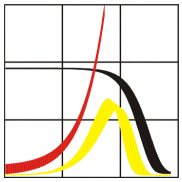

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Menopause and post-generative longevity:

Testing the 'stopping-early' and 'grandmother' hypotheses

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Abstract

The existence of menopause and post-generative longevity as part of the human females' life history is somewhat puzzling from an evolutionary perspective. The 'stopping-early hypothesis' states that, because human infants are so altricial, it is beneficial for women to cease reproduction at the age at which the risk of maternal death reaches a certain threshold. In contrast, 'the grandmother hypothesis' states that survival long past the age of menopause has been selected for because grandmothers significantly improve grandoffspring survival probabilities.

In this study, 'the stopping-early hypothesis' and 'the grandmother hypothesis' are tested for both the evolution of menopause and the evolution of post-generative longevity. This is done by simulating hypothetical life histories of women with and without menopause, with and without post-generative longevity, and with and without positive grandmother effects on infant survival.

Results indicate that neither the benefits accrued from maternal care of late born offspring, nor grandmaternal facilitation of infant survival, are adequate to account for the evolution of menopause. With respect to the existence of post-generative longevity, rather than menopause, however, some level of support is found for both the stopping-early and the grandmother hypothesis. The effects of removing post-generative grandmaternal care on long-term reproductive success are shown to be far greater than the effects of removing post-generative maternal care.

Introduction

Human female fertility rises gradually with age, peaks in the late twenties, then begins to decrease until menopause occurs during the late 40's or early 50's. At this point it is not possible for women to reproduce anymore, yet women can expect to live between another 19 and 22 years even in contemporary hunter-gatherer societies without modern medicine (Blurton-Jones et al, 2002). That human females typically have their last offspring so long before they die is peculiar.

Certainly, all female primates experience reduced fecundity at late ages (Bellino and Wise, 2003; Finch and Gosden, 1986; Gage, 1998; Harley, 1990) but whether or not they experience menopause is a controversial issue. Evidence regarding the termination of reproduction prior to death amongst primates kept in captivity, well fed and free from predation, is very mixed (Caro et al, 1995; Graham, 1979). Where an animal dies having not reproduced for some years it is unclear whether this represents menopause or just an increase in interbirth intervals with increasing maternal age. Amongst wild populations, the complete termination of reproduction well before death rarely, if ever, occurs (Pavelka and Fedigan, 1991). The issue is important because if fertility declines rapidly regardless of external conditions amongst females of all primate species, while other physiological functions show more flexibility and decline at slower rates when conditions are good, considerable weight is lent to the argument that female menopause and post-generative longevity are not themselves adaptations, but are either artifacts of modern life or by-products of other adaptations.

That menopause and post-generative longevity are artifacts of modern life that were not present for most of human evolutionary history (Washburn, 1981; Weiss, 1981) is in accordance with the lack of evidence from the fossil record of human

ancestors surviving to late ages. However, it can be argued both that the fossil record is not a reliable enough source of evidence for firm conclusions to be drawn from (O'Connell et al, 1999), and that the use of negative evidence is scientifically unsound (Turke, 1997). It has also been argued that because modern hunter-gatherer societies have life-expectancies well beyond the mean age at menopause means that the same was probably true for historical populations with similar lifestyles (Jamison et al, 2002; Pavelka and Fedigan, 1991; Turke, 1997). While all these arguments are valid, the fact remains that the 'modern-artifact' hypothesis can not be ruled out.

Hypotheses that post-generative longevity is a by-product of other adaptations are similarly difficult to refute. Wood et al (2002) argue that improved survival at younger ages has been selected for and that improved survival at elder ages is simply a by-product of this; a "fly-by" phenomenon. From this perspective there does not need to be any real benefit of post-generative longevity, there just has to be no real cost. It has also been proposed that female post-generative longevity is a by-product of selection for increased life span in males (Marlowe, 2000). Marlowe proposes that extended life span was selected for early in human evolutionary history but that the primary selection pressure was the improved status and mating opportunities that age affords men. Men achieve status with age, and reproductive opportunities with status. Thus a male can continue to increase his reproductive success into old age and increased longevity is beneficial. According to this hypothesis, female longevity is a by-product of selection for longevity in males but there has never been sufficient selective pressure on females to alter their reproductive life span.

The argument that an adaptationist explanation for the existence of menopause is not necessary rests on the assumption that changes to the female reproductive system, and the senescence of that system, is phylogenetically constrained. In all female

primates, the number of primary oocytes is established while the female fetus is still in utero and, unlike males, females are thereafter unable to develop more sex cells (Finch and Gosden, 1986). Throughout the female's reproductive life, oocytes are stimulated by a complex process of hormonal secretions and feedback loops to develop into ovums ready for fertilization. Most oocytes, however, die without being spent through ovulation and it is the rate of this process that both determines the length of the reproductive life span and regulates fertility throughout life (Finch and Gosden, 1986). The female reproductive system works as an interactive whole, rather than as related but separate functions. Therefore, to change the end point of this process, i.e. to select against menopause, would involve changing the whole system. Menopause occurs at the age predicted by phylogenetic-comparison with other primates (Alverez, 2000; Hawkes et al, 2000) yet death does not occur for many more years, perhaps as an artifact of civilization or perhaps as a by-product of selection for other adaptations. Either way, if neither menopause nor post-generative life span have been directly selected for then there is no reason to expect any significant long-term reproductive benefits to be associated with it.

However, it has also been argued that there is no clear reason why either the rate of oocyte depletion could not be slowed down or the initial stock of oocytes could not be increased with adequate selection pressure for longer reproductive life spans (Hawkes, 2002; Hill and Hurtado, 1991; Peccei, 2001; Shanley and Kirkwood, 2001). If this is true it suggests that menopause has been selected for some adaptive advantage it bestows. Or at least that there is no cost to menopause and no reason why it should have been selected against.

Williams (1957) proposed that it may have become advantageous at some point in history for females to stop dividing their energy between extant and potential

offspring and focus all remaining energy entirely on the extant. This followed from consideration of the increasing risk of maternal death with age, a risk that may be best avoided given the extreme altriciality of human infants. If a female dies during the birth of an infant, Williams supposed that the infant would also die and so would all other offspring still dependent on their mother. As such it may pay to cease reproduction at the point when the risk of maternal mortality (death caused by pregnancy or birth) reaches a certain threshold. This hypothesis has become known as the 'mothering' or 'stopping-early' hypothesis and has received wide support (Moss de Oliveira et al, 1999; Packer et al, 1998; Peccei, 1995; Shanley and Kirkwood, 2002).

However, as pointed out by Hill and Hurtado (1991), there are problems with using the increased risk of maternal death as an explanatory factor for menopause. Most maternal deaths (between 50 and 70%) are caused by sepsis and haemorrhage, both of which are consequences of the uterus weakening and becoming unable to contract fully following birth (Loudon 1992). In other words, most maternal deaths are consequences of reproductive senescence. If reproductive senescence, ending in menopause, had not been selected for in humans then it is possible that we would not observe such a steep increase in maternal mortality with maternal age because selection would have made the uterus stronger and more durable. Thus it could be seen that using the increasing likelihood of maternal death with age as an explanatory factor for reproductive senescence and menopause is somewhat circular.

Hawkes et al (1989, 1997, 1998, 2000) propose that female post-generative longevity has been selected for not only in order that females can ensure the survival of their offspring, but that they might also aid offspring's fertility and enhance grandoffspring survival. This follows from the observation that humans are cooperative breeders (Hawkes, 1997; Hrdy, 1999). Williams (1957) recognized that the extreme

alriciality of human infants means that mothers are essential for offspring survival in the first years of life. Hawkes et al argue that the extreme altriciality of infants has wider reaching effects than this; while infants are entirely dependent on their mothers, mothers are dependent on help from conspecifics (Hrdy, 1992). Father's are not expected, from an evolutionary point of view, to be the most dependable helpers given their lack of certainty regarding relatedness to offspring. Hawkes et al therefore propose that human females evolved longevity past the age of reproductive-senescence in order to help their daughters raise grandoffspring, an idea that has become known as 'the grandmother hypothesis'. Amongst the Hadza, for example, post-generative women are very productive in terms of foraging and Hawkes et al (1989) speculated that this is a strategy designed to reduce the workload of related kin who can then spend their effort on reproduction.

In support of this hypothesis, Shanley and Kirkwood (2002) showed that while the increased risks of maternal mortality with increasing age plus the altriciality of human infants is not great enough to account for the existence of menopause, if post-generative women also decrease their grandoffsprings' mortality by 10%, and increase their daughters' fertility by 10%, then women who experience menopause have greater reproductive fitness than those who do not.

It is important to note that the grandmother hypothesis as specified by Hawkes et al (1998, 2000) is not a hypothesis for the evolution of menopause but a hypothesis for continued longevity after menopause. Hawkes et al (2000) argue that age at reproductive senescence is in line with other primate life-histories and it is therefore post-generative longevity that is peculiar to humans and in need of explanation. Having said that, Hawkes (2002) does argue that it would be physiologically possible for reproductive life span to be expanded if there were sufficient selection pressure. This

argument is key because it makes subtle but important differences to what evidence could be considered supportive. If it is argued that human longevity is the given and menopause is an adaptation requiring explanation then it would be possible to refute the argument by demonstrating that women who cease reproduction at age 50 have lower reproductive success than hypothetical women who continue to reproduce until they die. Such evidence would not refute the hypothesis that menopause is a given and post-generative longevity is the peculiarity requiring explanation, however. To refute this hypothesis it would be necessary to show that women who provide grandmaternal care do not achieve significantly greater long-term reproductive success than women who do not provide grandmaternal care.

Results from studies of non-industrial societies provide mixed evidence for the grandmother hypothesis. Grandmothers seem to have only a very limited effect on the fertility of their daughters, but a significant influence on the survival of grandoffspring. Amongst the historical population of the Krummhörn in north-west Germany, the presence of the maternal grandmother is associated with increased parity progression ratios in only the largest families, but with decreased mortality risk of infants between 6 and 12 months of age in all families (Voland and Beise, 2002). This age is particularly significant as it is likely to be around the time that infants are weaned and thus there is a greater role for helpers other than the mother to play in caring for the infant. In contrast, paternal grandmothers double the risk of infant mortality during the first month of life. The authors interpreted this as being a consequence of stress imposed on the mother by the paternal grandmother.

That maternal and paternal grandparents have quite different effects on fertility and survival has been shown for other populations too. Amongst Gambian agriculturists, the presence of paternal mothers and fathers is associated with increased

parity-progression ratios while maternal grandparents have no effect on fertility (Sear et al, 2003). In contrast, maternal grandmothers do have significant positive effects on child health and survival, which paternal relatives (including fathers) do not (Sear et al, 2000, 2002). Similarly, maternal grandmothers had a positive (but non-significant) influence on child survival amongst a historical Japanese peasant village, while the presence of paternal grandmothers, and paternal and maternal grandfathers, were associated with increased risk of infant death (Jamison, 2002).

While these studies are very informative, they do not help to distinguish between the stopping-early and the grandmother hypotheses. Neither is it possible to know from such studies whether or not continued reproduction past the age that menopause occurs would result in greater reproductive success. In order to do this, modelling methods are required.

Hill and Hurtado (1991) compared the reproductive success of women who cease reproduction and spend the rest of their lives helping relatives reproduce, with the estimated reproductive success of the same women if they continued to reproduce past the age of menopause. The results suggest that a 50 year old Ache woman could achieve far greater long-term fitness through continuing direct reproduction than through ceasing reproduction and helping kin (Hill and Hurtado, 1999). Only by maximizing grandmother effects (the positive effect that the grandmother has for the survival of grandoffspring) to very high levels, can the cessation of direct reproduction with continued longevity be considered beneficial. This model included the increasing risk of maternal death with age but did not include the increasing risk of offspring death or non-viability with increasing maternal age. It is likely that if these extra costs to continued reproduction were considered then the benefits to ceasing reproduction would

appear larger, and the estimated benefits of continued reproduction would appear far smaller.

It is well established that the risk of unsuccessful pregnancies and post-natal death increases with maternal age, as does the risk of infants being born with congenital or non-congenital abnormalities (Astolfi and Zonta, 1999; Czeizel, 1988; Friede et al, 1988; Hollier et al, 2000; Lackmann et al, 1999; Loudon, 1992; Mostafa, 1991). These are both important variables that have not been considered to date with respect to the evolution of menopause and post-generative lifespan.

Almost certainly, the increasing risk of offspring death and offspring abnormalities are not independent of each other. The maternal screening hypothesis states that women vary over time in their "choosiness" of which fetuses to abort and which to carry. Younger women, with plenty of time and opportunity left to reproduce, will be choosier and more likely to abort fetuses as soon as there is some suspected abnormality. Older women, with less future opportunity, relax this screening system and as such are more likely to carry non-viable fetuses to term (Forbes, 1997). Thus elder women have more spontaneous abortions later in pregnancy and more abnormal births (Bernds and Barash, 1979; Forbes, 1997). It could be argued that when the likelihood of a conception being unsuccessful reaches a certain threshold it becomes preferable to expend all energies on extant offspring, just as when the likelihood of maternal death reaches a certain threshold it may be preferable to cease reproduction. This argument assumes that if menopause did not occur, yet women continued to live long past age 50 years, then the risk of offspring mortality would continue to increase with maternal age. It is possible, however, that the increased risk of unsuccessful pregnancy is a consequence, rather than a cause, of reproductive senescence. If menopause did not occur and females continued to live and to reproduce past the age of 50 years then the

pressure to reproduce would not increase so strongly with time and there would be less need for females to relax their screening process so early in life. It would still be necessary to relax the process with time as the probability of death would still increase with time, but the effect would not be so great at such young ages. Under these circumstances it would be much less likely that the benefits of ceasing reproduction to help kin reproduce would outweigh the benefits of direct reproduction.

The purpose of the current models is to test the stopping-early and grandmother hypotheses using the most recently available estimates of grandmother effects and including parameters that have not been considered previously. Namely, the increased risks of infant death and infants being born with congenital abnormalities with increasing maternal age. The hypotheses are tested separately as they apply to the existence of menopause and the existence of post-menopausal longevity.

Method

Three main models were created. In all models, age-specific fertility probabilities were based on that of the Ache, and mortality estimates were taken from model life-tables and altered according to maternal age-specific risks of offspring being non-viable, offspring mortality, and maternal mortality. The parameter values are described separately for each model below.

Data are from a mix of non-industrial populations. We are aware that mixing parameters of different populations may have pitfalls since life history parameters are interrelated. Nevertheless, we consider this approach preferable to using data from a single population for following reasons: Firstly, there does not exist complete and available data for an appropriate population. Secondly, no single population, from which most of the parameters could be extracted from, can be considered representative of human populations in the evolutionary past. Obviously, mixing characteristics from different populations does not necessarily provide a more realistic representatation of the evolutionary past, but neither is there any reason to assume that it provides a less realistic representation. Thirdly, by extracting parameter values from a variety of sources it is possible to use the best available data sources for each individual parameter.

All models are Monte Carlo simulations of 100,000 women's life histories, beginning at birth. For each life history, the age-specific probability of mortality is used to determine the woman's lifespan. For women who live until at least age 12, a random number generator is used in conjunction with the age-specific probabilities of various events, outlined below, to determine whether or not the woman gives birth, the infant is

viable, and the mother survives the birth. For every female infant that is born, the same process determines her life history. Four generations are simulated.

The measure of reproductive success in each model is the net replacement rate. This is calculated by dividing the number of great-granddaughters that survive to age 11 (one year before reproduction can begin) by the number of granddaughters that also do so. By comparing the net replacement rates of each model we can see which life history is best in terms of producing the most surviving descendants in following generations.

The first models simulate female life-histories as they are observed, with menopause, post-generative survival, and positive effects of grandmaternal presence on offspring survival. These models provide a control from which the later models can be compared.

In the second set of models, post-generative maternal and grandmaternal care are manipulated. In model 2a, menopause occurs and women continue to survive, but grandmother presence does not effect the survival probabilities of infants. Comparison of this model with model 1 provides a test of the grandmother hypothesis as it applies to the existence of post-generative longevity. In model 2b, there is no post-generative longevity and women die at the age at which they would have experienced menopause. This provides a test of the stopping-early hypothesis as it applies to the existence of post-generative longevity.

In the third set of models, menopause does not occur and females can continue to reproduce until age 100 (although it should be noted that the mortality schedule is not altered, so very few women actually survive to this age). Comparison of these models with model 1 provide tests of the hypotheses as they apply to the existence of menopause. In model 3a, the decline in fertility following its peak at age 34, and the

increase in maternal and infant mortality risks are estimated to be extreme (details below). Thus it is possible to estimate the reproductive success of women who do not experience menopause and for whom fertility declines steeply and the risks associated with birth increase steeply with age. In other words, this model assumes that menopause is avoidable (perhaps by an increase in the number of oocytes developed in utero), but that the rate at which the uterus wears out and chromosomal abnormalities increase could not possibly be any lower.

In model 3b the decline in estimated fertility and the increase in maternal and infant mortality are estimated to be far more gradual than in model 3a (details below). From this version of the model we can estimate the reproductive success of women who do not experience menopause and for whom the rate of reproductive senescence (the wearing out of the uterus and increase in chromosomal abnormalities) is slow. In this version of the model, it is assumed that if it were not the case that female fertility declines to zero by age 50 years, then neither would it be the case that maternal and infant mortality rates would increase so steeply preceding that age.

Model 1a and 1b (The control models)

Fertility data were from the Ache during the 'forest living' period, before the population came to live in a reservation (Hill and Hurtado, 1996, table 8.1). The Ache are a modern population of hunter-gatherers in Paraguay, characterized by relatively high fertility rates, particularly in later life. The age-specific fertility curve is shown in figure 1 (filled circles).

[Figure 1]

Age-specific mortality risks were taken from Model West for Females Level 8 (Coale and Demeny, 1983), shown in figure 2. Level 8 represents a medium-high level

of mortality where maximum lifespan is 100 years, mean life expectancy at birth is 37.50 years, and the mean life expectancy at age 50 (when menopause occurs) is 18.81 years. This level of mortality is the closest model to the mortality experienced by the Ache during the forest-living period (mean life expectancy at birth = 37.1 years, mean life expectancy at age 50 = 19.2 years).

[Figure 2]

The increase in mortality risk to an infant in its first year of life according to its mother's age was calculated by an event-history analysis using data from the Krummhörn region of Germany. All children born between 1720 and 1874 were included with the exception of those born to the wealthiest farmers (for full data description and methods see Voland and Beise, 2002). Results showed that the likelihood of an infant dying before reaching age 1 year decreased slightly between maternal age groups <20 years and 20-24 years but steadily increased thereafter (figure 3, solid line).

[Figure 3]

Offspring viability is considered separately for the sake of simplicity although in all models the effect of being non-viable is identical to death. To estimate the likelihood of offspring non-viability according to maternal age, only Down's syndrome is considered and the risk increases with both age and parity. It could be argued that excluding the increasing risks of other congenital abnormalities with maternal age is unfavorable to the grandmother hypothesis as it may result in an underestimate of the costs of reproduction at late ages. However, other congenital abnormalities which increase with maternal age, namely neural tube defects and inguinal hernia, do so at a far lesser rate than Down's syndrome. For example, the probability of offspring being

born with a nural tube defect increases by a factor of approximately 1.5 between women aged under 20 and over 40. In contrast, the probability of offspring being born with Down's syndrome increases by a factor of 96 over the same period (Cziezal, 1988). Consequently it is considered that, for the purposes of the current study, the inclusion of only Down's syndrome as a cause of non-viability is adequate and unlikely to significantly bias the results against the grandmother hypothesis. The probabilities of offspring being non-viable according to parity and maternal age are calculated from Kallen (1997).

Age-specific risk of maternal mortality was calculated from Ngom et al (1999). The data were from the Kassena-Nankana district in Ghana between 1983 and 1997 and provide the probability of death during childbirth for all women until age 49 (figure 4, solid line).

[Figure 4]

The increase in mortality risk to an infant in its first year of life according to the survival status of its maternal grandmother was calculated by event history analysis of historical data from the Krummhörn region (see Voland and Beise, 2002). Where women cease reproduction at age 50 but continue to live, the baseline risk of offspring dying by age 1 year (taken from the model life table) is decreased by 30% while the grandmother is alive. In cases where the mother dies within a year of the offspring being born then it is assumed that the offspring also dies if there is no grandmaternal help. If the mother dies while the offspring is under 1 year old but the grandmother is present and post-generative then it is assumed that the offspring suffers the same likelihood of death as when the grandmother dies but the mother is alive, i.e. the baseline level. This assumes that post-generative grandmothers can completely replace the role of the mother if she dies, which is a very favourable assumption for the grandmother

hypothesis. In reality this is very unlikely indeed that grandmothers could completely replace the role of the mother in the event of her death, particularly in the first few months while the offspring is still heavily dependent on breast milk. The assumption is removed in model 1b.

In model 1b, the baseline risk of infant mortality in the first year of life is doubled in cases where the mother dies but the grandmother is alive and post-generative (this still provides a significant grandmother effect, as with no grandmother the mortality probability for offspring when the mother dies is 1). Other than this, all parameter values are identical to model 1a.

Model 2a and 2b (Post-generative longevity tests)

In model 2a, grandmother effects are removed completely. If the mother dies while the offspring is under 1 year of age, the offspring also dies regardless of whether or not the grandmother is alive and post-generative. While the mother is alive, there is no added benefit to the grandmother being alive and post-generative also.

In model 2b, the mothering hypothesis is tested by raising the probability of death to 1 at age 50. This means that there is no post-generative lifespan as death occurs where menopause otherwise would have. This provides a test of the mothering hypothesis, as the only difference between this model and model 2a was the lack of maternal care provided to infants born to women at the end of their reproductive lives.

Model 3a and 3b (Menopause tests)

Model 3a was designed to simulate the life histories of women where menopause does not occur, women remain fertile for the entire life span and the risks associated with birth increase steeply with maternal age.

Fertility was estimated to decline by 70% between its peak at age 34 and age 80 (figure 1, crosses). This is similar to the decline of breathing capacity (Shock 1984). The increase in infant mortality with increasing maternal age was the same as in models 1 until age 50. To estimate the relative risks of infant mortality at maternal ages past 50 years, a polynomial curve was fitted to the existing data (figure 3, dotted line). Similarly, the increase in offspring non-viability with maternal age was the same as in model 1 until age 50. The probability of offspring being non-viable when born to a woman over age 35 years is approximately three times that of to women under age 35 years. Therefore, to estimate the probability of offspring being non-viable when born to women over age 55, the probabilities from age 35 were multiplied again by 3, and then again for women over 75 years (figure 5).

[Figure 5]

To estimate the risk of maternal mortality past age 50, a curve was fitted to the existing data and the observed pattern was assumed to continue such that maternal mortality risk rose steeply after age 50 years (figure 4, dotted line). If a mother died while an infant was under age 1 year, the infant also died. There is no possibility of grandmothers intervening because they are still reproductive.

Model 3b was the same as model 3a, but the parameters are altered so that reproductive functions senesce far more slowly. Fertility was estimated to decline by 40% between ages 34 and 80 years (figure 1, empty circles). This is similar to the decline of resting cardiac function (Shock, 1984).

The increasing risks of offspring mortality, offspring non-viability, and maternal mortality, were estimated to be very gradual by stretching the observed risks up until age 50 so that they reach age 100. This means that the highest probabilities of mortality

and non-viability that are observed in reality at age 50, occur instead at age 100. For example, the maximum maternal mortality risk of 0.06 observed for ages 45-50 amongst rural Ghanaian women was allocated to age group 95-100, and the risk of maternal mortality that is observed in Ghana at age 30-35 years (0.0157) was allocated to age category 50-52, and so on. An exponential curve was then fitted to this stretched data so that the maternal mortality risk at all ages could be calculated.

The probabilities of infant mortality, infant non-viability, and maternal mortality that are used in this model are illustrated in figures 6, 7 and 8.

[Figures 6, 7 and 8]

Results

The mean number of granddaughters and great-granddaughters that survive to age 11, and the net replacement rate that results from each model are shown in table 1. [Table 1]

The difference in net replacement rate between model 1a and 1b is very slight at only 0.003. This shows that whether the grandmother can replace the role of the mother in the event of her death completely, or can do so only partially, is not too important an issue. We compare the results of the other models with model 1b rather than 1a, however, because the assumption that grandmothers can replace the role of the mother only partially is considered to be more realistic.

When grandmothering effects are excluded altogether in model 2a, so that infant survival is not enhanced by the presence of the grandmother, the net replacement rate is reduced by 0.110, or 5 %, from model 1b which includes grandmother effects.

In order to assess the size of post-generative maternal care, we compare model 2b (no post-generative maternal or grandmaternal care) with model 2a (no grandmaternal care). The difference in the net replacement rate between these models is 0.005. This is 22 times smaller than the difference between model 1b (with maternal and grandmaternal care) and 2a (with post-generative maternal care, but no grandmaternal care). In other words, the effect of post-generative grandmaternal care is 22 times larger than the effect of post-generative maternal care on the net replacement rate.

Models 3a and 3b, which simulate the lives of women with completely hypothetical life histories where menopause does not occur and women reproduce for their entire life spans until a maximum age of 100 years, result in by far the highest

replacement rates: 4.259 for model 3a, and 4.844 for model 3b. This is 1.921 and 2.185 times as high as model 1b, respectively. Thus it is very clear that not experiencing menopause and continuing to reproduce until very late ages is by far the best life history in terms of numbers of descendents in following generations, despite the continually increasing risks of maternal death, infant death and infant non-viability.

Discussion

If it is considered that it is the existence of menopause that requires an explanation, then both the stopping-early and the grandmother hypothesis must be rejected on the basis of these results. Models 3a and 3b provided estimates of net replacement rates if menopause did not occur, and both result in far higher replacement rates than models 1a and 1b, where menopause does occur and post-generative women aid their offspring and grandoffspring's survival.

Model 3b resulted in a higher replacement rate than model 3a because fertility decreased at a slower rate and the probabilities of maternal and infant death were far smaller at later ages. But the fact that both models 3a and 3b resulted in far higher replacement rates than all other models shows that the issue regarding the likely rate of reproductive senescence under the hypothetical evolutionary scenario of menopause being selected against is not important. Whether the risks associated with maternity increase exponentially (model 3a), or very slowly (model 3b), the net replacement rate that could be achieved by such a life history far outweighs that achieved by the observed life history inclusive of menopause. Thus the increase in risks of child-bearing with increasing maternal age do not outweigh the potential benefits that could be accrued by continuing reproduction past age 50 years. As such these results contribute to the growing body of literature that refutes the 'stopping-early' or 'mothering' hypothesis as it was originally conceptualized by Williams (1957).

This does then raise the question, why is menopause not selected against? In addition to the physiological constraints against this discussed in the introduction, it must also be considered that the maximum number of offspring is not necessarily the optimal number of offspring. All organisms face a trade-off between the quality and

quantity of offspring, because for every unit of energy that is spent on offspring quantity, there is one less unit of energy expended on offspring quality (Levins, 1968; Smith and Fretwell, 1974; Stearns, 1992). In comparison to other species, humans produce few offspring of high quality. Thus we should not be surprised that amongst humans the maximum number of offspring is unlikely to ever have been the optimal number of offspring (Borgerhoff-Mulder, 2000; Kaplan, 1994; Mace, 1998, 2000). It is entirely plausible therefore, that menopause has not been selected against because there is no selection pressure to increase the number of offspring.

If it is considered that it is not the existence of menopause, but the continued longevity following menopause, that requires an explanation, then the results of this study have different implications for the stopping-early and the grandmother hypotheses.

The results show that the consequences of there being no post-generative longevity, such that late born offspring receive no maternal care, are very small. Female fertility is so low from age 40 years onwards that there are very few infants young enough to suffer mortality when their mother dies at age 50. If fertility is maintained at its peak level until age 50, the effect of there being no post-generative lifespan is far greater (results not shown). In contrast, the consequences of there being no grandmaternal care affects all grandoffspring from all daughters. As such, the effect of removing post-generative grandmaternal care from a population is far larger than the effect of removing post-generative maternal care.

That model 1b results in a net replacement rate only marginally less than that of model 1a shows that it is not the role of the grandmother in the event of the mothers death that accounts for the high replacement rate relative to model 2a (where there are no grandmother effects). Rather, it is the 30% reduction in mortality that grandmother

presence affords infants in the first year of their lives while mothers are also present, that results in women with post-generative life spans having greater long-term reproductive success than women without post-generative life spans.

The grandmother hypothesis as it was originally formulated (Hawkes et al, 1989), postulated that post-generative lifespan was selected for because post-generative women help their daughters increase fertility. The hypothesis was subsequently expanded to include the role that grandmothers have in reducing infant mortality. Indeed, the evidence from empirical studies suggests that grandmothers are more effective in reducing grandoffspring mortality than in increasing daughters' fertility (Voland and Beise, 2002; Sear et al, 2000, 2002, 2003). This is perhaps not suprising given the fact, as previously mentioned, that the maximum number of offspring has probably never been the optimal number of offspring. While there may never have been any pressure to increase numbers of offspring born, there is surely constant pressure to reduce offspring mortality and thus achieve the optimal number of offspring with minimal loss. The results of the present study suggest that this pressure may have been great enough to result in the evolution of post-generative longevity.

If the current models were altered such that elder infants suffered increased mortality risks in the event of their mother's and their grandmother's deaths, the negative consequences of there being no post-generative survival would be slightly larger. However, the purpose of these models was to provide conservative tests of the stopping-early and grandmother hypotheses, and given that the exceptional vulnerability of human infants is very much focused in their first year of life (in the mortality estimates used in the current models, mortality risk is reduced from 0.178 in the first year of life to 0.030 in the second year) it is considered that focusing the effect of maternal and grandmaternal deaths in the first year was appropriate. Furthermore, if the

effect of maternal and grandmaternal death affected elder as well as younger offspring, the ratio of the affects would not change; grandmaternal mortality would still effect more offspring that maternal mortality. As such the primary finding of this study, that the effect of post-generative grandmaternal care is greater than the effect of post-generative maternal care, would not be compromised.

There are some aspects of grandmaternal presence not included in the current models. Firstly, because only matrilines were considered, the negative effects that paternal grandmothers have on offspring survival (Jamison, 2002; Voland and Beise, 2002) were not included. Excluding the negative effects biases the models toward a favorable outcome for the grandmother hypothesis. On the other hand, there are likely to be other, beneficial effects of grandmother presence, not included in the models. Hawkes et al (1989) showed that post-generative Hadza women are particularly efficient foragers, providing the group with significantly more than their own share of food. Presumably, grandoffspring are beneficiaries of this food, to the betterment of their health and growth. This has yet to be shown for the Hadza, but it is true that the presence of a maternal grandmother is associated with improved growth and nutrition amongst the Gambian agriculturists (Sear et al, 2000). It is likely that this improvement in health and growth is transferred into improved fertility and survival later in life. If so, the effects may be large enough to provide further selection pressure for post-generative survival amongst human females.

In conclusion, if one accepts that it is post-generative life span, rather than menopause, that is the anomaly in human life history requiring explanation, the results of the current models provide support for the grandmother hypothesis, but little support for the stopping-early hypothesis.

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Table 1: Mean number of grandoffspring and great-grandoffspring that survive to age 11 in each model.

	surviving grandoffspring	surviving great-grandoffspring	net replacement rate
1a GM[1] can replace mother completely in event of death.	4.654	10.333	2.220
1b GM can replace mother only partially in event of death.	4.63	10.257	2.217
2a No GME[2]	4.441	9.419	2.107
2b No post-generative life	4.438	9.330	2.102
3a Fertility continues to age 100 with steep senescence	10.678	45.448	4.259
3b Fertility continues to age 100 with gradual senescence	18.492	89.580	4.844

[1] GM = grandmother

[2] GME = grandmother effect of reducing infant mortality risk

Figure legends

Figure 1. Age-specific fertility used in all models. Filled black circles show probabilities of birth used in all models until age 34, and models 1a, 1b, 2a and 2b thereafter. Crosses show probabilities of birth after age 34 used in model 3a. Empty circles show probabilities of birth after age 34 used in model 3b.

Figure 2: Age-specific mortality used in all models. Note that age is given in five year categories with the exception of the first year of life, and ages 1-5 years.

Figure 3. Risk of infant mortality used in model 3a. Diamond points joined by a solid line show the observed data. The dotted line shows the estimated values ($y = 0.0978x^2 - 0.789x + 2.25383$).

Figure 4. Age-specific probability of maternal mortality used in model 3a. The diamond shaped points joined by a solid line show the observed data, the dotted line shows the estimated values at later ages ($y = 2E{-}6x^{2.5699}$).

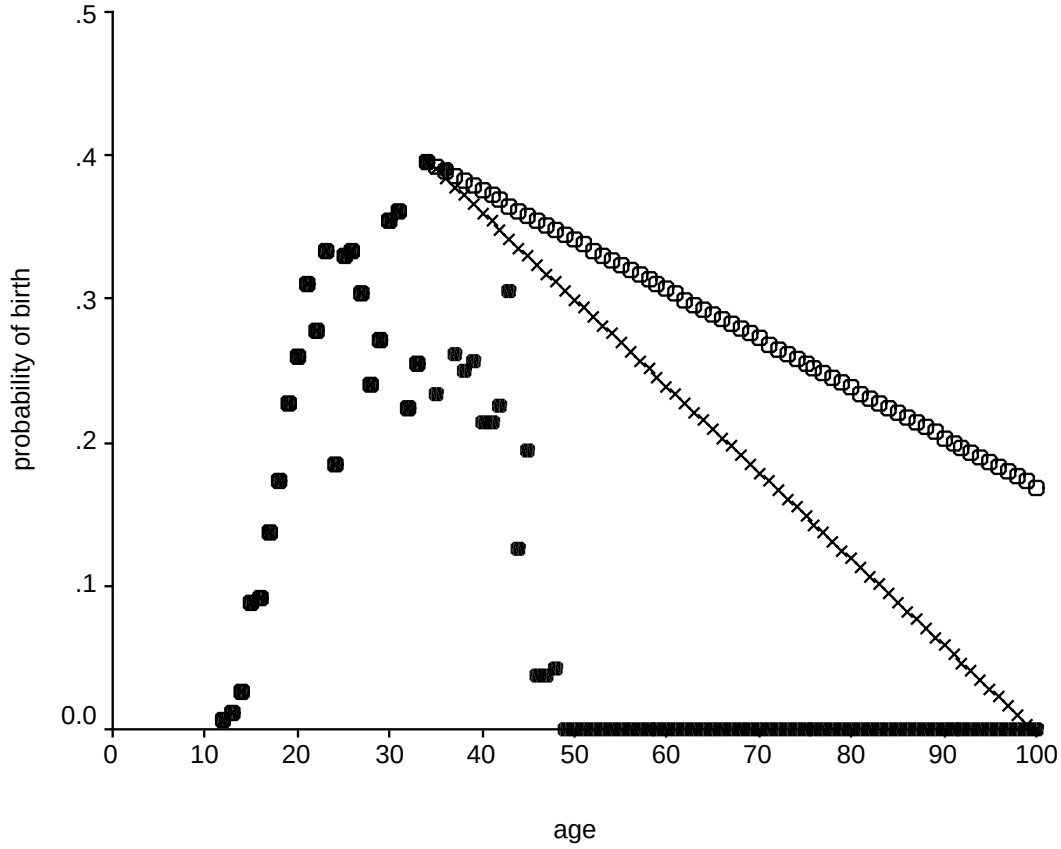
Figure 5. Maternal age and parity specific probability of Down's syndrome used in model 3a.

Figure 6. Estimated relative risk of infant mortality by maternal age used in model 3b.

Figure 7. Maternal age and parity specific probability of Down's syndrome used in model 3b. The black bars show the probabilities where maternal age is <35 years, the white bars 35-55 years, the striped bars 56-75 years, and the checked bars >75 years.

Figure 8. Age-specific maternal mortality probability used in model 3b. ($y = 0.0029e^{0.031x}$)

Fig. 1.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.3
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probability of birth
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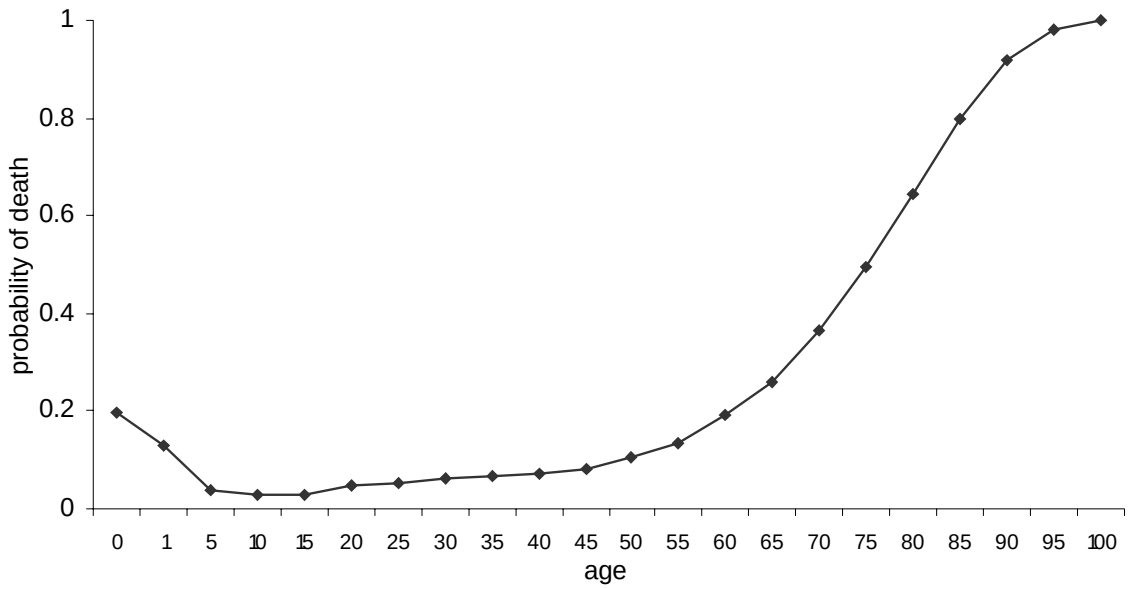
10
20
30
40
50
60
70
80
90
100
age

Fig. 2.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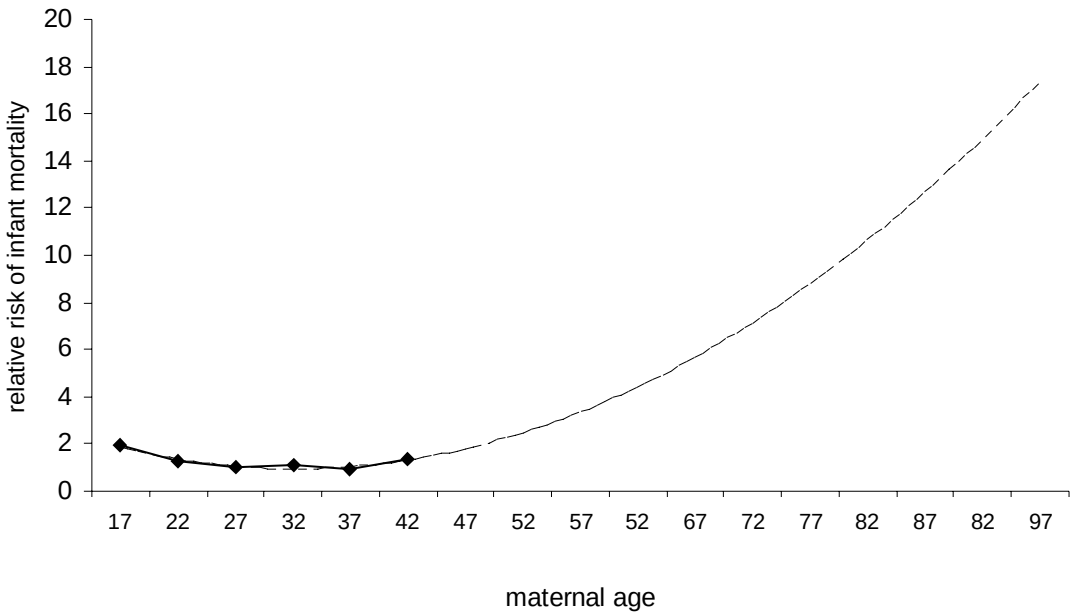
0.6
0.4
0.2
0
0 1 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100
age

Fig. 3.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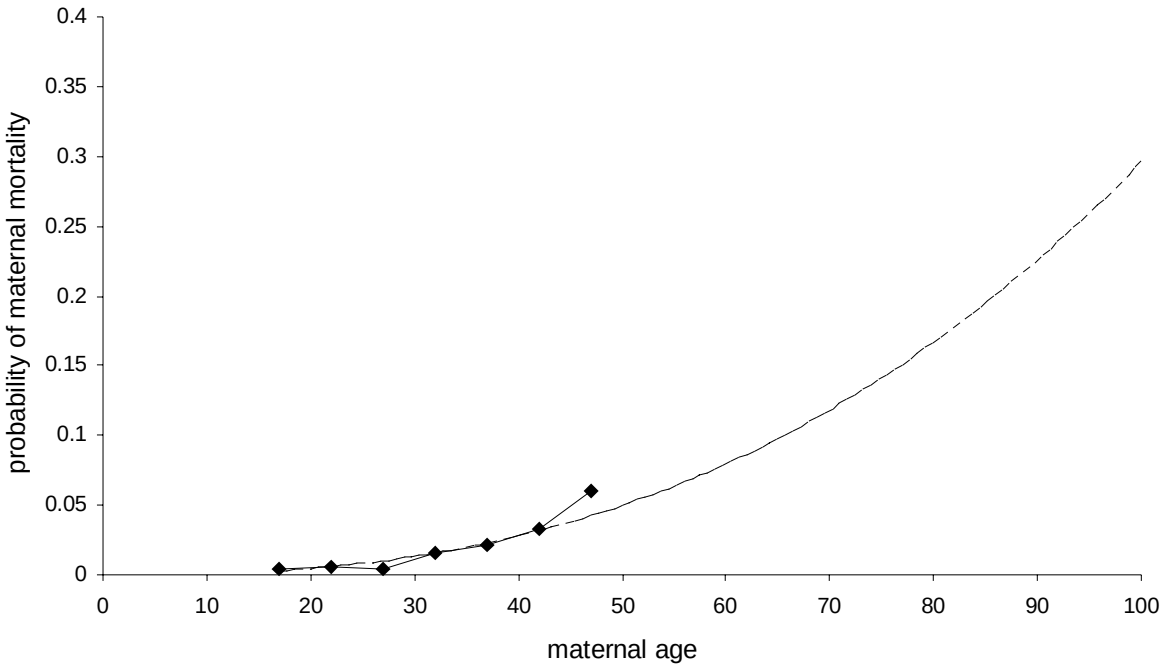
18
16
14
12
10
8
6
4
2
0
17
22
27
32
37
42
47
52
57
52
67
72
77
82
87
82
97
maternal age

Fig. 4.


probability of maternal mortality
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0.3
0.25
0.2
0.15
0.1
0.05
0
0
10
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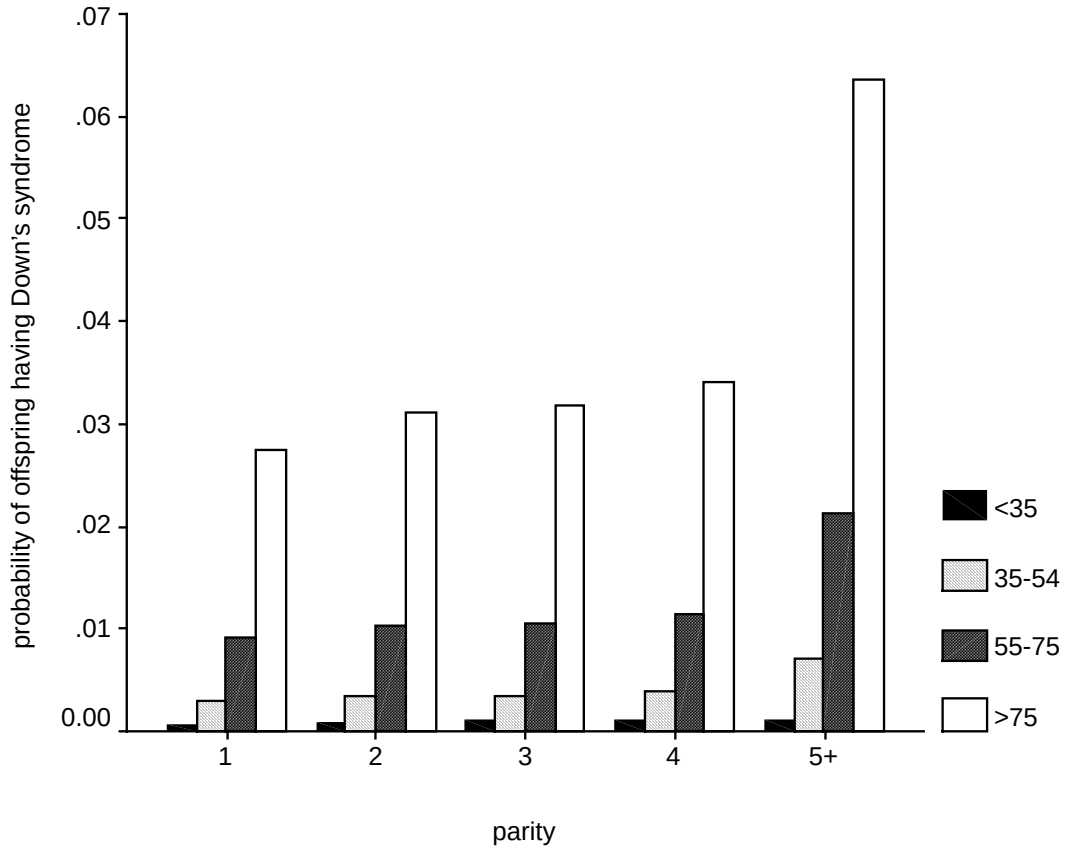
40
50
60
70
80
90
100
maternal age

Fig. 5.


.07
.06
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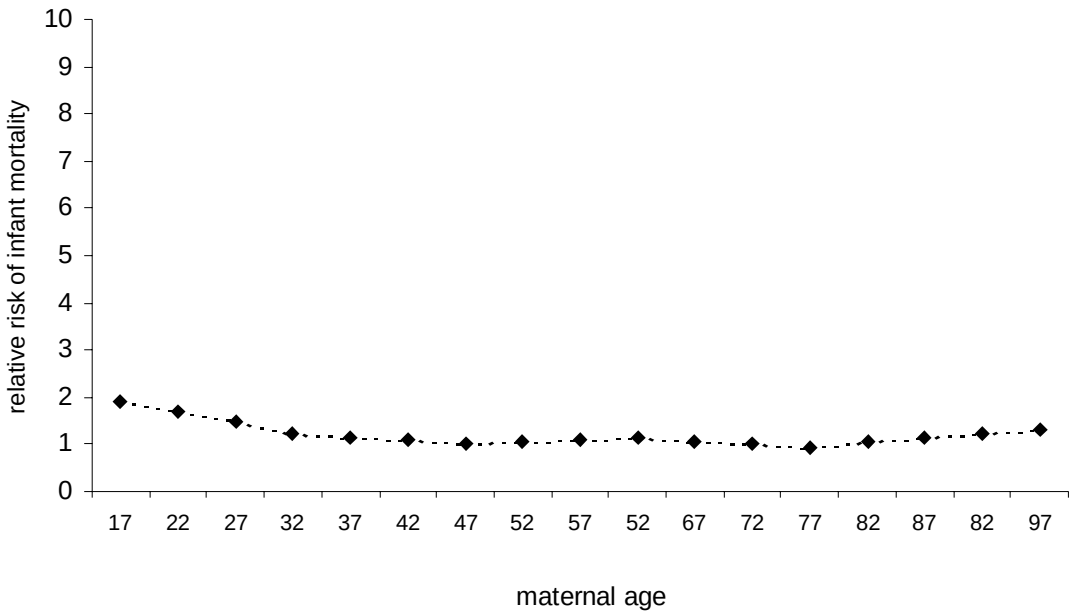
probability of offspring having Down's syndrome
1
2
3
4
5+
parity
<35
35-54
55-75
>75

Fig. 6


relative risk of infant mortality
10
9
8
7
6
5
4
3
2
1
0
17
22
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32
37
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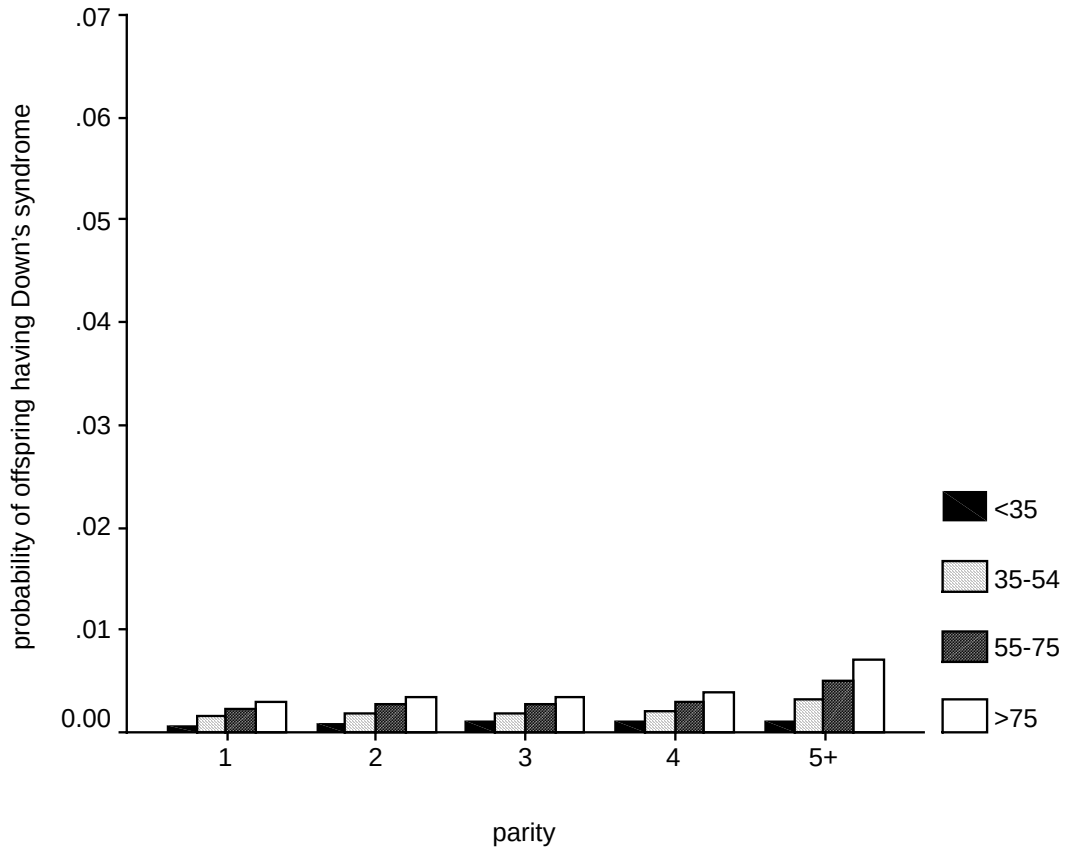
47
52
57
52
67
72
77
82
87
82
97
maternal age

Fig. 7.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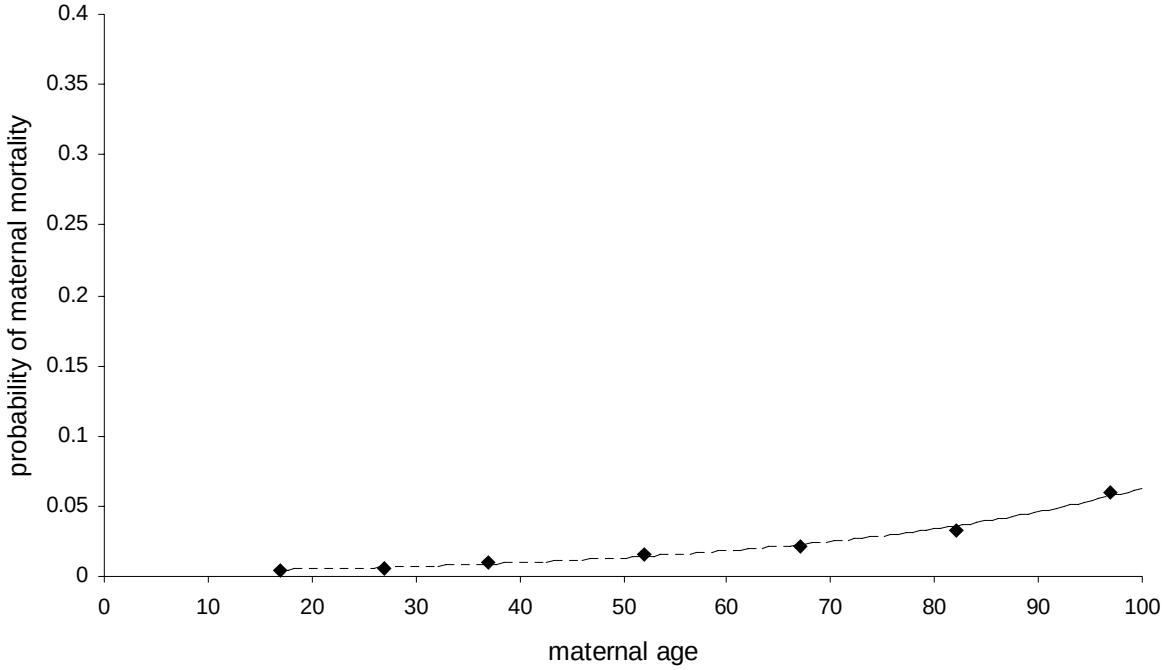
probability of offspring having Down's syndrome
1
2
3
4
5+
parity
<35
35-54
55-75
>75

Fig. 8.


probability of maternal mortality
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0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
0
10
20
30
40
50
60
70
80
90
100
maternal age