Size-dependent reproductive success in Gambian men: does height or weight matter more?

by

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Abstract

Size is an important component of life history analysis, as it is both a determinant and an outcome of life history decisions. Here, we present an investigation of the relationships between two components of size (height and weight) and life history outcomes for men in a rural Gambian population. This population suffered seasonal food shortages and high disease loads, and lacked access to medical care or contraception. We find that there is no relationship between height and mortality among adult men. Tall men also do not have more children than shorter men, though they do contract slightly more marriages than shorter men. Tall men, therefore, do not seem to have higher reproductive success in this Gambian population. Instead, weight (measured by BMI) appears to be a better predictor of life history outcomes, and ultimately reproductive success, in this population. Heavier men have lower mortality rates, contract more marriages and have higher fertility than thinner men.

Introduction

Size is an important component of life history analysis, as it is both a determinant and an outcome of life history decisions (Roff 1992; Stearns 1992). For example, adult size is often the result of a trade-off between growth and reproduction – in many species growth ends when reproduction begins, so that individuals who start reproducing early end up as small adults (Allal *et al.* 2004; Stearns and Koella 1986). Size then determines demographic outcomes in adulthood, such as mortality and fertility. Larger individuals tend to have lower mortality and higher fertility rates (Gaillard *et al.* 1989; Harvey and Clutton-Brock 1985). Size has costs, however (Blanckenhorn 2000). If reproduction only begins when growth ends, individuals who grow for long periods to become large adults may lose out to their competitors by starting reproduction relatively late (and also risk dying before they can begin reproducing). Larger individuals also need greater energy intakes to maintain their large bodies, requiring greater time spent in food acquisition. The aim of this paper is to explore the effects of size on life history outcomes, and ultimately reproductive success, in a population of Gambian men.

Size can be measured in more than one way. A simple measure might be the overall weight of an individual. However, in capital breeders (species which store energy for reproduction) such as *Homo sapiens*, weight confounds two variables. It is a composite of both stature (or the size of the skeleton) and body composition (individuals with both more stored fat and greater lean tissue will be heavier than those with less). Both overall stature and body composition are likely to have an impact on life history variables. Stature is an indicator of chronic energy availability – individuals who experienced low energy availability throughout their growth period will become smaller adults than those who had greater resource access during growth (Silventoinen 2003). But it is also correlated with life history outcomes for reasons unrelated to energy availability. For example, in many species, overall size affects mortality risk because smaller individuals are more at risk of predation than larger, hence providing one explanation for the correlation between size and mortality risk (Roff 1992). Body composition, on the hand, is a good indicator of the energy available during adulthood itself. Individuals with low body mass will have relatively few energetic reserves to devote both reproducing and maintaining body condition, and may have lower reproductive and higher mortality rates than those of greater body mass (Glazier 2000). Additionally, males with greater lean tissue, and therefore muscle mass, are likely to out-perform their weaker rivals in competition for mates. In our own species, these two components of size can be measured using height and BMI (body mass index, height/weight^2, a measure of fatness: Ferro-Luzzi *et al.* 1992).

Previous research on humans has shown that height is linked with life history outcomes. Taller individuals tend to have lower mortality rates than shorter individuals (Marmot *et al.* 1984; Waaler 1984), though the universality of this relationship has recently been disputed (Samaras *et al.* 2003). Analysis of cause-specific mortality suggests that taller individuals have lower mortality for some causes of death, such as cardio-vascular disease, but for other causes of death, including several cancers, there is either a positive association between height and mortality or no correlation at all (Barker *et al.* 1990; Leon *et al.* 1995; Smith *et al.* 2000; Song *et al.* 2003). Height has also been linked to marital and fertility outcomes in men. In Western populations, tall men have more marriages, are less likely to be childless and have more children than shorter men (Mueller and Mazur 2001; Nettle

2002; Pawlowski *et al.* 2000), which ultimately leads to a positive correlation between height and reproductive success.

A potential problem with a full understanding of the relationship between height and life history outcomes is that much of the research cited above has been done in modern Western populations (but see Costa 1993; Kemkes-Grottenthaler 2005; Murray 1997 for research on the height-mortality relationship in historical Western populations). Given that the relationship between height and mortality appears to depend on cause of death, and that the main causes of death are known to differ between environments, it is also possible that the height-mortality relationship may differ between the well-nourished West and populations living under greater environmental stress. The determinants of other life history outcomes, such as marital and fertility rates, also differ between environments. The little research done on height and fertility outcomes in non-Western populations suggests that ecology also needs to be taken into account when investigating the link between height and reproduction. Tall men, for example, have higher reproductive success, in terms of number of surviving offspring, in a !Kung population and among a rural Bantu-speaking population in Namibia, but among urban Namibian Bantu-speakers shorter men have higher reproductive success (Kirchengast 2000; Kirchengast and Winkler 1995).

Previous research on weight and mortality has also shown clear evidence of a correlation. Thin individuals have been shown to be at high risk of death in both developed and developing world populations (Engeland *et al.* 2003; Flegal *et al.* 2005; Hosegood and Campbell 2003; Rissanen *et al.* 1991; Rotimi *et al.* 1999). However, over-weight individuals also seem to have higher mortality than those of medium weight (Ajani *et al.* 2004; Engeland *et al.* 2003; Murray 1997). Having few energetic reserves to spend on maintaining body condition is risky, then, but there are physiological costs to excess energy storage, so that the optimum weight may be moderate, rather than excessive, fatness.

Weight and fertility outcomes are less well studied, at least in men, but there is considerable indirect evidence that they are linked. Energetic resources correlate with reproductive endocrine function in men both within and between populations (Bribiescas 1996; Bribiescas 2001), and this variation may affect male fertility. For example, disease load (which will be correlated with energetic reserves – men with greater energetic reserves have more energy available to devote to immune function) has the potential to affect male fecundity. Fevers have been shown to negatively affect sperm production (McFalls and McFalls 1984), and may adversely affect other requirements of successful reproduction, such as coital frequency. But weight does not only measure relative fatness, it is also an indicator of lean tissue and muscle mass (Norgan, 1990; Della Bianca *et al.*, 1994). More important than their effects on spermatogenesis and coital frequency may be the effect energetic reserves and disease burden have on investment in muscle tissue (Muehlenbein, 2006). Male reproductive success is heavily dependent on attracting, competing for and keeping mates, which is likely to be affected by muscle mass. Larger men with greater muscle mass have higher physical work capacities and are more productive than smaller men (Ferro-Luzzi, 1985; Spurr, 1988), and may therefore be more attractive as mates. The drawback of muscle tissue is that it is expensive to maintain, and men of higher body mass have greater energetic requirements than smaller men (see Della Bianca *et al.*, 1994 for evidence from this Gambian population). Men with high muscle mass might then suffer during periods of food shortage, so that muscularity might not be entirely beneficial in the variable environment in which these Gambian men.

Aims of this study

Much of the research looking at relationships between size (both height and weight) and life history variables such as mortality and reproductive outcomes has been carried out on Western populations. While these populations are certainly not uniform, they do share some rather unusual demographic and anthropometric characteristics when compared to most human populations throughout our history. Mortality and fertility rates are low, and individuals are relatively tall and fat (over-nutrition is now a more serious health problem than under-nutrition). The aim of this study is to examine the relationship between male height, weight and life history variables in a rural Gambia population, which suffered seasonal food stress and where medical care was absent.

Data

The data were collected from 4 villages in rural Gambia, largely by Ian A. McGregor, a medical doctor funded by the UK Medical Research Council (MRC). McGregor carried out a research programme in these villages between 1950 and 1980 (McGregor 1991; McGregor and Smith 1952). In 1974, another MRC-funded team, the Dunn Nutrition Unit (DNU), also began research in the area. Data on births and deaths have been collected systematically since 1950 for all villages. Anthropometric data were collected by McGregor during surveys undertaken at least once a year between 1950 and 1980. McGregor also collected additional demographic information from villagers during these surveys, including details of marriages for 2 of the 4 villages. Little medical care or contraception were available during the first 25 years of the study, though some medical treatment was available when McGregor and other researchers were present in the villages. During this period, the villages were agricultural, farming rice and millet as primary staples. The diet was essentially vegetarian. There were considerable seasonal and annual fluctuations in food availability and disease prevalence.

Data are still being collected today at this field site, but in 1975 the DNU established a permanent medical clinic in one of these villages (Keneba) leading to a dramatic change in the demography of these villages (Weaver and Beckerleg 1993). Much of the analysis that follows (except where otherwise stated) is confined to the period between 1950 and 1974. This restriction was used partly because of the dramatic change in demography as a result of the medical clinic but also for reasons of data availability and quality. Systematic surveys of all villagers were no longer carried out after 1980, and the focus of research shifted to women and children, so that relatively little information on adult men is available after this date. The quality of demographic data is also considered somewhat more questionable after 1980.

Between 1950 and 1980, men in this population were relatively short and light, but the majority were within the limits of BMI considered to be adequately nourished according to international standards. Table 1 shows descriptive statistics for all anthropometric and demographic variables used in this analysis. A mean adult height and BMI for each individual was calculated by taking the average of all measurements obtained from that individual after the age of 20 years (BMI fluctuates over time but the measurements taken from a single individual are highly correlated, so that this average figure should be good indication of the individual's overall condition). From these averages, mean height of men in this population was 168cm (about 5'6) and mean BMI 20.4. 86% of men had a BMI of between 18.5-24.9 (considered the cut-off points for adequate nutritional status); a small proportion were underweight (13% had a BMI below 18.5) but very few were overweight (0.8% had a BMI of 25 or more) and none obese (with a BMI of 30 or more). Individuals tended to lose weight during the rainy season, when food shortages were combined with an increase in agricultural workload and disease prevalence, but gained weight again during the 'harvest' season (McGregor, 1976). The great majority of anthropometric measurements were taken during the harvest season, when villagers were more likely to be available because of their lighter work burden.

Methods

To estimate the relationship between size and life history outcomes in men in this population, we first analysed the effects of both height and BMI on the probability of death during adulthood for men of all ages between 1950 and 1974. Then we investigated the impact of size on marital and fertility outcomes for those men nearing the end of their reproductive lives (defined as having reached the age of at least 50) by 1975, in order to determine whether size was related to variables correlated with reproductive success.

Mortality of all adult men

The effect of size on the probability of dying in adulthood (*i.e.* from the age of 21 years) was analysed using discrete-time event history analysis (EHA). We used age 21 to define adulthood as most men would have reached their final adult height by this age. Despite high child mortality in this population, men who survived childhood could expect to live relatively long lives: the median age at death for those who survived to age 21 was 67 years (Table 1). 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 (Steele *et al.* 1996; Yamaguchi 1991). 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 were censored in 1975) 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).

Height and BMI were entered into separate models to test for their effects on the probability of death in adulthood. Models were run separately for height and BMI since the two variables are not entirely independent (although in fact exploratory analysis demonstrated that the substantive results are unchanged whether the size variables are entered separately or simultaneously into statistical models). Non-linear effects of both measures of size were tested for by including quadratic terms, and by calculating quartiles of each anthropometric measure and including dummy variables for three of the four quartiles. EHA assumes proportional hazards: this assumption was tested by including interactions between age and both size variables. These interactions were not significant so were dropped from the final models. The models controlled for birth cohort. Exploratory models also controlled for village of residence, but village appeared to have little relationship to adult mortality risk so this variable from dropped from the final models.

BMI was included as a time-varying covariate. Few individuals were surveyed in every year between 1950 and 1980, so a mean BMI 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 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. All 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.

Height is clearly less variable with age than BMI, 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.

Marital and reproductive outcomes of men 50+

Sample

For the marriage and fertility analyses, we restricted our analysis to a sub-sample of the population: we only included men nearing the end of their reproductive lives (*i.e.* had reached at least the age of 50 years) in 1975, and only men from the two villages with marriage data[1]. Restricting the analysis only to older men was done for both theoretical and practical reasons. Restricting the sample to men nearing the end of their reproductive lives gives the best indication of how size impacts on overall reproductive success in this population. This restriction also reduces the impact of data limitations on the analysis. For example, we cannot use EHA to investigate marital success for men of all ages as we have very few dates of marriage in our dataset, so that we are limited to investigating the impact of size on the total number of marriages contracted. Including men of all ages in this analysis would introduce confounds due to censoring. In addition, the great majority of the children fathered by older men would have been produced before 1975 when demographic data collection was reasonably accurate, and before the medical clinic had a substantial impact on mortality and fertility rates (in this population only 17% of children were born to fathers of 50 or older). The fertility analyses were restricted to the sub-sample of men from the two villages with marriage data partly because the fertility information was thought to be more complete for these villages but also because the final regression model in this section includes number of marriages as a covariate. Restricting all analyses to men from the two villages with marriage data therefore means that the results of all regression models in this section can be directly compared.

[1] We have, in fact, run all the fertility analyses described here on samples using men from all villages and of all ages, and the results are very similar to those obtained from analyses using only the sample described here

Almost all men in this sample were reported to have been married at least once: only 2% had no recorded marriages (see Table 1 for descriptive statistics on marriage and fertility). No analysis was therefore performed on the likelihood of marriage. There was much more variation in the number of marriages men contracted, both because this is a highly polygynous society, and because divorce was not uncommon. Of this sample, 86% had been married more than once (not necessarily polygynously), and 62% had had more than 2 wives. The average number of marriages was 3.27 (note this is the average total number of marriages, rather than current number of wives). Childlessness was also relatively uncommon in this sample: only 5% of these men had no recorded children. The average number of children born was 9.35, of whom 5.14 survived to age 14. For those men who did have children the average age at first birth was 34 years.

Analysis

To investigate the impact of size on male marital and reproductive outcomes, we performed 5 separate linear regression analyses. The dependent variables are listed below for each model:

1. Model 1: number of marriages contracted
2. Model 2: age at first birth
3. Model 3: total number of children born
4. Model 4: number of children surviving to age 14
5. Model 5: number of children surviving to age 14 (controlling for number of marriages)

Each model was run twice: the first time to test for an effect of height; the second to test for an effect of BMI. Again, models were run separately for height and BMI since these two variables are not entirely independent (but again the results were similar if height and BMI were entered into the same models). The height and BMI measurements included in these models were average measurements. For each man, an average height and an average BMI was calculated from all measurements taken after the age of 20. Though BMI (and, to a lesser extent, height) clearly changes over time, repeated measurements from each individual are highly correlated. These average measurements were thought to adequately capture an individual's size. Additional models were run controlling for the average age at which an individual's measurements were taken, and the number of times an individual had been measured, in case biases might be introduced due to age at, or frequency of, measurement. These controls made no difference to the results, so were not included in the final models. As with the mortality analysis, exploratory models were run to test for non-linear effects of both height and BMI.

In all 5 analyses, the man's age at death or censoring was included as a covariate to control for the fact that not all men will have completed reproduction by the age of 50. Any man without a known date of death was censored at the date when they were last known to be alive. Age was estimated for individuals born before 1950 based on both physical characteristics, and social age group (all individuals belong to *kafo*s – groups of individuals of similar age – and age-ranking of each *kafo* is known). Some men in this sample will have begun reproducing before 1950 when systematic data collection began. McGregor attempted to reconstruct the fertility and marriage histories of men before 1950, but it is likely that data are less complete for events that occurred before 1950 than those that occurred after the demographic surveillance system had been set up. Birth cohort is also controlled for in all models to reduce the effects of this potential bias. There were some differences in marriage and fertility outcomes between the villages in this study (Billewicz and McGregor 1981), so a control for village was also included in all models.

Models 1-4 therefore included identical independent variables (either height or BMI, village, birth cohort and age at death or censoring) but each had a different dependent variable. Model 1 aimed to test the effect of size on the number of marriages contracted. Models 2 to 4 aimed to test for an effect of size on age at first birth, total number of children born and number of children surviving to age 14. The last outcome is considered to be the closest proxy of reproductive success. Model 5 aimed to test for an effect of size on reproductive success, but controlling for the number of marriages each man had contracted. Size can potentially affect male reproductive success in two ways: either by affecting the number of marriages a man can contract or by directly affecting fertility outcomes. Model 5 was used to determine whether there was an additional effect of size on fertility, even when controlling for the number of marriages.

Results

Mortality of all adult men

Table 2 shows the results for both event-history models of the probability of dying: one testing for an effect of height and one testing for BMI (control variables are not shown in either Table 2 or Table 3 for clarity). There was no relationship between male height and the probability of death in adulthood, regardless of whether height was modelled as a linear or non-linear function. BMI was significantly correlated with the probability of death. The relationship between BMI and mortality was not linear, as the best fitting model included terms for both BMI and BMI squared. Visual inspection of this relationship indicates that under-nutrition is a more serious risk to men's survival chances than over-nutrition. Figure 1 shows the model predictions of the probability of dying across the observed range of BMI in this population. Mortality risk did start to rise in men considered overweight by international standards (*i.e.* had a BMI of 25 or more), but there were very few overweight men in this population. Men at the very bottom of the BMI distribution (BMI of 13) showed much higher risks of death per year than those at the very top of the observed BMI distribution (BMI of 29).

Marital and reproductive outcomes of men 50+

Marriage

Figures 2a and 2b show scatterplots of number of marriages contracted against height and BMI respectively, and include the fitted univariate linear regression lines. These plots suggest that both size variables are positively correlated with the number of marriages contracted. The results of the regression models displayed in Table 3 confirm this. Table 3 shows the parameter estimates, standard errors and significance levels for the height and BMI variables in each of the 5 regression models run on marriage and fertility outcomes (control variables are excluded from the table for clarity). The results of Model 1 show that both height and BMI are significantly and positively related to the number of marriages men contracted, so that taller and heavier men had more wives than shorter and lighter men. Both relationships appear to be linear as including non-linear functions for either height or BMI did not improve the fit of the models.

Fertility

Figures 3a and 3b show scatterplots of reproductive success (number of children surviving to age 14) against height and BMI respectively, and also show the fitted univariate regression lines. These figures suggest that height is not significantly related to the number of surviving children, but that BMI is significantly and positively correlated with this outcome. The results of the linear regression analyses shown in Table 3 confirm this. Models 2-5 show that height was not significantly related to any of the fertility measures, though the results of all models were in the expected direction (*i.e.* taller men had a lower age at first birth, more children and more surviving children). Including non-linear terms for height did not result in significant correlations between height and any fertility outcome. BMI was, however, significantly related to all fertility outcomes: heavier men had more children, more surviving children and a lower age at first birth. Model 5 investigated the effect of size on number of children surviving to age 14, controlling for the number of marriages contracted. The results of this model suggest that BMI remains significantly correlated with number of surviving children, even when controlling for number of marriages, though the size of the parameter estimate is reduced slightly. As expected, number of marriages was also significantly positively related to the number of surviving children (the parameter estimate for number of marriages in the regression model which included BMI as the size covariate was 0.75, SE=0.17 , $p < 0.001$). Again, including non-linear terms for BMI did not improve the fit of the models.

Discussion

In the Gambia, male height was not correlated with either mortality or fertility outcomes, though tall men did contract slightly more marriages than shorter men. Weight instead appears to be a better predictor of reproductive success, as men with higher BMIs had lower mortality rates and both more marriages and more children (note that very few men in this population were overweight, so that heavier men were those within the range considered a healthy weight, rather than over-nourished men). There was some indication that the relationship between BMI and mortality was curvilinear in that, though under-nourished men had the highest mortality rates, men at the top of the BMI distribution also appeared to have slightly higher mortality than those of average BMI. Few men were at the upper end of the BMI distribution, however, so that the detrimental consequences of low weight were likely to be of more functional significance. The relationships between BMI and both marriage and fertility were linear.

These results contrast somewhat with research done on Western populations. In the West, taller men have higher reproductive success than shorter men, apparently because of their greater success on the marriage market. Though there was a small positive correlation between height and number of marriages in this Gambian population, there is little other evidence that height matters to either men or women on the marriage market. In Western populations there is frequently positive assortative mating for height, so that taller men marry taller women (McManus and Mascie-Taylor 1984; Sanchez-Andres and Mesa 1994). There is also a strong 'male-taller' norm: marriages in which the husband is shorter than the wife are rare (Gillis and Avis 1980). Both observations are indications that height matters when Western individuals are choosing a mate. There is no such evidence for either assortative mating for height or a male-taller norm in this Gambian population, suggesting height is not an important factor on the marriage market (Sear *et al.* 2004).

This lack of evidence for strong mate preferences for height in the Gambia suggests the small positive correlation between height and number of marriages may be brought about by factors other than female choice for tall men. Taller men may be wealthier in this population, and thus able to afford more wives. Regardless of the mechanism for this relationship, it is rather puzzling that the positive relationship between number of marriages and height does not translate into higher reproductive success for tall men (since number of wives is a significant predictor of reproductive success for men in this population). Though the relationships between height and number of marriages and between number of marriages and reproductive success are statistically significant, the r^2 values suggest the proportion of variance explained in each model is rather small ($r^2 = .10$ for the regression on number of marriages including height as a covariate; $r^2 = .20$ for the regression on number of children surviving to 14 including both height and number of marriages). Given that marriages are rather unstable in this society, and that a relatively high proportion of marriages did not produce children (~30% of marriages were childless), the number of marriages may not necessarily be a very strong predictor of reproductive success for men in this population. Further exploration of this dataset suggests that taller men may have been more likely to have childless marriages than shorter men (results not shown). The reasons for this are unclear, but may provide an explanation for the lack of an association between height and reproductive success, despite a positive correlation between height and number of marriages.

Even if we set aside the issue of marriage, the lack of a relationship between male height and reproductive success is perhaps surprising. We might have expected to see a positive correlation between height and both survival and reproduction in a food-stressed population such as this because tall height should correlate with relatively good environmental conditions in childhood. Good conditions in childhood should correlate with positive demographic outcomes in later life both because a good environment in childhood may well be correlated with similarly good conditions in adulthood, and because poor conditions in childhood can result in physiological changes which result in poor health and higher mortality in adulthood (Barker 1994; Gluckman *et al.* 2007). But human populations vary considerably in their overall level and variability of energy availability, disease load, prominent causes of death and speed of growth: all factors which may affect the height-mortality relationship. Speed of growth, for example, may be important because laboratory studies on rodents have suggested there may be costs to fast growth in that calorie restricted animals have slower growth but extended lifespans (Rollo, 2002). In this Gambian case, variability of energy availability may be important. In this variable environment, food resources, energetic expenditure and disease load all vary both seasonally and annually. Since larger individuals have higher energetic requirements, tall men may suffer during periods of food shortage because of their higher energetic requirements, cancelling out any health advantages that taller men might have. The other limited evidence from non-Western modern populations does indicate that there may be some variation between populations in the relationship between height and mortality. While some historical or developing country populations show a negative relationship between height and mortality risk (e.g. Costa 1993; Kemkes-Grottenthaler 2005), some show no relationship (Hosegood and Campbell 2003; Murray 1997), or a U-shaped relationship (for women in this Gambian population: Sear *et al.*, 2004). More information on the height-mortality relationship is needed across a range of populations so that a systematic analysis of the causes of this variation can be conducted.

In contrast to the height-mortality relationship, the association between BMI and mortality in Gambian men is more similar to that seen in the West: individuals of both low and high BMI have higher mortality rates than those of adequate BMI. Though this relationship is curvilinear in most populations, there has been some debate about what is the optimal BMI in terms of mortality risk. A few recent Western studies have shown that overweight individuals (i.e. those with a BMI of 25-29) may not have an increased mortality risk compared to those of adequate weight, and that only obesity (BMI>29) increases mortality risk (Flegal *et al.* 2005; Laara and Rantakallio 1996). This has led some authors to argue that the detrimental effects of over-weight are decreasing over time, perhaps because of improved health care (Flegal *et al.* 2005; Henderson 2005). That we see an increase in mortality risk for Gambian individuals who have a BMI of only around 24 or 25 is consistent with this argument. Relatively few Gambian men have such high BMIs, however. Of more relevance to this population is the detrimental effect of under-weight.

What BMI is measuring may also differ between environments. In the well-nourished industrial world, BMI is an indicator of fatness. In leaner populations, while BMI may also provide an indicator of fatness or energy reserves, differences in BMI at the lower end of the scale may reflect differences in lean tissue as well as fat reserves

(Norgan, 1990; Norgan, 1994; Shetty and James, 1994). The high mortality rates of men at the very bottom of the BMI distribution may result from the detrimental consequences not just of a lack of energetic reserves, but a lack of lean tissue as well. The slight increase in mortality risks to the heaviest men indicates that high body weight also has costs in this environment. As with tall men, the heaviest men in this population may also suffer during periods of food scarcity because of their higher energetic requirements.

The relationship between nutritional status and fertility is, to our knowledge, relatively little studied in men, although at least one previous study has found a similar positive relationship between BMI and fertility to that seen here (Kirchengast 2000). Such a positive relationship is to be expected, as nutritional status is likely to be correlated with both wealth and health, and therefore with marital prospects. Wealthy men will be able to afford more wives. Healthier men may also be more attractive as marriage partners to women (see Kurzban and Weeden, 2005 for an example that weight matters, at least when American women are choosing a partner). This analysis confirms that heavier men in the Gambia do have more wives than lighter men. This analysis also suggests that the BMI-fertility relationship may not be entirely mediated by number of wives, however, as even controlling for the number of marriages there is still a significant relationship between BMI and fertility outcomes. This provides evidence that there may be a direct link between BMI and male fertility, through the influence of energetic status on male physiology. An alternative (though not necessarily mutually exclusive) explanation is that there is an indirect link, if healthier men are married to healthier women, and healthier women have higher fertility.

Conclusion

In summary, this paper suggests that there is no association between male height and reproductive success in a rural Gambian population. Here, weight (BMI) is a better predictor of life history outcomes than height: heavier men have lower mortality, more marriages and higher fertility than lighter men, ultimately leading to higher reproductive success. Though the number of marriages men contract is a significant predictor of reproductive success, the relationship between BMI and reproductive success is not entirely mediated by the greater number of wives heavier men acquire. This suggests there may be a direct link between energetic availability and fertility in men. Given that there is clearly variation between populations in the relationship between size and life history variables, we suggest further research needs to be undertaken to explore these relationships across a variety of populations, in concert with further work on the mechanisms which underlie these relationships.

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References

Ajani U.A., P.A. Lotufo, J.M. Gaziano, I.M. Lee, A. Spelsberg, J.E. Buring, W.C. Willett and J.E. Manson. 2004. Body mass index and mortality among US male physicians. *Annals of Epidemiology* 14(10): 731-739.

Allal N., R. Sear, A.M. Prentice and R. Mace. 2004. An evolutionary model of stature, age at first birth and reproductive success in Gambian women. *Proceedings of the Royal Society of London, Series B, Biological Sciences* 271(1538): 465-470.

Allison P.D. 1984. *Event History Analysis: regression for longitudinal event data*. Sage Publications, Newbury Park.

Barker D.J.P. 1994. *Mothers, Babies and Disease in Later Life*. BMJ Publishing Group, London.

Barker D.J.P., C. Osmond and J. Golding. 1990. Height and mortality in the counties of England and Wales. *Annals of Human Biology* 17(1): 1-6.

Billewicz W.Z. and I.A. McGregor. 1981. The demography of two West African (Gambian) villages, 1951-75. *Journal of Biosocial Science* 13: 219-240.

Blanckenhorn W.U. 2000. The evolution of body size: What keeps organisms small? *Quarterly Review of Biology* 75(4): 385-407.

Bribiescas R.G. 1996. Testosterone levels among Ache hunter-gatherer men: a functional interpretation of population variation among adult males. *Human Nature* 7(2): 163-188.

Bribiescas R.G. 2001. Reproductive ecology and life history of the human male, *Yearbook of Physical Anthropology*, pp. 148-176.

Costa D.L. 1993. Height, weight, wartime stress, and older age mortality: evidence from the Union Army records. *Explorations in Economic History* 30(4): 424-449.

Engeland A., T. Bjorge, R.M. Selmer and A. Tverdal. 2003. Height and body mass index in relation to total mortality. *Epidemiology* 14(3): 293-299.

Ferro-Luzzi A., S. Sette, M. Franklin and W.P.T. James. 1992. A simplified approach of assessing adult chronic energy deficiency. *European Journal of Clinical Nutrition* 46(3): 173-186.

Flegal K.M., B.I. Graubard, D.F. Williamson and M.H. Gail. 2005. Excess deaths associated with underweight, overweight and obesity. *Journal of the American Medical Association* 293(15): 1861-1867.

Gaillard J.M., D. Pontier, D. Allaine, J.D. Lebreton, J. Trouvilliez and J. Clobert. 1989. An analysis of demographic tactics in birds and mammals. *Oikos* 56(1): 59-76.

Gillis J.S. and W.E. Avis. 1980. The male taller norm in mate selection. *Personality and Social Psychology Bulletin* 6(3): 396-401.

Glazier D.S. 2000. Is fatter fitter? Body storage and reproduction in ten populations of the freshwater amphipod Gammarus minus. *Oecologia* 122(3): 335-345.

Gluckman P.D., M.A. Hanson and A.S. Beedle. 2007. Early life events and their consequences for later disease: A life history and evolutionary perspective. *American Journal of Human Biology* 19(1): 1-19.

Harvey P.H. and T.H. Clutton-Brock. 1985. Life history variation in primates. *Evolution* 39(3): 559-581.

Henderson R.M. 2005. The bigger the healthier: are the limits of BMI risk changing over time? *Economics & Human Biology* 3(3): 339-366.

Hosegood V. and O.M.R. Campbell. 2003. Body mass index, height, weight, arm circumference, and mortality in rural Bangladeshi women: a 19-y longitudinal study. *American Journal of Clinical Nutrition* 77(2): 341-347.

Kemkes-Grottenthaler A. 2005. The short die young: The interrelationship between stature and longevity-evidence from skeletal remains. *American Journal of Physical Anthropology* 128(2): 340-347.

Kirchengast S. 2000. Differential reproductive success and body size in !Kung San people from northern Namibia. *Collegium Antropologicum* 24(1): 121-132.

Kirchengast S. and E.M. Winkler. 1995. Differential reproductive success and body dimensions in Kavango males from urban and rural areas in Northern Namibia. *Human Biology* 67(2): 291-309.

Kurzban and Weeden. 2005. HurryDate: Mate preferences in action. *Evolution and Human Behavior* 26(3): 227-244.

Laara E. and P. Rantakallio. 1996. Body size and mortality in women: A 29 year follow up of 12,000 pregnant women in northern Finland. *Journal of Epidemiology and Community Health* 50(4): 408-414.

Leon D.A., G.D. Smith, M. Shipley and D. Strachan. 1995. Adult height and mortality in London: early-life, socioeconomic confounding, or shrinkage. *Journal of Epidemiology and Community Health* 49(1): 5-9.

Marmot M., M.J. Shipley and G. Rose. 1984. Inequalities in death--specific explanations of a general pattern? *Lancet* 1(8384): 1003-1006.

McFalls J.A. and M.H. McFalls. 1984. *Disease and Fertility*. Academic Press, London.

McGregor I.A. 1991. Morbidity and mortality at Keneba, the Gambia, 1950-75, in R.G. Feacham and D.T. Jamison (eds), *Disease and Mortality in Sub-Saharan Africa*, Oxford University Press for the World Bank, Oxford, pp. 306-324.

McGregor I.A. and D.A. Smith. 1952. A health, nutrition and parasitological survey in a rural village (Keneba) in West Kiang, Gambia. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 46: 403-427.

McManus I.C. and C.G.N. Mascie-Taylor. 1984. Human assortative mating for height: non-linearity and heteroscedasticity. *Human Biology* 56(4): 617-623.

Mueller U. and A. Mazur. 2001. Evidence of unconstrained directional selection for male tallness. *Behavioral Ecology and Sociobiology* 50(4): 302-311.

Murray J.E. 1997. Standards of the present for people of the past: Height, weight, and mortality among men of Amherst College, 1834-1949. *Journal of Economic History* 57(3): 585-606.

Nettle D. 2002. Height and reproductive success in a cohort of British men. *Human Nature* 13(4): 473-491.

Pawlowski B., R.I.M. Dunbar and A. Lipowicz. 2000. Evolutionary fitness: tall men have more reproductive success. *Nature* 403(6766): 156.

Rissanen A., P. Knekt, M. Heliovaara, A. Aromaa, A. Reunanen and J. Maatela. 1991. Weight and mortality in Finnish women. *Journal of Clinical Epidemiology* 44(8): 787-795.

Roff D.A. 1992. *The Evolution of Life Histories*. Chapman and Hall, New York.

Rollo C.D. 2002. Growth negatively impacts the life span of mammals. *Evolution & Development* 4(1): 55-61.

Rotimi C., I. Okosun, L. Johnson, E. Owoaje, T. Lawoyin, M. Asuzu, J. Kaufman, A. Adeyemo and R. Cooper. 1999. The distribution and mortality impact of chronic energy deficiency among adult Nigerian men and women. *European Journal of Clinical Nutrition* 53(9): 734-739.

Samaras T.T., H. Elrick and L.H. Storms. 2003. Is height related to longevity? *Life Sciences* 72(16): 1781-1802.

Sanchez-Andres A. and M.S. Mesa. 1994. Assortative mating in a Spanish population: effects of social factors and cohabitation time. *Journal of Biosocial Science* 26(4): 441-450.

Sear R., N. Allal and R. Mace. 2004. Height, marriage and reproductive success in Gambian women. *Research in Economic Anthropology* 23: 203-224.

Silventoinen K. 2003. Determinants of variation in adult body height. *Journal of Biosocial Science* 35(2): 263-285.

Smith G.D., C. Hart, M. Upton, D. Hole, C. Gillis, G. Watt and V. Hawthorne. 2000. Height and risk of death among men and women: aetiological implications of associations with cardiorespiratory disease and cancer mortality. *Journal of Epidemiology and Community Health* 54(2): 97-103.

Song Y.M., G.D. Smith and J. Sung. 2003. Adult height and cause-specific mortality: a large prospective study of South Korean men. *American Journal of Epidemiology* 158(5): 479-85.

Stearns S.C. 1992. *The Evolution of Life Histories*. Oxford University Press, Oxford.

Stearns S.C. and J.C. Koella. 1986. The evolution of phenotypic plasticity in life history traits: predictions of reaction norms for age and size at maturity. *Evolution* 40(5): 893-913.

Steele F., I. Diamond and S. Amin. 1996. Immunization uptake in rural Bangladesh: A multilevel analysis. *Journal of the Royal Statistical Society Series a-Statistics in Society* 159: 289-299.

Waaler H.T. 1984. Height, weight and mortality: the Norwegian experience. *Acta Medica Scandinavica* Suppl. 679: 1-51.

Weaver L.T. and S. Beckerleg. 1993. Is health sustainable? A village study in the Gambia. *Lancet* 341: 1327-1330.

Yamaguchi K. 1991. *Event History Analysis*. Sage Publications, California.

Table 1: descriptive statistics and sample sizes for anthropometric and demographic variables used in the analyses

Variable	Mean	Range	N
Anthropometrics			9585 measures from 855 men
Height (cm)	168	127-196	
BMI	20.4	13-29	
% men underweight (BMI<18.5)	13.0		
% men overweight (BMI=>25)	0.8		
Adult mortality			855
Median age at death for those who survived to age 21 (years)	67	21-81	
Marriage			188
% ever-married	98		
Number of marriages	3.27	0-10	
Fertility			
% childless	5		188
Age at first birth (years)	34	14-62	156
Number of children born	9.35	0-36	188
Number of children surviving to 14	5.14	0-20	188

Table 2: parameter estimates with standard errors and p values from the event-history analysis of the probability of dying[2]

	Height			BMI		
Independent variable	**Estimate**	**SE**	**p**	**Estimate**	**SE**	**p**
Constant	-6.99	2.09	<0.01	3.95	4.14	NS
Age	0.07	0.01	<0.01	0.07	0.01	<0.01
Height	-0.003	0.01	NS			
BMI				-0.98	0.39	<0.05
BMI squared				0.02	0.01	<0.05
Number of deaths		172			172	
Number of survivors		683			683	

[2] Both models also control for birth cohort

Table 3: parameter estimates with standard errors and p values for height and BMI variables in all marriage and fertility regression models[3]

	Height			BMI		
Dependent variable	**Estimate**	**SE**	**p**	**Estimate**	**SE**	**p**
Marriage						
Model 1:						
Number of marriages	0.04	0.02	<0.05	0.16	0.06	<0.05
Fertility						
Model 2:						
Age at first birth	-0.03	0.10	NS	-1.27	0.32	<0.001
Model 3:						
Total no. children born	0.06	0.07	NS	0.90	0.26	<0.001
Model 4:						
No. children surviving to age 14	0.06	0.04	NS	0.52	0.15	<0.001
Model 5:						
No. children surviving to age 14 controlling for number of marriages	0.03	0.04	NS	0.41	0.15	<0.01

[3] All models control for age at death or censoring, birth cohort and village of birth. Model 5 also includes a control variable for number of marriages

Figure 1: model predictions of the probability of dying per year by BMI (predictions are for a man 40 years old, born between 1920-29)

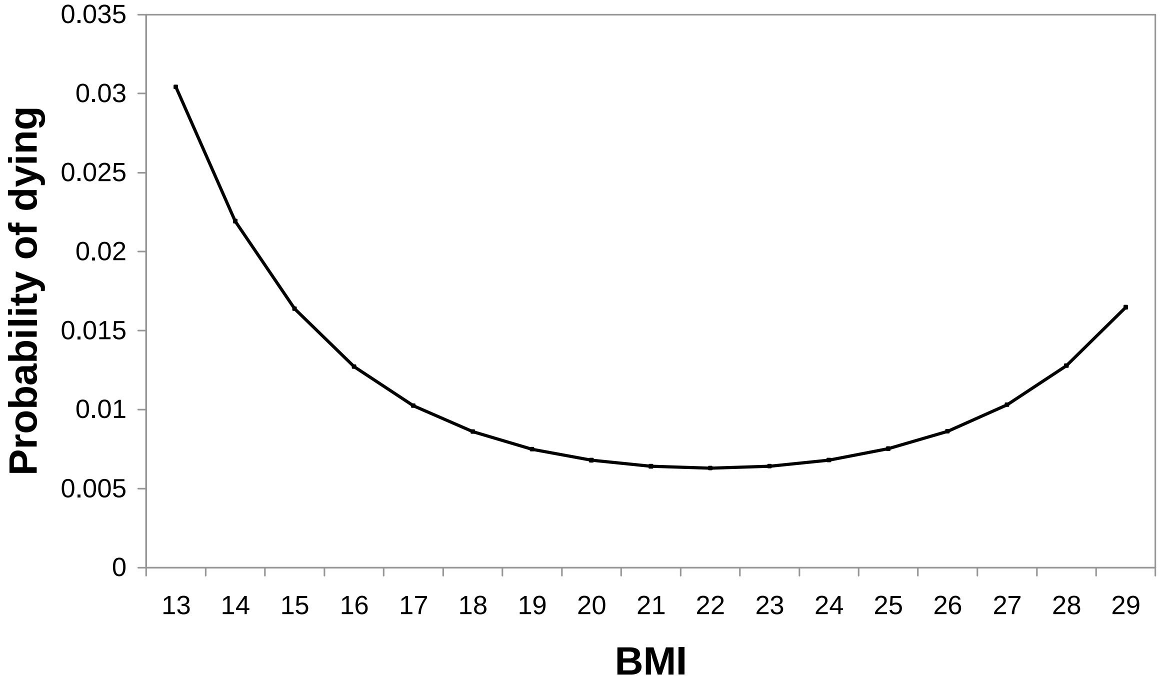
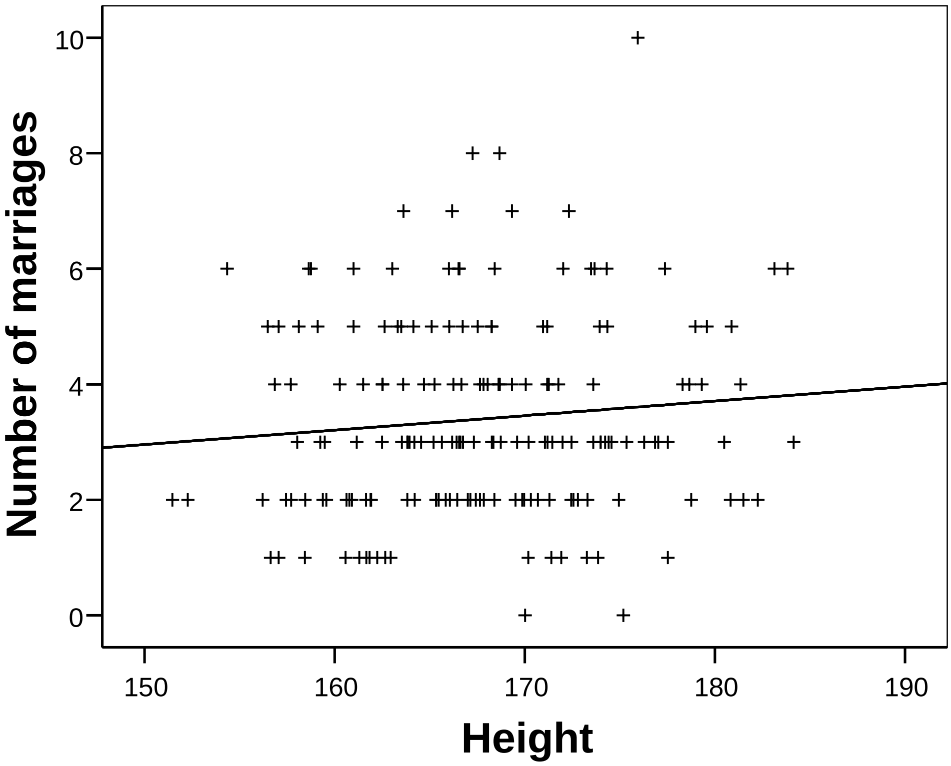


Figure 2: scatterplot, with fitted univariate regression line, of number of marriages against height (a) and BMI (b)

(a)



(b)

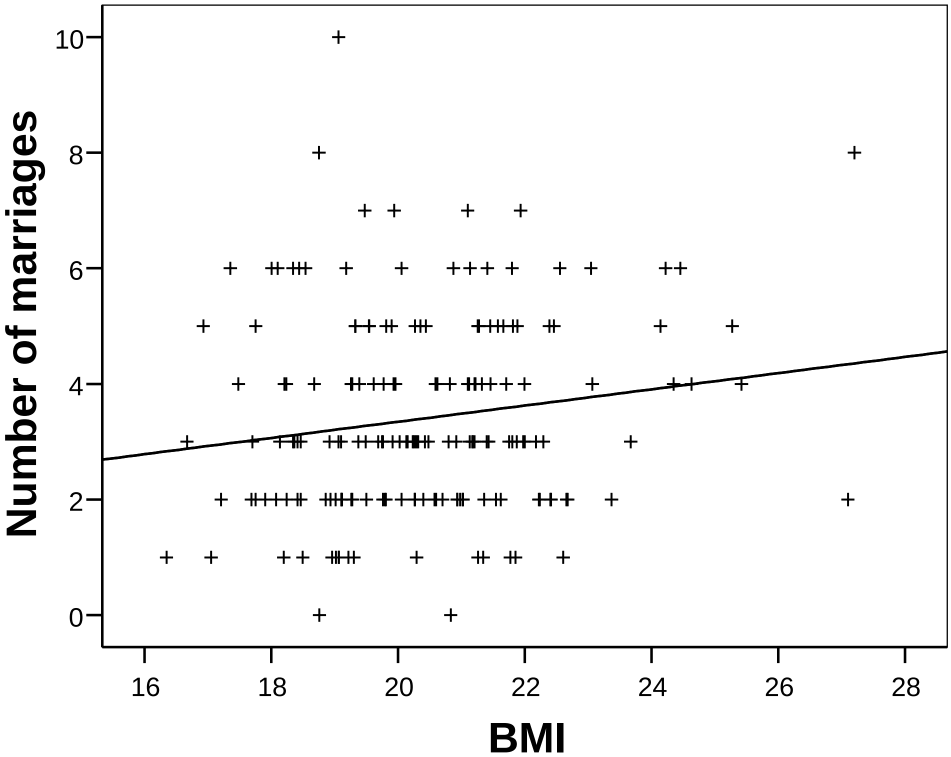
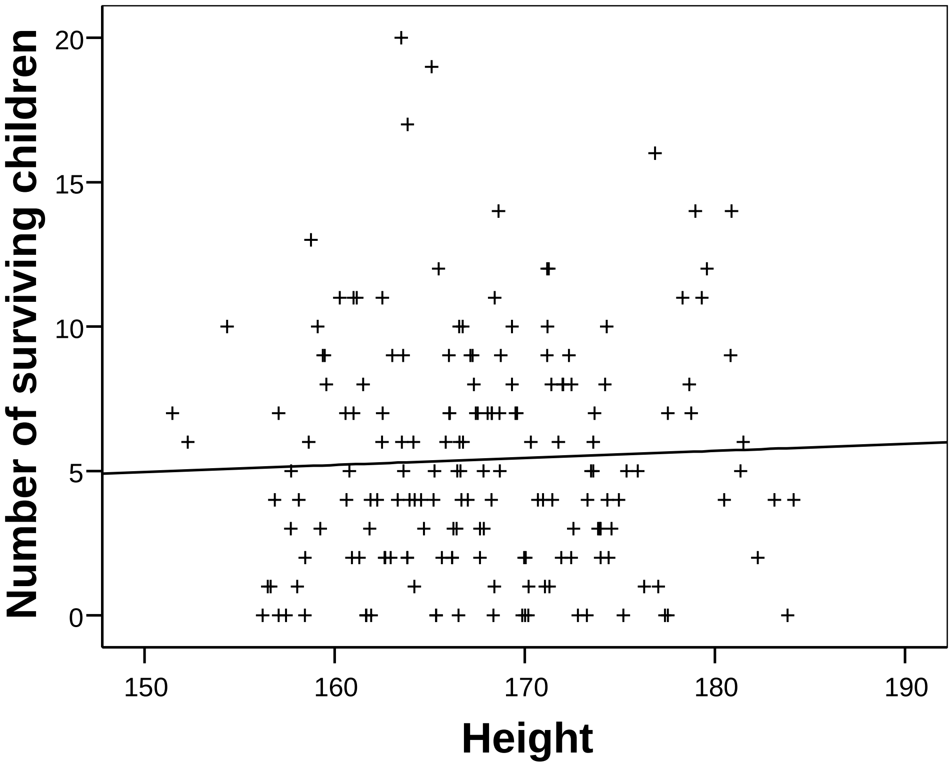


Figure 3: scatterplot, with fitted univariate regression line, of number of children surviving to age 14 against height (a) and BMI (b)

(a)



(b)