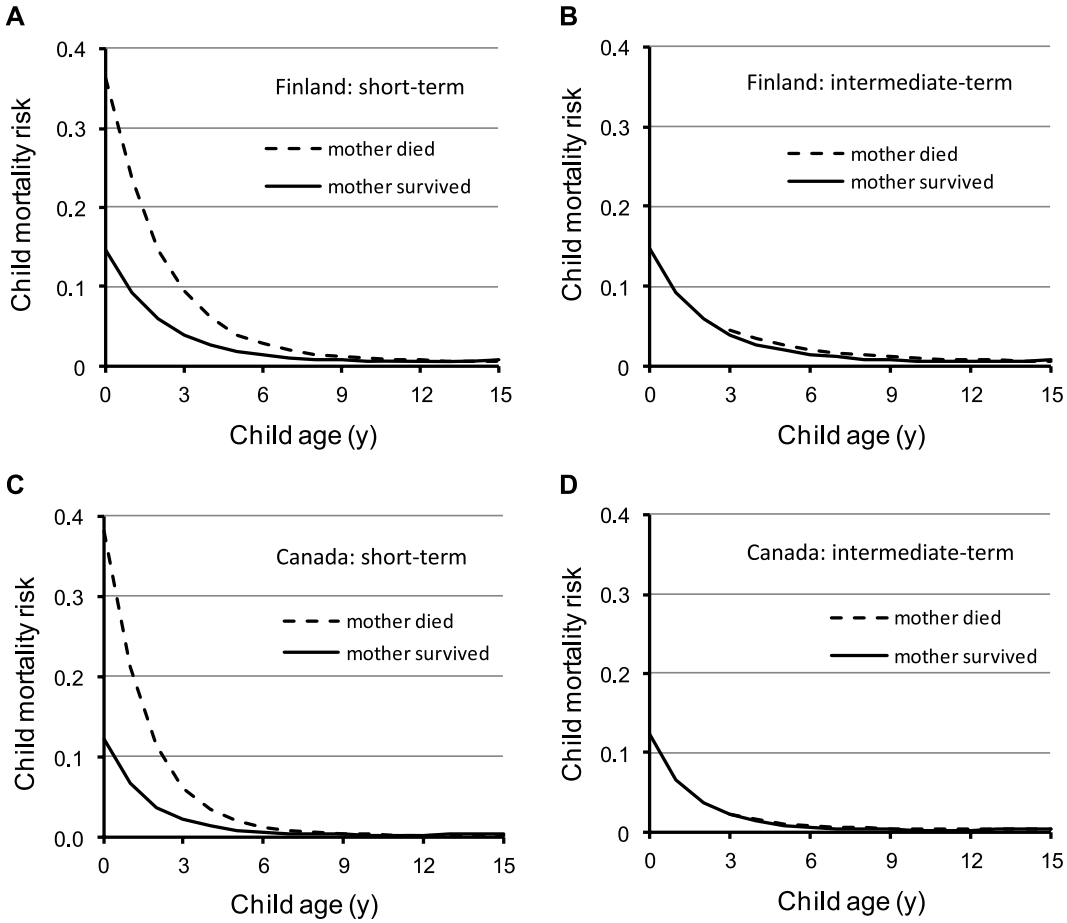

Figure 2. **Consequences of mother loss for offspring risk of dying during dependency in Finland (A, B) and Canada (C, D). Maternal death increased the offspring's probability of dying within the same or following calendar year (short-term effect) between birth and 15 years in (A) Finland ($\chi^2_1 = 27.74$, $P < 0.0001$) and (C) in Canada ($\chi^2_1 = 32.61$, $P < 0.0001$); in both cases offspring death probability decreased with increasing age (interaction between mother's presence and child age: Finland: $\chi^2_1 = 9.19$, $P = 0.0024$; Canada: $\chi^2_1 = 8.69$, $P = 0.0032$). In contrast, maternal death did not have any delayed effects on offspring survival between birth and 15 years (intermediate-term effect) in (B) Finland ($\chi^2_1 = 2.60$, $P = 0.11$) or (D) Canada ($\chi^2_1 = 1.07$, $P = 0.30$); in neither case was there an interaction with child age (interaction between mother's presence and child age: Finland: $\chi^2_1 = 1.91$, $P = 0.17$; Canada: $\chi^2_1 = 0.74$, $P = 0.39$). The sample sizes for which the discrete time survival analyses are based on are 7412 children and their 1482 mothers in Finland (A,B) and 9141 children and their 1143 mothers in Canada (C,D). A–D show predicted means from discrete time survival models.**

longevity, or reproductive success. Thus, we found no evidence for the assumption that mothers are required for several years or even decades to secure the survival of offspring (Peccei 1995, 2001; Kaplan and Lancaster 2003; Pavard et al. 2008). Our study is the first to comprehensively consider the effects of mother loss on both offspring survival and reproductive outcomes by investigating the effects of maternal death at various offspring ages on their survival across the whole preadulthood period, and on offspring adulthood longevity as well as reproductive success. As a consequence, we consider it unlikely that the need for prolonged maternal care can alone explain the evolution of menopause or the markedly extended postreproductive life span in human females.

We tested the two most important predictions of the Mother Hypothesis, but there are two predictions that we did not investigate. First, we did not directly consider the possibility that early life parity might have negative consequences on mortality risk late in life, despite the general expectation of a trade-off between these two life-history characters. Nevertheless, evidence for this trade-off is weak in humans generally (Helle et al. 2005; Hurt et al. 2006; Le Bourg 2007) and we have found no evidence to suggest that lifetime fecundity is negatively associated with maternal survival prospects following menopause in the Finns (Helle et al. 2004). Thus, the inclusion of lifetime fecundity in the current study is of limited value. Second, we did not investigate whether the quality of offspring produced decreases with increasing maternal age. It is well-known from contemporary populations that the risk of miscarriages, chromosomal abnormality, and obstetric complications in pregnancies increase with increasing maternal

Table 3. Effects of mother loss on child survival in Finland and Canada in the short term and the intermediate term. In the short term: results of the discrete time survival analysis of the effect of mother loss in the current or previous year on child survival in the current year. Time intervals were continuous (0–15 years) and categorical (0–2, 3–6, 7–15 years). In the intermediate term: results of the discrete time survival analysis of the effect of mother loss at least 2 years ago on child survival in the current year (3–15 years) and during the previous age interval (see years in parentheses) on the probability of child survival during the following age intervals. Time intervals were continuous (3–15 years) and categorical (3–6, 7–15 years). The reference groups in all analyses are children whose mothers were alive so odds ratios (OR) greater than 1 means that children in the other category (mother died) had higher mortality and vice versa.

	Finland			Canada		
Age interval	OR	χ^2_1	*P*	OR	χ^2_1	*P*
Short term						
0–15	3.35	27.74	<0.0001	4.32	32.61	<0.0001
0–2	6.01	27.32	<0.0001	7.40	29.08	<0.0001
3–6	1.49	1.27	0.26	1.37	0.46	0.50
7–15	1.90	3.18	0.075	1.84	1.50	0.22
Intermediate term						
3–15	1.30	2.60	0.11	1.25	1.07	0.30
3–6 (0–2)	1.28	0.75	0.39	1.47	0.70	0.40
7–15 (0–2)	1.21	0.23	0.63	0.85	0.09	0.76
7–15 (3–6)	1.25	1.06	0.30	1.63	1.73	0.19

age (Heffner 2004; Reddy et al. 2006; Djahanbakhch et al. 2007). Although such costs undoubtedly reduce the fitness benefits of reproduction late in life, it is unclear whether they are a contributing factor of menopause or an inevitable consequence (Cant et al. 2009). In addition, birth problems late in life will be insufficient to select for menopause unless they occur in tandem with both prohibitive risks of maternal mortality from childbirth and concomitant reductions in survival probability of multiple dependent offspring. Thus, consideration of any negative effects of maternal age on offspring quality would also be of limited value.

There are two difficulties with testing adaptive hypotheses for the evolution of menopause. It is impossible to test the predictions of hypotheses for menopause in the context in which it evolved. Additionally, all women experience menopause and the age at which it occurs varies markedly, making it impossible to compare the relative success of a menopausal versus nonmenopausal strategy or the effect of menopause onset on fitness. Given these problems, one should be cautious about interpretation of results from any study. Notwithstanding, the problems are alleviated in this study because it was conducted primarily on populations predemographic transition, without access to healthcare/medicine. Additionally, the populations lived in extreme environments; indeed, mortality and fecundity rates were in line with studies from modern hunter–gatherer societies (Peccei 2001). For example, the overall maternal mortality rate for the preindustrial Finnish population was 503 maternal deaths per 100,000 births, a frequency that is considered "high" by UNICEF (2008). Although significantly higher maternal mortality rates have been recorded (e.g., DeGroof et al. 1993–1000 deaths per 100,000 births in

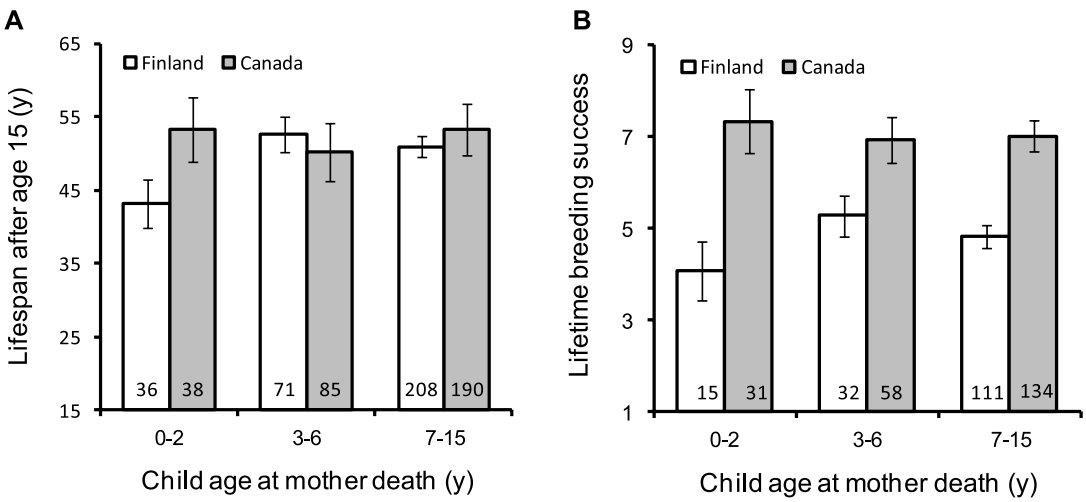

Figure 3. Consequences of early mother loss on adulthood life span and fitness. (A) In Finland, adulthood life span was shorter if the offspring had lost a mother during first two years of life compared to those offspring who lost their mother between 3–6 or 7–15 years ($F_{2,291} = 2.88$, $P = 0.058$, $n = 315$), although this effect was not apparent in Canada ($F_{2,303} = 0.96$, $P = 0.38$, $n = 313$). (B) In Finland, the offspring who lost their mother during weaning had lower lifetime breeding success than offspring who lost their mother during later age but also before maturity (0–2 vs. 3–15: $F_{1,151} = 3.80$, $P = 0.053$; 3–6 vs. 7–15: $F_{1,137} = 0.87$, $P = 0.35$), but not in Canada (0–2 vs. 3–15: $F_{1,218} = 0.04$, $P = 0.84$; 3–6 vs. 7–15: $F_{1,187} = 0.03$, $P = 0.87$). A-B show predicted means (±SE) from GLMMs.

Nigeria), most maternal mortality ratios in sub-Saharan African populations are consistent with the levels found in our study (Mace and Sear 1996) which are 200 times higher than in modern-day developed countries (Koblinsky 1995). Thus, the childbirth-associated maternal mortality of 1–2% by age 50 years in our study is in line with the rates from both historical and contemporary populations recording the highest probabilities of dying from childbirth in the world (Yerushalmy et al. 1940; Högberg and Broström 1985; Fortney et al. 1988; Knodel 1988; Loudon 1992; Mbizvo et al. 1993; Donoso and Villarroel 2003; Temmerman et al. 2004; Ujah et al. 2005).

The mean age at last reproduction in the Finns and Canadians was 38 years, and this is a general finding across populations and cultures (Robson et al. 2006). Despite this, the probability of dying from childbirth at this age approximated only 0.5% in both countries and values in other studies are similarly low (<3% after age 40: Yerushalmy et al. 1940; Högberg and Broström 1985; Fortney et al. 1988; Knodel 1988; Loudon 1992; Mbizvo et al. 1993; Donoso and Villarroel 2003; Temmerman et al. 2004; Ujah et al. 2005). Selection for ceasing reproduction at 38 would be expected if the risks of dying from childbirth increased exponentially after this age to a prohibitively high level soon after. For example, Pavard et al. (2008) suggested that an exponential increase in mortality risk from childbirth from 1.5% at 45 years would be sufficient to select for menopause. However, we found that fitting an exponentially increasing risk function through the available data from the mean age at last reproduction did not yield prohibitive risks of death in childbirth even by age 70 years (risks ca. 2–8% by age 70). Thus, we found no evidence to suggest that ceasing reproduction results from negative selection arising from increasing risks of dying from childbirth. Furthermore, even if this were the case, given that we show that death from childbirth only has a detrimental effect on the current child, there should be little selection against mothers attempting to produce another child irrespective of her risk of death. In other words, given that the probability of dying late in life as a consequence of childbirth is low, and only the fitness of the current child is jeopardized if mothers are lost from childbirth, a gene for menopause (selected for the reasons above) is unlikely to spread through a population when it competes against genes that allow offspring production throughout life.

The assertion that our results are unsupportive of the Mother Hypothesis is further supported by comparison of our empirical results with those of a previous quantitative model on the evolution of menopause (Shanley et al. 2007). This model, parameterized using a contemporary population of rural Gambians, had three components that were evaluated for their ability to select for menopause. These included: (1) increases of child mortality risks following maternal loss of 13.4-fold (for loss before age 1 year) and 11.7-fold (for loss between 1 and 2); (2) maximal estimates of maternal mortality risk from childbirth modeled as an exponentially increasing function equivalent to a doubling of risk every 1.4 years from the age of 45 years; and (3) benefits of grandmothering. Only the inclusion of all three factors was sufficient to select for menopause in their model, although at an unnaturally late age in life. In our study, children that lost their mother within the first two years of life had a 6- (Finland) to 7.5-fold (Canada) increase in dying. Furthermore, we found little evidence that the risk of maternal mortality from childbirth was as high as that estimated by Shanley et al. (2007). That the values used by Shanley et al. (2007) were markedly higher than those detected in this study and still theoretically insufficient to select for menopause, supports our contention that the Mother Hypothesis is unlikely to provide a predominant explanation for the evolution or maintenance of menopause in humans.

The question is, why should maternal mortality not have consequences for postweaned, but still dependent, offspring as assumed in the Mother Hypothesis? One explanation is that the father could compensate for maternal loss, and we found some support for this in Canada. Another is that other family members compensate for maternal loss among postweaned offspring. Although relatively unusual among mammals, and largely confined to callitrichids among primates (Russell 2004), humans are cooperative breeders, with both pre and postreproductive individuals caring for the offspring of the reproductive female (Blaffer Hrdy 1999, 2009; Foster and Ratnieks 2005; Mace and Sear 2005; Sear and Mace 2008). This compensation explanation is supported by the fact that we have found previously that grandmaternal presence is important for grandchild survival in both populations (Lahdenperä et al 2004b). Thus, the evidence appears to show that although maternal survival is essential for offspring survival and later success during weaning (see also Campbell and Lee 2009; Willführ 2009 for adulthood survival effects of early mother loss), this is not the case thereafter when other group members are able to compensate for maternal loss (Lahdenperä et al. 2004b; Sear and Mace 2008, this study). Indeed, it is noteworthy that in a large proportion of cooperative mammals mothers provide little in the way of care to offspring other than milk (e.g., Clutton-Brock et al. 2004; Russell 2004).

In conclusion, our data do not support the two primary predictions of the Mother hypothesis. Three other explanations for the evolution of menopause are possible. First, the Grandmother Hypothesis is likely to offer an explanation for the evolution of prolonged life span following menopause (e.g., Hawkes et al. 1998; Lee 2003; Lahdenperä et al. 2004b; Voland et al. 2005; Shanley et al. 2007; Sear and Mace 2008; Kaplan and Robson 2009), but is unlikely to offer the full explanation for the evolution of menopause. Second, the Male Longevity Hypothesis suggests that female menopause is an unselected consequence of strong selection for increasing longevity in men, resulting from

positive correlations among male age, resource accumulation, and reproductive success (Kaplan et al. 2000; Marlowe 2000; Tuljapurkar et al. 2007). Third, the Reproductive Conflict Hypothesis proposes that menopause results from reproductive conflict with younger generations, recently shown mathematically to select for the evolution of menopause in the older generation (Cant and Johnstone 2008; Cant et al. 2009). Reproductive conflict is universal among cooperative animals leading to temporal or permanent sterility in many insect, bird, and mammal species (Russell and Lummaa 2009) and thus could potentially have an important role also in the evolution of menopause. The predictions of neither of these latter two hypotheses for the evolution of menopause have been tested adequately and we urge future studies to do so. Such studies will not only further our understanding of the evolution of this paradoxical and unusual life-history trait, but will elucidate the ecological and social contexts under which cooperation and conflict among women, men, and offspring drive female life-history schedules.

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