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## **Adult health and mortality in the Gambia: a life history perspective on relationships between anthropometric status and mortality risk**

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### **Abstract**

Relationships between anthropometric status and mortality are of interest to a number of disciplines, including demography, nutrition and evolutionary biology. This subject has attracted considerable research on developed world populations, but relatively little in developing countries. Here, an analysis of the relationships between anthropometric measures and adult mortality is performed on data from a rural African population, and set within the framework of evolutionary life history theory. The anthropometric measures used are height, weight and haemoglobin level. For haemoglobin there is a linear negative relationship with mortality risk for both sexes: individuals with higher haemoglobin have lower mortality. BMI is broadly negatively related to mortality risk for both men and women, though overweight individuals suffer a slightly increased risk of death. The relationship between height and mortality differs between the sexes. For men, there is no relationship. For women there is a U-shaped relationship, with women of average height having the lowest mortality.

## **Introduction**

Life history theory is a branch of evolutionary biology which considers the allocation of energy over the life course (Roff, 1992; Stearns, 1992; Stearns, 2000). Its core assumption is that organisms will use the energy they acquire in the service of reproductive success. Since energy used for one purpose cannot be used for another (the 'principle of allocation'), much of life history theory is concerned with energetic trade-offs. How do organisms solve the problem of allocating energy optimally between growth, physiological maintenance and reproduction? The study of demography is a key constituent of life history analysis: demographic patterns are both a constraint on, and a response to, life history events. For example, an important component of life history theory is the study of mortality schedules (Promislow and Harvey, 1991). This is partly because survival is essential to reproductive success, and therefore organisms must devote considerable effort to staying alive long enough to reproduce. But also because many causes of mortality are extrinsic to the organism, so that mortality schedules are an important constraint on life history strategies and will help determine how an organism allocates energy between growth, maintenance and reproduction (Hill and Hurtado, 1996, p179).

Our own species should be a prime candidate for life history research. Not only is there a large literature in the social and medical sciences on the demography, nutrition and physiology of humans, but the human species is also unusual in its ecological range. Many of the life history studies in the non-human literature are comparative studies, explaining life history variation between species with reference to each species' ecological environment (e.g. Harvey and Clutton-Brock, 1985; Wilkinson and South, 2002). With humans we can investigate life history variation (and similarities) across a range of environments for the same species. The aim of this paper is to add to the sparse but growing life history literature for humans by investigating adult mortality in a subsistence agriculture community in Africa, with particular reference to the relationship between mortality and anthropometric measures (see Hill, 1993; Hill and Hurtado, 1996 for the most comprehensive example; Hill and Kaplan, 1999; Mace, 2000 for reviews of life history theory applied to human populations).

Two issues will be examined. Firstly, the relationship between size (in this case defined as height) and adult mortality will be determined. Body size has received much attention in

life history analysis (see Roff, 1992, Chapter 7; Stearns, 1992, Chapter 6). Between species, body size correlates with a number of life history traits, including mortality rates. Large species tend to have lower mortality rates and longer lifespans (Harvey and Zammuto, 1985; Gaillard et al., 1989). Humans are no exception to this rule. As a large bodied mammal, we have relatively low mortality and long lives (though our lifespan is even longer than would be predicted by our body size: Hill and Kaplan, 1999; Hill et al., 2001). Within species the relationship between size and mortality is less clear-cut. Large size confers some benefits, such as protection from predators, but there are also costs to large size, for example, the greater nutrient requirements of maintaining a large body (Blanckenhorn, 2000). A complicating factor is the speed of growth experienced during childhood, which is correlated with final adult height but may also have implications for mortality in adulthood. Laboratory studies on rodents have found that severe caloric restriction retards growth (resulting in a small bodied adult) but also lengthens lifespan, which suggests that fast growth may have negative impacts on subsequent mortality and lifespan (Rollo, 2002; Metcalfe and Monaghan, 2003).

The relationship between adult size (height) and mortality in our own species is already well studied. Secular changes in height have been shown to be correlated with mortality trends in both the US and the UK: as the average height of the population increases, so does life expectancy (Floud et al., 1990; Fogel, 1993). Within populations, tall height has also been correlated with lower mortality rate (Marmot et al., 1984; Waaler, 1984). However, the situation is not entirely straightforward. Cause-specific analysis of mortality suggests that though the incidence of some causes of death, such as cardio-vascular and respiratory disease, are inversely related to height; others, such as reproductive cancers, increase in frequency with height (Barker et al., 1990; Leon et al., 1995; Smith et al., 2000; Song et al., 2003). This has led to a questioning of the prevalent view that tall height is beneficial (Samaras et al., 2003). A drawback of this research is that much of it has been carried out in Western populations, where adequate nutrition and medical care are widely available. Less is known about the effect of height on mortality in the type of population which would have characterised most of human history, where food is in short supply and the important causes of death are acute, rather than chronic, diseases. Here, the relationship between adult height and all-cause mortality is investigated in a poorly nourished society without access to medical care.

The second goal of the paper is to analyse the relationship between current body condition and adult mortality. Adult height reflects a combination of an individual's genetic potential and past life history experiences. An understanding of how adult mortality varies by *current* body condition is also valuable for life history studies. Life history theory would predict a negative correlation between body condition and mortality rates: individuals with greater energetic reserves invest this energy in maintaining body condition, thereby prolonging life. But there are still questions to be resolved. For example, which measures of body condition are the best predictors of mortality? Given that life history theory is concerned with the allocation of energy, a measure of nutritional status will clearly be a good candidate. A measure such as body mass index (BMI: which measures weight for height) should be a good indicator of chronic energy deficiency (or excess: Bailey and Ferro-Luzzi, 1995). However, nutrition is not the only factor affecting energy balance. Also important are the demands being made on these nutritional reserves. An important component of body maintenance is the ability to fight off disease. While this may be partly genetically determined, immune defence is energetically expensive. There is a well-known synergy between malnutrition and infectious disease, with malnutrition exacerbating infection and vice versa (Beisel, 1982; Scrimshaw, 2003). Measures of immunological competence are widely used in the animal literature as an indicator of body condition, and have been correlated with a number of life history parameters (Sheldon and Verhulst, 1996; Lochmiller and Deerenberg, 2000; Norris and Evans, 2000; McDade, 2004). Here, haemoglobin level (a marker of anaemia) is used as a convenient indicator that should reflect both nutritional status and disease load (Wadsworth, 1992; Stephenson, 1993; Gilgen and Mascie-Taylor, 2001), and may therefore be a more sensitive indicator of body condition than BMI alone.

Another question to be resolved is the shape of the relationship between measures of body condition and mortality. As with height, much research has already been done on the relationship between BMI and mortality. A non-linear relationship is usually found (Wienpahl et al., 1990; Rissanen et al., 1991; Laara and Rantakallio, 1996; Yuan et al., 1998; Engeland et al., 2003; Kuriyama et al., 2004). Individuals with low BMI suffer high mortality rates, but so do those with high BMI. Again, most of this research is carried out in well- (if not over-) nourished societies, but one recent study that did investigate the relationship between BMI and mortality in a food stressed Bangladeshi population found a similar non-linear relationship to that seen in the West (Hosegood and Campbell, 2003).

Women in the lowest and highest quartiles of BMI suffered higher mortality rates than those of intermediate weight, though the consequences of low BMI were more serious than those of high BMI. This is notable because women in the highest quartile of BMI were well within the range considered to be of appropriate weight by internal standards (this quartile included women with a BMI >19.61; a BMI of >25 is needed before an individual is considered to be overweight). This raises the possibility that the relationship between body condition and mortality may depend on relative, rather than absolute, body condition. This is something of a puzzle for life history theory, since a superficial prediction would be that the greater the energy availability the better an individual can maintain body integrity and prolong life.

Studies of the effects of anaemia on adult mortality are relatively rare, although there is considerable research on anaemia in reproductive-aged (and particularly, pregnant) women (e.g. Isah et al., 1985; Tracer, 1997; Allen, 2000; Bentley and Griffiths, 2003). Anaemia is known to be associated with maternal mortality (Thonneau et al., 1992; McDermott et al., 1996; MacLeod and Rhode, 1998; Walraven et al., 2000; Brabin et al., 2001), and has also been correlated with higher non-maternal mortality rates for reproductive-aged women (McDermott et al., 1996), and with higher mortality of older individuals of both sexes (Izaks et al., 1999). For maternal mortality at least, this relationship may not be linear, as some studies report only an effect of severe, rather than mild or moderate, anaemia (Rush, 2000; Brabin et al., 2001).

Finally, the relationship between condition and mortality will be examined separately in both women and men to determine whether there are any significant sex differences. Potential differences between the sexes may be enhanced in the high fertility population studied here, where women have to bear the energetically expensive demands of pregnancy and lactation repeatedly during their reproductive lives. Previous research on whether the BMI-mortality relationship is the same in men and women is inconsistent. Some studies find a similar relationship between BMI and mortality for both sexes (Engeland et al., 2003); others find that the shape of the relationship differs between the sexes, but these results do not show any consistent pattern (Wienpahl et al., 1990; Dorn et al., 1997; Kuriyama et al., 2004). There may also be differences between the sexes in the relationship between anaemia and mortality. Levels of anaemia vary considerably between the sexes

(e.g. Kent, 1992), and anaemia appears to be a particularly important risk factor for maternal mortality, which will not affect men.

## **Data**

The data were collected from four villages in rural Gambia by Ian McGregor under the auspices of the UK Medical Research Council (MRC: see McGregor, 1991 for a full description of the study)<sup>1</sup>. A demographic surveillance system has been in place in these villages since 1950, recording all births and deaths. Anthropometric data was systematically collected at least annually from all available villagers between 1950 and 1980. In 1975 a permanently staffed research station was set up in the largest village, which included a medical centre that provided free treatment to villagers. This analysis is confined to the period between 1950 and 1974, as the medical clinic resulted in a rapid decline in mortality rates (Lamb et al., 1984; Weaver and Beckerleg, 1993; Sear, 2001). Between 1950 and 1974, these villages had little access to medical care, though the primary researcher (a medical doctor) did provide medical treatment to individuals as necessary during his visits to the area.

The population largely supported itself with subsistence agriculture between 1950-74, though some income was earned through the sale of groundnuts. This West African environment is very seasonal. During the rainy season, heavy workload, low food supplies and high disease transmission (particularly malaria) coincided, which adversely affected the health of villagers: adults routinely lost weight during the rainy season (McGregor, 1976). Before the advent of the medical clinic, both birth and death rates were high: women had around 7 children on average, but almost half died before the age of 5 years (Billewicz and McGregor, 1981).

The growth of children in these villages has already been comprehensively documented (McGregor et al., 1961; McGregor et al., 1968; Billewicz and McGregor, 1982). Children grew well relative to international standards initially, but growth-faltering began around the age of three months and growth thereafter lagged significantly behind that of Western children. Growth was strongly affected by season. Children grew much more slowly during the rainy season than the dry. The Gambian children had an extended growth period

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<sup>1</sup> Thanks to the Gambian Scientific Co-ordinating Committee and Ethical Committee for permission to use the data

compared to Western counterparts, but this was not sufficient to make up for their slower growth, so that adults in this population were relatively short and light. Anaemia was common and severe in childhood, and followed a similar seasonal pattern to that of nutritional status (McGregor et al., 1966). Malaria levels were also high during the rainy season, and haemoglobin levels were correlated with the presence of malaria parasites in the blood.

Between 1950 and 1980, 23,010 anthropometric measurements were taken from 2,096 adults in these villages (throughout this paper an 'adult' is defined as an individual 21 years or older, as growth had ended in virtually all individuals of both sexes by this age: Roberts et al., 1978). Rather more measurements were taken from women than men: 9,699 measurements were taken from 931 men, and 13,311 measurements from 1,165 women. The majority of measurements were taken during the dry season, as villagers were heavily involved in agricultural work during the rainy season.

Table 1 summarises the anthropometry of adults in these villages. Heights and weights were recorded in inches and pounds respectively and converted to centimetres and kilograms for this analysis. BMI ( $\text{kg}/\text{m}^2$ ) was then calculated from these converted measurements. Adults in these villages were in relatively poor nutritional condition relative to Western populations. They were comparatively short: the average male height was around 168cm, the average female height 158cm. These villagers were also relatively light, though the majority were within the weight range considered adequate by international standards. Mean BMI for both men and women was approximately 20. A minority of measurements are considered underweight by international standards, but only a tiny fraction are considered overweight. If an average BMI is calculated for each individual then 151 (13.1%) women and 125 men (13.4%) were underweight, but only 8 men (0.9%) and 34 women (2.9%) were overweight ( $\text{BMI} > 25$ ). None were obese, with a BMI of 30 or over (no man ever recorded a BMI of 30 or more, and only 0.2% of BMI measurements from women were greater than 30). This population did suffer from considerable iron deficiency, however. Around half of all haemoglobin measurements from women were below the cut-off for anaemia ( $< 12 \text{ g/dl}$ ). Men were slightly better off, but one-third of measurements from men were also considered anaemic ( $< 13 \text{ g/dl}$ ).

## **Exploratory analyses**

### *Adult mortality and sex*

Individuals in this population who survive to adulthood can expect to live into their late 60s: median age at death for women who survive to at least 21 years is 68, median age at death for men is 67. Mortality rates in adulthood are similar for women and men (Figure 1). Female mortality is a little higher during the reproductive years; male mortality is a little higher in older adulthood. Overall, there is no significant difference in the survival distributions of the sexes (log rank statistic = 0.31, df=1, p=0.57).

### *Correlations between anthropometric measures*

To test for correlations between anthropometric measures, regression models were run to determine the correlation between haemoglobin and height, and haemoglobin and BMI (no correlations between BMI and height were estimated, since the calculation of BMI includes height). Because most individuals contributed more than one measurement to the analysis, multi-level regression models were used to control for these repeated measures. These models controlled for age, birth cohort, and year and season of survey. Separate models were run for women and men. For women, there was a positive correlation between haemoglobin and BMI ( $\beta=0.043$ ,  $SE=0.009$ ,  $p<0.01$ ). This correlation was highly significant but relatively modest (every one point increase in BMI resulted in an increase in haemoglobin of 0.043, so that across the entire range of BMI observed in female subjects, haemoglobin would only increase by 1.17 g/dl). There was no association between haemoglobin and height for women. For men, haemoglobin level was positively correlated with both height and BMI, and these correlations were of slightly greater magnitude (for BMI:  $\beta=0.190$ ,  $SE=0.017$ ,  $p<0.01$ ; for height:  $\beta=0.049$ ,  $SE=0.007$ ,  $p<0.01$ . This translates into a change in haemoglobin of 3.12 g/dl across the observed range of male BMI, and 3.39 g/dl across the observed range of male height).

## **Methods**

To determine the relationship between all three anthropometric measures and mortality, the probability of dying in adulthood (*i.e.* from the age of 21 years) was analysed between 1950 and 1974 using discrete-time event history analysis (EHA). EHA models the probability of an event, in this case a death, happening over time. Such models have the two advantages of being able to deal with censored data, and can include time-varying covariates (Allison,



1984). Discrete-time models are used in this analysis as time to event (death) is recorded in years, which are relatively large units of time. When such large time units are used, discrete-time models are more appropriate because of the difficulty continuous time models have dealing with ‘ties’ *i.e.* several events occurring in the same time interval (Yamaguchi, 1991; Singer and Willett, 2003). The model takes the form:

$$\log \left( \frac{h_{it}}{1 - h_{it}} \right) = \alpha_t + \beta' \mathbf{x}_{it}$$

where  $h_{it}$  is the probability that individual  $i$  will experience the event at time  $t$ , given that the individual had not experienced the event prior to time  $t$ ;  $\alpha_t$  is a function of time and  $\mathbf{x}_{it}$  is a vector of covariates, which may be either time-constant or time-varying, with associated parameters  $\beta$ .

Individuals were both right-censored (those without a known date of death were right-censored at the age they were last known to be alive, and all individuals still alive in 1975 were censored in that year) and left-censored (those who reached the age of 21 before 1950 were only included in the analysis from the age they had reached in 1950).

BMI and haemoglobin were included in the models as time-varying covariates. Few individuals were surveyed in every year between 1950 and 1980, so a mean BMI or haemoglobin 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 over), assuming the individual had more than one measurement in the 5-year age block. These mean BMI and haemoglobin 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. Measurements taken within 12 months of death were excluded when calculating these 5-year means, to avoid a decline in body condition prior to death contaminating the results (but this only excluded a very small number of measurements – only 4 measurements were taken within 12 months of an individual’s death). For women, BMI and haemoglobin measurements taken during pregnancy were also excluded, as were measurements taken within three months after a

birth for the haemoglobin analysis (haemoglobin 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 haemoglobin, 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 21 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. These time-varying height measures were constructed using the same method as for BMI and haemoglobin.

The effects of height, BMI and haemoglobin on adult mortality were investigated for each sex separately. First, models were run for each sex which included only one anthropometric measure (Model I included only height, Model II only BMI and Model III only haemoglobin). Then a model was run for each sex which included all three anthropometric measures (Model IV). A final model was run which included all three anthropometric variables and interaction terms between each anthropometric variable and age (Model V), to determine whether any of the effects of anthropometry varied by age. Non-linear effects of all measures of anthropometric status were tested for by including quadratic terms. All models also controlled for birth cohort. A series of (time constant) dummy variables were constructed for 10 year birth cohorts and included in the models. In all models, only cases where all three anthropometric measures were available were included, so that the fit of the five models could be compared. The final sample size in each model was 1005 women (of whom 182 died) and 855 men (of whom 172 died).

## **Results**

The results of the event history analyses demonstrate a clear relationship between body condition and mortality for both sexes, but the nature of these relationships differs between different measures of body condition, and between men and women. Table 2(a) shows the results of all five models for women, Table 2(b) the results for men (models I, II and III include only one measure of anthropometric status – height, BMI and haemoglobin respectively; model IV includes all three anthropometric measures; model V includes all anthropometric measures and interactions between anthropometric measures and age).

Height shows the greatest differences between the sexes. For women there is a significant relationship between height and the risk of death, though this relationship is not linear. Models I and IV suggest a U-shaped relationship between female height and mortality, so that both tall and short women suffer higher mortality rates than women of average height. Figure 3(a) plots the model predictions of the probability of death for a 30-year old woman across the range of heights seen in the population (excluding extreme values). The lowest mortality is seen for women of approximately average height; both short and tall women suffer relatively high mortality risks. Model V suggests this relationship is not modified by age, as the interaction between age and height is not significant. For men there is no evidence that height and mortality are correlated, as neither linear nor non-linear functions of height are significantly related to mortality risk.

For BMI, the relationship between anthropometric status and mortality is very similar for both sexes. This anthropometric variable is significantly related to mortality risk for both men and women, and also shows a non-linear relationship with mortality. In this case, however, across most of the observed range of BMI an increase in BMI results in a decrease in mortality risk. It is only at high BMIs that mortality risk begins to rise (see Figure 3(b)). The shape of this relationship appears to be very similar for both sexes. Model V again suggests these relationships are not modified by age, as the interaction between age and BMI is not significant for either sex.

Haemoglobin shows a significant linear correlation with mortality for both women and men. The risk of death decreases as haemoglobin increases across the range of observed haemoglobin levels (in this case, a quadratic term did not improve the fit of the model). The shape of the relationship does differ somewhat between the sexes: the slope of the line is greater for women than men, so that the consequences of low haemoglobin are more severe for women than men (Figure 3(c)). This relationship is also likely to be more important for women than men as the distribution of haemoglobin differs between the sexes: women tend to have considerably lower haemoglobin levels than men. For women, this relationship is somewhat modified by age, as there is a significant interaction between age and haemoglobin in model V. The positive sign of this interaction suggests that the effect of haemoglobin on mortality risk is greatest for young women, but decreases as women age. The magnitude of this effect is small, however.

The relationships between different anthropometric measures and mortality appear to be independent of one another, despite the correlation between anthropometric measures reported earlier. The results for the final model including all three measures of anthropometric status were similar to those where only a single anthropometric measure was included in the model. Interaction terms between the anthropometric measures were included in preliminary models, but were not significant.

## **Discussion**

Size (height) is related to mortality in this population for women but not for men, though this relationship is not linear. The higher mortality of short women may be partly explained by maternal mortality. Short stature increases the risk of prolonged and difficult labour, primarily due to cephalopelvic disproportion (Sokal et al., 1991; Tsu, 1992; Moller and Lindmark, 1997), which is likely to result in high maternal mortality rates for short women in settings where modern medical care is unavailable. Though the numbers are too small to permit meaningful statistical analysis, the mean height of the 10 women known to have died from maternal causes before 1975 is 155.8cm, which is 2cm shorter than the mean height for the entire female population (157.8cm).

Short height is also an indicator of conditions an individual experienced during childhood, such as nutritional availability and prevalence of disease. If there is a link between poor conditions in childhood and poor conditions in adulthood (mediated, for example, by socio-economic status), then this may explain the correlation between short stature and high mortality. It has also been suggested that an adverse environment in early life (particularly during the foetal period) results in a higher incidence of disease in later life (Barker, 1994). Adverse early life conditions may result in both short adult height and high adult mortality rates, thus mediating the link between adult and mortality. Most of this research has focused on chronic diseases in Western populations, but an analysis of the effects of season of birth on adult mortality in this Gambian population suggests a similar association with infectious disease (Moore et al., 1997; Moore et al., 1999). Using a different sample to that analysed here, including data collected up until the present day, these authors found that individuals born during the ‘hungry’ season (when food was scarce and disease rife) have higher mortality in adulthood than those born during the ‘harvest’ season (when food was more plentiful and disease less so (Moore et al., 1997)). This mortality was primarily due to

infectious, rather than chronic, diseases (Moore et al., 1999). However, it has not proved possible to replicate this association between season of birth and adult mortality using similar longitudinal datasets from Senegal (Simondon et al., 2004) or Bangladesh (Moore et al., 2004), and the mechanism is unclear, as season of birth is not related to childhood immune function in this Gambian population (Moore et al., 2001). The jury therefore remains out on whether adverse early life experiences increase the frequency of infectious diseases, as well as the chronic diseases of affluence. Unfortunately, it was not possible to determine relationships between season of birth, height and adult mortality in the sample used here. Season of birth was known for few individuals in this sample, as most were born before 1950 when demographic surveillance began.

The increased mortality of taller women is harder to explain. Though studies in Western populations have shown increased mortality from certain cancers in taller individuals, cancer was probably a relatively minor cause of death among these Gambian women (though little information on cause of death is available during this time period, with the exception of some information on maternal deaths). In addition, 'tall' Gambian women were not particularly tall by Western standards. The tallest woman was around 178cm (5'10), and 95% of women were 167cm or less (approx 5'6). A potential cost to relatively tall height that could apply to this population is that taller individuals need relatively large amounts of energy to maintain their somatic tissue. There is considerable seasonal and also yearly variation in food availability and disease prevalence in this part of the world. Tall individuals may suffer more than shorter individuals during lean periods because of their greater energy requirements. However, this logic should apply to both men and women, yet the analysis suggests the correlation between height and mortality applies only to women. An alternative hypothesis is that the higher mortality of tall women could be linked to reproduction. The advantages of large size are thought to include more successful reproduction, and it has been shown previously in this population that taller women are more reproductively successful than shorter women (Sear et al., 2004). In particular, the survival of the children of tall women is markedly higher than that of the children of shorter women. Successfully raising many children to adulthood may create additional energetic stresses on taller women which ultimately lead to higher mortality rates.

The relationships between BMI and mortality, and haemoglobin and mortality for both sexes are rather more straightforward. This analysis suggests that mortality rates decrease

as both BMI and haemoglobin increase, as would be predicted by life history theory: individuals with access to greater energetic reserves use them to lower their mortality rates. The model suggests that relatively high BMI does lead to higher mortality rates, but that the consequences of low BMI are much more severe than those of high BMI. The increase in mortality among overweight individuals is relatively slight (Fig. 3b), and overweight individuals in any case are rare in this population. The increase in mortality at high BMIs (apparently a universal feature of all populations studied, whether in the developed or developing world) is perhaps surprising from a life history perspective, as theoretically the greater energy available the lower the mortality risks. Physiologically there are clearly costs to energy storage; for example, increased fat deposits around organs and in blood vessels impair their functioning, resulting in an increase in mortality risk in individuals with large fat stores. This is likely to be a maladaptive outcome of a previously adaptive response: when energy is less readily available, the ability to store fat is advantageous in protecting us from starvation during lean periods. This fat storage mechanism becomes maladaptive in the current climate of excess energy availability (together with other previously adaptive mechanisms which encourage us to consume as much energy as is available).

It is perhaps surprising that the relationship between BMI and mortality is so similar between the sexes, given that women have to bear the energetic costs of regular pregnancies and lactation (both sexes perform substantial amounts of productive labour in this community). But recent research by both nutritionists and evolutionary biologists has suggested that women do have a number of adaptations which allow them to mitigate the costs of reproduction (e.g. Peacock, 1991; Ellison, 1994; Dufour and Sauter, 2002; Ellison, 2003). Women appear to delay reproduction until they are in suitable condition to bear such a high energetic cost, for example. Such adaptations can be seen in the Gambian population reported on here: women with low BMI (and haemoglobin levels) have slower birth rates than those in better condition (Sear et al., 2003), and only those in relatively good condition are able to produce twins (Sear et al., 2001). Research by the MRC in this population after 1975, has also demonstrated that women are able to make metabolic adjustments which minimise the energetic stresses of pregnancy (Prentice and Whitehead, 1987; Poppitt et al., 1993; Prentice and Goldberg, 2000).

There seem to be no diminishing returns to increasing haemoglobin in terms of mortality rates (at least within the range of haemoglobin observed here), as there was no evidence for

a non-linear relationship between haemoglobin and mortality. This relationship is similar for both sexes, though the models indicate that the mortality costs of severe anaemia are greater for women than for men. This could be related to maternal mortality, as severe anaemia greatly increases the risk of maternal mortality, though mild or moderate anaemia does not appear to have the same effect (Rush, 2000; Brabin et al., 2001).

BMI and haemoglobin (and height, in the case of women) may all have independent effects on the probability of death because they measure slightly different things. BMI reflects the energy available for body repair and maintenance, including immune function.

Haemoglobin may reflect some genetic immunity to disease, particularly malaria. Though energy availability is very important for immune function, there are certain genotypes which are advantageous for protection against certain diseases (e.g. sickle cell trait and malaria). BMI is unlikely to pick up on such genetic factors, but haemoglobin will do. Similarly, the links between height and mortality are not yet established. Though energy availability may again be a factor, other factors are also involved. E.g. protection from certain cancers of short people may reflect hormonal differences between short and tall people.

### *Conclusion*

To answer the questions posed in the Introduction, there is clear evidence that current body condition has a significant impact on adult mortality rates for both sexes. Both nutritional status (BMI) and haemoglobin level (an anthropometric measure which is affected by disease load) were independently related to mortality risk, suggesting that both are good indicators of current body condition. The relationship between measures of body condition and mortality is essentially linear: as body condition increases, the probability of death decreases. Though there do seem to be some costs to energy acquisition among those individuals who acquired sufficient energy to become overweight, this accounts for a very small proportion of the population. The condition-mortality relationship is similar for both sexes, despite some differences in condition between the sexes (for haemoglobin but not BMI), and differences in the association between these anthropometric measures and age.

Ecology also seems to matter for the relationship between size and mortality, though perhaps less so for the relationship between current body condition and mortality. The reverse J-shaped correlation between BMI and mortality, for example, appears to be the

common pattern across both well nourished and poorly nourished populations (in relative, if not absolute, terms). The size-mortality relationship is more complicated. Broadly speaking, the all-cause mortality for men in Western populations decreases with increasing height. This may not be the case in populations with lower nutrient intakes, since no relationship between height and mortality was found for men in this study or in a nineteenth century population (Murray, 1997). Relationships between height and mortality for women appear more variable. The U-shaped relationship observed here has been found in Western populations (Laara and Rantakallio, 1996; Engeland et al., 2003), but so has a linear decrease in mortality with height (Jousilahti et al., 2000; Smith et al., 2000). Additionally, the study of Bangladeshi women reported earlier found no effect of height on mortality (Hosegood and Campbell, 2003). As discussed in the Introduction, large size brings both costs and benefits, and these may differ between environments. It is also affected by previous life history experiences, which may include not only conditions encountered during early life but also life history events such as age at first birth. Growth and reproduction are both energetically costly, so a trade-off between height and age at first birth is often found. As seen in this Gambian population, tall women tend to have later first births than shorter women (Allal et al., 2004). Complex relationships between energy availability, growth, reproductive events and height may make it difficult to draw general conclusions about the relationship between height and mortality. More research on this relationship for both sexes across a range of societies is needed to determine if any consistent patterns emerge, and whether these have any functional significance.



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**Table 1: Summary of adult anthropometric data<sup>1</sup>**

	<b>Women</b>	<b>Men</b>
<b>Height (cm)</b>		
Number of measurements	13,290	9,680
Mean $\pm$ SD	157.8 $\pm$ 5.6	168.0 $\pm$ 6.7
Range	122.5-178.4	127.6-196.8
<b>BMI (kg/m<sup>2</sup>)</b>		
Number of measurements	11,598 <sup>2</sup>	9,674
Mean $\pm$ SD	20.7 $\pm$ 2.3	20.4 $\pm$ 1.8
Range	12.4-39.7	13.5-29.9
% Underweight (<18.5)	16.1	13.5
<i>Mildly (17-18.49)</i>	12.6	11.4
<i>Moderately (16-16.9)</i>	2.6	1.5
<i>Severely (&lt;16)</i>	0.9	0.6
% Overweight (25+)	3.8	1.5
<i>Obese (30+)</i>	0.2	0
<b>Hb (g/dl)</b>		
Number of measurements	10,986 <sup>3</sup>	9,653
Mean $\pm$ SD	11.8 $\pm$ 1.7	13.5 $\pm$ 2.1
Range	2.0-17.1	2.7-20.0
% Anaemic (<12 for women, <13 for men)	48.4	33.5
<i>Mildly (10-11.9/12.9)</i>	35.7	26.8
<i>Moderately (7-9.9)</i>	12.1	5.7
<i>Severely (&lt;7)</i>	0.6	1.0

<sup>1</sup> Means and standard deviations are presented here for the whole sample of measurements, rather than for individuals (calculating a mean summary measure for each individual, and then averaging these summary measures results in similar means, however)

<sup>2</sup> Excluding measurements taken during pregnancy

<sup>3</sup> Excluding measurements taken during pregnancy and within 3 months of giving birth

**Table 2: results of event-history analysis on the probability of dying. Models also control for birth cohort**

**(a) Women**

Variable	Model I	Model II	Model III	Model IV	Model V
Constant	67.88 (35.70) <sup>†</sup>	3.22 (2.77)	-3.46 (0.60)**	82.36 (36.48)*	97.22 (38.06)*
Age	0.06 (0.01)**	0.06 (0.01)**	0.07 (0.01)**	0.06 (0.01)**	-0.19 (0.13)
Height	-0.96 (0.45)*			-0.97 (0.46)*	-1.04 (0.48)*
Height squared	0.003 (0.001)*			0.003 (0.001)*	0.003 (0.001)*
BMI		-0.85 (0.25)**		-0.87 (0.26)**	-1.07 (0.30)**
BMI squared		0.02 (0.01)**		0.02 (0.01)**	0.02 (0.01)**
Haemoglobin			-0.35 (0.05)**	-0.34 (0.05)**	-0.67 (0.14)**
Height*age					0.001 (0.001)
BMI*age					0.003 (0.002)
Hb*age					0.007 (0.003)*
AIC	1799.04	1782.84	1750.84	1737.84	1734.40
-2 log likelihood	1783.04	1766.84	1736.84	1715.84	1706.40
Number of deaths			182		
Number of survivors			823		

† p<0.1, \* p<0.05, \*\* p<0.001

**(b) Men**

Variable	Model I	Model II	Model III	Model IV	Model V
Constant	-6.99 (2.09)**	3.95 (4.14)	-4.41 (0.60)**	4.81 (4.64)	8.22 (8.79)
Age	0.07 (0.01)**	0.07 (0.01)**	0.07 (0.01)**	0.07 (0.01)**	-0.02 (0.15)
Height	-0.003 (0.01)			0.005 (0.013)	-0.01 (0.05)
BMI		-0.98 (0.39)*		-0.91 (0.39)*	-0.91 (0.39)*
BMI squared		0.02 (0.01)*		0.02 (0.01)*	0.02 (0.01)
Haemoglobin			-0.24 (0.03)**	-0.23 (0.03)**	-0.14 (0.13)
Height*age					0.0003 (0.001)
BMI*age					0.003 (0.003)
Hb*age					-0.002 (0.002)
AIC	1638.13	1626.37	1591.43	1589.52	1594.30
-2 log likelihood	1624.13	1610.37	1577.43	1569.52	1568.30
Number of deaths			172		
Number of survivors			683		

\* p<0.05, \*\* p<0.001

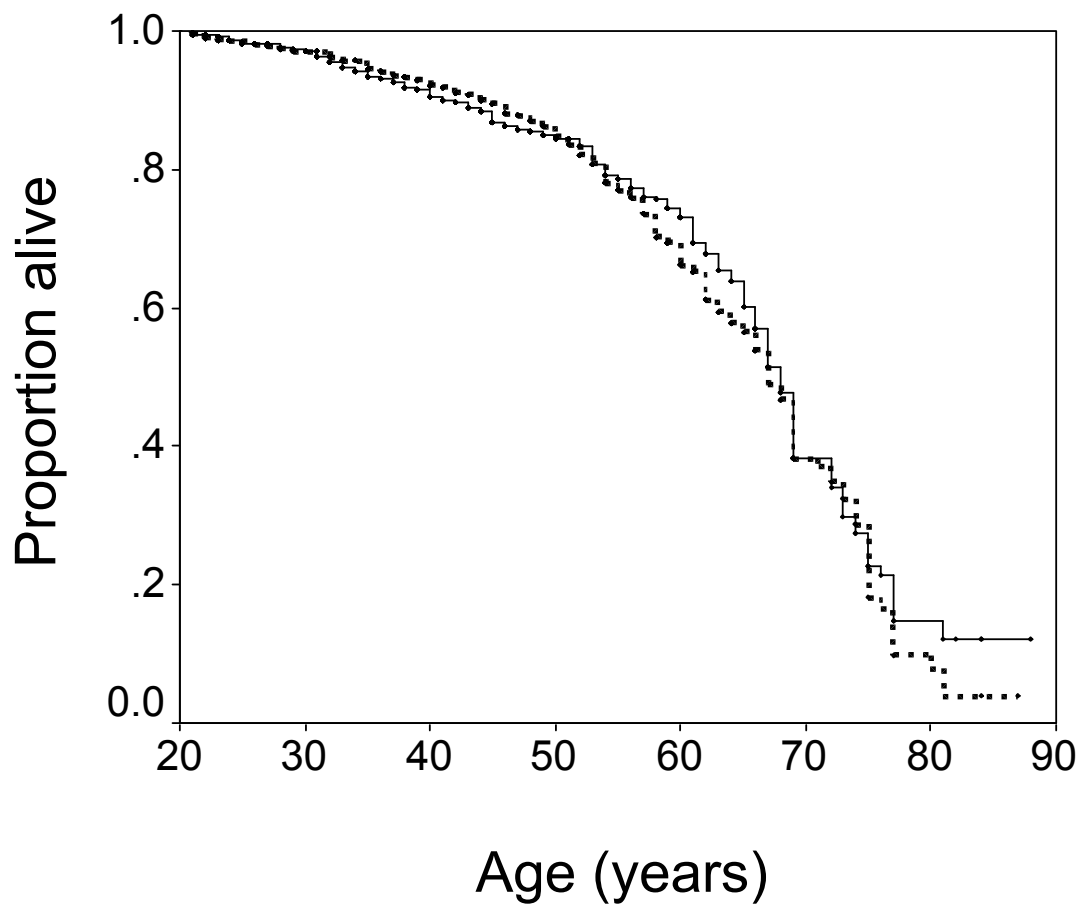


### Figure legends

Figure 1: Kaplan-Meier plot showing survival function of women (solid line) and men (dotted line) who survived to at least 21 years. N=1125 women, of whom 208 died; N=968 men, of whom 201 died.

Figure 2: Model predictions of probability of dying per year by height (a), BMI (b) and haemoglobin (c). Solid line represents women, dotted line men.

Figure 1



**Figure 2 (a)**

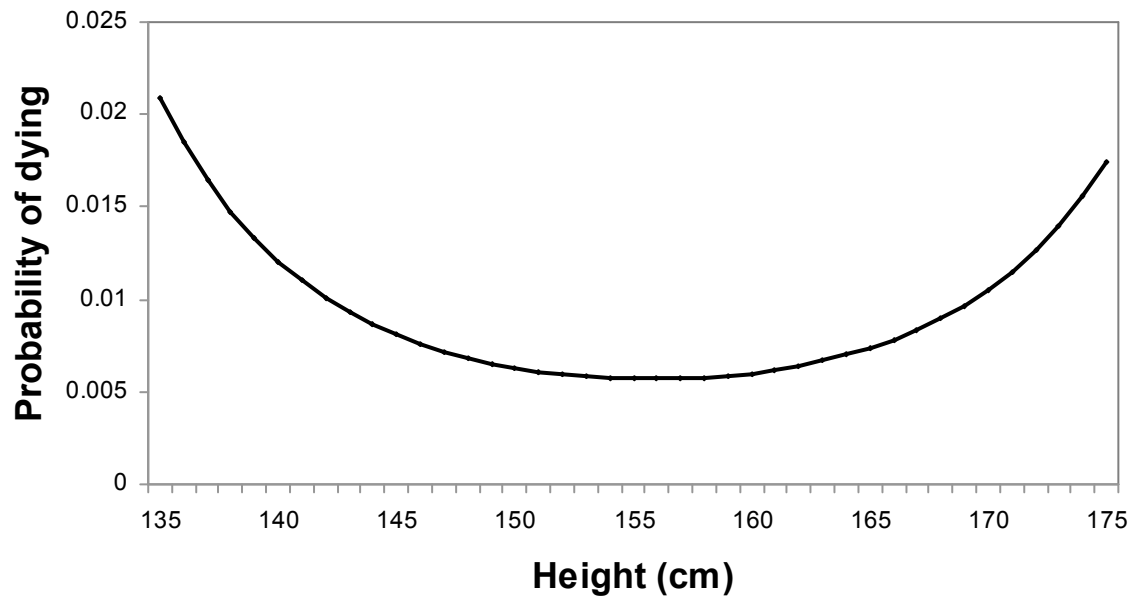


Figure 2 (b)

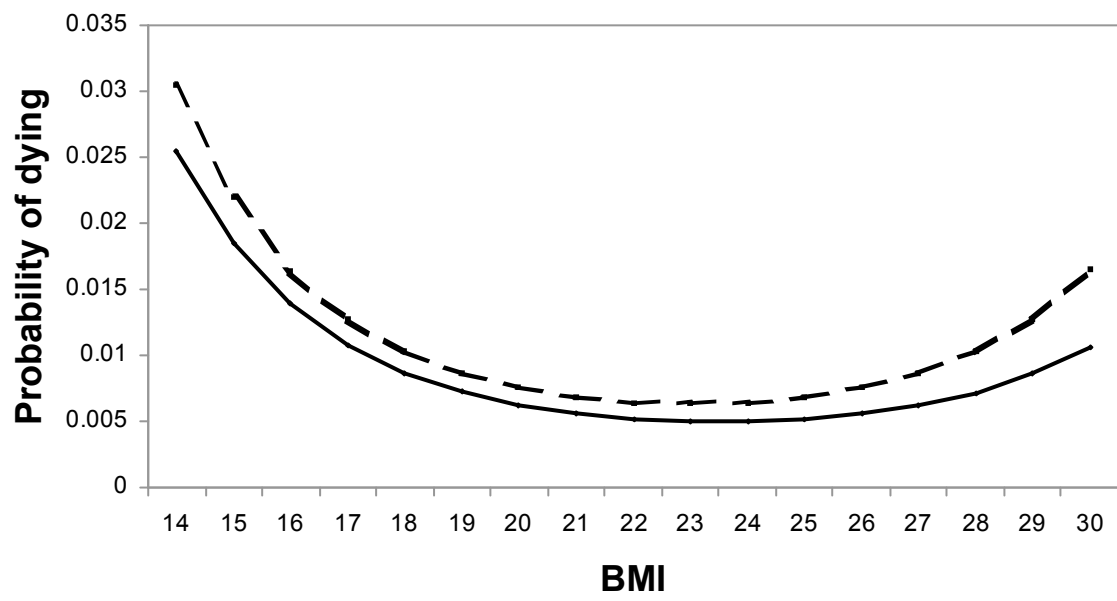


Figure 2 (c)

