Published in *History of the Family*, 2022
https://doi.org/10.1080/1081602X.2022.2123842

Nutritional status and adult mortality in a mid-20th century Gambian population: do different types of physical 'capital' have different associations with mortality?

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Abstract

Measures of nutritional status are often used as markers of health, at both individual- and population-level. Different measures of nutritional status – such as height or weight, for example – may have different associations with health outcomes because they reflect both current nutritional status and the accumulation of past health experiences, but the weighting of past and present experiences differs between different measures. Here, we present an analysis of a longitudinal dataset, collected between 1950-74 in four Gambian villages, to investigate whether three different measures – height, body mass index (BMI) or haemoglobin level – are associated with adult mortality. We interpret these different measures as indicators of different types of physical 'capital' that vary in their liquidity. Adult height represents 'illiquid' capital, indicative of nutritional status in the past, during different periods of development. BMI, determined both by past childhood experiences and recent nutrition and health exposures, represents capital of intermediate 'liquidity'. Haemoglobin represents 'liquid' capital, determined largely by recent environmental exposures. We find, not unexpectedly, that the more 'liquid' measures of capital show clearer associations with adult mortality: for haemoglobin there is a negative relationship with mortality risk for both sexes: BMI is also broadly negatively related to mortality risk for both men and women, though overweight individuals suffer a slightly increased risk of death. For men, there is no relationship between the 'illiquid' measure of height and adult mortality; but for women, there is a U-shaped relationship, with women of average height having the lowest mortality.

Introduction

There is considerable research on associations between anthropometric measures, such as height and weight, and mortality risk in children as well as in adults in higher income countries. These measures are markers of (past and/or present) nutritional status and health, so that associations of these measures with mortality are often found. Data on such measures can be easily collected and so can act as useful indicators of elevated mortality risk in children and adults, which can be managed with suitable interventions. There is substantial evidence from lower and middle income countries that mortality is higher in children of shorter height and lower weight, for example, though these associations may vary somewhat by age of child and context (e.g. Billewicz and McGregor, 1982; McDonald et al, 2013; Schroeder et al, 1994). The evidence on associations between anthropometric status and adult mortality is more likely to come from higher income contexts than (contemporary) lower income contexts, and is rather more mixed than for children (e.g. Sear, 2010; Lee et al 2018).

For adults, while low and high body weight both seem to be associated with higher mortality risk, the lowest risk of mortality may be seen at different body weights in different contexts (Wienpahl et al., 1990; Rissanen et al., 1991; Laara and Rantakallio, 1996; Yuan et al., 1998; Engeland et al., 2003; Kuriyama et al., 2004). Overweight (BMI >24.9 and <30) and obesity (BMI >30) are often used in clinical settings as evidence of potentially elevated disease and mortality risk, but mortality rates may be relatively low for overweight individuals in at least some higher income contexts. There is also evidence that mortality risk may begin to increase at relatively low BMIs in some lower income contexts (Hosegood and Campbell, 2003). These findings suggested the lowest mortality risk is found at higher body weights in contexts where body weight is, on average, higher. The relationship between height and mortality is also somewhat contested in adults (Samaras et al., 2003). Many studies do show that taller height comes with reduced mortality risk, at least up to a point (Marmot et al., 1984; Waaler, 1984), but this is not universal. When cause-specific mortality is examined, it seems that tall height might come with elevated risk of some diseases, such as many cancers, but lower risk of cardiovascular disease (Barker et al., 1990; Leon et al., 1995; Smith et al., 2000; Song et al., 2003; Nüesch et al, 2016; Wells et al 2020). The third measurement we use – haemoglobin level – has been less studied in association with mortality, but 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).

Beyond their clinical significance, relationships between anthropometric status and mortality have been of interest to several academic disciplines (e.g. Stulp & Barrett, 2016). In historical demography and economic history, height data is often used as a marker of population health, given that it is available for many historical populations (though often from biased samples; men are more often represented than women in such data, for example)(Fogel, 1986; Costa, 1993; Harris, 1997; Murray, 1997; Wilson 2019). Height data can therefore be used to track population health over time, and to compare between populations. It has also been used as a marker of early life conditions, given it is determined by conditions during childhood (Schellekens and Van Poppel, 2016). Adult height has relatively high heritability (Jelenkovic et al, 2016), but its variability also reflects conditions during development, as demonstrated by studies of foetal or post-natal exposure to famine or malnutrition (Huang et al, 2010; Portrait et al, 2017; Mwene-Batu et al, 2020). The somewhat variable associations seen between height and mortality for adults at the individual-level may perhaps be less of a concern when heights are used as markers of population health because, at the population-level, associations between height and health or mortality may be more reliably positive (Floud et al., 1990; Fogel, 1993), making average population-level height a useful marker of physical health (Perkins et al, 2016; but see Deaton, 2007).

In this paper, we adopt a unifying framework which considers all three components of nutritional status as indicators of different types of embodied capital (Kaplan et al 2003), which vary according to the timescale (resolution) with which they are gained and lost within and between life-spans. Our hypothesis is that the costs and benefits of physical capital may vary according to their according to their liquidity, which refers to the

timescale over which physical capital can be gained or lost from the body (Wells 2010), which in turn shapes their capacity to promote survival and fitness.

First, we consider stature, the magnitude of which reflects long-term experience as well as individual genotype. On the one hand, a proportion of variability in stature can be attributed to complex multi-genic factors, hence the genotype of an individual at the present time reflects selection having favoured the associated phenotype in ancestors. On the other hand, stature is also sensitive to early life experience, and so reflects the interaction between maternal phenotype (as the provider of nutrition during foetal life and infancy) and the external environment (as the source of factors impacting on maternal provisioning or offspring health). In the Gambian population studied here, environmental factors acting on recent matrilineal experience are likely to be of particular importance. However, whilst adult stature can respond flexibly to early life experience, this flexibility becomes more constrained after 2 years as linear growth comes under the influence of growth hormone rather than nutrition, and so becomes a relatively fixed or 'illiquid' component of capital, though adolescence may be another period when nutrition is influential (Prentice et al, 2013). Stature then has benefits (to health and fitness) and but also costs, in terms of physiological stress and larger energy requirements. Benefits include stature acting as a marker of good quality infant and childhood environment, greater physical work capacity and reproductive robusticity. Costs include higher adult levels of IGF1 (Insulin-like Growth Factor 1), a protein which predispose to cellular ageing.

Second, we consider BMI, which is a component of capital that reflects genes (also under complex multi-genic influences), early life experience but also recent nutritional experience. BMI indexes both lean mass (which is strongly determined by growth during foetal life and early post-natal life) and also fat mass, which is more strongly influenced by childhood weight gain and adult energy balance. BMI is therefore more flexible than stature, which is essentially fixed once linear growth has ceased, though flexibility in BMI pertains more to fat mass than lean mass. For our analyses, a difficulty is that between-subject BMI variability may reflect lean more than fat, whereas within-subject variability may reflect fat more than lean. In a population with high body weight, BMI may most strongly represent physiological costs (excess adiposity), whereas in a population with low-to-medium body weight, BMI may reflect physiological benefits (organ function, reproductive capacity and physical work capacity). However, the key point here is that

adult BMI incorporates variability in weight which fluctuates, in contrast to adult height which does not. Therefore, BMI represents a component of capital of intermediate liquidity.

Finally, we consider blood haemoglobin content, which primarily reflects recent nutritional and disease experience (Wadsworth, 1992; Stephenson, 1993; Gilgen and Mascie-Taylor, 2001; Orish et al, 2013). Although haemoglobin phenotype is influenced by genetic factors, including, in particular, sickle cell disease and other haemoglobinopathies, the primary environmental factor is iron intake. Regardless of genotype, levels of haemoglobin must respond to dietary intake, and the data indicate considerable variability. We consider higher levels of haemoglobin to represent a high quality diet, and low exposures to infections, and hence to act as a marker of 'liquid capital', sensitive to recent rather than developmental experience.

We therefore consider the associations of mortality with components of capital that are characterised by either high, intermediate or low liquidity (see Figure 1).

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). 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; Rayco-Solon et al., 2004). Between 1950 and 1974, these villages had relatively little access to medical care, though the primary researcher (a medical doctor) did provide medical treatment to individuals 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 (see also Wells et al, 1993). 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 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 and blood biochemical 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 largely ended in 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 (kg/m^2) was then calculated from these converted measurements. Adults in these villages 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 kg/m^2. A minority of measurements are considered underweight by international standards (18.5 kg/m^2; PMID 3148462), but only a tiny fraction are considered overweight (BMI>25 kg/m^2). 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. None were obese, with a BMI of 30+ kg/m^2 (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 g/dl). Men were slightly better off, but one-third of measurements from men were also considered anaemic (<13 g/dl).

Methods

To determine the relationship between all three nutritional 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. Event history analysis 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*; α_t is a function of time and x_t is a vector of covariates, which may be either time-constant or time-varying, with associated parameters β.

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 affecting 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 associations between height, BMI and haemoglobin and adult mortality were investigated for each sex separately. First, models were run for each sex which included only one nutritional 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 nutritional measures (Model IV). A final model was run which included all three nutritional variables and interaction terms between each nutritional variable and age (Model V), to determine whether any of the associations varied by age. Non-linear associations of all measures of nutritional 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. Exploratory analyses tested whether results were robust to alternative model specifications, including models run with and without birth cohort variables, and models run with and without variables for calendar time. Substantive conclusions were the same regardless of whether or not birth cohort or calendar time were included.

In all models, only cases where all three nutritional 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

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 years, median

age at death for men is 67. Mortality rates in adulthood are similar for women and men (Figure 2; in line with Billewicz and McGregor's (1981) observation using data from two of these villages that sex differences in mortality were minimal). 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 nutritional measures

To test for correlations between nutritional 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 (β=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: β=0.190, SE=0.017, p<0.01; for height: β=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). Despite some collinearity, parameter estimates were similar, and substantive conclusions the same, whether nutrition variables were entered into models separately or simultaneously.

Event history analysis of associations between nutrition and adult mortality

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 men, Table 2(b) the results for women (models I, II and III include only one measure of nutritional status – height, BMI and haemoglobin respectively; model IV includes all three nutritional measures; model V includes all nutritional measures

and interactions between nutritional 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 40-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 nutritional status and mortality is very similar for both sexes. This 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 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 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 association between haemoglobin and mortality risk is greatest for young women, but decreases as women age. The magnitude of this association is small, however.

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

Discussion

Our results show that, roughly speaking, there is a negative association between the more 'liquid' measures of nutritional status – BMI and haemoglobin – and adult mortality risk: higher BMI and haemoglobin is associated with a lower probability of dying, for both sexes. This suggests that low values on these measures are markers of poor health. Our 'illiquid' measure of nutrition – height – demonstrated more complex associations with mortality. Height was only associated with adult mortality for women, not men, and the relationship between height and mortality for women is not linear. It's worth noting that there was no clear secular trend in mortality during the time period studied here: both Billewicz and McGregor's (1981) description of the demography of two of these villages, and our own exploratory analyses (not shown) demonstrated no clear links between calendar time and mortality; Billewicz and McGregor (1982) note that there were few notable secular trends in living conditions over this time period. Mortality fluctuated in this population between and within years – it was a very seasonal population, in which food stress and disease load were higher during the 'hunger' season each year; then alleviated during the 'harvest' season. Child, but not adult, mortality was higher in the 'hungry' season (Billewicz and McGregor, 1981). There was also year-to-year variation in food availability and disease load, which affected child mortality, though such fluctuations are harder to detect in adult mortality. We did not attempt to explore any interactions between calendar time (or birth cohort) and our nutritional status variables, both because there were not clear secular trends, and also because our data don't allow to fully disentangle period effects, from cohort or age effects; we only study mortality over a 24-year time period, and so don't have full lifecourse data for our birth cohorts. We did, however, check our substantive conclusions were robust to alternative model specifications (including or excluding birth cohort and calendar time, for example).

The higher mortality of short women may possibly be at least 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 household resource access), then this may explain the correlation between short stature and high mortality. However, the association between height and mortality was not attenuated when we included height, BMI and haemoglobin simultaneously in models, which ought to effectively control for poor condition in adulthood. Instead, adverse environment in early life (particularly during the foetal period) may result in long-term physiological changes which are associated with a higher incidence of certain diseases in later life, which may partly explain associations between height and adult mortality (Barker, 1994). Most of this research has focused on the association between adverse early life conditions and higher rates of chronic diseases in high income 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 have higher mortality in adulthood than those born during the 'harvest' season (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, but has also been observed elsewhere: in different Dutch samples from the nineteenth century, both women (Thompson et al., 2022) and men (Thompson et al, 2020) were observed to experience higher mortality at taller heights. When cause-specific mortality is investigated, studies in high income populations have shown increased mortality from certain cancers in taller individuals, but 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 given height ranges observed in different populations: 95% of women were 167cm or less (approximately 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. Thompson and colleagues (2020) suggest that in high mortality populations, such as their historical population, selection effects may explain the higher mortality of taller individuals; since those who experienced poor conditions in childhood and ended up as shorter adults, would only have survived their poor childhood if they were particularly robust. 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 could ultimately lead to higher mortality rates (but see further discussion below about physiological strategies which might mitigate these costs of reproduction).

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: 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 higher or lower income world) can partly be explained by the physiological 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 Sauther, 2002; Ellison, 2003). Women appear to delay reproduction until they are in suitable condition to bear such a high energetic cost, for example. Research 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). Finally, women store fat in metabolically less active adipose tissue depots (gluteo-femoral fat) which reduces the cardiometabolic risk associated with higher adiposity (Karastergiou et al, 2012).

For haemoglobin, there is a more consistently positive association between higher haemoglobin and lower mortality rates, with no evidence of increased mortality at high

haemoglobin levels. 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).

We conclude with the observation that results from our analysis, combined with other published studies, suggests that ecological factors, including historical context, seem to affect the relationship between height and mortality, but perhaps matter less for the (shape of 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), probably related to universal penalties for low lean, and high fat, mass, regardless of context. The absolute BMI level at which mortality risk is lowest, however, varies between populations, and is lower in populations in which BMI is lower on average.

The height-mortality relationship seems to be more variable across different contexts (see also Sear, 2010). Broadly speaking, all-cause mortality for men in high income populations often decreases with increasing height, where deaths are predominantly from non-communicable diseases (e.g. Waaler 1984; Engeland et al., 2003; Peck and Vagero, 1989; Jousilahti et al., 2000; Song et al., 2003; Ong et al., 2013); though occasionally no association is found even in high income populations (e.g. Liao et al., 1996). Negative associations between height and mortality for men are not so common in populations with lower nutrient intakes (but see Alter et al., 2004), and where a higher proportion of deaths are from infectious causes, such a historical populations or contemporary lower income populations: a number of studies have found no association (Murray, 1997; Alter et al., 2004; Wilson 2019) or positive (Thompson 2020) or U-shaped (Wilson, 2019) associations between height and adult mortality for men. Relationships between height and mortality for women appear to be even more variable; while negative associations between female height and adult mortality are also quite commonly seen in high income populations (Waaler, 1984; Peck and Vagero, 1989; Jousilahti et al., 2000; Smith et al., 2000; Song and Sung, 2008), null associations (Liao et al., 1996) U-shaped associations (Laara and Rantakallio, 1996; Engeland et al., 2003) and positive associations have also been observed (Rohrmann et al., 2017). In lower or middle income and historical populations, null (Hosegood and

Campbell, 2003; Wang et al., 2011), positive (Thompson et al., 2022) and U-shaped (this study) relationships have been observed, but, as far as we're aware, not negative associations.

Tall height brings both costs and benefits, and these may differ between environments. Height 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, women with earlier first births tend to have shorter height in adulthood than women with later first births (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, which hold universally in all populations (see Sear et al., 2004; Sear 2006 for more discussion of associations between nutritional status and reproductive outcomes for women and men respectively in this population). However, an overarching conclusion is that ecology affects the relationship between height and mortality because different ecologies result in differing patterns of mortality by cause. Prior to the epidemiological transition, the relationship may be quite variable between populations, given differing patterns of causes of death. Over historical time, associations between height and mortality may become more consistently negative, given the clearer associations between taller height and lower rates of deaths from most noncommunicable diseases, particularly for men. For women, associations between height and mortality may have an extra layer of complexity because of interactions between height, reproductive factors and health (just as one example, height is positively associated with risk of death from reproductive cancers, and reproductive cancers are more prevalent in populations with lower fertility: Green et al., 2011).

Conclusion

There is clear evidence that nutritional status in this population is associated with adult mortality rates for both sexes. Both nutritional status (BMI) and haemoglobin level were independently related to mortality risk. The relationship between these measures of body condition and mortality is roughly linear, at least over the range of values within which most people fall: as body condition increases, the probability of death decreases. There do seem to be some costs to energy acquisition among those individuals who acquired

sufficient energy to become overweight, but this accounts for a very small proportion of the population. These relationships are similar for both sexes, despite some differences in average values between the sexes (for haemoglobin but not BMI), and differences in the association between these nutritional measures and age. Height shows somewhat different associations with mortality, both in that significant associations are seen only for women and that the relationship is U-shaped rather than linear. These three measures of nutritional status - BMI, haemoglobin and height – may all have independent effects on the probability of death because they are, as our framework suggests, indicators of different types of capital, and measures of 'liquid' capital are more strongly and consistently associated with mortality than 'illiquid' capital.

Acknowledgements

Thanks to the Keneba Data Steering Committee for permission to use the data.

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Table 1: Summary of data on adult nutritional status[1]

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

[1] 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)
[2] Excluding measurements taken during pregnancy
[3] 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)†	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 1. Schematic diagram illustrating how the human body incorporates different components of nutritional status in the form of 'embodied capital'. These capital components vary in the rate at which they may be gained or lost from the body (liquidity). Illiquid components of adult capital carry a strong signal of nutritional status during development and cannot change in adult life, whereas liquid components of capital reflect recent exposure to factors such as dietary supply and infection, and hence either short term accretion or depletion.

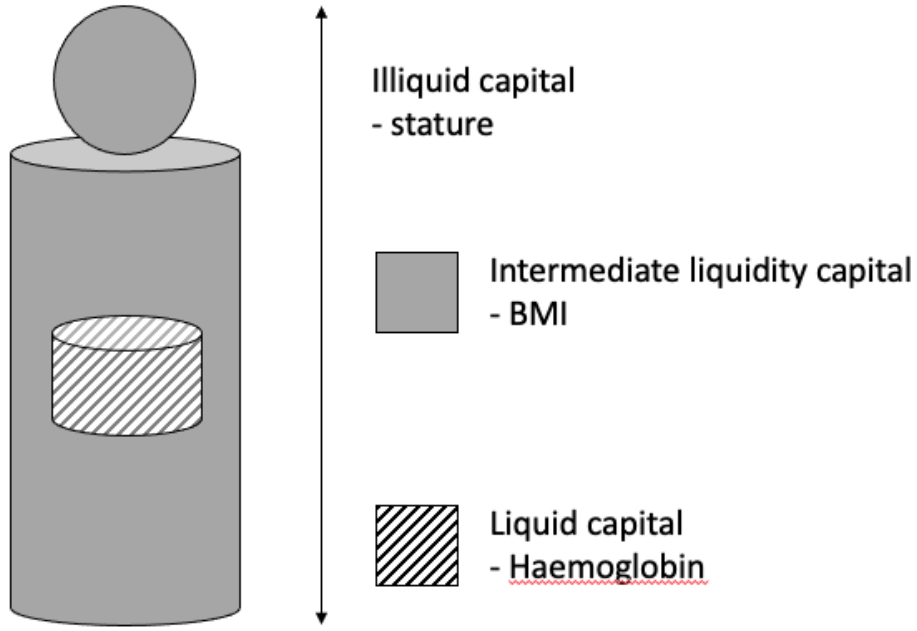


Figure 2: Kaplan-Meier plot showing survival function of women (solid line) and men (dotted line) who survived to at least 21 years. N=1125 women, of whom 208 died; N=968 men, of whom 201 died.

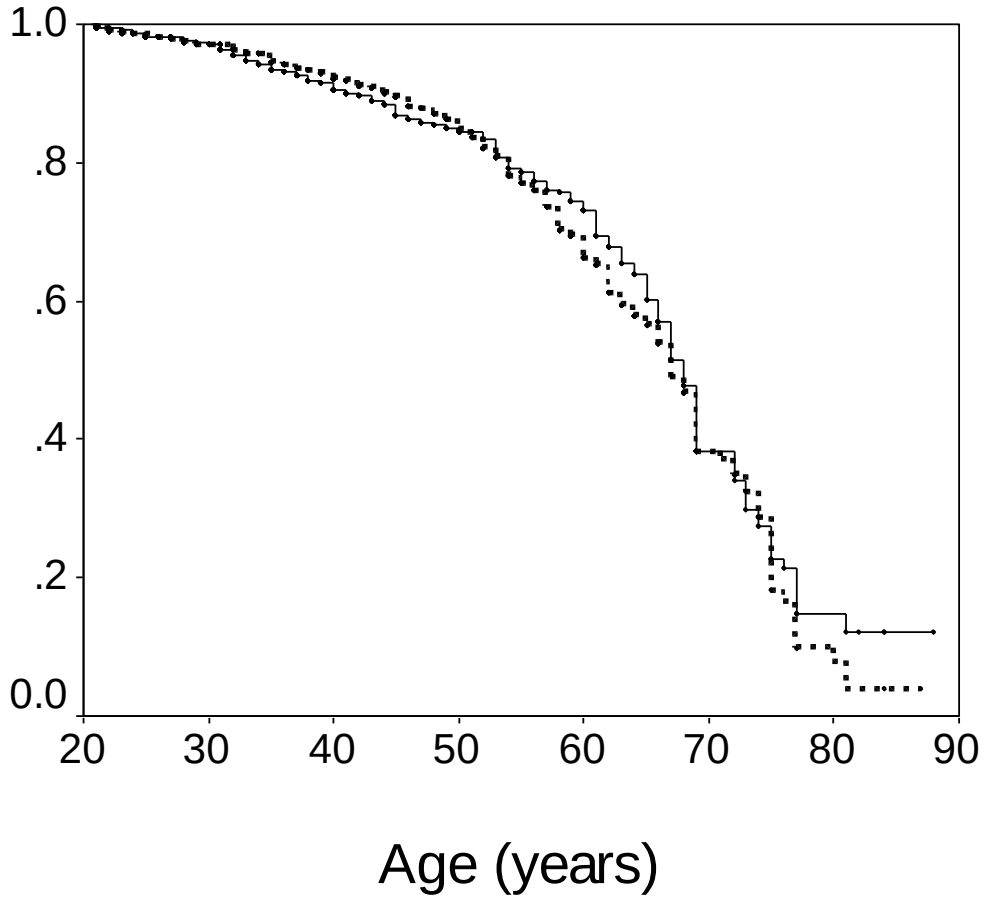


Figure 3: Model predictions of probability of dying per year by height (a), BMI (b) and haemoglobin (c). Solid line represents women, dotted line men (men not plotted in 3a). Model includes all three nutrition measures, a BMI squared term, and interaction between haemoglobin and age, age and birth cohort; predictions calculated at age 40, 1920s birth cohort, and at average values for nutrition variables not being plotted.

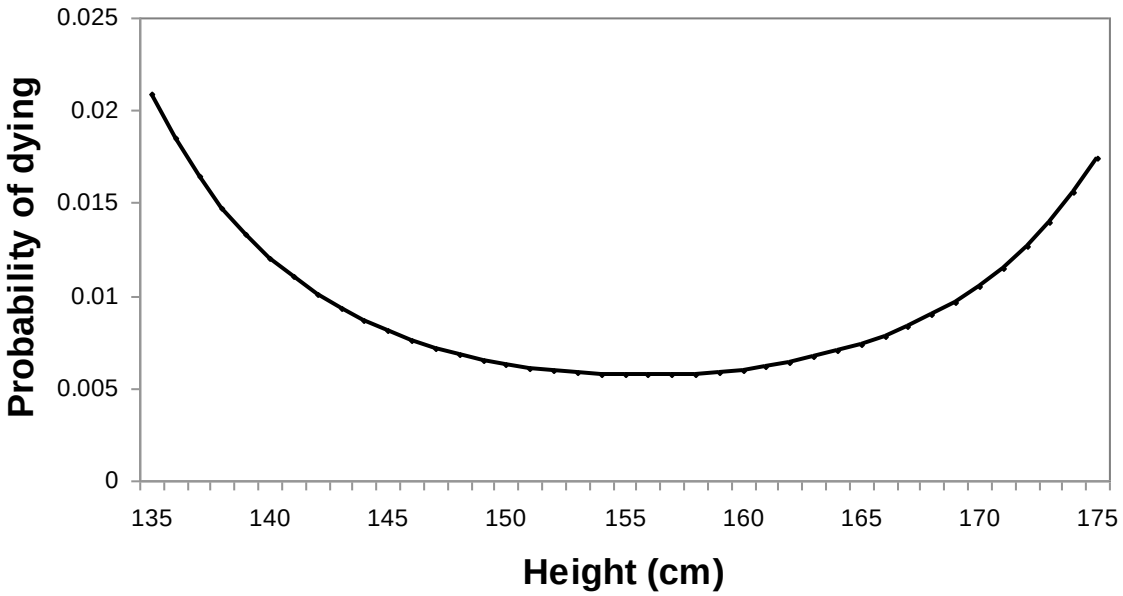


Figure 3 (b)

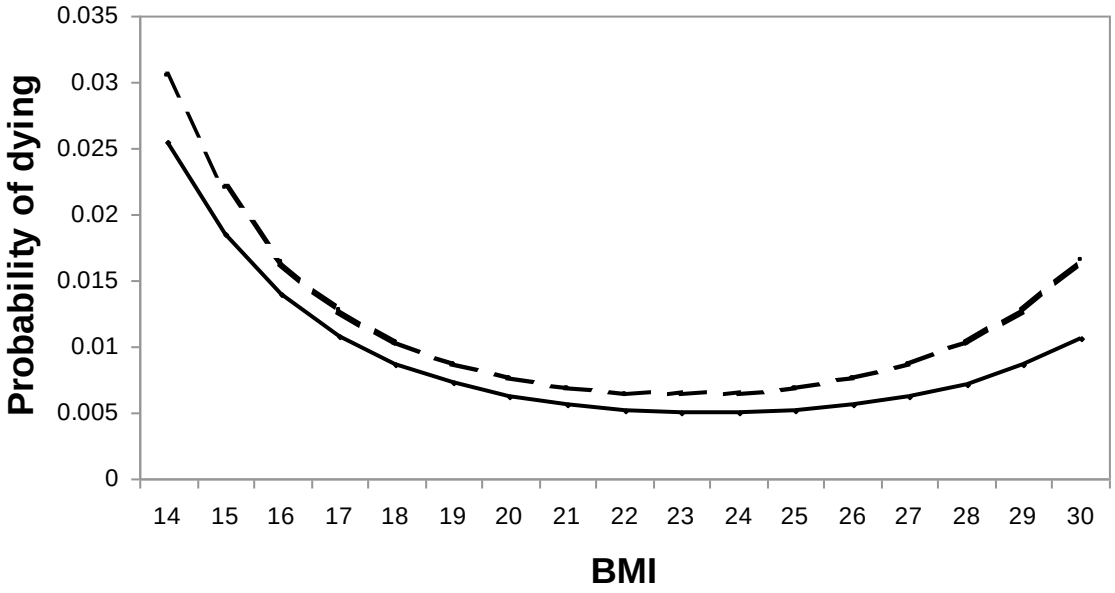
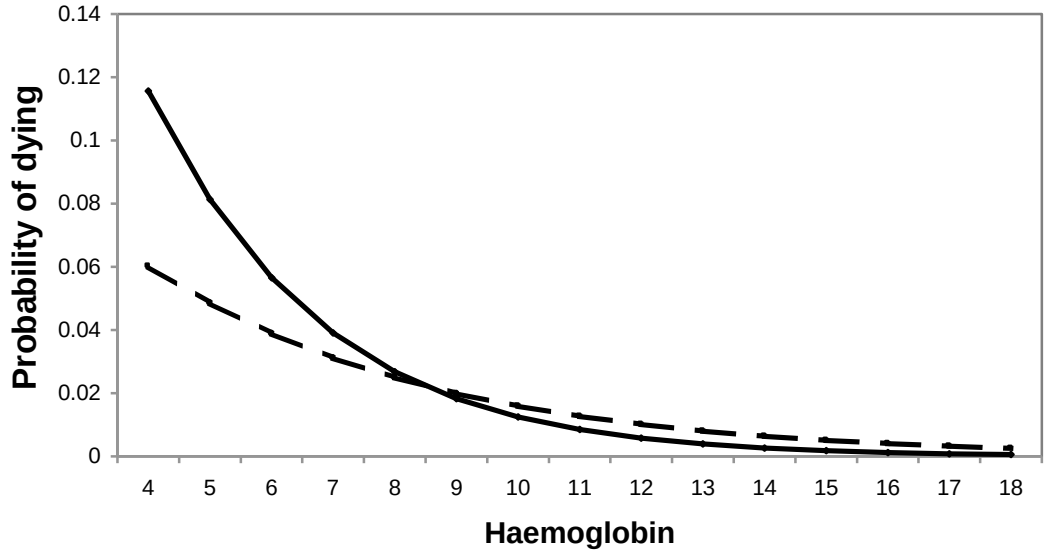

Probability of dying
0.035
0.03
0.025
0.02
0.015
0.01
0.005
0
14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30
BMI

Figure 3 (c)


0.14
0.12
0.1
0.08
0.06
0.04
0.02
0
Probability of dying
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
Haemoglobin