AMERICAN JOURNAL OF PHYSICAL ANTHROPOLOGY 132:632–641 (2007)

The Impact of Reproduction on Gambian Women: Does Controlling for Phenotypic Quality Reveal Costs of Reproduction?

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KEY WORDS life history; mortality; sub-Saharan Africa

ABSTRACT Life history theory predicts that where resources are limited, investment in reproduction will cause a decline in body condition and ultimately may lower survival rates. We investigate the relationship between reproduction and mortality in women in rural Gambia. We use a number of different measures of reproductive investment: the timing of reproduction, intensity of reproduction, and cumulative reproductive investment (parity). Though giving birth is clearly a risk factor for increased mortality, we find limited evidence that the timing, intensity, or cumulative effects of reproduction have a survival cost. Instead, there is some evidence that women who have invested heavily in reproduction have higher survival than women with lower reproductive investment: both high parity and late age at last reproduction are associated with high survival. The only evidence for any cost of reproduction is that women who have given birth to twins (considered a marker of heavy investment in reproduction) have higher mortality rates than other women, after the age of 50 years. A potential confounding factor may be differences in health between women: particularly healthy women may be able to invest substantially in both reproduction and their own survival, leading to the positive correlations between survival and both parity and age at last birth we observe. To control for differences in health between women, we reanalyze the relationship between reproduction and mortality but include variables correlating with health in our models (height, BMI, and hemoglobin). Even when controlling for health, the positive correlation between investment in reproduction and survival remains unchanged. Am J Phys Anthropol 132:632–641, 2007. ©2007 Wiley-Liss, Inc.

Life history theory predicts that where resources are limited, investment in reproduction will cause a decline in body condition and ultimately may lower survival rates (e.g. Reznick, 1985; Roff, 1992; Stearns, 1992). This is because energy allocated to reproduction cannot simultaneously be used for repair and maintenance of somatic (i.e. non-reproductive) tissues. Reproductive effort should therefore be negatively correlated with survival rates. It has been possible to demonstrate costs of reproduction using experimental manipulation of reproductive effort in non-human species. For example, some bird species show a decline in immune response if their brood size is artificially increased (Gustafsson, 1994; Moreno et al., 1999). This suggests a proximate mechanism through which reproductive effort could lead to higher mortality rates, and a correlation between increased reproductive effort and higher mortality has also been observed among bird species (Daan et al., 1996). However, identifying these costs is problematic in observational studies (the only option when studying our own species), because of variation in quality between individuals (e.g. Wendeln and Becker, 1999). Individuals of high quality (i.e. those in good health with access to relatively high levels of resources) may be able to invest relatively heavily in reproduction but still maintain good body condition, leading to a positive association between reproductive effort and survival.

Previous research on human populations on the relationship between reproductive effort and survival risks has given mixed results (the following discussion relates to women only: reproductive investment is easier to identify and measure in women than men, and consequently there is a larger literature on costs of reproduction in women rather than men). A recent thorough review of the evidence for a relationship between number of births and mortality risk in post-reproductive women found inconsistent results (Hurt et al., 2006). There was some evidence that mortality actually decreased with increasing parity in natural fertility populations. This suggests that individual variation in quality may be masking potential costs of reproduction and leading to a positive correlation between reproduction and survival. In contrast, there were indications that in contracepting populations women with many children did suffer higher mortality rates. These findings are somewhat counter-intuitive given that the costs of reproduction are predicted to be more obvious in populations with limited resources (and limited resources characterize most natural fertility populations), rather than well nourished contracepting populations. Overall, these authors concluded that the evidence for costs of reproduction was weak: there were few statistically significant results, and different methods of analysis resulted in inconsistent outcomes. In contrast, a population-level (rather than individual-level) analysis of the relationship between longevity and fertility came to the opposite conclusion. Thomas et al. (2000) conducted an analysis of 153

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Received 30 May 2006; accepted 30 November 2006

DOI 10.1002/ajpa.20558
Published online 1 February 2007 in Wiley InterScience (www.interscience.wiley.com).

populations worldwide, and found the negative correlation between fertility and longevity predicted by life history theory. However, as the authors themselves point out, socio-cultural factors (not controlled for in the model) have the potential to confound such cross-cultural analysis, making it difficult to be sure that their findings do indicate a cost of reproduction across populations.

The studies described above used the total number of children born as a useful measure of reproductive effort, but several studies have investigated the impact of other components of reproduction on mortality risk. A number of studies have found that women who have a relatively late age at last birth tend to have lower mortality risks in later life than those who finish reproducing earlier (Doblhammer, 2000; Muller et al., 2002; Smith et al., 2002; Dribe, 2004; Grundy and Tomassini, 2005; Helle et al., 2005). Again, this suggests that observational studies are more likely to find positive correlations between reproductive effort and survival, than any costs of reproduction. However, several studies have also shown that women who had a late start to reproduction also had lower mortality in later life compared to women who started reproduction earlier (Doblhammer, 2000; Korpelainen, 2000; Smith et al., 2002; Grundy and Tomassini, 2005). This may indicate that a strategy of delayed reproduction leads to the lowest mortality risks. The proximate mechanisms by which these relationships are brought about are not yet clear, though the greater longevity of women with late births may reflect slow ageing among these women. Slow ageing may, in turn, reflect better health (or higher quality) on the part of these women.

As well as the timing of reproduction, the intensity of reproduction may also be important. The intensity of reproductive effort is less well studied than either cumulative effort (parity) or scheduling of reproduction, but various authors have found that mortality rates are increased by short inter-birth intervals (Grundy and Tomassini, 2005), giving birth to twins (Helle et al., 2004) and to sons (Helle et al., 2002). All three are thought to be indicators of particularly intensive reproduction. However, as with parity, the relationship between these alternative components of reproductive effort (both timing and intensity) and mortality is not always clear-cut. It is always possible to find studies which observe no effects of these measures of reproductive investment on mortality risk: see, e.g., Dribe (2004), Helle et al. (2005) or Le Bourg et al. (1993) for age at first birth; Mueller (2004) for age at last birth; Beise and Voland (2002) for number of sons; and Menken et al. (2003) for the pace of reproduction. Overall, therefore, there is some evidence for costs of reproduction in women, but this is by no means universal, and several studies observe positive correlations between reproductive investment and mortality rather than evidence that reproduction is detrimental to survival chances.

Most of the previous work on the costs of reproduction in women has not been able to control for variation in quality between individuals, perhaps providing an explanation for why costs of reproduction have been so hard to demonstrate consistently. An exception is provided by Doblhammer and Oeppen (2003), who attempted to control for individual variation between women using a new statistical technique (simultaneous equation modeling): this study did reveal costs of reproduction in women but not men in a historical British population using this method. The aim of the study described here is to test the hypothesis that investment in reproduction increases the risk of death for in women in a natural fertility population, controlling for individual variation in quality between women. The impact of all components of reproductive effort on women's mortality risk in rural Gambia is explored: the intensity of reproduction, timing, and cumulative reproductive investment. The analysis attempts to control for individual differences in quality between women by including variables for anthropometric status (height and body mass index: BMI) and anemia (hemoglobin level) in the statistical models.

Quality refers to a suite of traits which affect an individual's fitness. A large component of quality will be access to energetic resources, but quality will also encompass other characteristics, such as the ability to fight off disease [which will be related to energetic reserves, since individuals with greater energetic reserves will be able to devote more energy towards immune defense, but also has a genetic component, e.g., certain human genotypes are known to be more efficient at providing protection against malaria than others (Hill et al., 1991)]. A measure of overall health (if health is defined as the absence of disease[1]) may therefore be a better indicator of quality than simply a measure of energetic reserves, though the two will be correlated. Even health may not wholly capture a woman's quality, since other factors are likely to affect her fitness, such as factors which affect the ability to acquire mates and successfully conceive. Health, however, is likely to be large part of a woman's quality, and has the advantage of being relatively easy to measure and control for in the statistical models used here.

We consider the three measures (height, BMI, and hemoglobin) we include as control variables in our models to be good indicators of a woman's health, and should therefore indicate her quality. Height is frequently used as a measure of a population's health, as it is affected by both energetic availability and prevalence of infectious disease during childhood (Silventoinen, 2003). Height is therefore a measure of long-term health and energy availability. Height has already been shown to correlate with some components of fitness in this Gambian community, such as child mortality, suggesting it may be a measure of the quality of a woman (Sear et al., 2004). BMI and hemoglobin level are short-term measures of energy availability and health. BMI should indicate available energetic reserves (Ferro-Luzzi et al., 1992). Hemoglobin is affected by both nutritional status and disease load, and may be a better indicator of overall health than BMI (Wadsworth, 1992). Infections with both malaria and parasitic worms have been correlated with low hemoglobin levels (Gilgen and Mascie-Taylor, 2001; Olsen et al., 1998); and low hemoglobin has been correlated with both adverse reproductive and economic outcomes, such as low birth weight (Allen, 2000) and reduced labor productivity (Gilgen et al., 2001). Relatively tall, well nourished women with high hemoglobin levels should therefore be those with relatively plentiful access to resources who are successful at resisting disease, or women of high phenotypic quality. Though it is recognized that these measures of health may not necessarily capture entirely a woman's quality, these measures are thought to be good proxies, and will be referred to as indicators of quality throughout this paper. By including variables which correlate with individual quality, trade-offs between reproduction and survival may be revealed, which would otherwise be masked by differences in quality between women.

[1]With apologies to the WHO, which defines health as "a state of complete physical, mental, and social well-being, and not merely the absence of disease or infirmity" (WHO 1946).

DATA

The data were obtained from four villages in rural Gambia. The UK Medical Research Council has been funding research in this region of the Gambia since 1950. Research in this area was established by Ian McGregor, who set up a demographic surveillance system in 1950, which continues to operate today (see McGregor 1991 for a full description of the study and study site). McGregor also systematically collected anthropometric data from these villagers, collecting data on height, weight, and hemoglobin level from all villagers during anthropometric surveys, which were conducted at least annually between 1950 and 1980. McGregor (a medical doctor) offered villagers medical treatment as necessary during his visits to the area, and in the early years of his study attempted some village-wide treatments to eradicate certain parasitic infections. Overall, however, this population was without systematic access to medical care and contraception until 1975, when the MRC set up a permanent medical clinic in one of the villages, as part of a permanent research station which continues to operate in the village today. This clinic had a significant impact on the demography of these villages: child mortality dropped rapidly, and fertility also declined, although at a slower rate than mortality (Lamb et al., 1984; Weaver and Beckerleg, 1993; Sear, 2001).

This analysis was confined to the period between 1950 and 1980, during the period when anthropometric data was collected systematically and when the population was largely a natural fertility population. Adult mortality patterns and fertility were slower to change than child mortality due to the influence of the clinic, and so including 5 years of data after the clinic was established is unlikely to affect the results substantially. During most of this period, both fertility and child mortality were high: women gave birth to around 7 children on average, but more than 40% of these children died before the age of 5 years (Billewicz and McGregor, 1981). The economy of these villages was largely based on subsistence agriculture. Women were responsible for a substantial proportion of the subsistence farming; men also contributed to this subsistence work and did a little cash-cropping of groundnuts. It was a very seasonal environment. Individuals tended to lose weight during the rainy season, when high workloads were combined with food scarcity and high disease loads, but gained weight again during the dry season (McGregor, 1976). Adults were not well nourished by Western standards, but the majority of individuals had BMI measurements within the range that the WHO considers to indicate adequate nutritional status. Average BMI for adults of both sexes was $\sim$20. More than 80% of women had an average BMI between 18.5 and 24.9 (considered to indicate adequate nutritional status); 13% of women were malnourished (BMI $<$ 18.5), but very few were overweight (only 4% had an average BMI of 25 or greater). Although the majority of women were within the range of BMI considered adequate by international standards women were, on average, anemic. The mean hemoglobin concentration for women was 11.8 g/dl: a value of less than 12 g/dl is considered to indicate anemia. Most of these anemic women were mildly (10–11.9 g/dl, 38% of all women) or moderately anemic (8–9.9 g/dl, 11%); only 1.9% had a hemoglobin level less than 8 g/dl, the cut-off for severe anemia.

METHODS

The aim of this research was to test the hypothesis that reproductive investment increases adult female mortality risk. Discrete-time event-history analysis (EHA) was used to analyze the probability of an adult woman dying. EHA analyses the probability of an event, in this case a death, happening over time (in practice, a discrete-time EHA is simply a logistic regression with time entered as a covariate. Here, the binary outcome variable is dead/alive and the time covariate is the woman's age). This technique has the dual advantages of being able to include both censored cases and time-dependent variables, such as parity (Allison, 1984; Singer and Willett, 2003). The sample included all women who survived to at least age 15 by 1980. All births have been systematically recorded in these villages since 1950. Attempts were made to reconstruct birth histories for women who began their reproductive careers before 1950, but the fertility histories of older women are unlikely to be complete. The impact of reproductive investment may also differ in younger women, who are still experiencing reproductive events, and older women, whose physiological investment in reproduction has ended (though they are likely to be still caring for children and grandchildren). We therefore divided the sample into two groups: reproductive-aged women (defined as those aged between 15 and 49) and post-reproductive women (aged 50 and older). For the younger sample, we excluded any women born before 1920, in order to include only women with the most accurate birth histories. We also excluded childless women from both samples. In a strongly pro-natal society such as this, where all women marry and contraception is not used, childless women are likely to be those who are physiologically incapable of reproducing and therefore a rather unusual group of women.

Women were included in the analysis from the age of 15 (for the younger sample) or from age 50 (for the older sample) until they died, or were censored. All women with no recorded date of death were given a right-censoring date of either the last occasion they were reported to have been alive in the population, or in 1980 if they were known to have survived beyond 1980. Additionally, for the younger sample, women who were known to have survived until the age of 49 were censored at that age. All women who reached the age of 15 before 1950 were left-censored at the age they were in 1950. All models controlled for both village and birth cohort, as there were slight differences between villages and cohorts in mortality rates.

Reproductive-aged women

We ran two models of the effects of reproduction on the probability of dying for reproductive-aged women. The first model tested whether direct measures of reproductive investment affected her mortality risk, and included only women who ultimately gave birth at least once during their reproductive lives. The two measures of reproductive investment we included in this model were whether the woman gave birth in a particular year, and her parity, a cumulative measure of reproductive investment. Both variables were coded as time-varying covariates. We use a non-standard definition of parity in this analysis. Parity was defined as the number of deliveries a women had experienced (i.e., twins were counted as a single birth). Strictly speaking, parity should only include livebirths but we included stillbirths in the number of deliveries because, physiologically, a stillbirth is little different from a neonatal death. We therefore use the term "parity" in this analysis as a convenient shorthand for "number of deliveries, including stillbirths." Dummy variables were used to indicate parity, in order to test for a nonlinear relationship between parity and risk of dying.

TABLE 1. Descriptive statistics for reproductive variables in the reproductive-aged women models

	All women	Women of parity 2 or greater
Birth year		
Yes	28	29
No	72	71
Parity		
1–2	40	24
3–4	26	34
5–6	18	23
7–8	10	12
9+	6	7
Age at first birth		
<18		31
19–20		30
21+		33
Missing		6
Woman has ever given birth to twins		
Yes		4
No		96
Last birth was		
Male		46
Female		48
Missing		6
Previous interbirth interval		
<3 yrs		55
>3 yrs		35
Missing		10

Figures show the percentage of cases in each category.

TABLE 2. Descriptive statistics for reproductive variables in the post-reproductive models

	All women	Women of parity 2 or greater
Completed fertility		
Below average	34	
Average	35	
Above average	31	
Age at first birth		
Younger than average		35
Average		37
Older than average		28
Age at last birth		
Younger than average		22
Average		37
Older than average		40
Woman has ever given birth to twins		
Yes		2
No		98
Proportion of sons		
<0.5		40
0.5		19
>0.5		41
Pace of reproduction		
Less than average		30
Average		32
Greater than average		38

Figures show the percentage of cases in each category.

The second model, as well as including the two direct measures of reproductive investment described earlier, also tested whether measures of reproductive timing and intensity affected a woman's risk of dying. This model included only women who had had at least two births. Age at first birth was included as a time-constant measure of reproductive timing. Three variables of intensity were included: whether a woman had ever given birth to twins, whether her previous birth had been a son, and the length of the interbirth interval between her two preceding births (this last variable necessitated restricting the second model to women who had had at least two births). All intensity variables were time-varying. Early first births, twins, sons and short interbirth intervals are all assumed to indicate relatively heavy investment in reproduction. Table 1 shows descriptive statistics for the variables used in both models.

Post-reproductive women

Again, we ran two models of the probability of dying for post-reproductive women: one including a direct measure of reproductive investment (completed fertility), and the second including measures of the timing and intensity of reproduction. The first model included all parous women aged 50 and older, and tested whether a measure of cumulative reproductive investment affected mortality risk. The measure of cumulative reproductive investment used was completed fertility, which has the same definition of parity described earlier for reproductive-aged women, but is used only for those women who have completed childbearing. Because some of these older women do not have complete fertility histories, instead of using absolute family size we constructed a relative measure of family size. There are differences in the total number of births reported by women according to their birth cohort: older women report smaller completed family sizes than younger women (suggesting they have incomplete birth histories). We therefore calculated whether women had below average, average, or above average completed fertility for her birth cohort, by dividing each 10-year birth cohort of women into three roughly equal groups according to their completed family size. Dummy variables for below average and above average parity were then included in the model.

The second model was restricted to those women who had had at least 2 births, and tested for the effects of timing and intensity of reproduction (again, including a variable for the length of birth intervals necessitated restricting the model to those women who had had 2 or more children). For the timing of reproduction, relative measures of age at first and last birth were used, rather than absolute age at first and last birth. For each birth cohort, women were coded into three categories of age at first birth: younger than average age, average age, and above average age at first birth. Age at last birth was similarly divided into three groups by birth cohort. Dummy variables for below and above average age at first birth, and for below and above average age at last birth were then included in the model. Three measures of reproductive intensity were included in the model: whether the woman had ever given birth to twins, the proportion of her children that were male, and her pace of reproduction. Pace of reproduction was calculated by dividing a women's reproductive span (the difference between her ages at first and last births) by her total number of deliveries. This was then coded as a relative measure by dividing women into birth cohorts and assigning women a status of below average, average, or above average pace of reproduction for her birth cohort. Table 2 shows descriptive statistics for the variables used in both models.

Controlling for phenotypic quality

As discussed in the Introduction, a number of studies have found it difficult to find convincing evidence for costs

TABLE 3. Results of the event history analysis modeling the relationship between reproductive investment and mortality risk in parous reproductive-aged women[a]

	Model I			Model II		
Variable	Parameter estimate	SE	Odds ratio	Parameter estimate	SE	Odds ratio
Constant	−6.87	0.69*		9.30	3.68*	
Age	0.08	0.02**	1.08	0.07	0.02**	1.07
Birth year						
No (reference)			1.00			1.00
Yes	0.97	0.24**	2.61	0.79	0.29**	2.20
Parity						
1–2 (reference)			1.00			1.00
3–4	−0.73	0.27**	0.48	−0.45	0.30	0.64
5–6	−1.25	0.35**	0.29	−0.86	0.39*	0.42
7–8	−1.88	0.51**	0.15	−1.42	0.53**	0.24
9+	−1.30	0.50*	0.27	−0.64	0.54	0.53
Height				−0.06	0.02**	0.94
BMI				−0.17	0.06**	0.84
Hemoglobin				−0.27	0.07**	0.76
Number of women	973			924		
Deaths	90			75		

*$P < 0.05$.
**$P < 0.01$.
[a] All models control for village and birth cohort.

of reproduction, perhaps because women vary in phenotypic quality. If women who are high quality are able to both produce many children and maintain good body condition, then a positive, rather than the predicted negative, correlation may be seen between reproductive effort and mortality. To attempt to control for variation in quality between women, we ran each model in two versions. The first version included only the reproductive measures described earlier (with controls for village and birth cohort). The second included the reproductive measures and also three variables which were thought to indicate a woman's phenotypic quality or health status: height (in cm), BMI (weight/height^2), and hemoglobin level (in g/dl).

BMI and hemoglobin were included in the models as time-varying covariates. Few individuals were surveyed in every year between 1950 and 1980, and so a mean BMI or hemoglobin measurement was calculated for each individual for 5-year age blocks (for the ages 21–24, 25–29, 30–34, etc., up to the age groups 70–74, 75 and above), assuming the individual had more than one measurement in the 5-year age block. These mean BMI and hemoglobin measurements were then entered into the model as time-varying in 5-year age blocks. If no measurements were taken in a particular age block, the mean of the 2 measurements in the immediately younger and older age blocks was calculated and included in the model for the age block with missing data. If no measurements had been taken in the older age block then the value of the younger age block was entered into the block with missing data. BMI and hemoglobin measurements taken during pregnancy were excluded, as were measurements taken within 3 months after a birth for the hemoglobin measure (hemoglobin declines during pregnancy and takes a few months after birth to return to pre-pregnancy levels).

Height is clearly less variable with age than either BMI or hemoglobin, though does show a decline in older adults. Height was therefore included as time-constant until the age of 49 years and time-varying for older individuals. A mean height was calculated for each individual using all measurements collected between the ages of 15 and 49, and this measurement was included as the individual's height for ages under 50 years. From the age of 50 onwards, height was included as a time-varying covariate.

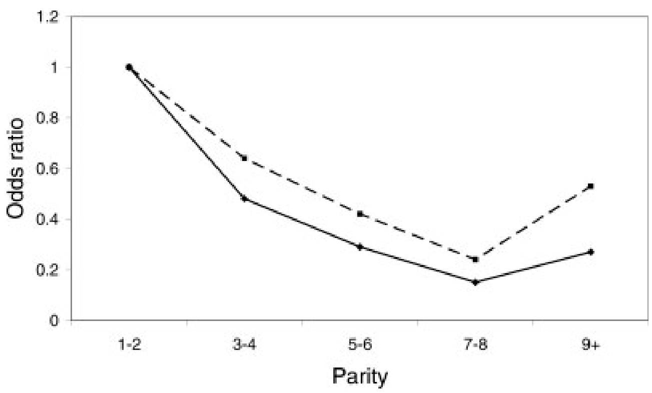

Fig. 1. Odds ratios for the probability of dying by parity, for reproductive-aged women (solid line shows results of model with only reproductive variables, dotted line the same model but including controls for health).

These time-varying height measures were constructed using the same method as for BMI and hemoglobin.

RESULTS

Reproductive-aged women

Table 3 shows the results of the first model for reproductive-aged women. The odds ratios (OR) can be interpreted as the increase (if the OR > 1) or decrease (if OR < 1) in the odds of the event happening for individuals in a particular category compared to those in a reference category. For continuous variables, the OR is the change in the odds of the event happening for a single unit change in the predictor variable, e.g., the OR for age is 1.08, which means that for every year a woman ages, her odds of death increase by 8%. The results presented in this table show that, for these women, giving birth is clearly a risk factor for increased mortality: women who give birth in a particular year have higher odds of dying than those who had not given birth in that year. Parity is also correlated with mortality risk, but we do not see the linear positive relationship that might be expected if costs of reproduction

TABLE 4. Results of the event history analysis modeling the relationship between reproductive investment and mortality risk in reproductive-aged women of parity 2 or greater

	Model I			Model II		
Variable	Parameter estimate	SE	Odds ratio	Parameter estimate	SE	Odds ratio
Constant	−7.10	0.91**		4.13	4.28	
Age	0.08	0.02**	1.08	0.08	0.03**	1.08
Birth year						
No (reference)			1.00			1.00
Yes	0.89	0.28**	2.43	0.86	0.30**	2.37
Parity						
2 (reference)			1.00			1.00
3–4	−0.72	0.31*	0.49	−0.56	0.35	0.57
5–6	−1.21	0.40**	0.30	−1.01	0.45*	0.36
7–8	−1.86	0.56**	0.16	−1.59	0.60**	0.20
9+	−1.32	0.57*	0.27	−0.90	0.62	0.41
Age at first birth						
<18	−0.15	0.39	0.86	−0.08	0.40	0.92
18–19 (reference)			1.00			1.00
21+	0.31	0.32	1.36	0.13	0.34	1.14
Ever given birth to twins						
No (reference)			1.00			1.00
Yes	0.35	0.55	1.43	0.45	0.56	1.57
Sex of most recent birth						
Female (reference)			1.00			1.00
Male	0.03	0.25	1.03	0.16	0.27	1.17
Length of preceding birth interval	−0.01	0.01	0.99	−0.01	0.01	0.99
Height				−0.03	0.02	0.97
BMI				−0.17	0.07*	0.84
Hemoglobin				−0.33	0.08**	0.71
Number of women		808			786	
Deaths		68			60	

* $P < 0.05$.
** $P < 0.01$.

were in evidence. There is a broadly negative relationship between parity and mortality risk. Women of parity 1 or 2 have significantly higher odds of dying than women of all higher parities. Inspection of the odds ratios suggests this negative relationship is reverse J-shaped rather than strictly linear (see Fig. 1). The odds of dying decrease from parity 1 to 2 up to parity 7 to 8 but then increase for women of very high parity (9 and above). Controlling for health variables appears to make little difference to the relationship between parity and risk of dying (Model II in Table 3), although as might be expected mortality risk is reduced with higher anthropometric status and higher hemoglobin levels [see Sear, (Size, body condition and adult mortality in rural Gambia: a life history perspective, submitted) for a full discussion of the relationship between health and adult mortality in this population]. Figure 1 shows the odds ratios calculated for each parity group for both Model I (without controlling for health) and Model II (controlling for health). This figure shows there may be some slight attenuation of the effects of parity when health is controlled for (as the odds ratios are slightly closer to 1), particularly at the highest parities. But overall this effect is rather small: the relationship between risk of death and parity is similar whether health is controlled for or not.

Table 4 shows the results of the second EHA model for reproductive-aged women, including only women of party 2 and higher. Again, we see that giving birth is a risk factor for dying, and that there is a reverse J-shaped relationship between parity and the risk of death. However, no other measure of reproductive investment appears to affect mortality rate: there is no correlation between age at first birth and the probability of dying, nor are mortality risks affected by whether the women had recently given birth to twins, to a son, or had recently had a short interbirth interval. Again, controlling for health makes little difference to the results.

Post-reproductive women

For post-reproductive women, the evidence for any costs of reproduction is similarly inconclusive. In the first model, including all parous women over the age of 50, there is little evidence that completed family size (relative to birth cohort) has any effect on the risks of dying (Table 5). Including health variables in the model makes no difference to the results: the odds ratios are virtually identical when health variables are included in the model. In the second model, including post-reproductive women who had had at least two births, two reproductive variables appear to be significantly related to mortality risk (Table 6). Women who gave birth at a relatively late age have lower odds of mortality than those who stopped reproducing earlier, again suggesting a negative relationship between reproductive investment and the risk of mortality. However, mothers of twins did have significantly higher odds of mortality than women who had only given birth to singletons. Once more, controlling for health made no difference to the results.

DISCUSSION

This analysis has shown that while giving birth is clearly a risk factor for mortality, there is little evidence that reproduction is costly in the long-term in this population. For reproductive-aged women the association between parity and mortality risk is actually negative, with women of higher parity having lower mortality risks than those of

TABLE 5. Results of the event history analysis modeling the relationship between reproductive investment and mortality risk in parous post-reproductive women

	Model I			Model II		
Variable	Parameter estimate	SE	Odds ratio	Parameter estimate	SE	Odds ratio
Constant	−8.29	0.83**		−1.14	3.80	
Age	0.07	0.01**	1.08	0.07	0.01**	1.08
Completed fertility						
Below average	−0.28	0.27	0.76	−0.27	0.30	0.76
Average (ref)			1.00			1.00
Above average	−0.44	0.26	0.65	−0.47	0.29	0.63
Height				−0.02	0.02	0.98
BMI				−0.09	0.05	0.92
Hemoglobin				−0.21	0.09*	0.81
Number of women	317			306		
Deaths	93			80		

* $P < 0.05$.
** $P < 0.01$.

TABLE 6. Results of the event history analysis modeling the relationship between reproductive investment and mortality risk in post-reproductive women of parity 2 or greater

	Model I			Model II		
Variable	Parameter estimate	SE	Odds ratio	Parameter estimate	SE	Odds ratio
Constant	−8.67	0.93**		−1.33	4.76	
Age	0.07	0.01**	1.08	0.08	0.02**	1.08
Age at first birth						
Below average	0.07	0.29	1.08	0.45	0.32	1.56
Average (ref)			1.00			1.00
Above average	0.11	0.32	1.11	0.18	0.37	1.19
Age at last birth						
Below average	0.05	0.29	1.05	−0.15	0.33	0.86
Average (ref)			1.00			1.00
Above average	−0.82	0.31**	0.44	−1.08	0.36**	0.34
Twin mother						
No (ref)			1.00			1.00
Yes	2.26	0.67**	9.61	2.29	0.55*	3.63
Proportion male	−0.02	0.49	0.98	−0.41	0.56	1.51
Pace of reproduction						
Below average	0.30	0.31	1.36	0.34	0.37	1.40
Average (ref)			1.00			1.00
Above average	−0.10	0.31	0.91	0.12	0.36	1.13
Height				−0.02	0.02	0.98
BMI				−0.06	0.06	0.94
Hemoglobin				−0.32	0.10**	0.72
Number of women	275			265		
Deaths	78			67		

* $P < 0.05$.
** $P < 0.01$.

lower parity. Women of very high parity may suffer slightly higher mortality risks than those of medium parity, but this effect is small. In post-reproductive women, completed fertility is not related to mortality, but age at last birth does show an association with mortality risk: women with later last births have lower mortality risks after the age of 50 than those with earlier last births. This observation replicates the findings of several similar studies (Doblhammer, 2000; Muller et al., 2002; Smith et al., 2002; Dribe, 2004; Grundy and Tomassini, 2005; Helle et al., 2005). It may be that this association results from the correlation of reproductive senescence and somatic senescence: women who can achieve late last births are those who age slowly and are therefore relatively long-lived. This suggestion is supported by the finding that late age at menopause has also been linked with higher post-menopausal survival (Ossewaarde et al., 2005). These findings that women who have invested heavily in reproduction actually have relatively low mortality risks suggest that there are indeed differences in quality between women in this population: women of high quality can invest in both reproduction and somatic maintenance, thereby lowering their mortality rates.

The only women who appear to suffer costs of reproduction, in terms of higher mortality rates, are mothers of twins. After the age of 50, women who have given birth to twins have significantly higher mortality rates than those who have only given birth to singletons. We have previously shown that twin mothers in this population are particularly heavy investors in reproduction (Sear et al., 2001). Not only is a twin birth energetically costly in itself, but twin mothers also have shorter birth-intervals and a larger total number of births than mothers of singletons. Twin mothers on average had 10 deliveries in this population (counting a twin birth as a single delivery); singleton mothers only 7. This analysis suggests these twin mothers are paying the price for heavy reproductive effort in terms of higher mortality risk during their post-reproductive years. Similar results have also been found in historical Finland and contemporary Britain (Helle et al., 2004; Grundy and Tomassini, 2005). In Finland, the higher mortality of twin mothers appeared to be due to a higher probability of dying from infectious disease. This suggests that the costs of reproduction are mediated by an impairment in immune function, as has been observed for several bird species (e.g. Gustafsson, 1994; Ardia, 2005).

But twin mothers are scarce in this population. For the majority of Gambian women, reproduction appears to have relatively few long-term costs. The lack of any long-term cost of reproduction in this population is also supported by a previous analysis of fertility rate (Sear et al., 2003). The costs of reproduction need not be confined to an increase in the risk of death, but may show up as any reduction in later reproductive output. We have already analyzed the fertility rates of these Gambian women to determine whether women who had previously invested heavily in reproduction suffered any reduction in fertility compared to women who had invested less heavily in reproductive effort, controlling for age. We found the contrary: women of high parity actually had a *higher* fertility rate than those of lower parity (controlling for age), whereas such women might be expected to slow down their fertility rates with age if they were suffering costs of reproduction. This analysis of fertility rate found no effect of other measures of reproductive investment, such as age at first birth. It also controlled for the measures of quality used here (height, BMI, and hemoglobin), but again found no substantial effect of including variables for health on the analysis.

Despite this lack of long-term costs, reproduction clearly has short-term costs in the higher risk of death in the year that a woman gives birth (i.e. maternal mortality). Maternal mortality is not widely discussed in the life history literature on costs of reproduction, perhaps because it is rather lower in non-human species than in humans. The two human characteristics of bipedal locomotion and large brains combine to cause human females particular problems during childbirth. The narrow pelvis required for bipedal locomotion means that the birth canal is rather a tight fit for a large-brained baby, resulting in relatively difficult labors (Trevathan, 1987; Rosenberg, 1992). Even in our species, in terms of the probability of dying per birth, a maternal death is relatively rare. Using data from the Gambia in the 1980s (from a different population to that analyzed here), Graham et al. (1989) estimated the probability of dying in childbirth to be around 1% per birth; similar studies in populations with little access to medical care find even lower estimates (e.g. Mace and Sear, 1996). In high fertility populations the *lifetime* risk of maternal mortality is, of course, much higher. In Graham et al.'s study, the lifetime risk of dying in childbirth was estimated to be 1 in 17; recent WHO estimates suggest that women in sub-Saharan Africa as a whole have a similar lifetime risk of maternal mortality, 1 in 16 (AbouZahr and Wardlaw, 2003). And in relative terms, maternal mortality does cause a high proportion of deaths to reproductive-aged women. In certain populations up to a half of all deaths to reproductive-aged women may be caused by pregnancy and childbirth (AbouZahr and Wardlaw, 2003). This significant risk of maternal mortality is a very real cost of reproduction.

Finally, the results of the analyses presented here were very similar whether or not measures of health were included in the models. This suggests that the negative relationship between reproduction and mortality is not mediated by the three control variables included in this analysis. This is somewhat surprising given that the variables used are thought to be good indicators of health. Height, BMI, and hemoglobin should all correlate with aspects of nutritional status or disease load, and all have been shown to affect reproductive and survival outcomes in this and other studies. Controlling for all three measures should be a particularly thorough method of controlling for health or quality, as each correlates with a slightly different aspect of health. It may be that costs of reproduction are only obvious under conditions of more extreme resource stress: some studies have shown that reproduction only has long-term effects in women in the lower socio-economic strata of a population (Tracer, 1991; Lycett et al., 2000; Dribe, 2004). The Gambian women studied here were mostly within the range of nutritional status considered to be adequate by international standards (BMI 18.5–24.9). They may therefore be able to devote energy to reproduction without sacrificing their own body condition too much. Women do appear to have a number of adaptations which allow them to optimally manage the allocation of energy between reproduction and survival (Poppitt et al., 1993; Prentice and Goldberg, 2000). There is a negative correlation between BMI and length of interbirth intervals in this population, for example, suggesting that women only attempt a reproductive bout when they are in sufficiently good condition to bear the costs (Sear et al., 2003). This careful allocation of energetic reserves, together with adequate food resources in this population, may mean that any long-term costs of reproduction are not sufficiently severe to show up as increased mortality rates. Alternatively, it may be that the measures of health used here are not good proxies of individual quality. We have assumed that these three measures between them adequately capture a woman's health, and that health correlates with quality. If either of these assumptions is incorrect, then controlling for these three measures will not adequately control for variation in individual quality between women. It may be that there are other factors which underlie the positive correlation between reproductive investment and adult survival chances, not controlled for in the models used here. For example, work by reproductive ecologists has shown that it is energy balance and energy flux rather than simply energetic reserves which affect reproductive function in women (Ellison, 2003); e.g. high levels of energy expenditure have been shown to suppress ovarian function even in well nourished women (Jasienska and Ellison, 1998). Women may differ in more ways than height, BMI, and hemoglobin, and these differences may be substantial enough to prevent any potential costs of reproduction being revealed.

CONCLUSIONS

In summary, this study tested the hypothesis that reproductive effort would increase the risk of death for women, by testing for correlations between adult female mortality and measures of reproductive effort in a natural fertility population without access to medical care. There is clear evidence for short-term costs of reproduction (i.e. the act of giving birth increases mortality significantly), but little evidence for long-term costs of reproduction in this population. On the contrary, women who had invested relatively heavily in reproduction (those of high parity and who had a late age at last birth) had lower mortality rates than those who had invested less in reproductive effort, once maternal mortality had been controlled for. Such positive correlations between reproductive and somatic effort may be due to individual differences in quality between women obscuring life history trade-offs. However, an attempt to control for such individual differences by including variables in the analysis thought to correlate with nutritional status and health made no difference to the results. This may be due to these women being reasonably well-nourished and therefore not suffering substantial long-term costs of reproduction. Alternatively, the measures of nutritional status and health may not sufficiently control for individual differences between women. The only evidence for long-term costs of reproduction is that mothers of twins do suffer high mortality rates in post-reproductive life. These women are particularly heavy investors in reproduction, but form a relatively small proportion of the population. Overall, though short-term costs of reproduction are obvious in this population, the inconclusive evidence regarding long-term costs illustrates the difficulty of demonstrating life history trade-offs in an observational study.

ACKNOWLEDGMENTS

The author thanks the Gambian Scientific Co-coordinating Committee and Ethical Committee for permission to use the data. The author also thanks two anonymous reviewers whose suggestions improved the text.

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