Anthropometry and mortality - a cohort study of rural Bangladeshi women

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Thesis submitted for the degree of Doctor of Philosophy,
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London School of Hygiene and Tropical Medicine, April 1997

Abstract

Many authors suggest that low anthropometric levels are associated with higher mortality risk in adults. In developing countries however there have been few opportunities to test this hypothesis. In addition, there is increasing interest in the role of women's nutritional status in their own health and survival as distinct from its impact on infant outcomes.

This thesis describes the results obtained from a longitudinal historical follow-up of a cohort of 2,314 rural Bangladeshi women over a period of 19 years (1975-1993). The demographic, socio-economic, and anthropometric characteristics of the study cohort are described with reference to the methods of data extraction, preparation and validation. The risk of mortality associated with different levels of the anthropometric indicators (height, weight, arm circumference and body mass index) were analysed using Cox's proportional hazards models. In addition to the basic survival models, the effects of confounding, early mortality, missing data, and young subjects, on the estimates are discussed.

A significant association between BMI and mortality (p=0.009) was found in adjusted analyses which used categories that distinguished the women in the highest and lowest 10% of the cohort BMI distribution. Women with BMI levels between 10% and 90% and >90% had hazard ratios of 0.45 (95% confidence intervals 0.27,0.73) and 0.55 (0.25,1.22) respectively, when compared to women with BMI <10%. The strength of the association between BMI and mortality risk was reduced after adjusting the models for early mortality (<4 years), (p=0.068). No significant associations were found between height, arm circumference and mortality risk.

In conclusion, these data provide no evidence that these anthropometric indicators would be useful in population-based screening programmes in rural Bangladesh to identify women at higher mortality risk. The findings are considered with respect to the study's methodological constraints and comparisons with other studies in order to produce recommendations for those working in research and health programmes in women's nutrition.

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Acknowledgements

Dreaming up the acknowledgements page is fine until it needs to be written - now I'm stumped for words which can adequately thank my colleagues, family and friends.

To my supervisors, Oona Campbell and Sharon Huttly, who gave a scruffy MSc student access to their offices, the enormous benefits of their experience, and their guidance when I lost the thread - thank you. To other colleagues in LSHTM whose interest in Bangladesh and women's nutrition was always welcome, in particular, Carine Ronsmans, Felicity Cutts, Sarah Salway, Jane Pryer and Ann Ashworth Hill.

I would like to thank the researchers in ICDDR,B, Michael Strong, Khayrul Khan and Mr Chakraborty who helped me participate in the world of the Matlab DSS, but also the many others who made my stay in Bangladesh a heady time of learning and laughter: Marufa Hossain, Gerri, Marijke, Akiko, James, Ali and Abdhul.

A huge thanks to the Coffee Crew whose friendship meant so much more than a 10.30 caffeine fix, Helen Edwards, Julia Mortimer, Jacky Saul, Lisa Schmidt, Joanie Wilkinson and Jane Wooders. One up, no more downs ... To my fellow Republicans of the Portacabin - keep banging the bins: Mostafa Hosseini, Nelson Gouveia, Maria Pastor, Najwa Rizkallah.

Finally to my long-suffering Folks who having coped with many a tearful piano exam stayed the course, through whoops, wheezes, commas and pips - thanks a bundle. Next time it's an embroidery GCSE.

With no small p.s. I would like to thank the Medical Research Council, UK, who have generously funded me through both my MSc and PhD. Without their assistance I would not have been able to work on this thesis at all.

Reducing the high levels of chronic and acute undernutrition of children and adults has been identified as a priority in many developing countries (WHO,1995a). Until recently, the health of adults was relatively neglected by both health programmes and research, in contrast with child health (Feachem et al,1992). Interest in women's health has led to increasing concern about the consequences of undernutrition in girls and women. For many decades interest in women's nutritional status in developing countries predominantly focused on the role of maternal nutritional status as a potential determinant of fetal and infant outcomes, recently there has been increasing concern that the nutritional status of women may have important consequences for their own survival (Merchant and Kurz,1993).

Many of the existing and proposed programmes which seek to improve food availability and security at the societal level, or identify and supplement adults with low nutritional status, are constrained by i) the limited evidence regarding the determinants and consequences of undernutrition, and ii) the lack of consensus in the appropriateness and practical use of measures and indicators of chronic and acute energy deficiency. The paucity of information has been recently recognised and several documents have been produced which extensively review the available evidence for functional consequences of low levels of anthropometry, in particular body mass indices, and the uses and interpretation of anthropometry in measuring undernutrition (IDECG,1994; Shetty and James,1994; WHO,1995a.WHO,1995b).

All these papers highlight the lack of evidence to support suggestions that low levels of anthropometry are associated with increased mortality risk, or that adult anthropometry is an useful screening tool in development and health programmes. The WHO Expert Commitee report (1995a) identified only one study examining the risk of adult mortality associated with BMI, a retrospective study of adult men in India. All suggest that there is a need for more studies which examine the association between specific measurements of anthropometry and the risk of mortality, but acknowledge the considerable difficulties in obtaining reliable data on nutritional status and mortality in developing countries.

Study objectives and hypothesis

The objectives of this study are -

to examine the association between four commonly used anthropometric indicators of nutritional status (height, weight, body mass index and mid-upper arm circumference), and mortality in a cohort of reproductive aged, rural Bangladeshi women.

The study hypothesis is -

that women with low levels of anthropometry have an increased risk of mortality independent of other demographic and socio-economic factors.

Study design

The demographic surveillance system in Matlab, a rural area of Bangladesh, conducted by the International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDR,B), offered an unique opportunity to obtain data on the survival of a cohort of adult women for whom retrospective anthropometric data was available. The women had been subjects in a detailed nutritional study in 1975-1980, and were also participants in an ongoing routine surveillance system which collects monthly data on vital events, including births, deaths, marriages and migration. An additional advantage of conducting the study in this area, is the availability of population based data on mortality, fertility and socio-economic characteristics.

The thesis is organised as follows. Chapter 1 is a review of the literature which has described the uses and interpretation of anthropometric measurements, and studies, mostly from developed countries, which have examined the relationship of anthropometry with total and cause-specific mortality. In Chapter 2 the study design and the sources of data are described. The demographic and socio-economic characteristics of the cohort are also presented. Chapter 3 presents the results of life-table survival analyses examining the pattern of mortality over the period of follow-up. In Chapter 4, the approaches used in preparing and validating the anthropometric data are described, and the nutritional status of the cohort is compared with other data on women in Bangladesh. The results of

proportional hazard models test the role of anthropometric indicators as risk factors for mortality are presented in Chapter 5. The findings and limitations of the study are discussed in Chapter 6 with recommendations for programmes and future research. The references cited are presented at the end of the relevant chapter.

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Chapter 1 The relationship between anthropometry and mortality - a literature review

Chapter introduction

This chapter summarises the literature which informs this thesis. The use of anthropometric indicators to predict mortality risk has long interested both researchers and health providers, but this study is one of the first to examine the relationship between anthropometry, height, weight, body mass index (BMI) and arm circumference, and women's mortality, using longitudinal data from a developing country. Studies of similar design and methodology have tested the effect of height and BMI on mortality risk in cohorts from developed countries, mostly from Northern Europe and the United States. Consequently, the background literature which informs the design of this thesis and the subsequent analysis needs to be considered with caution since there are many nutritional, demographic, socio-economic and mortality differentials between cohorts, and the populations from which they are sampled.

The first section of this chapter sets out the basic principles of using anthropometry to measure nutritional status. Each of the anthropometric indicators is defined. The features of nutritional status that they measure, their correlation with other anthropometric measures, and general limitations are also detailed. For BMI, the levels commonly used to define both under- and overweight individuals are described.

In the second section, each anthropometric indicator is discussed separately with respect to: i) the types of studies which have been conducted, ii) some of the principle limitations of previous studies, iii) a review of a number of selected studies in developed countries, and iv) a review of anthropometry literature from developing countries which offers insight into the possible relationships in areas with different socio-economic, demographic, nutritional and mortality patterns, for example, studies examining the relationship between BMI and morbidity.

For this Chapter, the published literature was identified using i) key word searches of the main reference databases, BIDS, MEDLINE and POPLINE, for papers written in English; and ii) by following-up citations in relevant papers and reviews.

SECTION 1.1 Anthropometry - its applications and interpretation.

Anthropometry measures the body at its highest level of organisation. The most commonly collected measures, also known as indicators, are height and weight. Measures of anthropometry are typically collected i) to make comparisons between individuals, ii) as an indicator of current nutritional status, or iii) to monitor growth or weight changes in individual(s) over time.

The term anthropometry and nutritional status are often incorrectly considered as synonymous. There are numerous definitions of nutritional status which place emphasis on clinical status, function consequences, or energy balance (Himes,1991). Where possible this thesis uses only the term anthropometry, and in discussing the relationship between anthropometry and mortality, the specific aetiological or functional consequences.

Anthropometry can only measure part of the components of the body as an indication of the body's past or current energy balance. It is important to avoid equating low anthropometric measures with poor nutritional status. For example; two individuals of similar heights may have very different levels of calorific intake and energy expenditure, and therefore, be in different states of energy balance. They may also have very different types of dietary composition. A useful discussion of the scientific legacy of this misrepresentation of anthropometric indicators is found in Payne (1992).

The level of anthropometric indicators may be raised or elevated in response to situations which could be seen in terms of advantage and disadvantage. Changes in anthropometry, such as weight, can be the result of a myriad of nutritional status determinants, including the ratio of energy intake to energy expenditure. Some determinants of anthropometry, for example age-related changes in height, are not immediately consistent with a decline in nutritional status.

Anthropometric indicators have been used to broadly classify grades of functional nutritional status. In particular, BMI through its correlation with fat mass ('leanness'), has been suggested as an indicator to distinguish chronic energy deficiency (or undernutrition), and obesity (overnutrition).

The following section examines the features and interpretation of each of the anthropometric indicators used in this study.

1.11 Height

Standing height measures body stature from the heel to the crown of the skull. Essentially it is the length of the skeleton. The determinants of attained adult height has been the subject of debate for many decades, focusing principally of the relative contributions of genetics and nutritional/ environmental experiences during the intra-uterine period and childhood. Tanner's (1989) review of the evidence for interaction between heredity and environment in the control of growth seems to reflect the general consensus at present. There are numerous studies demonstrating correlations between individual's height and parental height, and between generations in respect to the tempo of growth and development. However, the environmental conditions operating during pregnancy and childhood may markedly alter growth tempo and achieved adult height, depending on the timing, duration and severity of growth moderating 'insults'. Improvements in nutrition in childhood are often able to 'over-ride' the retardation of growth due to earlier malnutrition and disease. Certainly the body appears to have a strong ability to respond to improvements in its environment, and 'catch-up' growth can often be observed. Secular trends in increasing height have been reported in many developed and developing countries (Van Wieringen,1986; Riley,1994; Popkin,1994). This appears to be related to increasing calorie intake by younger cohorts and a decline in the incidence of infectious diseases, such as diarrhoeal and parasitic diseases.

However, the contribution of genetics and chronic illnesses during growth does not permit the assumption that individuals with relatively short stature were disadvantaged during childhood. Adult height may change in response to ageing and as a result of some specific

conditions, for example kyphosis. The height of adults is maximised on reaching the end of the second period of rapid growth around puberty. Height in the absence of skeletal deformity remains constant during early adulthood and starts to decline gradually in late adulthood due to vertebral collapse, changes in the vertebral discs, loss of muscular tone and postural changes (Svanborg et al,1991; Noppa et al, 1980). Longitudinal studies in developed countries suggest that maximal height is attained at the end of the third decade of life and declines with increasing velocity from the end of the fourth decade (Friedlander et al, 1977; Noppa et al, 1980). In their study of 20,000 women and men in Norway, Friedlander et al (1977) observed reductions of around 1mm/y in each five years age group between ages 45 and 69 years. By age, 80-84 years, height was declining at a rate of 2.2 mm/y. The pattern of height changes with ageing in developing countries has not been examined.

1.12 Weight

Weight is the sum of all components of the body including bone, muscle, fluid and adipose tissue. Its diurnal fluctuations are indicative of the influence of water, energy intake, and expenditure. Weight differences between individuals cannot be simply ascribed to differences in nutritional status since height is an important determinant of weight. A taller woman will typically be heavier than a shorter woman because she has more bone, muscle and tissue mass. However, weight changes in an individual are interpretable since weight responds to changes in body composition. Seasonal changes can produce fluctuations in both the weight of an individual and also the distribution of weight in a population. Illnesses which induce anorexia, elevated metabolic rates, or preferential catabolic loss of lean tissue will also cause a decline in body weight. Conversely, excess caloric intake, decreased energy expenditure, genetic predisposition, pregnancy and lactation can result in increased body weight (Abrams and Berman, 1993; WHO,1995b). Weight is therefore affected by past and current nutritional experiences but should be considered with reference to height.

1.13 Arm circumference

Recently mid-upper arm circumference (MUAC) has become more widely used as an indicator of nutritional status in adults. Its increased use is partly due to the relative ease of measuring arm circumference in field based studies and increased evidence suggesting a correlation with body weight and body mass index (Krasovec and Anderson,1990). MUAC measures the circumference of the upper arm which is determined by the amount of bone, fat and muscle mass. Except in individuals where biceps and triceps muscles are excessively developed, arm circumference reflects the amount of adipose tissue present and is determined by past and current nutritional experiences. Arm circumference is less responsive than body weight to short-term changes in health and nutrition which may make it a stable indicator of current nutritional status. From a single study in the US, arm circumference in women showed a slow steady rise throughout adult life (WHO,1995a).

Several studies in developed and developing countries have tested the correlation between MUAC and BMI (discussed in Section 1.4). MUAC appears to be a reasonable predictor of BMI for the lowest and highest BMI categories, but has poor sensitivity and specificity in predicting mid-range BMI levels (WHO,1995a). No relationship was observed between height and MUAC.

1.14 Body Mass Index

Body mass indices are measures which adjust weight for height. Body mass indices can be calculated as weight as a percentage of reference weight at a given height (weight-for-height), or by adjusting an individuals weight for their height. BMI calculated as weight [kg] / height squared $[m]^2$ is referred to as the Quetelet index. A Ponderal index (the cube root of weight [kg] / height [m]) or a Rohrer index (weight [kg] / height cubed $[m]^3$) are occasionally used (Krasovec and Anderson, 1990).

BMI, calculated as the Quetelet index, has been shown to be highly correlated with % fat and is relatively independent of height in many population studies (Shetty and

James,1994). It is one of the most widely used nutritional status indicators, particularly since it has been used as defining criteria for obesity and chronic energy deficiency. Given its wide spread and frequent use in other national and cohort studies, this thesis uses BMI calculated as Quetelet's index (kg/m^2), as its anthropometric measure of body mass. In reviewing the literature, BMI refers to the Quetelet index unless otherwise stated.

Body mass indices provide information on the composition of the body. Although not measuring fat mass or fat percentage directly, they are related to body composition and are often referred to a indicator of 'leanness'. Thus, BMI approximates to low weight, fat mass and fat-free mass, and is considered a good indicator for assessing the percentage body fat in epidemiological studies (Kushner,1993). An obese woman, where excess body fat is stored, will have a high BMI measurement, in contrast to the much lower BMI of a woman with chronic energy deficiency where stored fat and possibly lean tissue mass have been metabolised to produce energy. Table 1.1 presents the BMI levels currently being used to define chronic energy deficiency and obesity.

Table 1.1

Suggested levels of BMI associated with differing body composition types (James et al,1988; Ferro-Luzzi et al,1992; WHO,1995a).

Chronic energy deficiency				
	Grade 1 :	BMI	17.0 - 18.49	(mild thinness)
	Grade 2 :	BMI	16.0 - 16.99	(moderate thinness)
	Grade 3 :	BMI	< 16.0	(severe thinness)
Normal range: BMI	18.50 - 24.99			
Obesity				
	Grade 1 :	BMI	25.00 - 29.99	(overweight)
	Grade 2 :	BMI	30.00 - 39.99	(overweight)
	Grade 3 :	BMI	≥ 40.00	(overweight)

BMI is therefore an indicator of the past and present nutritional experiences of an adult and will be affected by some of the same determinants which influence weight, such as illness and energy intake, but it is more stable and distinguishes different aspects of body composition.

In some, mainly US, publications, reference is made to body weight which is an inclusive term for BMI or relative weight i.e. weight relative to some internal or external standard weight. In this thesis, where distinction is important, BMI (Quetelet index) or relative weight are used to avoid confusion.

SECTION 1.2 Evidence for relationships between anthropometric measurements and adult mortality.

There is strong evidence from numerous cohort studies that BMI is independently associated with the risk of mortality during adulthood. The pattern of mortality with BMI is generally J-shaped. The nadir mortality risk is associated with BMI levels below average. However, all the studies observed cohorts from developed countries. No prospective data examining long-term mortality risk associated with levels of BMI was identified from developing countries. The evidence for a relationship with respect to height and mortality is less convincing. Again prospective data was only available from developed countries. The relationship between arm circumference and all cause mortality risk does not appear to have been examined in any studies.

There is therefore no direct evidence to support a hypothesised relationship between anthropometry and mortality in a rural Bangladeshi cohort of adult women. The evidence from developed countries should be reviewed with an awareness of the many potentially important differentials in anthropometry and mortality determinants which could result in different associations between anthropometry and mortality. The studies from developed countries offer an understanding of anthropometry mortality relationships, particularly at higher BMI and height. Importantly, the studies published over the last 50 years contain important insights into the methodological constraints and interpretational difficulties of anthropometry mortality relationships.

1.21 BMI and mortality

The long-standing interest in weight and height as risk factors for mortality can be seen in the early use of life insurance statistics from Canada and the US (Simopoulos and Van Itallie,1984). In 1959, the Build and Pressure Study used mortality data collected by

insurance companies to define 'desirable' weights and heights. These were derived from the mortality rates associated with different weight and height levels (Build and Pressure Study,1959). In 1979, the Build Study examined insurance data and observed that relative weight, i.e. weight as a percentage of the population average, had a U-shaped relationship with mortality (Build Study,1979). Tables of 'desirable' weights and heights were modified repeatedly over the 1980s and 1990s. A feature of these insurance statistics studies is the large number of people which were included, nearly 4.2 million people during 1950-1979. However, the subjects included are not a representative cross-section of the entire population, the sample being based on middle-class individuals considered to be in good health following screening for serious medical impairments (Simopoulos and Van Itallie,1984).

In addition to the insurance statistics studies, many small and large cohort studies have examined the mortality risk associated with BMI levels, or relative weights (Sjöström,1992). These cohorts were initiated specifically to identify potential risk factors for mortality. For example, the American Cancer Society (prospective study) followed 750 000 men and women from 1959 until 1973 (Lew and Garfinkel,1979). Other studies utilised secondary anthropometric and mortality data (retrospective study), for example, the Norwegian Experience used compulsory TB screening data from 1967 to 1975, which contained weight and height information on 85% (1,718,000 men and women) of the Norwegian population (Waaler,1988). The American study found a J-shaped relationship between BMI and mortality, the Norwegian study a U-shaped relationship. For both studies the nadir mortality was found at levels of BMI below the average.

There has been a change in the focus of recent published studies. Many have examined the role of obesity in determining or predicting mortality, particularly cadio-vascular and allied causes of death, and several studies have focused specifically on sub-groups, the elderly, women and blacks, in response to the larger body of studies with white, middle-aged male cohorts.

1.211 Constraints of previous prospective studies examining BMI and mortality

From the literature it is possible to observe how changes in epidemiological understanding has lead to many of the earlier papers being criticised for important sources of potential bias in both their design and in their analyses. Review articles considering the body of evidence for a relationship between BMI and mortality were published in 1984, 1987, 1992 and 1993 (Simopoulos and Van Itallie,1984; Manson et al,1987; Sjöström,1992; Kushner,1993). Each successive review raises more methodological criticisms about previous study design and interpretation. There are five main criticisms of these studies:

1. Failure to adjust for smoking

Several early studies did not control for cigarette smoking, including the insurance studies and the Norwegian Experience study described above. For some studies using historical data, information on smoking was unavailable (Hoffmans et al,1988). Smoking prevalence has been observed in several studies to be highest among low BMI subjects (Sidney et al,1987). In the Framingham Study, cigarette smoking almost completely confounded the relation between thinness and mortality (Garrison et al,1980). Failure to adjust for smoking in analyses, may elevate the risk of mortality observed among low BMI subjects given the association between smoking and mortality. Cohort studies with only non-smoking subjects have observed mortality to be lowest among low BMI individuals (Lew and Garfinkel,1979).

2. Failure to adjust for pre-existing illness

Low BMI measured at enrolment into a prospective study may be due to weight loss caused by a clinical or sub-clinical illness. Consequently, the risk of mortality associated with low BMI may be overestimated. To minimise this potential bias, a few studies have used very careful screening examinations at the time of enrolment, excluding those where unintentional weight loss had recently occurred (Lew and Garfinkel,1979). More typically, studies have adjusted for mortality during the early years of the follow-up period. In several studies, the pattern of mortality associated with BMI centiles was found

to differ by interval of the follow-up period (Hoffmans et al,1988;Vandenbroucke et al,1984).

3. Small sample sizes and/or short-term studies

A graph is presented in Sjöström's review, plotting cohort size and the follow-up period in relation to 40 employee, community, or random population studies findings, i.e. whether an association between BMI and mortality was found (Sjöström,1992). Negative studies, where no association was found, were clustered among the short term and/or small studies. All studies with >20,000 subjects, and 20 out of 21 cohorts with >7,000 subjects, found a positive association between high BMI levels (obesity) and mortality. A minimum of 5 years follow-up in larger studies appeared to be necessary to show a positive relationship, and in smaller cohorts this period needed to be much longer. Even with a 35 year follow-up, the Minnesota study of 279 men found no association between obesity and mortality (Keys et al,1971).

4. Inappropriate control for intermediate risk factors

In some early studies a crude association between high levels of BMI and mortality was found. However, obesity was discounted as an important risk factor for mortality, when, on adjusting for conditions such as hypertension and diabetes, statistical significance was lost (Chapman and Massey,1964). Several papers, reviewing the studies (Manson et al,1987; Sjöström,1992; Kushner,1993), argue that this is an erroneous interpretation since hypertension and diabetes are not confounders but are intermediate risk factors that are, in part, caused by obesity. If the aim of the analyses is to examine the overall risk of mortality due to BMI, it is inappropriate to control for conditions which are the effects of high BMI levels and links in the causal pathway by which obesity exerts its influence on mortality risk.

5. Misclassification bias

An issue raised in recent papers, is that misclassification biases may be causes for a reduced internal validity of population studies (Sjöström,1992; Folsom,1993). This is principally a concern that BMI levels cannot discriminate between different sub-groups of obese individuals based on adipose tissue distribution. For example, women of the same level of obesity as measured by their BMI may have adipose tissue distributed in the lower body (hyperplastic obesity) or in the upper body (hypertrophic obesity) (Hartz et

al,1984). If BMI levels alone are considered in relationship to mortality risk, then any increased mortality risk among a particular sub-group would be diluted in the statistical analysis. Since the only measure of adiposity available for the thesis cohort was BMI, this area of research was not presented in detail in reviewing the literature.

1.212 *Review of selected cohort studies which examined BMI and mortality risk in developed countries.*

Comprehensive summaries of the earlier papers are presented in the reviews of Simopoulos and Van Itallie (1984) and Manson et al (1987). The papers reviewed in this section were selected through the following criteria:

a) In the light of the four important sources of potential bias discussed above, only papers which address these issues in their methodology were considered. In particular these were papers which adjusted analyses for early mortality during the follow-up period.

b) The papers considered BMI as one of their risk factors of interest, and in which risk associations with absolute levels of BMI could be identified. These papers are the most comparable with the thesis data, design and methodology. Several other papers reported relative weight to an internal or external standard (Garrison et al,1983); or focused on other measures of adiposity, for example weight-hip ratio (Lapidus et al,1984).

c) The paper reported the all cause mortality risk associated with BMI, again for their comparability to the thesis cohort. Some papers, particularly those focusing on obesity and mortality, report only coronary heart disease mortality (Hubert et al,1983; Johnson et al,1986;Manson et al,1990; Fitzgerald and Jarrett,1992).

Table 1.2 presents a summary of the design of each study, with a note on the general finding with respect to BMI and mortality. The term, U- or J-shape relationship with mortality, is used in this review to denote the position of the nadir and zenith mortality risk.

Table 1.2

Summary of recent cohort studies which report all-cause mortality risk associated with BMI

	Data source Reference	Country	Number of subjects (sex)	Age at entry, y	Years of follow-up	Weight mortality association
A	Army medical data Hoffmans et al (1988)	Holland	78,612 (m)	18 y in 1950	32 yrs from 1950	Yes - nadir <average BMI†
B	Population based health survey data Rissanen et al (1989)	Finland	22,995 (m)	25-79 y in 1966-1972	med. 12 yrs	Yes -nadir wide around average BMI
C	Population based health survey data Rissanen et al (1991)	Finland	17,159 (f)	25-79 y in 1966-1972	med. 12 yrs	Yes - nadir < average BMI
D	Framingham Heart Study Harris et al (1988)	US	597 (m) 1126 (f)	65 y in 1957-1981	mean 9.5 yrs	Yes - nadir < average BMI for both sexes
E	Kaiser Foundation Health Plan Wienpahl et al (1990)	US	2,453 (m) 2,731 (f) (m)+(f) black only	30-79 (m) 40-79 (f) in 1964-1973	15 yrs	(Men) Yes - nadir < average BMI (Women) No - flat relationship, non-significant
F	Seventh Day Adventist Lindstead et al (1991)	US	8,826 (m)	≥30 yrs in 1960	26 yrs	Yes - positive relationship Direct monotonic pattern
G	US Railroad workforce study Yao et al (1991)	US	3,043 (m)	mean 49.3 y in 1957-1960	29 yrs	Yes - nadir around average BMI
H	Charleston Heart Study Stevens et al (1992)	US	452 black (f) 738 white (f)	≥35 y in 1960 or 1963	25-28 yrs	(Whites) Yes - nadir < average BMI (Blacks) No - flat relationship
I	Iowa Women's Health Study Cohort Folsom et al (1993)	US	41,837 (f)	55-69 y in 1986	5 yrs	Yes - nadir around average BMI

† Did not adjust for smoking in the analysis since the data was not collected during the medical examination.

All studies observed a significant relationship between BMI and mortality, except among black women (Wienpahl,1991;Stevens,1992). The general mortality risk relationship observed was that of a J-shape, with increased risk for the leanest and heaviest subjects, the risk being higher with high BMI than with low BMI. The lowest mortality was observed for individuals with BMI around or moderately below the cohort average.

From the papers reviewed in Table 1.2 several important aspects of the BMI mortality association can be highlighted: 1) age-related differences; 2) racial differences; 3) causes of mortality at different BMI levels; 4) weight loss in low BMI subjects; 5) confounding factors; 6) minimum mortality risk and BMI levels.

1. Age appears to change the levels of BMI associated with highest mortality risk

In both women and men, the association between high levels of BMI and mortality is strongest among younger ages. In addition, comparison between studies seems to suggest that as age increases, the BMI levels associated with lowest mortality risk also increase.

Among the cohort of 18 year old Dutch army applicants, the risk ratio of obese men (BMI ≥26), to men with BMI 19.00-19.99, was 1.95. In the Norwegian experience, a large population study found men and women aged 25-34 years with BMI >31 had a mortality risk two times higher than subjects of average weight (Waaler,1984).

From the results of the Finnish female cohort, aged 25-79 years, Rissanen et al (1991) conclude: *'Both thinness and overweight are detrimental to the longevity of initially healthy young and middle-aged non-smoking women, but through differing disease patterns. BMI is not an important predictor of mortality in old women.'*

Other authors have questioned the interpretation that obesity is not an important determinant of mortality in the elderly (Harris et al,1988; Sjostrom,1992; Hoffmans et al,1988). They suggest that age should be considered with respect to the weight changes and levels during adulthood. Harris et al (1988), concluded from the Framingham cohort study of 1,723 non-smoking men and women aged >65 years, that higher BMI levels were associated with

increased all-cause and cardiovascular mortality. Relative to men with BMI between 23.0 and 26.1 and women between 24.1 and 26.1, those at the 70th percentile of BMI (men ≥28.4, women ≥28.7), had a relative risk of mortality of 1.6 and 1.9 respectively. Interestingly, the study repeated the analysis, comparing the mortality risk where individuals had been in the 70th BMI percentile at both age 55 and age 65, and those who were only overweight at age 65. The relative risk among the latter was considerably lower at 1.00. This may suggest that age is a possible marker for the length of exposure an individual has to a high or low body weight.

Exposure to high or low body weight is also referred to in Hoffmans et al (1988) in the discussion of the young male Dutch army medical cohort data. They observed that the best survival rate is observed among men with a BMI of 19.00-19.99. The higher relative mortality risk of overweight men (BMI ≥ 25) and underweight men (BMI ≤17.99), compared to men (BMI 19.00-19.99) was only demonstrable after a follow-up of more than 20 years.
They suggest, similar to Harris et al (1988), that the duration of over- and underweight may play an important role in establishing the adverse affects. Alternatively, the negative effect of over- and underweight may not become evident for many years because at young ages a very lengthy induction time is involved.

Both papers on the Finnish study also report that the length of follow-up and the initial age of subjects were important in relationship between BMI and mortality (Rissanen et al,1989; Rissanen,1991). They suggest that the discrepancy between the numerous studies where the relationship between BMI and mortality was reported to be non-linearly U- or J-shaped, linear or non-existent, may in some part, be due to the age distribution and mortality pattern of the population studied. On reviewing 40 studies Sjöström (1992) concludes that obesity has some impact on mortality up to 80 years of age.

In addition to the risk of mortality associated with BMI being reduced in the elderly, the BMI range associated with mortality may be shifted upwards (Kushner,1993). The nadir of mortality risk for both males and females in the older Framingham cohort (age ≥65 years), lie at higher BMI levels than the younger Finnish male and female cohort (age 25-79 years). Rissanen et al (1989), in a 12 year follow-up of 22,995 Finnish males found that the

minimum mortality for men aged ≥75 years was associated with a BMI range of 28-31, whereas in younger men, 25-75 years, it was at a lower range of BMI (22.0-24.9). Data on 17,159 female subjects in the same Finnish study showed similar age differentials. The most favourable life expectancy for women >65 years was observed in a BMI range of 27-31 compared to a range of 21.0-26.0 for younger women (Rissanen et al,1991). There is some supporting evidence from other actuarial sources which indicate higher survival for overweight older people, although the BMI levels were not specified (Mattila et al,1986).

Explanations for the upward shift in favourable BMI with older ages include age related differences in fat patterning, and/or age-related differentials in the relative risk of cardiovascular or non-cardiovascular mortality. Dissociation between body mass and body fat patterning has been suggested as a possible explanation for age-related, gender-related and racial-differences in the strength and the shape of the relationship between BMI and mortality. For the cohort described in this thesis, there are no indicators of fat patterning other than BMI. Consequently, this review seeks only to introduce this relatively recent area of interest.

Several studies have discussed the limitations of BMI as an predictor of mortality. Folsom et al (1993) argue that body mass may be the wrong dimension for assessing the risk of death, particularly in the light of recent evidence suggesting that excessive abdominal adiposity may be more hazardous than overall body size. Other anthropometric measures of central body fat, for example waist-hip ratio (WHR) have been suggested as better correlates of abdominal visceral adipose tissue volume and obesity-related metabolic abnormalities (Folsom et al,1993, Yao et al,1991, Stevens et al,1992). However, the use of WHR as a correlate of underweight related mortality is not discussed.

Folsom et al (1993) collected data on both BMI and waist/hip circumference in the Iowa Women's Health study cohort of 41,837 women aged 55-69 years. BMI was associated with mortality in a J-shaped relationship with the nadir of mortality risk at BMI 27.03-30.71. WHR was strongly and positively associated with the risk of death in a monotonic pattern. They observed that the highest relative risk of death was among women in the lowest quintile of BMI and the highest quintile of WHR.

Stevens et al (1992) in their study of 1,190 women in the Charleston Heart Study, among white women BMI had a J-shaped relationship with mortality, the nadir being in the 2nd quartile. As in the Iowa cohort, there was a positive association between abdominal circumference and all-cause mortality, and also for CHD mortality in white women. No association between BMI or WHR and mortality was found for black women. This is discussed below. Yao et al (1991) also observed a U-shaped relationship between total, central and peripheral body fat and all-cause mortality, and direct relationship with CHD mortality. In reviewing studies with specific reference to mortality and overweight, Kushner (1993) states: *"The WHR could be a marker of genetic, hormonal, or life-style factors that are more proximally related to CHD risk. Nonetheless, failure to take body fat patterning into account is a significant confounding variable in all studies addressing the relationship between excess body weight and mortality."*

2. Black women show no association between BMI and mortality

Wienpahl et al (1990) report the findings of a 15 year follow-up of 2,731 black women aged 40-79 who had been members of the Kaiser Foundation Health Plan. Women under 40 had been excluded to minimise the confounding effect of pregnancy or lactation on body weight, interestingly this possible source of bias has not been noted in the major reviews as a possible limitation of other younger female cohort studies. Regression analysis of BMI and all-cause mortality was essentially flat over the entire BMI range. The risk of death for women with a BMI >25.8 was 1.00 in comparison with women whose BMI was 23.5-25.8.
The results for black male members of the Health Plan was a J-shaped significant relationship between BMI and mortality. This lack of association between BMI and mortality for black women was also observed in Stevens et al (1992) and Johnson et al (1986).

Stevens et al (1992) compared the BMI/ mortality relationships found in a 25-28 year follow-up of 452 black and 738 white women in the Charleston Heart Study. Black women in the cohort had a significantly higher BMI (mean = 27.4) than white women (mean = 24.5). Black women were nearly twice as likely to be obese as white women, 25% and 46% respectively. The relatively higher risk of obesity among black women in the US has been shown in other

population based studies (Kumanyika,1987). White women showed a significant mortality hazard at the 85th percentile relative to the 15th percentile of BMI, of 1.5 ($p<0.001$). In contrast the hazard for black women was non-significant 1.1 ($p>.2$). This racial differential was repeated when CHD mortality alone was examined, white women had a hazard of 1.7 ($p<0.003$), whereas black women showed no significant association with BMI levels, 1.1 ($p>0.2$). In studies of black men, CHD mortality showed a U- or J-shaped relationship with mortality (Wienpahl et al,1990; Stevens et al,1991)

With specific reference to underweight, 15% of white women were underweight (≤19.1) compared with 12% of black women in the Charleston Heart study follow-up. The effect of adjusting for mortality within the first 5 years of follow-up was most noticeable in the lowest quartile of BMI in black women. The death rate decreased from 19.4 to 13.7 per 1000 person-years when early deaths were excluded. After adjusting for mortality in the first 5 years of follow-up, the lowest BMI quartile (15.1-21.6) had no statistical difference in mortality risk compared with the other higher BMI quartiles. The lowest BMI quintile in the Kaiser Foundation study (16.6-23.5) also showed no difference in mortality risk to the higher quintiles.

Racial differences in fat patterning have been offered as a possible explanation for the lack of association between BMI and mortality among black women (Weinpahl et al,1990; Stevens et al,1992; Folsom et al,1993; Kushner,1993). The concept of fat patterning is similar to that proposed for gender-differentials. Studies have shown that with BMI held constant, black women are more centrally obese, i.e. relatively more excess trunk or upper body fat than extremity or lower body fat compared with white women (Stevens et al,1992; Blair et al,1984; Kumanyika,1987). However, there is no supportive evidence for this hypothesis. In the Charleston Heart study, fat patterning was not associated with all-cause or CHD mortality in black women, but was associated with CHD mortality, but not all-cause mortality in white women. Fat patterning was not examined in the Kaiser Foundation study.

With respect to racial differences and BMI mortality associations, no study was identified which presented results separately for women or men of Asian origin, although several authors report a higher prevalence of centrally-distributed obesity in people of South Asian

descent living in the USA and the UK (McKeigue et al,1992; Hodge et al,1996). Centrally-distributed obesity has been suggested as a possible determinant of the higher risk of non-insulin-dependent diabetes mellitus, glucose intolerance, hyperinsulinaemia and coronary heart disease, observed for Asians in comparison with other ethnic groups.

3. Different causes of excess mortality among those with low and high BMI

It is suggested that the causes of death at the extreme levels of BMI may be considerably different. Mortality at low BMI appears to be dominated by digestive and pulmonary disease. At high BMI it is related predominately to cardiovascular disease, diabetes mellitus, gallbladder disease, and cancer of the breast, colon, endometrium, and prostate (Kushner,1993;WHO,1995a).

Rissanen et al (1989) discuss the discrepancy between the relationship between BMI and mortality observed by different studies with respect to mortality patterns. They suggest that the mortality pattern in the population, and the age distribution of cohorts may be important in determining the shape of the relationship. They argue that where cardiovascular deaths predominate, the contribution of overweight to overall mortality is substantial, but the impact of overweight may be concealed when non-cardiovascular causes predominate.

4. Mortality associated with low BMI levels, with reference to weight loss

The Seventh Day Adventist study focused particularly on the mortality risk associated with BMI levels defined as underweight (Lindstead et al,1991). This 26-year follow-up of 8,828 male Adventists included 439 who were very lean (BMI <20). In this cohort, men with low BMI showed a similar risk ratio to those whose BMI was between 20.0 and 22.3. The highest mortality risk was observed in those with the highest BMI >27.5.

Lindstead et al (1991) raise the issue of life-style habits. Adventists generally do not use tobacco, alcohol or eat meat, in particular pork. This concept of 'healthy' low BMI levels is in contrast to other studies, where low BMI was suggested to be predominantly due to existing

illness. Stevens et al (1992) found that black women with low BMI were observed to have a higher risk of dying in the early period of follow-up.

If low BMI is a surrogate for conscious 'healthy' life choices, it has potentially a very different predictive relationship with mortality, than low BMI due to illness, nutrient deficiency, or poverty. This suggests that behavioural factors may confound the relationship between BMI and mortality. However, the direction of the relationships are complex and poorly addressed in the studies reviewed.

Another related issue is the role of weight loss or weight cycling among individuals with low BMI. This issue was examined in detail since earlier studies have shown that Bangladeshi women in Matlab experienced marked seasonal fluctuations in their weight (Huffman et al,1985).

Of the nine papers reviewed, only the Framingham study examined the relationship between BMI change and subsequent mortality. This is mirrored by limited studies in the general literature (Kushner,1993). As described above in point 1), Harris et al (1988) argues that men and women who were overweight (>70% percentile) at both 55 years and 65 years had a much higher relative risk of mortality, than those who were less than the 70th percentile at 55 years but had increased their BMI in later life. In addition, they examined the mortality risk of gaining or losing weight from age 55 to age 65. Those subjects who lost 10% of their BMI were at almost twice the relative risk of death compared with those with a gain of 0%-9%.

In a study of Honolulu men, Rhoads and Kagan (1983) predicted future mortality based on BMI levels. Men aged 25 in the lowest quartile of BMI had the lowest mortality in middle age, provided that they did not lose weight in the intervening years. Unintentional weight loss in low BMI individuals has been found to increase the mortality risk from coronary heart disease, and is suggested to be important in the increased risk associated with low BMI (Hamm et al,1989; Sidney et al,1988; Pamuk,1992; Kushner,1993; Blair et al,1993).Hamm et al (1989) analysed data on 2,107 men, aged 40-55 from the Western Electric Study, followed-up for over 25 years. They observed that the risk of death from coronary heart disease was two times higher in men who gained and lost more than 10% of their weight during any 5-

year period, compared with men with less than a 5% weight change. Adjusting for BMI in the analysis reduced the relative risk of death to 1.8. The risk was not significantly raised for cancer mortality. Weight cycling in non-obese individuals, as distinct from stable low BMI was also found to be associated with an increased risk of coronary heart disease (Blair et al,1993). These studies suggest that there may be a threshold where weight loss among low BMI individuals places them at a higher risk of mortality.

There are many problems with using longitudinal studies to examine the health risk of weight change. Changes in weight are usually self-reported and as discussed in section 1.2, weight has been found to generally increase in older adults. There may be little information available regarding the cause of weight loss or gain, which may be important where weight loss is involuntary i.e. due to illness, or deliberate i.e. after the adoption of different life-style behaviour (Kushner,1993).

5. Confounding factors and mortality risk associated with BMI

Rissanen et al (1989) suggest that a host of other factors may conceal the relationship between BMI and mortality. Body weight is determined by a complex interaction of individual and social characteristics and diseases, many of which have separate effects on longevity.

Table 1.3 presents the different confounding factors which the nine studies have considered in some or all of their analyses. Some studies based on cohorts whose original focus included an interest in nutritional determinants of morbidity or mortality, for example the Iowa and the Framingham studies, have many more clinical measures. These were not available for the retrospective cohorts, for example, the study of Dutch recruits is the only one which cannot control for smoking.

Table 1.3

Confounding variables used as adjustments in different models in nine selected studies

	Data source Reference	Confounders used in analyses adjustment	Weight mortality association
A	Army medical data Hoffmans et al (1988)	age education health status resting pulse rate	Yes - nadir <average BMI†
B	Population based health survey data Rissanen et al (1989)	smoking age region social class physical activity serum cholesterol blood glucose diastolic blood pressure	Yes -nadir wide around average BMI
C	Population based health survey data Rissanen et al (1991)	smoking region age	Yes - nadir < average BMI
D	Framingham Heart Study Harris et al (1988)	smoking age serum cholesterol level blood glucose level systolic blood pressure cardiovascular disease diagnosed before 65 yrs	Yes - nadir < average for both sexes
E	Kaiser Foundation Health Plan Wienpahl et al (1990)	smoking age education alcohol antecedent illness	(Men) Yes - nadir < average (Women) No - flat relationship, non-significant
F	Seventh Day Adventist Lindstead et al (1991)	smoking history age region race marital status educational level alcohol use medical illness exercise level coffee intake dietary pattern	Yes - positive linear relationship with BMI Direct monotonic pattern
G	US Railroad workforce study Yao et al (1991)	smoking age blood pressure serum cholesterol	Yes - nadir around average BMI
H	Charleston Heart Study Stevens et al (1992)	smoking age education	(Whites) Yes - nadir < average BMI (Blacks) No - flat relationship
I	Iowa Women's Health Study Cohort Folsom et al (1993)	smoking age marital status educational level alcohol use estrogen use	Yes - nadir around average

Age and smoking were used in virtually all analyses. The effect of adjustment on crude mortality risk is not always presented. Rissanen et al (1989) found that adjusting for smoking

habits substantially increased the relative excess mortality of high BMI men, and decreased the mortality in men with low BMI. This suggests that smoking related mortality is higher among men with the lowest BMI. Also adjusting for social class and physical activity slightly increased the relative excess mortality of those with higher BMI.

As discussed in section 1.211, several authors have questioned the appropriateness of adjusting BMI mortality models for markers of hypertension and diabetes such as blood pressure and blood glucose, since hypertension and diabetes may not be confounders but intermediate risk factors for mortality (Manson et al,1987; Kushner,1993). In the analysis of the Framingham cohort, the significance in the higher mortality risk associated with BMI above the 70th percentile was considerably reduced after adjusting for variables including, serum cholesterol, blood glucose level and systolic blood pressure.

6. BMI levels and minimum mortality risk

The BMI centiles associated with the lowest risk in the nine studies are presented in Table 1.4. Since the studies use differing categories and statistical methods, this table is necessarily an approximation.

A crude comparison between the studies, without regard to age, would suggest that in the USA, Finland and Holland, minimum mortality risk is associated with BMI around 19 - 27 for men, and 22-27 for women, with the important exception of black women. However, in older women and men, favourable BMI levels may be marginally higher, men 23-27 and for women 24-27. However, of the nine studies reviewed although identifying one or more centiles with relatively lower mortality risk, most studies avoid making recommendations for weight-height standards for the general population. Their caution appears to be due to the numerous questions and constraints which the authors raise.

Table 1.4

Approximate BMI levels associated with lowest mortality risk in nine selected studies. (Cut-offs refer to limits of the centiles used)

	Data source Reference	**BMI range associated with lowest mortality**	**Notes**
A	Army medical data Hoffmans et al (1988)	men - 19.0-19.9	Lowest quartile was <19.0
B	Population based health survey data Rissanen et al (1989)	men - 22.0-24.9	Men with 19.0-21.9 had only a relative risk of 1.1 compared to reference
C	Population based health survey data Rissanen et al (1991)	women - 22.0 - 25.2	Note quintile II had the lowest mortality, study calculates this in 10 year age groups. Q2 increases its upper limits with increasing age.
D	Framingham Heart Study Harris et al (1988)	men - 23.0-25.2 women - 24.1 - 26.1	
E	Kaiser Foundation Health Plan Wienpahl et al (1990)	black men 28.0 (95% CI 25.6,30.4) black women - no statistical significant relationship observed	Statistical modelling may have biased this estimate upwards, but suggest that it would be no less than BMI 24
F	Seventh Day Adventist Lindstead et al (1991)	no nadir - lowest mortality is in the lowest BMI quintile men - <22.3	Monotonic relationship
G	US Railroad workforce study Yao et al (1991)	men - 23.3 - 27.5	Based on cumulative percentage dead
H	Charleston Heart Study Stevens et al (1992)	white women - low mortality at the 15th percentile <20.2 black women - no significant association observed	
I	Iowa Women's Health Study Cohort Folsom et al (1993)	women - 25.03-27.43	3rd quintile

The nadir mortality risk found in the studies are comparable with those of recommended levels for adults from national and international policy making bodies. These are shown in Table 1.5.

Table 1.5

Recommended BMI levels for adults†

Source of recommendation	Sex	Age group (years)	Recommended BMI (kg/m^2)
US Departments of Agriculture and Health and Human Sciences (USDA/DHHS)	Both	19-34 >=35	19-25 21-27
National Academy of Sciences (NAS)	Both	19-24 25-34 35-44 45-54 55-64 >=65	19-24 20-25 21-26 22-27 23-28 24-29
National Center for Health Statistics (NCHS)	Male Female	20-74 20-74	20.7-27.8 19.1-27.3
World Health Organization (WHO)	Both	adults	20-25
Ministry of National Health and Welfare Canada (Canadian)	Both	20-65	20-27

†Table modified from Kushner (1993)

1.213 Studies examining BMI and mortality risk in developing countries

Only two studies were identified which examined the relationship between BMI and mortality in developing countries. Murray et al (1992) present a short conference abstract reporting their study of 403 Masai adults over a period of 12 years. They collected anthropometric data, mortality and morbidity data. Undernutrition was defined as BMI <18.6 for men, and BMI <16.8 for women. At the start of the study, clinical and laboratory evidence for overt and occult disease were used to exclude those with existing disease. Adults were re-examined at 4,8 and 12 years and a history of health events obtained. Unfortunately, they present combined results for males and females. None of the 5 deaths (all automobile accidents) were among the undernourished adults. The small number of deaths does not allow meaningful statistical analysis.

Naidu and Rao (1994) analysed nutritional data from India available from the National Nutrition Monitoring Bureau surveys and studies. They found that 49% of the adult rural Indian population had a grade of chronic energy deficiency (CED), defined as BMI <18.49

(James et al,1988). They cite the results of an earlier unpublished study which examined the relationship between mortality and BMI status in men (NIN Report, 1989-90). Mortality increased from 12 per 1000 population with 'normal' BMI of >18.49, to 33 per 1000 population in BMI <16.0 (grade III CED). However, they do not present details of the study methodology. Caution needs to be used in interpreting these results because there was no indication that illness on entry into the studies, or early mortality, was controlled for in the analysis. The section of the WHO Expert Committee (1995a) report on anthropometric indicators discussing mortality and BMI, reports only the Naidu and Rao (1994) paper, and concludes:

".... so it is difficult to assign any causal significance to the relationships. Nevertheless, if it can be assumed that immune competence is compromised in those with a low BMI, susceptible low-weight individuals may succumb to the prevalent life-threatening diseases. More prospective epidemiological studies in this field are needed in the developing world."

Other recent studies have focused on the relationship between BMI and morbidity. Their findings are briefly reviewed, principally because they were conducted in Bangladesh and Nepal, and highlight some of the functional consequences of low BMI.

1.214 Studies examining BMI and morbidity in developing countries.

There are a few studies which examine the impact of low BMI on morbidity in developing countries. None of these studies report specific causes of morbidity, but focus on days of self reported illness, where severity is defined by working days lost or days spent in bed due to illness.

For women in a small study in Rwanda, the number of days of illness increased markedly with decreasing BMI (Francois, 1990; WHO,1995a). Women with BMI <17.0 had 77 days of illness per year compared with 14 days for women of BMI 18.7-23.8. In rural Sarawak a similar inverse relationship between BMI and reported illness was observed for both women and men. However, the ability of BMI cut-off values to predict illness did not achieve a high specificity or sensitivity (Strickland and Ulijaszek,1994). A larger study in Brazil found a U-shaped relationship between days ill in bed and BMI for both women and men, which was

most marked for women (de Vasconcellos,1994). 4.5% of women with BMI <16.0 spent 8-14 days in bed due to illness compared with 0.9% of women with BMI 18.5-19.9.

A study of urban slum households in Kulna, Bangladesh (Pryer,1993) recorded the number of working days lost during one month by men. There was an inverse association between days lost and BMI. Below a BMI of 16.0, 55% of men had lost one or more working days, compared with 35% among those with BMI 16.0-17.0. Men below 16.0 had a 5.9 times higher risk of losing working days than men with a BMI above 18.5. Pryer comments that it was not possible to ascertain whether a low BMI predisposed men to work-disabling morbidity, or whether the illness itself was a contributory factor to a low BMI status.

There appear to be few papers which examined the relationship of BMI or body weight with specific causes of morbidity in developing countries. Murray et al (1992) in their study of adult Masai, reported cause specific morbidity for adult men and women combined, by BMI status over a 12 year period. Morbidity data collected included malarial attacks/year, other infections and parasites/year, malignancy/year, major trauma/year, miscellaneous/year, mean days of incapacity per person per year. They observed that infections, in particular malaria, were significantly less in the undernourished adults. Mean annual time of incapacity was also lower in the undernourished adults. There was no difference between BMI groups with respect to the other causes of morbidity. The authors suggest that the morbidity data suggests that chronic undernutrition is potentially a protective adaptation for the Masai, who live intimately with their environment, and may be advantageous for survival.

Studies of BMI and its relationship with illness episodes or work loss provide evidence, at a very general level, which has been important for the current interest in understanding the functional consequences of low BMI in developing countries. The research covers a wide range of possible determinants and consequences, from energy requirements and basal metabolic rates, to economic productivity. This thesis does not have any information on these factors, and therefore this literature is considered only in the discussion of findings in Chapter 6.

1.22 *Height and mortality.*

Adult height has long been considered a marker of nutritional experiences during childhood, and consequently at a population level, height has been used as a proxy for the economic and nutritional situation. Records were made of European heights and weights in the 18th and 19th centuries. In 1865 Beddoe identified weight and height as possible indicators of social and racial differences in Britain. He obtained anthropometric and socio-economic data on 3,498 men from England, Ireland, Scotland and Wales, and observed that heights and weights were higher among public service and professional men than among lower social status occupations (Riley,1994). Historical anthropometry data has been used by demographers, historians, nutritionists and economists, in population based correlational studies to examine the relationship between height and other factors, including mortality, food availability and prices and labour productivity (Kunitz,1987; Gage and O'Connor,1994).

In general, the results of these studies have indicated that gains in stature have often occurred at the same time as increases in income, life-expectancy, material possessions and security. Therefore, many authors have suggested that lower adult height will be an indicator of higher mortality risk (Floud,1993). This has been countered by others who refer to evidence from cross-national comparisons and studies of immigrant communities in the USA (Walker et al,1994). For example, the Japanese achieved life expectancies equivalent to those in Western countries whilst their height remained considerable lower (Riley,1994). In 1960-1980 Sweden and Japan had the world's highest life expectancies, though the Swedes were amongst the tallest in the world and the Japanese among the smallest. A few correlation studies have included data from developing countries (Gage and O'Connor,1994).

Correlation studies in this review are not considered in detail because height is not linked to mortality in particular individuals; and therefore it is not possible to test the hypothesis that short stature is associated with increased mortality risk. It is also not possible to consider the role of potential confounding factors, such as socio-economic status (Hennekens and Buring,1987).

There are a few cohort studies which have analysed the relative mortality risk associated with different levels of height. None of these studies used data from developing countries and most found some slight association between adult height and the mortality risk, which in some studies was eliminated after adjustment for socio-economic status. Although the strength of the association with mortality was not consistent, the relationship pattern was typically inverse and direct. (See Section 1.222). Unlike the literature on BMI and mortality, there are no extensive reviews of prospective studies focusing on height, mortality relationships, and the papers have less cohesive cross-referencing. Many recent studies have focused on specific causes of death, notably coronary heart disease, and do not report all-cause mortality (Notkola et al,1985; D'Avanzo,1994). However, many of the limitations which were identified in Section 1.211 are relevant to cohort studies on height and mortality.

An area of interest discussed in many of the cohort or ecological studies, and other relevant papers, is the importance of height determinants in the mortality risk associated with height. This is highlighted by a paper entitled 'Deprivation in infancy or in adult life: which is more important for mortality risk?' (Ben-Shlomo and Davey Smith,1991). Some studies have been able to attempt to adjust their mortality risk analyses for indicators of childhood and adult socio-economic status. Interest in this aspect has been stimulated in the last decade, in part due to the large number of publications by Barker and colleagues in the MRC group at Southampton University. Their general hypothesis is that a baby's fetal nutritional experiences, which are manifested in fetal and infant growth, are important pre-determinants for the development of mortality risk factors, including raised blood pressure, fibrinogen concentration, factor VIII concentration and glucose intolerance ((Barker,1994;Goldberg and Prentice,1994;Paneth and Susser,1995). Barker et al have not examined the direct relationship between adult height and mortality but they consider that adult height is a proxy for circumstances in infancy and childhood. Other authors view the relationship differently, suggesting that short height may also be a marker for deprivation in adulthood (Ben-Schlomo and Davey Smith,1991). For the thesis cohort, no information is available on birth weight, childhood growth or socio-economic status and therefore this review does not attempt to review this literature in detail. In Chapter 6, these concepts are raised again where pertinent to the discussion of the thesis findings.

1.221 Limitations of previous cohort studies examining height and mortality.

There are several methodological and conceptual constraints in using prospective studies to analyse the association between height and mortality.

1) Failure to adjust for smoking

The failure to adjust for smoking in some retrospective studies, i.e. the Norweigian experience, may overestimate the mortality risk associated with shorter height. Shorter adult height has been shown to relate to lower adult socio-economic status (Evelynth and Tanner,1990); and a higher prevalence of smoking is observed in low socio-economic status compared with those of higher socio-economic status (Davey Smith et al,1990). Therefore smoking may confound the association between height and mortality.

2) Small sample sizes and/or short follow-up durations

As discussed in Section 1.211, small sample sizes may result in small numbers of individuals in each category of height and wide confidence intervals for estimates of mortality risk. Studies using short follow-up durations, particularly those with small samples, may have insufficient numbers of deaths with which to observe statistically significant associations between height and mortality.

3) Illness and stunting

Growth may be retarded during fetal life and/or childhood due to conditions that predispose adults to a higher risk of mortality. These conditions can be of hereditary, congenital, infectious, or accidental origin. They include, all chronic diseases of the vital organs e.g. heart, lung, liver, kidney and brain pathology, and some congenital biochemical pathologies, e.g. phenylketonuria; and infections, e.g. malabsorption due to persistent diarrhoea. Inclusion of these individuals could overestimate the mortality risk due to shorter heights (Leon et al,1995). It may be difficult for studies to identify individuals with stunted growth on enrolment, and may only be able to exclude individuals with current severe illnesses. In a study of Swedish adults, the shortest group had the largest proportion of individuals reporting long-standing illness (Peck and Vågerö,1989).

4) Age-related changes in height

There is considerable evidence from longitudinal studies in developed countries that height declines with age. Typically the decline starts in the late 40s, with the rate of decrease increasing in older ages. (See Section 1.11). Therefore, in longitudinal studies where height was measured only once at the beginning of follow-up, the inclusion of women whose heights had already declined may over-estimate the mortality risk associated with lower heights. No studies were identified where repeated height measures were collected throughout the follow-up period, allowing mortality risk to be adjusted for age-related changes in height. Leon et al (1995) also considers the possible role of illnesses in causing 'shrinkage' in height. However, no supporting evidence was available.

5) Inappropriate control for confounding

Socio-economic status in both adults, usually measured by current occupation, and childhood, as measured by father's occupation, have been shown to be strongly associated with adult mortality (Marmot et al,1984). Height has been found also to be associated with both the levels of childhood and adulthood socio-economic status (Schumacher and Knussman,1979; Evelynth and Tanner,1990; Peck,1992; Riley,1994). Consequently, any association between height and mortality may be strongly confounded by child or adult socio-economic status. The presence of confounding would over-estimate the mortality associated with lower levels of height.

Identifying appropriate indicators of socio-economic status is very difficult. Many studies are constrained by the limited information collected by previous studies or surveys. For example, the largest study, a retrospective study of 1.7 million Norwegians, did not adjust for socio-economic status, since no data was available (Waaler,1984). Other studies have adjusted for adult socio-economic status, but they had no information on childhood status (Leon et al,1995). Even where adjustment is made, the use of indicators such as occupation grade in the Whitehall civil servant cohort study may not accurately discriminate individuals and reflect the complexity of socio-economic circumstances in adulthood. Therefore, residual confounding may continue to over-estimate the association between height and mortality.

Another complexity in the pathways through which socio-economic status confounds the association between height and mortality, is through the possibility that adult height itself, may be associated with socio-economic status. For example, taller adults are more likely to be socially and economically upwardly mobile, independent of their childhood socio-economic status, and therefore, benefit from the lower mortality risk associated with higher socio-economic status (Peck,1992; Riley,1994; Leon et al,1995).

1.222 Review of selected cohort studies in developed countries.

Few cohort studies were identified which reported all cause mortality. Other studies presented results for a few specific causes of death, primarily CHD, or risk factors for CHD (D'Avanzo et al,1994;Notkola et al,1985). The four studies which examine the association between height and all-cause mortality are summarised in Table 1.6.

Table 1.6

Summary of cohort studies which report all-cause mortality risk associated with height

Data source Reference	Country	Number of subjects (sex)	Age at entry, y	Years of follow-up	Adjustment for childhood socio-economic status	Height mortality association
Whitehall study Leon et al (1995)	UK	18,403 (m)	40-64 in 1967,1969	max. 20 yrs	No	Slight evidence for an inverse association after adjusting for adult socio-economic status. Risk is not significant after 10 yrs of follow-up. Strong inverse association with respiratory disease, moderate inverse association with CHD.
Norwegian experience Waaler (1984)	Norway	1.7 million (m+f)	15+ yrs in 1963-1975	max. 16 yrs	No	Inverse association with height. Strong inverse association with obstructive lung disease, medium inverse association with cardiovascular disease.
National Central Bureau of Statistics Peck and Vågerö (1989)	Sweden	12,695 (m+f)	16-74 in 1980-81	6 yrs	Yes	Slight evidence of an inverse association after adjusting for childhood + adult socio-economic status. Strong inverse association with CHD.

Peck and Vågerö (1989) Terciles calculated as:
(men) short group 145-174cm; medium group 175-180cm; tall group 181-206cm
(women) short 129-162cm: medium 163-167cm; tall 168-180
Leon et al (1995) mortality expressed as the rate ratio associated with a six inch difference in height

The studies reviewed above, when considered in the light of the limitations discussed in Section 1.221, do not present convincing evidence for a strong relationship between height and mortality. In all of the studies strong crude associations were considerably reduced on adjusting for either socio-economic status (childhood or adult) and duration of follow-up period. Stronger associations were observed between height and specific causes of death, CHD and respiratory causes.

Waaler (1984) found that mortality in those 185-9cm tall was half the rate of that found in those 150-5cm tall. The association was a strong inverse relationship between height and mortality, except possibly for the very tallest group. Although Waaler's sample size is impressively large, and his findings are widely cited, his results have recently been criticised as too simplistic (Riley,1994). In the models only height and survival by age group are considered. No adjustment is made for childhood or socio-economic status, which all other studies reviewed found to be independently associated with both height and mortality.

Leon et al (1995) re-examined the relationship between height and mortality among men in the UK Whitehall civil servant longitudinal study, in the light of the current interest in early life experiences and adult health. The cohort of 18,403 men aged 40-64 was initiated between 1967 and 1969, and followed until 1987. Significant inverse relationships were observed between height and all cause mortality, respiratory disease and circulatory disease. In contrast taller men had a higher risk of aortic aneurysm compared with shorter men. No consistent direction of effect was observed with different specific malignant neoplasms. Adjustment for socio-economic status, as defined by civil servant grades, reduced the risk associations, but did not remove the statistical associations. In this study, height effects by period of follow-up were analysed, and the height effect demonstrated a pronounced attenuation with length of follow-up. For coronary heart disease there was no significant association with height by more than 15 years of follow-up. Although the results of the study by Leon et al (1995) could be considered to offer evidence supporting the fetal and childhood origin of adult disease hypothesis, their discussion contains several important reservations which are raised below.

Peck and Vågerö (1989) report the results of a study of 12,695 adults, aged 16-74 years. They were randomly selected from the Swedish population and information obtained on their height, socio-economic status in childhood and adult life, self-perceived health, self reported long-standing illness, and mortality during a six year follow-up. The short height category was defined as between 129-162cm and 145-174cm for women and men respectively. Both men and women in the short group had a 20% excess risk of dying during the follow-up period, compared with the tallest group. When the ratio was adjusted for adult and childhood socio-economic status, the excess risk declined to 15%. They also collected information on self-reported illness. Short men and women were more likely to report currently poor health and long-standing illness than taller groups. However, adjusting for socio-economic status removed the differences between the tallest and shortest groups, particularly for women. Adults of medium height were much less likely to report ill health than tall and short adults.

Several issues are identified from the studies reviewed above 1) importance of sample size and duration of follow-up: 2) relative importance of confounding by childhood or adult socio-economic status; 3) height reduction and pre-existing illness; 4) causes of mortality; 5) gender differentials; 6) minimum mortality risk and height levels.

1) Sample size and duration of follow-up

The studies selected observed large samples, 1.7 million in the Norwegian study, and with the exception of the Swedish study, they had long follow-up periods. Therefore, the reservations about identifying mortality associations in prospective studies with small sample sizes, as discussed in Section 1.211 for BMI studies, would not be expected to be a factor in these studies failing to observe strong height mortality associations.

2) Childhood or adult deprivation

The relative importance of the role of childhood and adult deprivation in determining the increased mortality risk associated with short heights is discussed by all authors, with differing conclusions. However, not all studies were able to adjust their analyses for both childhood and adult socio-economic status, and therefore were unable to test the hypothesis.

Using the Norwegian data, Waaler (1984) suggested that taller individuals are more likely to be socio-economically mobile and advantaged regardless of their childhood status. This is supported by evidence for a independent relationship between adult height and socio-economic status (Barker,1990; Davey Smith et al,1990). Therefore, given the strong evidence for the importance of socio-economic status in determining mortality, mortality risk would be overestimated in the shorter individuals (Notkola et al,1985;Davey Smith et al,1990; Leon et al,1995).The strength of the height mortality association should consequently be reduced if the risk was adjusted for adult socio-economic status, and indeed this was observed by all four studies. In their interpretation of the Whitehall study results, Leon et al (1995) conclude that:

> *"..these Whitehall data provide little support for the contention that circumstances in infancy and childhood influence susceptibility in adulthood to disease in general."*

Peck and Vågerö (1989) draw the opposite conclusions from their study in Sweden, arguing for a potentially important role of childhood influences on adult mortality. They used father's and respondent's occupation as proxies for childhood and adult socio-economic status respectively. Adjustment for present status only reduced the differences in mortality between height groups, and controlling for both current and past socio-economic status further reduced the differences between height groups, but did not eliminate them. The authors concluded that there was a residual association between height and mortality dependent on factors in the childhood environment which could not be controlled for using only father's socio-economic group. Alternatively there might be genetic factors which contributed to both short stature and a higher risk of early adult mortality.

Despite the interpretation by Peck and Vågerö (1989) that their findings only show a small risk of early adult mortality associated with height, they go on to estimate that the excess mortality risk for the shorter tercile of the general population would be 5%, and the reduced risk of the taller tercile would be 10%, after controlling for current socio-economic status. They argue that these deaths would be directly linked to risk factors rooted in the childhood environment, and suggest that their interpretation is supported by other studies by Waaler (1984), Marmot (1986), and Notkola (1985). They concur with Forsdahl's (1977) hypothesis that living conditions during childhood, which also affect

height, can precondition adult health but have little supportive evidence, Waaler (1984) does not present empirical evidence; Marmot (1986) presents ecological data; and Notkola (1985) reports only CHD mortality.

Although all the papers reviewed have discussed the issue of childhood verses adult socio-economic status in confounding the association between height and mortality, it may be suggested that none of the studies are of an suitable design, or have enough information on changes in socio-economic status, height, and their relationship to mortality, to clarify this issue.

3) Height reduction and pre-existing illness

The height association with mortality observed in the Whitehall study was reduced to a non-significant level when the analyses were adjusted for the duration of the follow-up (Leon et al,1995). In the period 0-9 years of follow-up the rate ratios (95% CI) was 1.23 (1.11,1.37), by 10-14 years the rate ratios were reduced to 1.09(0.97,1.23) and by 15+ years to 0.98 (0.86,1.13). The authors suggest that this may result from either differences in maximum attained height (as in point 1) or 'shrinkage'.

'Shrinkage' is described as a situation whereby adults with chronic morbidity leading ultimately to death, might experience a reduction in their height, and consequently overestimate the mortality risk associated with shorter heights. Leon et al (1995) offer an example: *"This 'shrinkage' might be due to postural changes, some of which may be organic, such as kyphosis among men with obstructive lung disease."* They observed that men reporting pre-existing disease at entry to the Whitehall study were on average ¼ inch shorter at the start of follow-up than other men. However, no study collected repeated measurements over the follow-up period and therefore, are unable to assess the importance of 'shrinkage'. Another difficulty in testing the plausibility of 'shrinkage' as a source of potential bias is that height also declines in older age (Noppa et al,1980). This was discussed in Section 1.11.

4) Height relationship with specific causes of mortality

The relationship between shorter height and increased risk of CHD and respiratory disease mortality is more strongly significant than its relationship with all cause mortality in all studies shown in Table 1.6. In the Whitehall study the strongest effect of height was

observed on respiratory disease mortality, with slightly weaker inverse associations with cardiovascular diseases (Leon et al,1995). These findings were similar to those in the Norwegian population (Waaler,1984) and the Swedish population sample (Peck and Vågerö,1989). In contrast, the cohort of Swedish conscripts aged 18 found an increased risk of death from cardiovascular and respiratory diseases with increasing height. This study had a considerable younger average age at measurement compared with those studies where an inverse relationship between height and CHD were observed.

This strong inverse effect of height on CHD mortality, after adjusting for adult and/or childhood socio-economic status, was also shown in other prospective studies in Italy and Finland (Notkola et al,1985;D'Avanzo et al,1994). Notkola et al (1985) in their study of 1,711 men in rural Finland found that some of the higher mortality risk (coronary death, myocardial infarction, ischemic heart disease) among landless and small farmers could be explained by smaller heights. They suggest that this may be a function of nutritional deficiency in childhood or some unknown heredity factor being connected with small height. However, adjustment for childhood socio-economic status did not affect the increased risk in the shortest group. The authors end their paper with a set of open conclusions which include the possibility of residual confounding or alternatively, a genetic factor associated with short height which increases the risk of CHD.

D'Avanzo et al (1994) proposed that shortest adult height can be an indicator of unfavourable living conditions in childhood which, in turn may be associated with an increased risk of coronary heart disease and other diseases. They conducted a case control study in Italy on 429 women with acute myocardial infarction (AMI) and aged under 75 years, and 863 controls. The excess risk of AMI after adjusting for age, BMI, smoking and family history, was 30-40% in the lowest tercile of height (<159cm) compared with the highest tercile (165-180cm). The association was stronger in elderly women i.e. earlier birth cohorts, and heavier women. They interpret the reduced strength of the association between mortality and height in younger women as an indication that the role of environmental factors in childhood and adolescence in determining adult height is less importance in recent years than in earlier generations.

5) Gender and ethnic differentials

Studies including both male and female subjects did not report marked gender differentials in the relationship between height and mortality. All studies reviewed used cohorts of homogeneous ethnicity, therefore there was no comparable information on ethnic differentials in the relationship, as discussed with respect to BMI (see Section 1.212).

6) Minimal mortality risk and height levels

The relationship between height and mortality has also been considered as providing evidence for decreasing mortality risk with increasing height levels, and a challenge to theories which proposed height adaptations of people to their environment. Since the pattern of association observed was typically a direct inverse relationship, the older literature appears to suggest a prevailing perception that tallness equals health, with the assumption that short stature is a good marker for populations which are not 'thriving'. This perception is now challenged by several authors who argue that the existing studies have not adequately examined the roles of environmental factors on anthropometry and health or ill health in different populations. The various reference standards for growth and adult stature were not devised with concurrent clinical examinations to see whether taller children and adults were healthier (Simopoulos and Van Itallie,1984). Riley (1994) has also criticised studies which consider height, without reference to weight.

Walker et al (1994) in their paper 'Maximal growth potential for adult stature: is this aim desirable?' argue that there is little evidence to support suggestions that tall height benefits an individual. They stress that in all populations there are wide ranges of nutritional status which are compatible with good health and that researchers are too ready to 'blame' single risk factors for high mortality in populations. These are similar notes of caution to those discussed by Davey Smith and Phillips (1990).

Samaras and Storms (1992) examined hospital records on 373 men in San Diego, USA with information on height, weight and age at death. The authors state:

> *" while short stature due to malnutrition or illness is undesirable, our study suggest that feeding children for maximum growth and physical development may not add to, and may, indeed by harmful to their long term health and longevity."*

De Waard (1992) and Willett (1989) suggest that the high energy and fat intake during growth periods in childhood which can lead to taller statures, are also risk factors for adult diseases such as breast cancer. The hypothesis is the reverse of that proposed by D'Avanzo et al (1994) for the risk of AMI discussed above.

1.223 The relationship between height and maternal mortality in developing countries.

Many authors have suggested that short height is associated with maternal mortality by being a risk factor for obstructed labour (Krasovec and Anderson,1990; WHO,1995a;WHO,1995b). The WHO Expert Committee on anthropometric indicators (1995b) states:

> *"Measuring a woman's height provides a proxy indicator of childhood growth and skeletal pelvic structure and a good predictor of the risk of cephalopelvic disproportion and obstructed labour, which is a major cause of maternal death in developing countries."*

Heights of pregnant women have been used as an antenatal screening tool in several maternity- care programmes in developing countries. For example, in the MCH-FP programme in Matlab, Bangladesh, heights were measured antenatally, and women with heights <1.50m were considered 'high risk' pregnancies (Fauveau,1994). In a WHO Collaborative study, data from developed and developing countries on maternal anthropometry (height, weight, weight gain and arm circumference) and pregnancy outcomes, were used in a meta-analysis to examine their associations (WHO,1995b). Maternal anthropometric indicators showed none, or only weak, associations with complications of pregnancy and labour, in contrast to the stronger associations with fetal growth. The strongest association was observed between the risk of assisted delivery and women in the shortest quartile of height (OR=1.61).

The studies are limited when suggesting a possible relationship between height and maternal mortality because, 1) the outcome observed was morbidity not mortality and; 2) the absence of studies in Asian populations. These constraints are discussed below.

1. Most studies observed maternal morbidity not maternal mortality

Of the prospective studies conducted in developing countries, only one was identified which observed mortality as the outcome of interest (Tsu et al,1992). Tsu et al (1992) report the results of a population based case-control study in Zimbabwe. Maternal height <1.60m was associated with a significant relative risk of mortality of 2.0 compared with taller women. On adjusting the risk for additional intrapartum factors, including fetal head position, pelvic capacity and birth weight, women <1.60m had a significant relative risk of mortality of 1.9 compared with taller women.

Other studies examined the relationship between height and the risk of cephalo-pelvic disproportion (CPD), assisted delivery, operative delivery, caesarean section (Gebbie,1966;Philpott and Castle,1972;Stewert et al,1979;Harrison et al,1984;Kasongo Project Team,1984;Liljestrand et al,1985;Aitken and Walls,1986;Sokal et al,1991;Kwaukume et al,1993). The majority of these studies observed an association between shorter height and a higher risk of CPD, assisted or operative delivery. However, extrapolating the significant association between height and delivery complications to maternal mortality might be constrained by other intermediate factors, for example, differential health service utilisation and elective interventions. For example, in the absence of appropriate interventions, CPD may result in a fetal death without a concomitant maternal death.

2. All studies identified were conducted in central and southern African countries

Although height is used in antenatal screening protocols in Asian countries, no published prospective studies were identified which evaluated the relationship between level of height and mortality risk for Asian populations. Obstructed labour is also an important cause of maternal mortality in Asia (Freedman and Maine,1993). The relationship between obstructed labour and height observed in one population should be cautiously applied to another, such as rural Bangladeshi women, since there may be marked differences in women's heights, their pelvic position and size, birth weight and fetal dimensions.

1.23 Arm circumference and mortality

No paper was found which presents data relating arm circumference to mortality in adults. As discussed in Section 1.3, studies of arm circumference have been limited to examining its correlation with BMI with a view to its use as a relatively easy indicator in population surveys. Although in use as a screening tool in antenatal programmes (Krasovec and Anderson,1990), there appears to have been no evaluation of its predictive value with respect to maternal mortality.

Chapter summary

Section 1.1 presented an overview of the principal characteristics of the four anthropometric indicators, height, weight, arm circumference and BMI, used in this thesis. With the exception of arm circumference, there is a wealth of literature considering different aspects of their biological determinants, correlation with other nutritional factors, and uses in health programmes. At present there is a large amount of work focusing on the use of BMI in identifying individuals with extreme body composition, i.e. chronic energy deficiency and obesity.

Given the dearth of data from developing countries relating these anthropometric indicators to mortality risk, Section 1.2 discussed the evidence from developed countries. The limitations of these studies was specifically highlighted, since some issues were useful in informing the design, analysis and interpretation of the thesis. From the body of literature reviewed, there appears to be strong evidence for an association between high levels of BMI and mortality, but less certainty about the association with the low BMI levels observed in cohorts from the US and Europe. Also the relationship between height and mortality is not convincing, although several studies observed a lower mortality risk among taller individuals. The papers discussing height may be loosely characterised as having intricate discussions over largely untestable hypotheses.

A special note was made of two areas of anthropometry research which have been conducted in developing countries these are: the relationship between BMI and morbidity as measured by days of illness; and height as a risk factor for obstructed delivery and maternal mortality.

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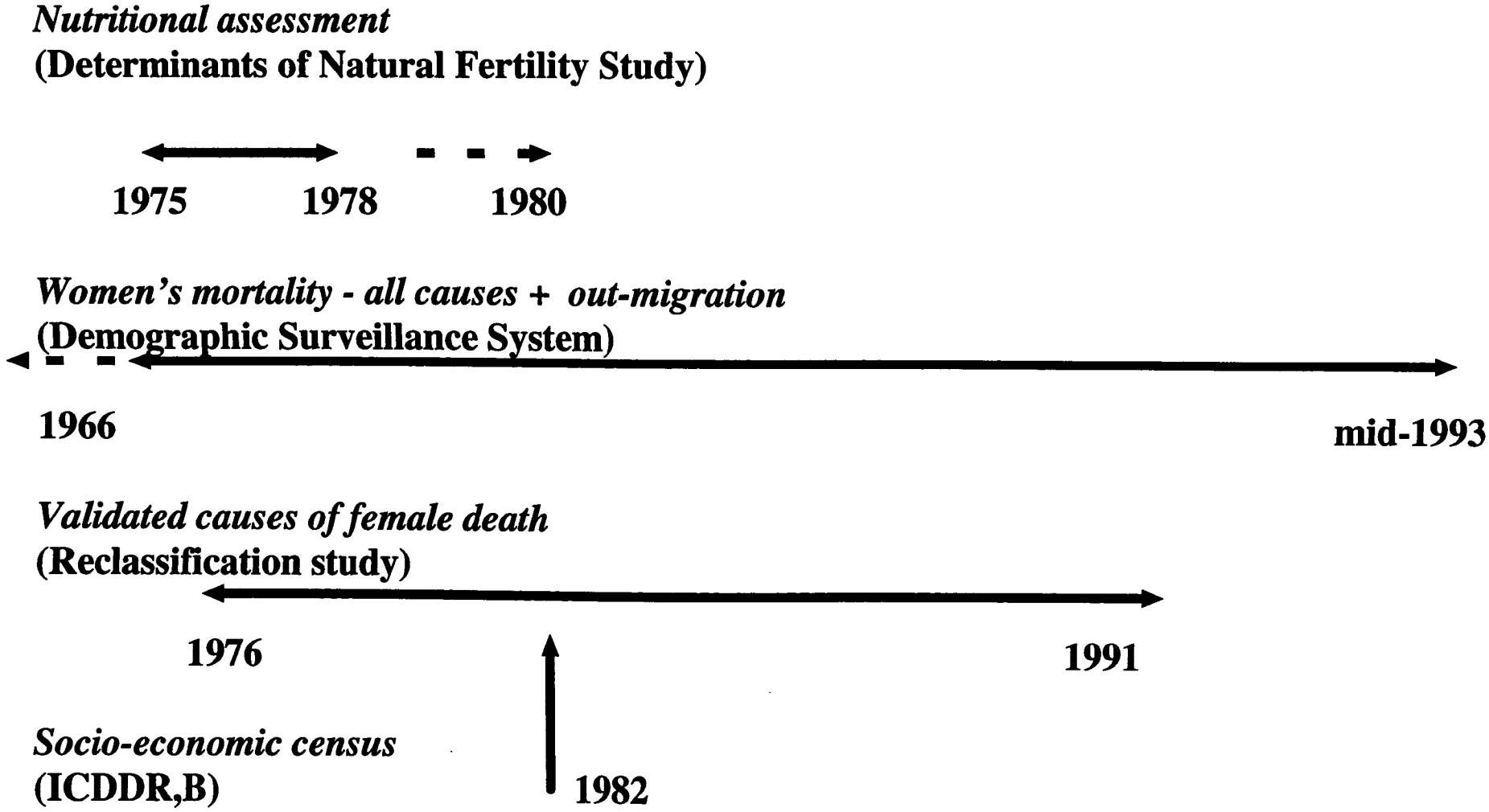
Chapter 2 Methods and population

Chapter introduction

The subjects used in this thesis are a sub-sample of 2,314 women drawn from a historical cohort of women from the Determinants of Natural Fertility Study (DNFS). The DNFS was a longitudinal study investigating the role of nutrition in determining fertility conducted in Matlab, Bangladesh between 1975 and 1980. In this thesis women were tracked from their date of entry into the DNFS until mid-1993 using data from the Demographic Surveillance System (DSS) administered by the International Centre for Diarrhoeal Disease Research (ICDDR,B) in Matlab. The DSS is a surveillance system monitoring all births, deaths, marriages and migrations for approximately 200,000 people, among them members of the DNFS cohort. Cause of death data were also extracted from a 1976-1991 study of all reproductive aged female deaths in the DSS area. Additional socio-economic data were obtained from the 1982 census of the DSS population. The data sets are illustrated in Figure 2.1. The first section of this chapter discusses the methodology used in the extraction, preparation and validation of the study data. In addition, it gives a brief description of the design and objectives of the primary sources of data. In the second section the geographical, economic and social features of both the Matlab area and the cohort itself are described.

Figure 2.1
Dates of data collection for the various data sets used in this study



SECTION 2.1 Data extraction, preparation and validation.

This section describes the methods used to collect, link and validate the secondary data. The nutritional, vital status, cause of death, and socio-economic data are discussed individually because they are obtained from separate sources, and the method of validation consequently also differs. A detailed description of the objectives, the design and important publications of the three sources of data used in this study, the DNFS, the ICDDR,B DSS data, and a retrospective adult female mortality study in Matlab, are given in each relevant section.

2.11 Data extraction, linkage, coding and processing

The main phase of secondary data preparation began in August 1994 and was completed by February 1995. The data extraction and preparation was supervised by the author in the ICDDR,B offices in Dhaka and Matlab, Bangladesh.

The linkage of data sets is facilitated by a unique registration identification number (RID) given to all individuals being followed by the DSS (Box 2). Due to this potential for linkage, all studies or censuses being conducted in collaboration with ICDDR,B, Matlab, record RID information. Since all of the women enrolled in the DNFS were also participants of the DSS, the RID allowed the cohort to be linked to various DSS databases and censuses.

The DNFS data in ASCII format and the file code plan were provided by the Population Sciences Division of ICDDR,B. The dataset contained all the enrolment and monthly visit data for the entire period of the DNFS study. For ease in handling repeated measures of nutritional and fertility data most data was handled and analysed using DBASE IV and STATA 4.0 for DOS.

The DSS data are stored in a relational database maintained on a mainframe computer system. The processing and extraction of data from the system requires specialist programming. Data was extracted in ASCII format and later transferred into DBASE IV and STATA 4.0 for DOS formats. The central database contains basic information on

participants currently in the DSS, this file identified the vital status data of cohort survivors as of mid-1993. Other information about the cohort was extracted from supplementary databases on marriage, outmigration, mortality, birth, and the 1982 socio-economic census. Some of the coding schemes used by the DSS have been modified several times. Therefore, some extracted data contained a mixture of codes, for which all code plans used were obtained and the data standardised.

2.12 Criteria for inclusion

The DNFS cohort was the sample frame from which the thesis sub-sample was drawn. The inclusion criteria reflects both the objectives of this thesis, i.e. the selection of women with anthropometric data and vital status information; but also the requirement for a valid linkage number.

An individual was considered eligible for inclusion in the cohort if she:

i) was a subject enrolled in the DNFS;

ii) had a valid registration identification number (RID);

iii) had a starting date for entry into the DNFS study;.

iv) had at least one anthropometric measure (height or weight or arm-circumference) recorded by the DNFS whilst not pregnant or in the post-partum period.

36 women without an accurate registration identification number were excluded because it was not possible to identify them in other data sets. In addition, one woman without an entry date for the DNFS was excluded since it was not possible to establish a precise follow-up period. Following these steps each anthropometric measurement was cross-matched with the fertility status at the time of measurement. Ninety-five women without a non-pregnancy measure of either weight, height, or arm circumference were excluded. Consequently, 2,314 women were included in the study cohort.

2.13 Nutritional status data

The principal reason for using the DNFS data set was the availability of anthropometric data on a large group of adult women, with the potential to identify vital events over the subsequent 18 year period. The general objectives and design of the DNFS study are

discussed in Box 1. Nutritional status data, including weight, arm circumference and wrist circumference, were collected monthly over a period of 29 months between November 1975 and May 1978. Height was measured only once at enrolment. All 2,446 women in the DNFS had some missing monthly measurements due to staggered starting dates, late arrival of measuring equipment, temporary absences, out-migrations or deaths. Consequently, there was a maximum number of 27 completed measurement rounds for weight and arm circumference.

Box 2.1

Determinants of Natural Fertility Study (DNFS)

The DNFS examined several aspects of the relationship between nutritional status and fertility, including the relationship between non-pregnancy nutritional status and fertility, nutritional changes during pregnancy and lactation, birth-interval dynamics and maternal weight, and the relationship between nutritional status and fetal mortality. 2,446 women from 14 villages in the Matlab DSS area, were recruited into the study between October 1975 and January 1980. All married women in the villages were eligible unless they reported menopause, or if parous, had not had a birth in the preceding five years, or who had not had a menstrual period in the preceding six months.

On entry into the study, data were collected on demographic and socio-economic factors, pregnancy histories, and current pregnancy status. Each woman was visited monthly by field workers to obtain information on their reproductive status, spousal absence, breast feeding, illness, fetal, still births or child deaths. For the first two and a half years measurements of weight, arm circumference and wrist circumference were collected during these monthly visits. Weight measures were not initiated until January 1976. Height was measured once at enrolment. In addition, hematocrit, protein and albumin were also measured every two months during the first year of the study.

The bibliography below contains some of the key papers which have been published using the DNFS data:

Chen LC et al (1974) *A prospective study of birth interval dynamics in rural Bangladesh.* Population Studies. 28(2):277-296.

Ford K, Huffman SL, Chowdhury AKMA, Becker S, Allen H, Menken J (1989) *Birth-interval dynamics in rural Bangladesh and maternal weight.* Demography. 26(3):425-437.

Huffman SK, Chowdhury AKMA, Mosley WH (1978) *Postpartum amenorrhoea: how is it affected by maternal nutritional status?* Science. 200:1155-1157.

Huffman SL, Chowdhury AKMA, Chakraborty J, Mosely WH (1978) *Nutrition and post-partum amenorrhoea in rural Bangladesh.* Population Studies. 32(2):251-260.

Huffman SL et al (1979) *Difference between postpartum and nutritional amenorrhoea.* Science. 202:921-923.

Huffman SL, Wolff M, Lowell S (1985) *Nutrition and fertility in Bangladesh: nutritional status of nonpregnant women.* Am J Clin Nutr. 33:144-154.

Huffman SL (1985) *Responses to determinants of natural fertility reconsidered. Population Studies.* 39:163-168.

John AM, Menken JA, Chowdhury AKMA (1987) *The effects of breastfeeding and nutrition on fecundability in rural Bangladesh: a hazards-model analysis.* Population Studies. 41:433-446.

Miller JE, Rodriguez G, Pebley AR (1992) *Do reproductive patterns affect maternal nutritional status?: an analysis of maternal depletion in Bangladesh.* (in press)

Miller JE, Rodriguez G, Pebley AR (1993) *Lactation and mother's post-partum weight change: an analysis of maternal depletion.* (in press)

In the analyses of relationships between anthropometry and mortality presented in this thesis, repeated anthropometric measures are not appropriate, and a single summary measure for each woman was therefore calculated. The specific aspects of preparing and validating the anthropometric data are described in Chapter 5.

The reason for excluding these measurement rounds is that during both pregnancy and the post-partum period significant changes in the levels of both weight and arm circumference are observed. Several of the papers published using the DNFS data have reported detailed information on these changes (Ford et al,1989;Miller et al, 1993). However, seasonal changes in weight and arm circumference were included in the calculation of anthropometric summary measures.

The use of Cox's proportional hazard models for the later survival analyses presented in Chapter 5 was the principal rationale for not including 30 women from the DNFS in the study cohort who although at least one anthropometric measure was available, were lost to follow-up at an unknown time. In the Cox's models data must be available for all covariates, and for these 30 women no socio-economic data was available from the 1982 census, and their time to loss of follow-up is unknown. The possible bias which excluding these women may have had on the profile of anthropometric measures was tested by comparing their nutritional status indicators with those of other women who had outmigrated. The anthropometric data were prepared using the methodology described in Chapter 4. The results are presented in Table 2.1 and suggest that there are no significant differences between the nutritional status of those excluded and other women who outmigrated.

Table 2.1

t-test comparisons of mean levels of anthropometric status indicators between women with and without a date of outmigration

	Outmigration date known	**No.**	**Mean**	**Standard deviation**	**t-test p value**
Height	Yes No	315 27	1.477 1.470	0.048 0.049	t=0.74 p=0.4572
Weight	Yes No	275 23	40.713 39.25	4.775 4.309	t=1.42 p=0.156
Arm circumference	Yes No	278 26	22.094 21.765	1.471 1.500	t=1.09 p=0.2770

2.14 Ascertainment of vital status

The follow-up of the cohort women was conducted through the Matlab DSS, an overview of which is presented in Box 2. The RID numbers of the cohort women were matched into the DSS data base, and a list obtained of 1,861 women who were currently resident in the DSS as of mid-1993. For women who were no longer registered in the DSS, the mortality and migration databases were searched. 126 women were recorded as having died prior to mid-1993 and their date of death and cause of death information were extracted. The remaining 327 women were identified in the migration database.

The main method of validation for the vital status information was consistency checks. The mortality and migration files were checked to ensure that women who were registered with the DSS in mid-1993 were not also recorded as having died or out-migrated. There were no duplicates with respect to mortality, probably since this is a check that the DSS runs itself.

Where there were inconsistencies in the migration history these cases were checked against the record books held in the field office in Matlab and the last outmigration date confirmed. Inconsistencies were found for some women where the migration file recorded them as out-migrating twice without an intervening in-migration date. In addition, a few women who had returned to the DSS area in the early months of 1993, were registered in the DSS, but the migration file had not been updated.

The DSS database was also used to obtain women's DSS registered date of birth. In rural Bangladesh age reporting is generally very inaccurate. An approximate age was assigned to most adults and children on enrolment into the DSS at its inception in 1966.No information was available on age for 50 women in the DSS but the DNFS appear to have estimated their ages at the time of enrolment, consequently for all women the ages recorded by the DNFS were used to ensure consistency.

Box 2.2

ICDDR,B Demographic Surveillance System, Matlab

In 1966 a registration of births, deaths, marriages, divorces, in- and out-migrations and internal migrations in the Matlab area, was initiated following a census by ICDDR,B. Initially, the DSS covered 132 villages, but by 1992 it included 149 villages with a total population of 205,770(ICDDR,B, 1995). In 1977 the Maternal and Child Health-Family Planning and Health Services Project (MCH-FP) was launched in 70 of the villages in the DSS, the remaining villages were designated as a comparison area. The DSS continued to operate in both areas.

Individuals are allocated a permanent 10-digit registration identification number (RID) which relates to their village, household, their kinship relation to the head of household, and the period in which they were enrolled. In addition, they are assigned a flexible current identification number (CID) which is altered following internal migration, or a census, and can be used to locate individuals.

Vital events are recorded during bi-monthly household visits by community health workers (CHW) which are made in the comparison area and the MCH-FP area. Senior Health Assistants check the event forms and the data are entered in both record books, and the DSS computer database. Any inconsistent records identified by routine validation of the database, are referred back to the field for further checking.

Key references describing the Matlab DSS are:

Fauveau V (1994) (ed) *Women, children and health.* Special publication No 35. Pub: Pioneer Printing Press, Dhaka.

Nahar L, Sarder AM, Van Ginneken JK, Kham MKA (1996) *Demographic surveillance system - Matlab. Volume 26. 1993 population census.* Scientific report no 78. Pub: ICDDR,B, Dhaka.

Zimicki S, Nahar L, Sarder AM, D'Souza S (1985) *Demographic surveillance system - Matlab. Cause of death reporting in Matlab. Source book of cause-specific mortality rates 1975-1981.* Vol 13. Scientific report no 63. Pub: ICDDR,B. Dhaka.

2.15 Cause of death data

Data was obtained from the DSS on the causes of death for all cohort women who died whilst registered in the DSS. The DSS archives were also searched to obtain the original DSS death reports. 124 of the 126 cohort deaths were found, and reports written in Bengali were translated. The various approaches used by the DSS to identify and classify causes of death are described in Box 3. In 1986 the DSS modified its death classification code scheme without updating historical computerised mortality records. Consequently, the DSS data contain a mixture of death codes for cohort women.

Box 2.3

Classification of causes of death in the Matlab DSS

All deaths of individuals registered in the DSS are recorded and when a death is notified, procedures are initiated to identify the cause of death. Prior to 1986, ICDDR,B health assistants and/or supervisors, without formal health training, completed a death registration form (Appendix 2.1). This was based on their own personal impressions of the events leading up to the death, and they were allowed to suggest their own diagnosis. They wrote a few unstructured summary lines in English; many forms are both poorly written and present little relevant information. The assistants were also responsible for coding the causes of death from a list of 29 broad classification categories (Appendix 2.2).

In 1986 the format of death registration forms and the training of staff was modified. Information obtained from interviewing the deceased relatives and care providers was summarised on the form in Bengali (Appendix 2.3). Additional information from laboratory tests, X-rays and specialist's reports was noted. Classification of the cause of death was made by paramedical staff on the basis of these forms. The coding system was expanded (Appendix 2.4), modelled on the "basic tabulation list" of the World Health Organisation (WHO) International Statistical Classification of Diseases, Injuries and Causes of Death (Fauveau,1994).

Publications discussing the classification of deaths in the Matlab DSS:
Fauveau V(1994) (ed)*Women, children and health.* Special publication No 35. Pub: Pioneer Printing Press, Dhaka
Zimicki S, Nahar L, Sarder AM, D'Souza S (1985) *Demographic surveillance system - Matlab. Cause of death reporting in Matlab. Source book of cause-specific mortality rates 1975-1981.* Vol 13. Scientific report no 63. Pub: ICDDR,B, Dhaka.

In addition to the DSS causes of death, information about mortality in a sub-sample, 73 of the 126 cohort women who died, was available from a retrospective adult female mortality study conducted in Matlab DSS between 1976 and mid-1991, this reclassification study is described in Box 4. Those not included in this mortality study were older or younger than 15-44 at their time of death or had died after mid-1991. Three women should have been included in the reclassification study but were not. For these 73 women, two sources of causes of death are available - the DSS code and the reclassified code.

To prepare the cause of death data for descriptive analyses in this thesis, there was a need to standardise the coding system across and within the data sources, and to develop a rationale for accepting or disregarding conflicting classifications of cause of death. In order to standardise causes of death codes, a new code plan of 23 broad groupings of causes of death, was defined, these are shown in Table 2.2. All other DSS or reclassification codes plans were matched to these new codes and the files updated.

Box 2.4

Adult female mortality study in Matlab

This retrospective study investigated all deaths to women aged 15-44, recorded by the DSS in the Matlab area between 1976 and mid-1991. The principal objectives of this study were to identify all deaths which resulted directly or indirectly from maternity-related causes.

All deaths were checked against pregnancy outcome and death records. In the comparison area, if no pregnancy or pregnancy outcome was reported, the relatives of the women were interviewed. A copy of the questionnaire used is presented in Appendix 2.5. In the MCH-FP area, the MCH-FP records were also checked for pregnancy or pregnancy outcome reports, and if no record was found, relatives and the local community health worker were interviewed. Although the focus of this study was maternal deaths, the researchers investigated all causes of death, and recoded all deaths independently of the DSS. Both deaths where the cause of deaths was altered following investigation, and those accepted, had codes updated to the post-1986 code system. In addition, deaths identified as a maternal death were given a special code, with an additional comment on the timing of the death, i.e. during pregnancy, during delivery or during the post-partum period. In presenting the findings, the authors used a definition of maternal death based on that of the American Medical Association, where a maternal death is one that occurs while a woman is pregnant or within 90 days of a pregnancy terminating irrespective of the outcome or duration of pregnancy.

The bibliography below indicates some of the key papers published using data from this study:

Fauveau, V., Koenig, M.A. & Chowdhury, A.I. (1988) *Causes of maternal mortality in rural Bangladesh, 1976-1985.* Bulletin of the World Health Organization 66(5), 643-651.

Fauveau, V., Wojtyniak, B., Koenig, M.A., Chakraborty, J. & Chowdhury, A.I. (1989) *Epidemiology and cause of deaths among women in rural Bangladesh.* Int J Epidemiol 18(1), 139-145.

Fauveau, V. & Blanchet, T. (1989) *Deaths from injuries and induced abortion among rural Bangladeshi women.* Soc Sci Med 29(9), 1121-1127.

Koenig, M.A., Fauveau, V., Chowdhury, A.I., Chakraborty, J. & Khan, M.A. (1988) *Maternal mortality in Matlab, Bangladesh: 1976-85.* Studies in Family Planning 19(2), 69-80.

Zimicki S, Nahar L, Sarder AM, D'Souza S (1985) *Demographic surveillance system - Matlab. Vol 13. Cause of death reporting in Matlab. Source book of cause-specific mortality rates 1975-1981.* Scientific report. No 63. Pub: ICDDR,B, Dhaka.

Table 2.2

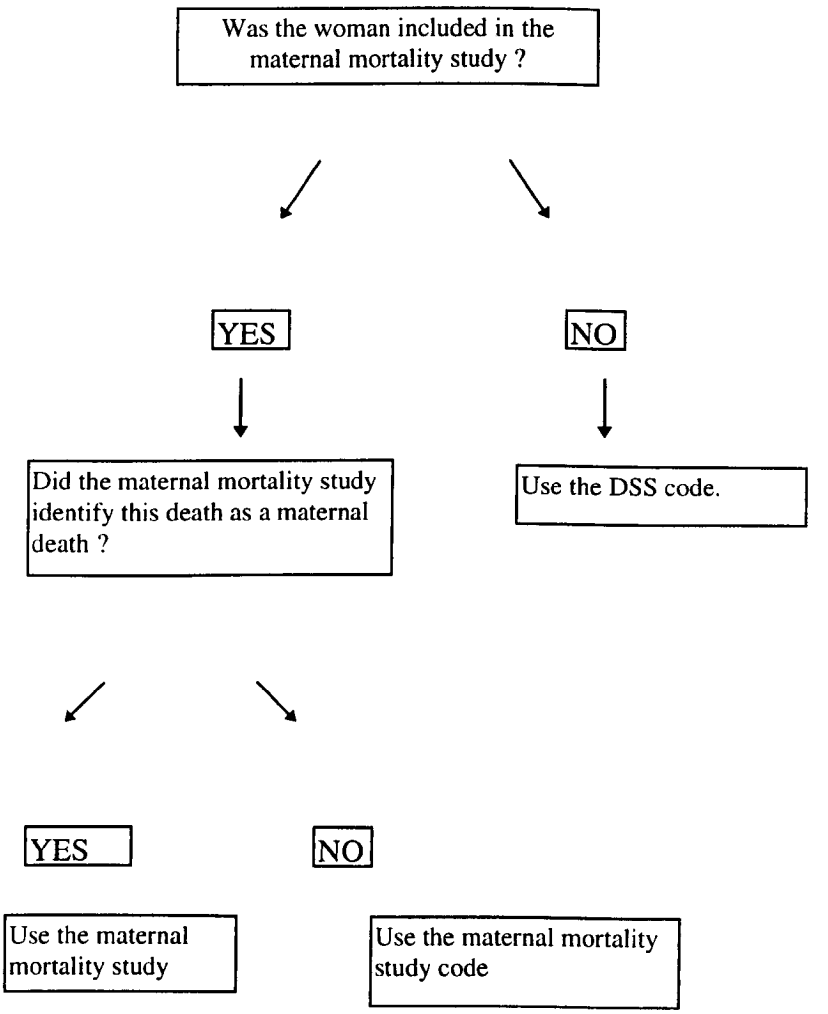
Cause of death categories used to classify female deaths in the cohort

Cause of death	Code
Spontaneous abortion	1
Induced abortion	2
Toxaemia, eclampsia	3
Obstructed labour	4
Haemorrhage	5
Infection puerperium	6
Post-partum tetanus	7
Complications post-partum	8
Diarrhoea, dysentery, cholera	9
Liver, ulcer, hepatitis	10
Respiratory tract infection, including T.B	11
Cardio-vascular disease, stroke	12
Suicide	13
Homicide	14
Accident, snake-bite, burns	15
Fever	16
Rheumatism	17
Tumours	18
Paralysis and malnutrition	19
Diabetes	20
Impossible to specify	21
Oedema and ascitis	22
Complications of health care providers	23

For the 73 women with two classifications of their cause of death, a simple algorithm classifies women by a single cause of death. Zimicki (1986) conducted a case-study examining the cause of death classification system of the DSS. She suggested that the lack of provision for multiple causes of death in the coding system; the lack of a structured protocol to aid coding of deaths; and the limited medical training of the health assistants, results in a poor quality of cause of death data from the DSS. In contrast, in the female mortality study, doctors coded the cause of deaths from structured and in-depth interviews conducted by an experienced interviewer. Given the limitations of the DSS classification protocol, the algorithm used in assigning duplicate causes of death gives precedent to the reclassified data, the algorithm is presented in Figure 2.2.

Figure 2.2

Algorithm used to classify causes of death among the cohort



2.16 Socio-economic and demographic data

A small number of demographic and socio-economic data were collected by the DNFS at the time of enrolment and were available in the data file. In addition, ICDDR,B has several data sets which contain important socio-economic and demographic information about members of the study cohort. Censuses of the DSS population were conducted in 1974 and 1982. The 1982 census had a special focus on socio-economic data and collected information on a large number of household and individual characteristics. Variables available include housing, sanitation, employment, education, and land ownership.

The 1982 census collected information at the family level and not by individual. In the DSS in addition to the unique RID, each individual has a changeable Current

Identification Number (CID). The CID is a 9 digit number allowing individuals to be located in the field, because encoded within the number is information on the village, the *bari*, the family and the individual. Consequently, to obtain the family information on the cohort, their RIDs were matched to their CIDs at the time of the 1982 census and the demographic and socio-economic data extracted.

The 1982 census data was available for 2,268 cohort women, and was completely missing for 46 women. These 46 women had outmigrated from the DSS area before 1982, presumably with their families. Data was available however, for other women who had outmigrated or died before the 1982 census, because other family members were still living in the area at the time of the census.
Validation of the socio-economic data was constrained by i) its secondary nature, i.e. no field checks were possible; ii) the different years in which the cross-sectional data were obtained, DNFS (1975-1978) and the 1982 census; iii) the absence of considerable amount of missing data from the 1982 census.

An initial comparison of variables collected both by the DNFS and the DSS, for example, education, found that some individuals had differences even in variables which might not be expected to alter throughout adulthood. In addition, although a larger range of variables are available from the 1982 census, more data are missing. For example, the education status is available for all women from the DNFS compared with only 2,263 women in the 1982 census.

Since the socio-economic data was required primarily to describe the cohort and ensure the adjustment for potential confounding of the statistical models, where data was available from both sources, the DNFS was used because of its completeness and because the information had been collected directly from the woman herself rather than from the head of household.

In this section the data sources and the methods used to extract, validate and prepare the data have been described. The following section, 2.2 describes the characteristics of the cohort, primarily with respect to the socio-economic and demographic factors obtained from the DNFS and the 1982 census.

SECTION 2.2

Socio-economic and demographic characteristics of the study cohort

Cohorts, by definition are groups of selected individuals. Any analyses and subsequent interpretations should consider both the cohort's demographic and socio-economic characteristics, and the representativeness of the cohort to the population from which they are drawn. Assessing the representativeness of the cohort, with respect to demographic, socio-economic and mortality characteristics, is facilitated by DSS data in the Matlab area. Comparison of the cohort with women living in other areas of Bangladesh is however, constrained by the absence of comparable data on Bangladeshi women as a whole.

In this section the geographical, demographic and socio-economic features of the cohort and the Matlab area in which they lived are described. The mortality and nutritional profiles of the cohort are the focus of separate Chapters, 3 and 4 respectively.

2.21 The Matlab area -

a summary of the geographical, economic and social context

The Matlab area may be considered to be topographically representative of many delta areas of rural Bangladesh. Most areas are low-lying with alluvial soil, criss-crossed by rivers and canals which flood during the monsoon. The main methods of travel are by foot, boat or rickshaw. By the end of the follow-up period in 1993 road access with other areas had improved, and Matlab town is now connected by tarmac roads to the towns of Chandpur, Comilla and Dhaka City (Map 2.1). In the outlying areas travel is predominantly by country boats and foot.

Bangladesh has three prominent seasonal periods, the monsoon period, July-September (average rainfall, 152cm; temperature, 23°-38°C); the dry season, October-February (13°-29°C); and a hot dry season, March-June (26°-38°C). The majority of land in Matlab is used for rice cultivation, with some smaller scale cultivation of millet and vegetables, mainly potatoes and onions. The 1987 Statistical Year Book of Bangladesh reports that the

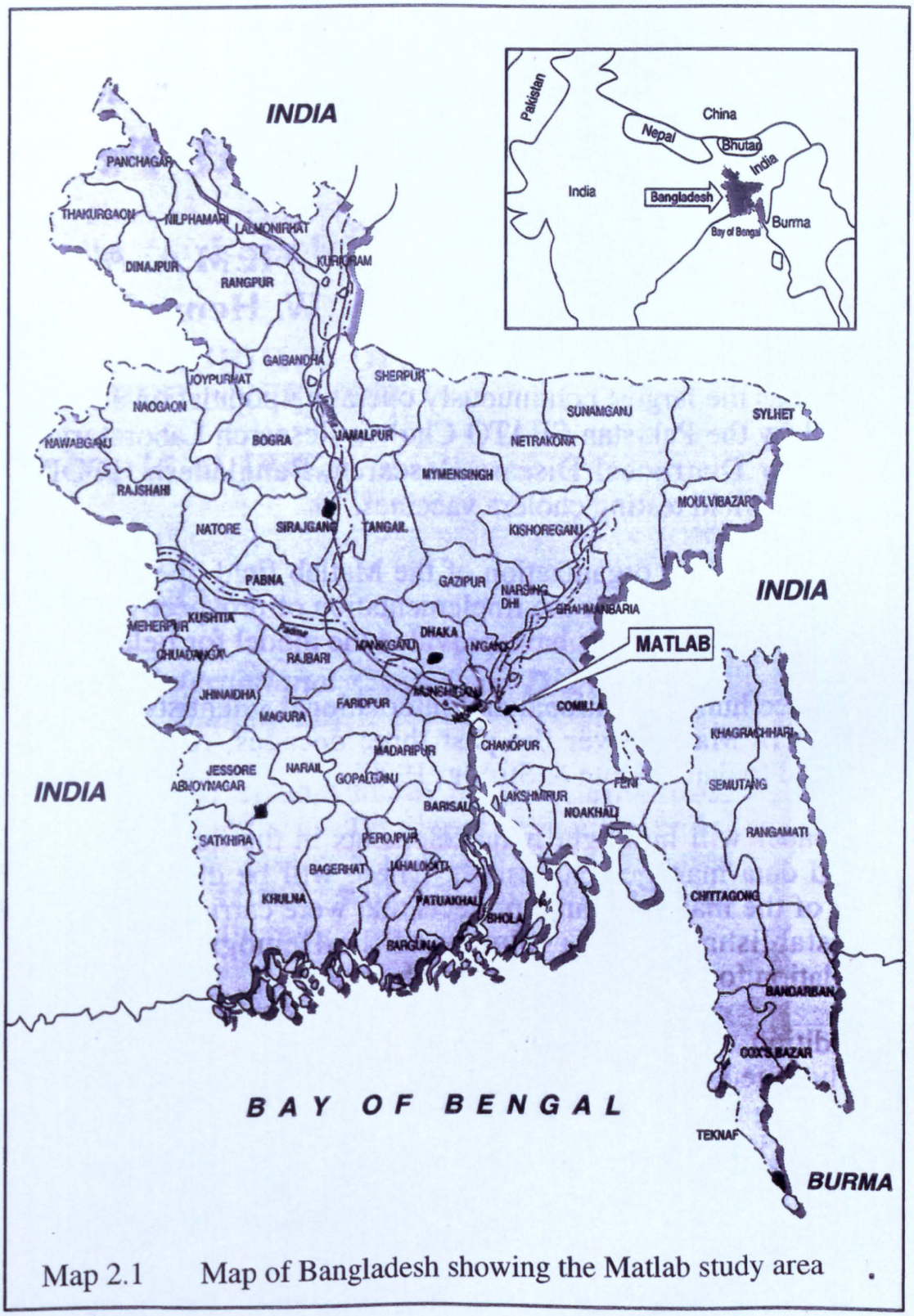
Comilla district, which includes the Matlab area, is the third largest producer of rice and has one of the highest per capita production values of any district.

The four villages from which the majority of women in the cohort were selected, Uddamdi, Baispur, Gazipur and Fatepur, border the large Gumti River close to the Matlab bazaar and the Chandpur road, which makes them relatively accessible by country boat or rickshaw (Map 2.2). Several of the larger villages lie within an embankment area, built to reduce the seasonal flood surges and improve agricultural productivity.

Matlab's proximity to Dhaka, which was one of the attractions of its selection as a field station, has encouraged the development of some manufacturing and light industry, notably garment and storage facilities. The presence of several large mills in the area has also been a source of regular, salaried employment. The town or bazaar of Matlab is a focal point for marketing, with permanent shops and a daily market for local produce. By 1993 there was a large mixed college offering higher education, and there is now a girls school.

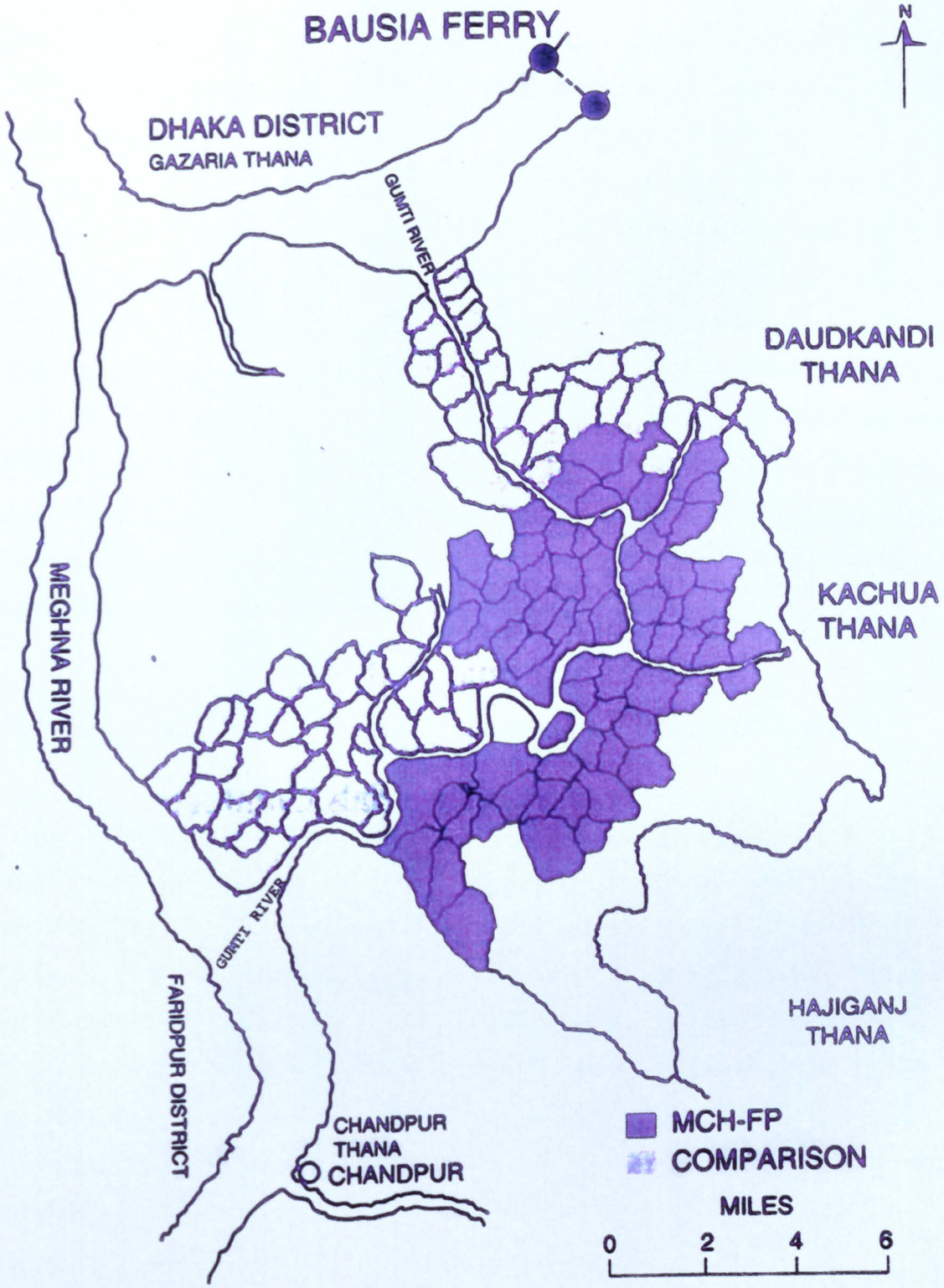
In Matlab the majority of people live in *baris*, which is a group of households sharing a communal courtyard. A household usually consists of a nuclear or extended family, which is related through the male line to other households in the *bari*. A household is considered economically independent but many families in Bangladesh participate in communal sharing of resources that extend beyond the immediate family. Several books provide additional contextual information on aspects of Bangladesh society (Maloney et al,1981; Chen,1983; Hartmann and Boyce,1990; Fauveau,1994:Wood,1994).

The presence of the ICDDR,B research centre, hospital and field health services in the area since 1966, has created semi-skilled and professional employment opportunities, with jobs specifically for women. In 1995 approximately 14 women family planning visitors (LFPV)/ midwives, and 130 women community health workers (CHW) were employed by ICDDR,B in Matlab (Vanneste,1995). Several husbands of women in the DNFS were employed by ICDDR,B. The presence of the research centre has also encouraged other surveys and studies in this area, with short term opportunities for health workers and interviewers. A non-governmental organisation, Bangladesh Rural Advancement Committee (BRAC) has also been active in the Matlab area since 1992, supporting employment generation schemes among the poorest families (BRAC-ICDDR,B,1994).



Map 2.1 Map of Bangladesh showing the Matlab study area

[Source: Fauveau, ed (1994)]



Map 2.2 Matlab area showing villages of the demographic surveillance system (DSS), 1978 [Source: Fauveau, ed (1994)]

The Matlab DSS population is divided into two areas, a comparison and a MCH-FP area, see Box 2.2. All women in the cohort were living in 14 villages in the comparison area at the time of their enrolment in the DNFS. The two major health care providers in the Matlab DSS area are ICDDR,B and the government. ICDDR,B targets most of its specific programmes at residents of the MCH-FP area (i.e. hospital treatment for acute respiratory infections, and maternity related complications; four rural health centres providing basic curative care; and, an intensive programme of home based family planning services, antenatal and delivery care and EPI. The ICDDR,B diarrhoeal hospital however, treats all residents of the Matlab area. Consequently, in the comparison area the governmental health care services are the only free services available. These services consist of a *thana* hospital serving a population of 475,000, an extended programme of immunisation (EPI) and family planning services. In addition, to the formal sector, there are a large number of informal health care providers, including traditional healers and pharmacists.

2.22 Socio-economic and demographic characteristics of the cohort

As described in section 2.15, data on the socio-economic and demographic characteristics of the cohort are available from the DNFS and the DSS 1982 census. The DNFS collected the information directly from each woman at the time of enrolment. For the women followed in this study, this was between 1975 and 1978. The 1982 census information was obtained in a single interview with a senior household member, the data refers to a family rather than an individual. In the descriptive statistics which follow the source of the data is noted. Where data on a single variable was available from both sources, the DNFS was used. Table 2.3 presents the summary statistics for the demographic and socio-economic variables for the 2,314 women in the cohort.

Table 2.3

Summary statistics for socio-economic and demographic factors obtained from the DNFS and 1982 DSS census

Variable	Categories	Number of women n = 2,314	% of women[a]	Summary statistics	Source of data
Age	10-14	93	4.0	mean = 27.5	DNFS
	15-19	494	21.3	±9.1	
	20-29	739	31.9	range 10 - 57	
	30-39	716	30.9		
	40-49+	272	11.8		
Religion	Muslim	2052	88.7		DNFS
	Hindu	262	11.3		
Parity	1-2	544	23.5	mean = 3.96	DNFS
	3-7	1020	44.1	±3.17	
	8+	367	15.9	range 0-19	
	nulliparous	383	16.6		
Number of living children	0	49	2.1	mean=3.64	DNFS
	1-2	643	27.8	±2.17	
	3-5	829	35.8	range 0 - 11	
	6-11	413	17.8		
	nulliparous	380	16.4		
Education in years	none	1767	76.4		DNFS
	1-5	471	20.3		
	6-15	76	3.3		
Household head years of education	0	2073	89.6		DSS 1982 census
	1-5	41	1.8		
	6-16	153	6.6		
	missing	47	2.0		
Husband's occupation	Cultivator	713	30.8		DNFS
	Service	290	12.5		
	Mill worker	259	11.2		
	Fishing	223	9.6		
	Agriculture labourer	169	7.3		
	Businessman	141	6.1		
	Mobile business Self-employed	119	5.1		
		102	4.4		
	Boatman	98	4.2		
	Daily labour	83	3.6		
	Student/unemployed	62	2.7		
	Disabled	23	1.0		
	Professional	11	0.5		
	Other	9	0.4		
	Unknown	6	0.3		
	Domestic labour	4	0.2		
	Beggar	2	0.1		
House-hold size, number of individuals	1-4	684	29.5	mean= 6 ±3	DSS 1982 census
	5-6	665	28.7	range 1 - 31	
	7+	919	39.7		
	missing	46	1.9		

Table 2.3 (continued)

Variable	Categories	Number of women n = 2,314	% of women[a]	Summary statistics	Source of data
Items owned	0-10	1484	64.1	range 0 - 63	DSS 1982 census
	11+	783	33.8		
	missing	47	2.0		
Land owned (acres)	0	590	25.5	range 0 - 200	DSS 1982 census
	1-4	600	25.9		
	5-12	493	21.3		
	13+	584	25.2		
	missing	47	2.0		
Boats owned	none	797	34.4	range 0 - 6	DNFS
	1	1268	54.8		
	2+	249	10.8		
Cows owned	0	1432	61.9	range 0 - 9	DSS 1982 census
	1-2	463	20.2		
	3+	373	16.1		
	missing	46	1.9		
Structure of house wall	pucca and other	324	14.1		DSS 1982 census
	tin	1913	82.7		
	missing	77	3.3		
Dimension of house	0 - 187 sq. ft.	773	33.4	range 0 - 1365	DSS 1982 census
	188 - 294 sq. ft.	745	32.2		
	295 +	747	32.3		
	missing	49	2.1		
Availability of water	tubewell or other	1439	62.2		DSS 1982 census
	tank and river	828	35.8		
	missing	47	2.0		
Distance to water	< 15 yards	801	34.6		DSS 1982 census
	15 + yards	1394	60.2		
	missing	119	5.1		

[a]Percentages may not equal 100 due to rounding.

Women included in the cohort had a mean age of 27.5 years with a mean parity of 3.93. 16.4% were nulliparous and many parous women would not have completed their family size. The mean number of living children, 3.64 was lower than the mean parity due to infant and child mortality. The majority of women were Muslims, 88.7%. Most women lived in large house-holds, 39.7% lived in house-holds of 7 or more individuals. The majority of women had received no education (76.4%) and of the 547 women with some education, 86.1% had only 1-5 years of schooling. Fewer heads of households had attended school (10.4%) than women in the cohort, over half the families (51.4%) were either landless or owned less than 5 acres. 38.1% of husbands worked in agriculture. The largest single occupational group among husbands was that of cultivator (30.8%). There is a low level of ownership of cows or boats and 84% of houses were built of pucca, similar to wattle and daub.

Two variables which have potentially important effects on socio-economic status at an individual level are age and religion. Table 2.4 examines the age groups and religious

affiliation with respect to the lowest category of education, items and land owned. The number and percentage of women are presented.

Table 2.4

Percentage of women without education, land or items, by age group and religion, with statistics

	No schooling (%)	**No land owned (%)**	**No items owned (%)**
Age			
10-14	51 (54.8)	27 (29.4)	25 (27.2)
15-19	365 (67.1)	128 (26.7)	104 (21.7)
20-29	552 (74.7)	186 (25.7)	160 (22.1)
30-39	593 (82.9)	182 (25.9)	137 (19.5)
40+	234 (86.0)	67 (24.8)	62 (23.0)
Test for trend χ^2	$\chi^2 = 47.25$ *p = 0.0000*	$\chi^2 = 0.40$ *p = 0.53*	$\chi^2 = 0.56$ *p = 0.46*
Religion			
Hindu	234 (89.3)	108 (41.9)	61 (23.6)
Muslim	1533 (74.7)	482 (24.0)	427 (21.3)
Relative risk p value	*RR = 1.20 (1.14,1.26) p = 0.0000*	*RR = 1.75 (1.49,2.07) p = 0.0000*	*RR = 1.12 (0.88,1.42) p = 0.36*

Two trends are observed with respect to age. Older women are much less likely to be educated than younger women, the test for trend results suggesting that there has been a consistent increase in the percentage of girls who attend school (χ^2 = 47.25,p=0.0000). For example, women aged 20-24 and 40+ are 2.43 and 5.07 times more likely to be uneducated than women aged 10-14, respectively. In contrast, landlessness is highest among the youngest women, 10-14 years, however, there was no significant trend in the proportion of landlessness by age. Hindu women are 1.20 times more likely to have never attended school (p=0.000), and have nearly twice the risk of being landless (RR=1.75, p=0.000), compared with Muslim women. There was no significant difference between the two groups in the ownership of items. The percentage of landlessness by religious group should also be put in the context of husband's occupation. The single largest occupation for Muslim husbands was as cultivators (33.9%), whereas for Hindus, 63% were fishermen.

A complication of considering ownership of assets such as land, housing or household items by age, is the relationship between age and duration of marriage. Older couples, even those

who had not inherited land, would have more years to secure the financial means to purchase assets, for example land which is highly valued in Bangladeshi society.

2.23 Comparisons of the cohort socio-economic characteristics with district and national data

The age range of the cohort is important when comparing the cohort data with that from other studies. The cohort included women between 10-57 years, with a mean age of 27.5 years. Many studies of adults consider ages between 15 and 65 years, or those focusing on reproductive age women, typically use the age range 15-49 (Feachem et al,1993).

One of the criteria for inclusion in the DNFS was that women were married. In rural Bangladesh between 1966-1971, the median age of first marriage was 15 years, most women marrying shortly after the onset of menarche (Aziz,1978;Chen et al,1974;Cain et al,1979). However, the pattern of nuptiality in Bangladesh has changed markedly over the last few decades, and generally, women marrying at very young ages, <15 years, are from families with very low socio-economic status (Shaikh,1984; Begum and Chowdhury,1985).

The 1974 census of Matlab estimated the proportion of Hindus in the area to be 16%. The cohort has a lower proportion of Hindus (11.4%). In Matlab, the DSS data show that Hindus have been shown to have lower fertility rates than Muslims, at all ages (Fauveau,1994). In addition, tensions between Muslims and Hindus in Bangladesh in the last few decades have resulted in many Hindus out-migrating, many moving to West Bengal in India, further decreasing the proportion of Hindus in Bangladesh, and lowering the socio-economic status of Hindus relative to Muslims. In the DSS area, Hindus declined from 15.9% in 1974 to 12.7% in 1993. Between 1982 and 1993 the Hindu population declined by 0.4% per year, primarily due to out-migration (Nahar et al,1996).

Hindus in the cohort were much more likely to be landless than Muslims. Many Hindu families in the cohort, lived in predominantly Hindu fishing villages. Their houses are often sited on marginal and easily flooded land. Fishing has a low status in Bangladesh

society and without land many Hindus are reliant on small net and line fishing, rather than fish farming.

Comparing the education status of the cohort with other sources of data is complicated by the use of two different terms - education and literacy. Attendance at school is not a proxy for literacy nor in converse, literacy a proxy for attendance at school. For many children the length of time spent at school is insufficient to become literate, and in religious schools the language taught for writing may be Arabic and not Bengali. In both the DNFS and the DSS, education refers to attendance at school and not literacy. Many other surveys have used tests for literacy, for example, the ability to write ones name, or read several lines of text. The low literacy rate in Bangladesh, particularly of women, has been reported in numerous papers and reports (Lindenbaum et al,1985). The 1987 Statistical Yearbook of Bangladesh reports literacy among women to be 15% (Bangladesh Bureau of Statistics,1987). The 1974 DSS census in Matlab reported literacy rates to be 18.5% for both sexes and all ages (Fauveau,1994). A study on a sample of mothers of the 1980 birth cohort in Matlab, identified 26.8% of women as literate (Shaikh et al,1990).

In Bangladesh, schools vary markedly in the standard and format of the education provided. UNICEF (1990) reported that daily attendance in primary schools averaged around 50% of those enrolled, with attendance declining further during peak agricultural seasons. They also noted a high rate of school drop-out for girls and boys combined, 24% in the first year after enrolment. Girls had a higher rate of drop-out than boys. The difference between school attendance and literacy, suggests that although 23.6% of the women from the DNFS had attended at least one year of school, a lower proportion will be literate. In Table 2.4, age was shown to be strongly related to probability of attending school, with decreasing proportions never attending school in the younger birth cohorts. The educational status of study cohort, appears to be comparable with that of both Matlab and Bangladesh in general, given the age profile and the period in which the DNFS was conducted.

The range of husband's occupations are characteristic of Matlab, where the traditional occupations are agriculture and fishing (Fauveau,1994). 38.1% and 9.6% of cohort husbands were working in these occupations respectively. In addition, it is likely that many of the other men would supplement their incomes by agricultural work either on

family land or on a daily wage basis during the harvest periods. The 1974 census found 55.4% of household heads were working in agriculture, and a further 5.3% earned the majority of their income from land they owned (Islam and Becker, 1979).

11.1% of cohort husbands are engaged as mill workers, generally garment and wood mills. Although the wages will vary between the different grades of employment, mill workers have more secure employment than agriculture workers, and are often members of workers unions. Comparison of Matlab with other areas of Bangladesh with respect to employment is constrained by features geography favouring different land use, transport, nearness of markets etc. In addition, most Bangladeshis work in the informal sector, many doing several different, often unskilled jobs, either concurrently or occasionally.

One difficulty in comparing the number of individuals living in a household, is the complex domestic arrangements in rural Bangladesh. Several families, either nuclear or extended, may live within each *bari*. In 1974, only 44% of households were simple nuclear families, consisting of a husband and wife and their unmarried children (Fauveau,1994). A household may also have kinship relationships, but is defined primarily as an independent economic unit. The extent to which ownership of property and land, leadership, and labour is shared between the families or households varies (Fauveau,1994).

The 1974 and 1982 census collected information on the number of household items owned by families, including quilts, radios, lamps and watches. Among the cohort only 22% of households did not own any of the listed items. Data from the 1974 census found that in Muslim nuclear family households, 55% possessed none of the items (Fauveau,1994). The higher ownership rate among the cohort households is probably due to the wider availability of these items by 1982. It could also be related to a difference in the proportion of extended family households in the cohort, but this information was not available. In the 1974 census, larger numbers of extended families owned a combination of these items (Islam and Becker,1979;Bhuiya et al,1986).

Several of the other economic indicators collected by the 1982 census can only easily be compared with the 1975 and 1982 DSS census data, and other ICDDR,B studies in Matlab, because other surveys used different socio-economic indicators. Shaikh and

Becker (1985), in their study of socio-economic status in the DSS, among women aged 15-44 with births between 1974-1977, reported that 52.6% of households owned no cows and 47.3% owned no boats. This compares with 63.1% and 34.6% respectively in the cohort. The interpretation of cow and boat ownership as a indicator of socio-economic status is potentially difficult. In some areas of Matlab the land is higher with fewer canals, in these areas more travel will be by foot or rickshaw. Similarly in low lying areas, the limited grazing areas may make keeping cows impractical. The villages from which the majority of the cohort were selected, are in lower lying western areas, and this may explain a higher percentage of boat ownership but a lower percentage of cow ownership. In addition, owning cows and boats may not be a good indicator of socio-economic status in all families. For example, families with males working exclusively in non-manual work, would be unlikely to require boats, instead choose to pay boatmen or ferries for transport. The importance of boat ownership will also depend on occupation, for fishermen ownership of a boat will be a considerable asset. In addition, fishermen would also be more likely to buy additional boats to rent out when they have the economic capacity, since they have the maintenance skills and a local community of potential clients. Cow ownership also has a more complex relationship with economic status, for example, where families rent out their land, their tenants may own cows, but the land owners benefit indirectly through rent.

The high use of tubewells by households in the cohort (43.0%) is similar to that of (52.9%) found by Shaikh et al (1990) in a study which also used DSS Matlab 1982 census data. The Matlab area has been targeted by NGO and Governmental projects to provide tubewells over the last two decades (DHS,1994).

2.24 The socio-economic and demographic representativeness of the cohort

There are several aspects of the inclusion criteria used for selecting the DNFS cohort, which should be considered for their possible influence on the representativeness of the cohort, with respect to socio-economic and demographic characteristics. One of the main objectives of the DNFS was to ensure the selection of a cohort which maximised the number of fertile women who would be likely to have a pregnancy during the follow-

period. Consequently, women were not included if they were using contraception. Generally, contraceptive use was low in the comparison area in the 1960 and 1970s, a survey in 1968-1969 found fewer than 4% of eligible women to be using a modern method of contraception (Chen et al,1974). By 1984 and 1990, surveys found that modern methods of contraception were being used by 12.6% and 20.0% of eligible women respectively (Koenig et al,1987; Khan et al,1989; Koenig et al,1992). Therefore, women in the cohort were unlikely to be very different from other similar aged women on the basis of their non-contraceptive use. No information was available regarding subsequent contraceptive use by the DNFS women.

The inclusion of only married women, is equally unlikely to bias the profile, in a population where by age 45-49, only 0.1% of women were never married; with the possible exception of very young women aged 10-14 and 15-19. In the 1974 DSS census, only 3.3% and 59% respectively, were ever married. Very young brides have been shown in this cohort to be more likely to be disadvantaged with respect to their material wealth. Although possibly not affecting the socio-economic representativeness of the cohort, since only 4% of the sub-sample selected from the DNFS were of age 10-14, they may affect the generalisability of later results with respect to nutritional status and mortality, since women this young may not have completed their potential growth. The inclusion of very young married women, also results in a slightly different age profile to other studies of adult reproductive aged women, where the age range is typically 15-44 years old.

The exclusion of women who although parous, had not had a birth in the last five years may introduce a selection bias for younger reproductive aged women. As discussed in section 2.23, it may be suggested that some socio-economic and demographic variables, for example land ownership, education and parity, may have a direct association with age. Younger women with young children would also not yet have any financial assistance from them, which is typical in Bangladeshi society. Consequently, the cohort might have a lower socio-economic status than the general population of adult women in the Matlab area.

An important constraint in considering the representativeness of the cohort are two limitations of the demographic and socio-economic data. The first that socio-economic status is very difficult to assess in rural Bangladesh. Since *baris* and groups of households

may co-operate in financial, labour, food and land operations, the variables related to households units may be poor indicators of the material realities of individuals or families. In addition, the influence of other distant family members are often important and some aspects of economic and social status may often be unquantifiable, for example, financial contributions made to other relatives, or payments to agents to secure work overseas. For families without economic reserves, even events requiring modest expenditure, such as illness or building repair, may have a major impact on their economic status.

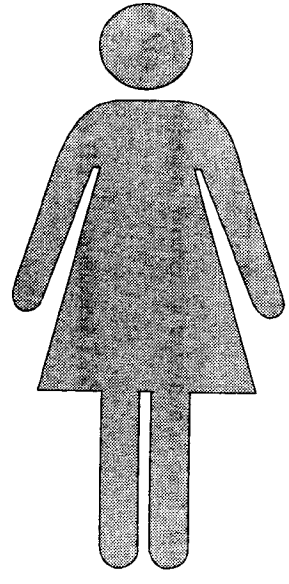
The second limitation is the cross-sectional type of data available. Many socio-economic and demographic characteristics may change over time. However, no information was available for comparison from earlier (childhood and early adulthood) or later years of the follow-up. No papers were identified which discuss the degree of social mobility in this population. Generally, the practice of arranged marriages means that women do not marry into families with very different economic levels, however, economic status of couples may have altered during adulthood.

Chapter summary

The first section of this chapter described the sources of data used in this thesis, and the methods used to link, extract and validate the data. The presence of a historical cohort for which detailed nutritional status data are available, in an area with routine mortality reporting, allows us unique opportunity to examine the relationship between anthropometry and mortality, over a period of 18 years. The key feature that allows the linkage between the data sources is the unique DSS RID. The DSS has also encouraged other studies in the Matlab area. Consequently, we were able to draw upon the socio-economic and mortality data from several other studies. Validation was made through consistency checks both between and within, datasets.

In the last section the socio-economic and demographic profile of the cohort and the Matlab area were described. An important question for this study is whether the cohort is representative of other women in the DSS. Figure 2.3 summarises the profile of the cohort and their environment.

Figure 2.3
Socio-economic and demographic characteristics of the cohort and the Matlab area



2,314 married women
Mean age = 27.5
Parous = 74%
89% are Muslims
76% had no education
21% had 1-5 years of schooling
34% do not own boats
62% do not own cows
26% do not own land
83% have pucca houses
62% use tubewells
70% live in families of 5+ members
38% of their husband's work full-time in agriculture
All women were living in the DSS comparison area

Matlab area 55km south-east of Dhaka.
Low lying delta area experiencing seasonal flooding
One large bazaar in Matlab town
Road and river transport to Comilla and Dhaka city
Most internal transport by boat or foot
Demographic Surveillance System run by ICDDR,B
Government and ICDDR,B hospital
Primary economy is agriculture and fishing
Most land used for rice cultivation

This chapter suggests that there is no evidence of marked bias in their socio-economic or demographic characteristics when compared with other data from the DSS area. They would be expected to be representative of similarly aged women living in the Matlab area in the last two decades.

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Chapter 3 Patterns and causes of mortality

Chapter introduction

This chapter examines the levels, patterns and causes of mortality in the study cohort, and compares the findings with population based data from the surveillance system in the same area of Bangladesh. The first section discusses the use and calculation of life tables to describe mortality. In the second section, various life table techniques are used to examine the pattern of the study women's period and cohort age-specific mortality rates. The third section compares their mortality levels and patterns with those of the ICDDR,B DSS population based data from the same geographical area. In section four the causes of death are described and compared with DSS data. Where annual DSS population level statistics are used they are obtained from ICDDR,B DSS scientific reports (see References).

SECTION 3.1 Methodological approaches to describing mortality patterns in cohort studies

Vital statistics were available from the DSS for 2,314 women from the DNFS cohort from 1975 until mid-1993. During this period 126 women died and 327 women were recorded as having migrated out of the DSS area. 1,861 women were reported to be alive by the latest updated DSS records of mid-1993. The outmigrants are considered lost to follow-up since it was not possible to establish whether they were alive by mid-1993. Table 3.1 summarises these outcomes:

Table 3.1

Survival outcomes of the cohort by mid-1993

Survival outcome by mid-1993	No.	%
Surviving	1,861	80.42
Dead	126	5.45
Out-migrated	327	14.13
Totals	2,314	100%

Several simple approaches are available to summarise mortality in each year of follow-up, for example, crude death rates (CDR)[1]. Although easily calculated, crude death rates are a poor measure of mortality since they do not make any adjustment for the age profile of the population or consider the exact mortality risk of individuals who are lost to follow-up during a year. Age-specific mortality is considered below, and loss to follow-up in section 3.13.

3.11 Age-specific mortality

Age-specific mortality patterns have two important aspects:

i) Age is a marker of temporal periods. In a given year two adults of a similar age will have had their childhood and adult years in the same decades. In societies undergoing a health transition there will be changes in the prevalence and magnitude of risk factors. These risk factors include: health service provision, health seeking behaviour i.e. vaccination and delivery care, environmental factors i.e. sanitation and water supply, schooling, and cultural mores, i.e. *purdah* and women's employment. Women of different birth cohorts, will have been exposed to events at different ages. For example, improvements in emergency obstetric care facilities will not reduce mortality risk in older women who have finished childbearing.

ii) In addition, to grouping women by year of birth (birth cohort), women can be categorised by their exact ages. Age groups can distinguish mortality risk due to causes of death whose incidence or case-fatality rate are age-specific, for example, certain cancers, neurological diseases, or maternal deaths. For example, only women in the reproductive ages, could be at risk of dying of obstetric complications. Obviously age in itself does not adjust for other differences in risk factors between similarly aged women. The exposure to risk factors and disease case fatality are often modified by socio-economic status and biological determinants. However age groups are a vital stratification of any mortality analysis.

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[1] *Crude Death Rate* = $\frac{\text{deaths to cohort women in a single year}}{\text{mid-year population of the cohort}}$ x 10,000

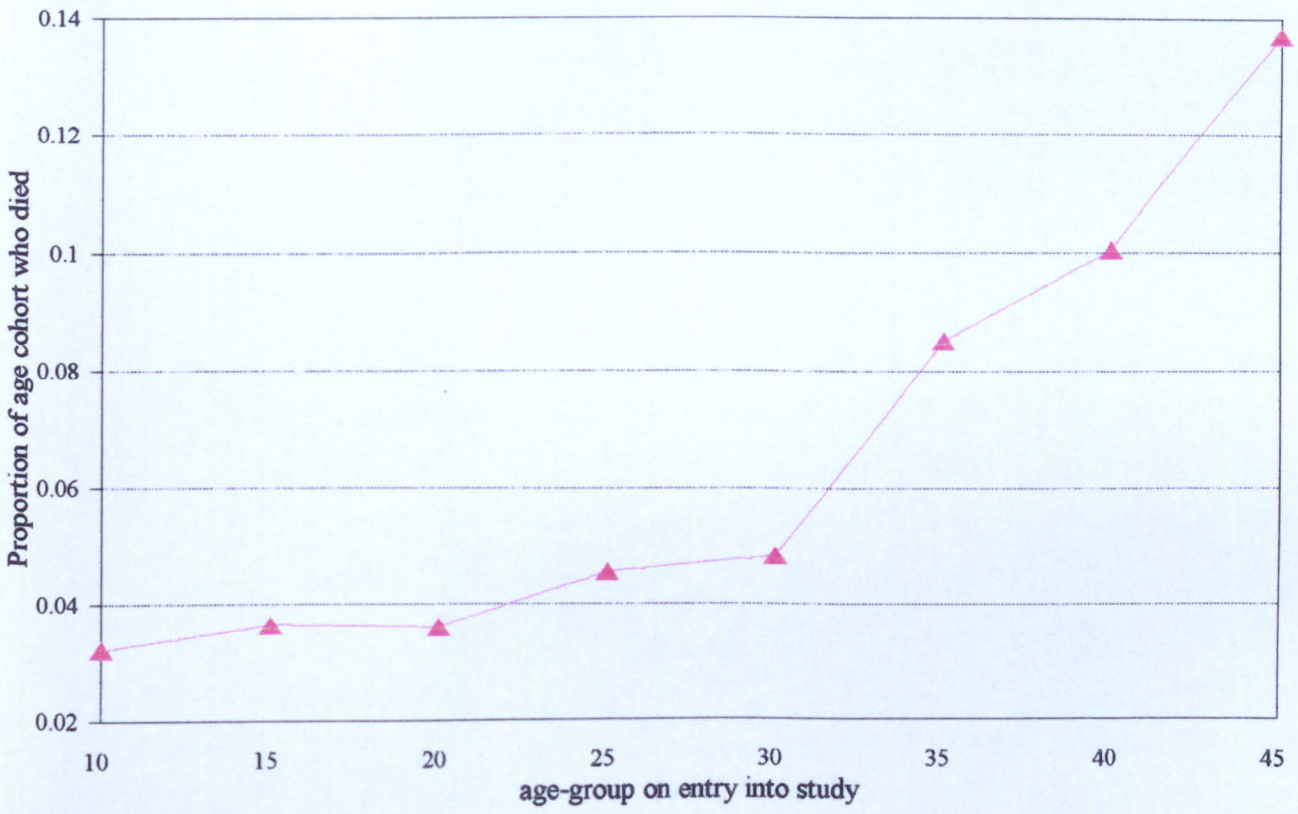
The age profile of this cohort is discussed in Chapter 2. The small sample size and deaths in the cohort require women to be aggregated into 5 year age groups, and the follow-up mortality observed in 5 year periods. However, the staggered enrolment of the DNFS presents a difficulty in assigning women to appropriate age groups. Women in the sub-sample were enrolled in the cohort late in 1975, 1976, 1977 and early 1978. A woman aged 20 years entering the study in 1977 would have been 18 years old in 1975. Consequently assigning her age group on the basis on her entry age would place her in the 20-24 age group. If her age in 1975 is considered, she would be placed in the 15-19 age group. In Tables 3.2 to 3.7 women were allocated to their age group on the basis of their age on entry into the study for two reasons. Firstly, it is conceptually implausible to treat all women as having entered the study in 1975 if their real entry date was later, since they are not at risk of dying during the period before their entry. In addition, an age adjustment back to 1975 would place 3 women in the age group 5-9 years which would have too large a standard error to be meaningful. The potential misclassification of women to age groups by the method used, needs to be balanced against the very limited confidence in the ages reported for the cohort. Age misreporting is potentially a much more important source of error than the method of age group categorising. Table 3.2 and Figure 3.1 show the proportion of women in the thesis cohort who died in each age group.

Table 3.2

Proportion of women dying by mid-1993, by age group

Age group	Population	Deaths	Proportion of age-group who died between 1975 and mid-1993
10-14	93	3	0.0322
15-19	494	18	0.0364
20-24	389	14	0.0359
25-29	350	16	0.0457
30-34	434	21	0.0483
35-39	282	24	0.0851
40-44	199	20	0.1005
45-49	73	10	0.1369

Figure 3.1 Proportion of women dying: 1975-mid 1993



As expected the proportion of women who died increased with increasing age. The highest proportion of deaths occurred in the last age group, 45-49, where 13.7% of the cohort had died during the follow-up period. In comparison, only 3.6% of the 15-19 year age group, who would have been 35-39 in 1995 had died. The graph shows the sequential increase in the proportion dying with each increasing age group. The degree of increase in the proportion of deaths increases after the age group 30-34. It rises from 4.8% in age group 30-34 to 13.7% in the 45-49 age group. By 1995, the oldest age group would have been the 65-69 age group.

3.12 Principles in calculating abridged life tables

Life tables are probability tables with which to describe the survival probabilities of populations with adjustments for age and loss to follow-up in more detail. Although derived from age-specific death rates (ASMRs), several functions can be subsequently calculated to provide estimates of survival between one age group and another, life expectancies, and the patterns of survival in synthetic cohorts. In summary, life tables provide a complete description of mortality at every age for which data is available, enabling patterns of survival to be compared with other life tables based on different populations.

Life tables can be either generational (cohort) or relational (period), depending on the type of data available (Newell,1988). A cohort life table uses data from a cohort study where individuals enter the study at the same age or time, and are followed up over x number of years with the date of death or loss to follow up being recorded. A period life table is produced from cross-sectional data, for example from a survey. The principal assumption made in a period life table is that the risk experiences of young women will be similar to that of older women once they reach the same age.

A life table may be constructed using single year age groups, (a complete life table), or using 5 year age groups (an abridged life table). The latter is more commonly used in demographic analyses, either because the date of death or loss to follow-up is unreliable, or because they provide a more concise, and therefore, more easily interpreted picture of mortality. Since the numbers of deaths in each period, when disaggregated by single years are very small five year age groups are used in this study.

In Table 3.3 the cohort is presented in a stylised approach which can be used to conceptually understand the different approaches which can be used in analysing the mortality over time of this cohort. Each letter represents the ASMR for each age-group in each period. Where the discussion refers to their summation, the population and the number of deaths over the given period are summed, and then the number of deaths are divided by the total population. The period of follow-up has been extended to the end of 1994. The modelling of expected mortality in the last years is described after Table 3.4.

Table 3.3

A stylised table of 5 year age groups and 5 year follow-up periods†

Age-group at entry	1975-1979	1980-1984	1985-1989	1990-1994	Period calculations 1975-1994	Cohort calculations
10-14	*a*	-	-	-	**1**	
15-19	*b*	*i*	-	-	**2**	
20-24	*c*	*j*	*q*	-	**3**	
25-29	*d*	*k*	*r*	*y*	**4**	
30-34	*e*	*l*	*s*	*z*	**5**	**9**
35-39	*f*	*m*	*t*	*aa*	**6**	**10**
40-44	*g*	*n*	*u*	*bb*	**7**	**11**
45-49	*h*	*o*	*v*	*cc*	**8**	**12**
50-54		*p*	*w*	*dd*		**13**
55-59			*x*	*ee*		**14**
60-64				*ff*		**15**
						16

† All ASMR (a to ff) are the calculated as the number of deaths/mid-period population, multiplied by 5 for each 5 year period

Period life tables

The ASMRs could be calculated for each period of follow-up (period rate). For 1975-1979 the 10-14 age group would equal a , 15-19 age group = b etc. To produce a single period life table over the entire period of follow-up, 1975-1994, the ASMRs would be calculated as the sum of the ASMRs of the women who at some period of follow-up were alive and in each age group. Any deaths or censored cases in one age group must be subtracted from the population that progresses into the next age group. The ASMR for the 10-14 age group over the 1975-1994 period would be a = (1), the ASMR of 15-19 age group would be b + i = (2), the ASMR of the 20-24 age group would be c + j + q = (3) etc.

Cohort life tables

For some analyses it is useful to look specifically at the ASMRs of a birth cohort over time independent of other birth cohorts. Birth cohort ASMRs can be calculated for the entire period of follow-up. For the cohort of women born in 1961-1965 i.e. those who were 10-14 in 1975, the ASMR over the whole period would be the sum of a + i + q + y = (9); for those born in 1956-1960 i.e. those who were 15-19 in 1975 the ASMR over the whole period would be the sum of b + j + r + z = (10); etc. To avoid confusion between the terms cohort life tables and cohort, referring to the sample of women included in the study, women are referred to as the sample where appropriate.

3.13 Calculation of other functions of life tables

In this section other life table functions based on the period mortality rates summed over the entire period of follow-up are described. For each age group, the increment (enrolment) and decrement (death, loss to follow-up, or end of follow-up) events are calculated for each year of follow-up. The following life tables were constructed on the basis of this schedule. Table 3.4 presents the abridged period life table whose calculations are explained below. The last period has been adjusted to the end of 1994.

Table 3.4

A period life table combining the mortality through each age-group between 1975-1994

age	N_x	C_x	D_x	pop at risk(E_x)	${}_nq_x$	${}_np_x$	l_x	L_x	95% CI† on ${}_nq_x$
10-14	93	2	1	92	0.010869	0.989130	1000	4972.	0.00028, 0.061
15-19	584	31	7	568.5	0.012313	0.987686	989.	4915.	0.0050, 0.025
20-24	935	58	4	906	0.004415	0.995584	976.	4873.	0.0012, 0.011
25-29	1223	65	18	1190.5	0.015119	0.984880	972.	4826.	0.009, 0.024
30-34	1497	55	24	1469.5	0.016332	0.983667	957.	4750.	0.01, 0.024
35-39	1326	46	16	1303	0.012279	0.987720	942.	4682.	0.007, 0.02
40-44	1167	20	19	1157	0.016421	0.983578	930.	4615.	0.009, 0.026
45-49	914	28	20	900	0.022222	0.977777	915.	4526.	0.014, 0.034
50-54	497	12	19	491	0.038696	0.961303	895.	4388.	0.023, 0.06
55-59	239	6	10	236	0.042372	0.957627	860.	4211.	0.02, 0.078
60-64	64	4	5	62	0.080645	0.919354	823.	3953.	0.026, 0.19
							757.		

† The 95% confidence intervals for ${}_nq_x$ are calculated from statistical tables for the Poisson distribution (λ), as an approximation to the binomial distribution thus avoiding negative CI (Scientific Tables,1962).

The calculation and interpretation of the functions presented in Table 3.4 are:

N_x = population of each 5 year age group

93 women were aged 10-14 at some time during the follow-up. The 15-19 age groups, includes women aged 10-14 years who survived the first 5 year of follow-up and became 15-19 years old (i.e. did not die or were lost to follow-up during the first 5 years) added together with the number of women who were 15-19 years old in the first 5 year follow-up period. Consequently the age-groups represent all women who at some time during the follow-up were of the specific ages.

D_x = number of women of each age group who died

The follow-up was extended to the end of 1994 by predicting mortality in the last 18 months, to allow the last period to be comparable with the previous 5 year periods. Although the period of follow-up was described by the DSS as valid until mid-1993, no deaths or out-migration was recorded in 1993. Consequently, the mortality expected in 1993 and 1994 was based on the probability of dying (${}_nq_x$) observed in 1990, 1991 and 1992. This resulted in a total of 17 deaths being added to age groups in the final period.

C_x = number of women of each age group who were lost to follow-up (out-migrated)

C_x is the censored cases. These are all women who are lost to follow-up whilst they were in each age group. In this thesis, all women censored were outmigrants from the DSS area for whom date of outmigration was known.

E_x = effective population at risk, the population at risk in each period adjusted for censored cases

It is important to adjust N_x for cases whose outcome is not known. They should not be completely removed them from the data because they do contribute information up to the time they are lost to follow-up. In general, where the study has a long follow-up period censored cases are usually considered to have contributed a half-person interval of risk to the interval during which they were lost to follow-up. An examination of the monthly pattern of out-migration by this cohort supports the validity of this approach with this data set since there is no monthly bias. E_x is calculated by the following equation:

$E_x = N_x - (C_x / 2)$

q_x = the probability of dying between exact ages x and x+1

This is calculated from the equation:

$$q_x = \frac{\text{deaths during the period of persons age x at the start of the 5 year follow-up } (D_x)}{\text{effective population aged x at the start of the 5 year period } (E_x)}$$

A q_x of 1.0 would indicate that all women aged x died before reaching the next age group.

p_x = the probability of surviving between exact ages x and x + n.

p_x is the complement of q_x and is calculated by the equation: ${}_np_x = 1 - {}_nq_x$

A ${}_np_x$ of 1.00 would indicate that all women aged x survived into the next age group

l_x = the number of persons alive at exact age x

This function is different to others described above in that it considers the survival to each age group of an artificial cohort known as the radix. At each age the value of l_x would be the number of a synthetic cohort who would survive to an age if they experienced the same mortality as the study cohort. The radix is set at 1000 in this life table. The age-

group of 10-14 has a l_x of 1000 since all women in the cohort survived to that age. The number of women who reach the age 15-19 would be the number of radix 'women' aged 10-14 who survived given the probability of survival p_x. The calculation of other l_x values for older age groups uses the formula:

$$l_{x+} = l_{x-n} * {}_nP_{x-n}$$

Therefore in Table 3.4, for the age group 15-19 the calculation would be:

1000 * 0.989130 = 989.13

Therefore the value of l_x is only meaningful when related to the radix.

L_x = the number of person-years lived between exact ages x and x + n.

Each woman who survives through a 5 year interval contributes 5 person years. However, those who die during that interval do not contribute the full person years. Assumptions can be made regarding the contribution of women who die in a similar way to the censored cases. In this cohort one could calculate the precise contribution since the date of death is known, however this complexity is not required because each interval is of 5 years and therefore, any seasonal pattern to the deaths is shared over 5 years. It is usual to consider women who died to have contributed half the person years of the interval. This weighting is called a_x and is 0.5 in this table. Because these are 5 year intervals the life years lived in a singe year need to be multiplied by 5. The formula for L_x in each age group interval is:

$nLx = n(l_{x+n} + {}_na_x * {}_nd_x)$

or : $[n (l_x + l_{x+n})] /2$

where ${}_na_x = 0.5$ and $n = 5$

In Table 3.4 the calculation of the number of person-years lived from age 15 to age 20 is:

${}_5L_{15} = [5 (l_{15} + l_{20})] /2$ $\qquad$ $4912 = [5 (989 + 976)] /2$

Note that the L_x column was calculated on a spreadsheet and therefore was calculated using decimal places removed from the table for ease of display.

The last open-ended interval requires special consideration because it is not known how many more years the 60-64 year age group will live. In very old women, (80+) the assumption is that they would all die. However, this is unlikely to be true in a cohort where from published data on females in this area, the average life-expectancy of women

aged 60-64 was 15.5 years in 1992. In Table 3.4, to calculate the ${}_5L_{65}$ of this cohort the l_{65} which would result if the ${}_5p_{60}$ is applied to the l_{60}.

SECTION 3.2 Results of period and cohort life tables calculated for the thesis sample.

3.21 Results and interpretation of the period life tables

This section describes the period mortality of the cohort as shown previously in Table 3.4. As would be expected of a sample of women selected from a population where there is no evidence of major adult epidemics, the probability of dying between one age group and the next, ${}_nq_x$, generally increases with age, with some slight exceptions in age groups 20-24 and 35-39. Figure 3.2 presents the ${}_nq_x$ values by age group, with their upper and lower 95% confidence limits. The probability of dying before reaching the next age group increases more markedly as women enter the older age groups. This is most striking between the 55-59 and 60-64 age groups. The probability of dying between 60-64 is 0.081, a two fold increase in the probability of dying compared to the probability of dying between 55-59.

The lx column of the life table provides a visual representation of how the ASMRs experienced by the study cohort would theoretically affect a cohort of 1000 women aged 10-14 over the same time period. In Figure 3.3 the survival of the synthetic radix cohort of 1000 women is shown for each age group.

The curve indicates that survival decreases with increasing age. The decrease in survival is largest between subsequent older age groups. The consistency of the curve suggests that the cohort was not subject to any unexpected age-specific mortality. It is expected that there is a higher probability of women aged 10-14 reaching age 40-45 than reaching the older ages of 60-64.

Figure 3.2

Period probabilities of dying (nqx): 1975-1994

+ upper estimate for 60-64 = 0.18

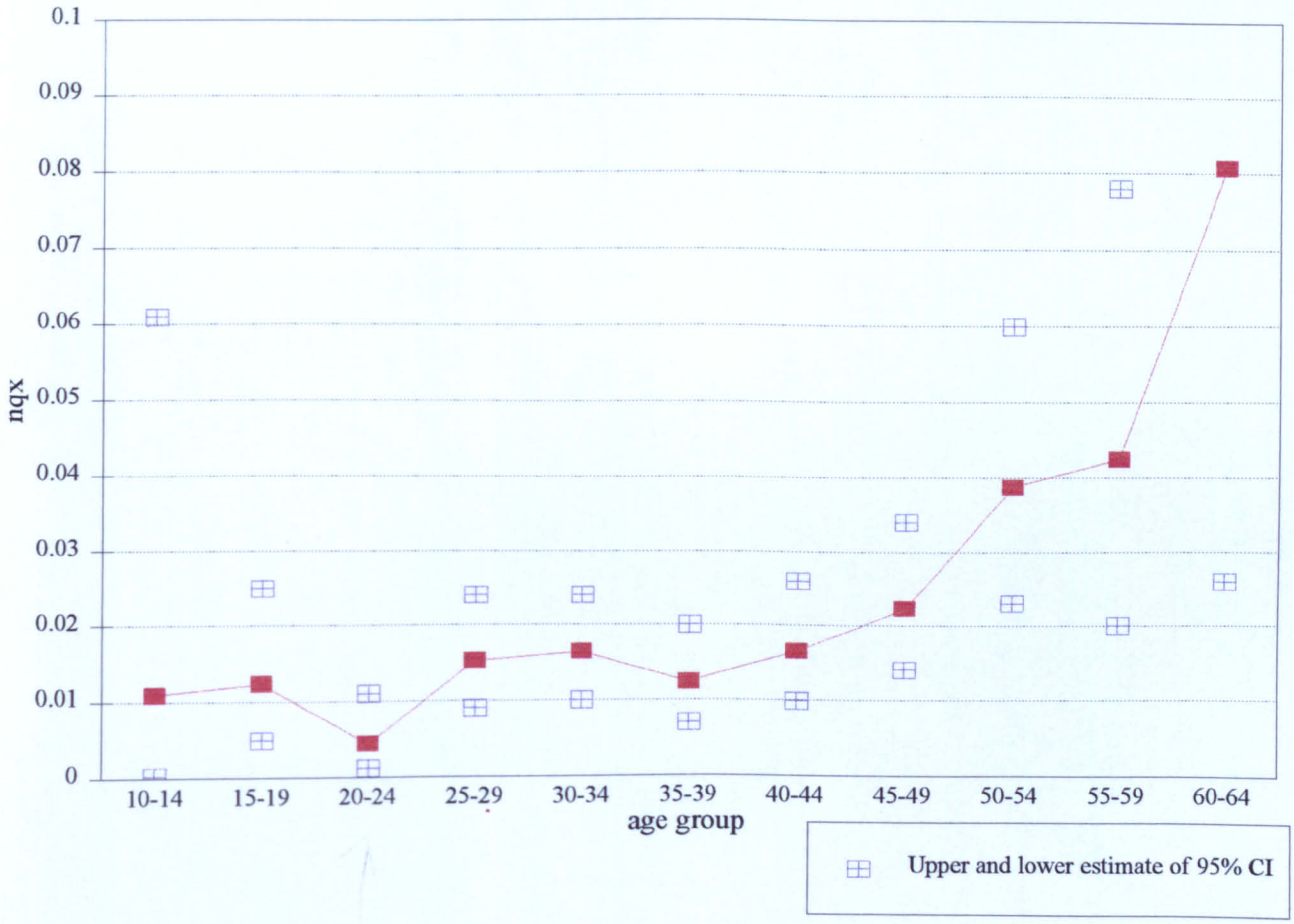
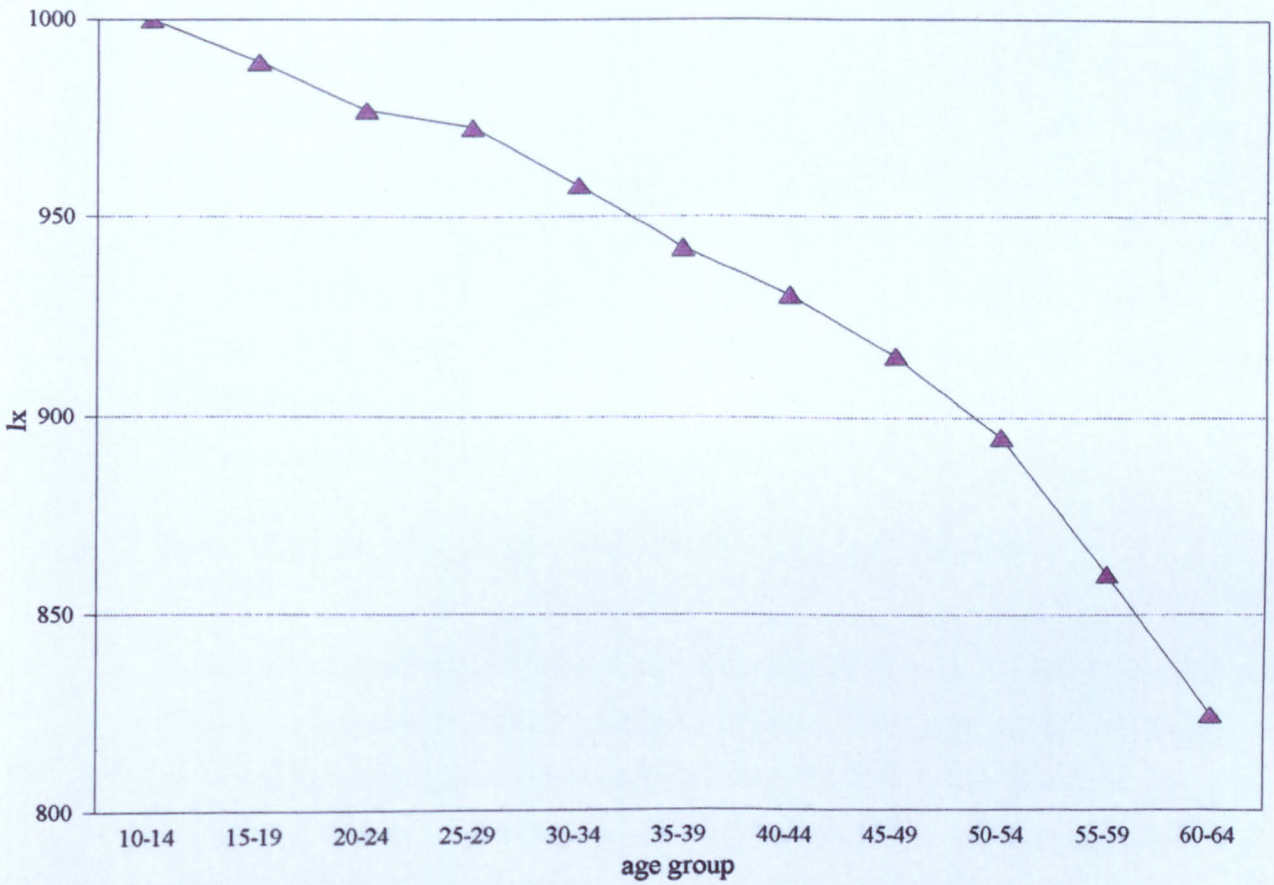


Figure 3.3 Radix survival (l_x) for 1000 women: 1975-1994



The lx column allows us to calculate the probability of any woman in the cohort of a given age reaching any other age. This is useful in comparing the mortality of this cohort with population based data from either Bangladesh or any other country. For example:

The probability of a woman aged 10-14 years old surviving to age 60-64 is calculated as:

$$_{50}p_{10} = l_{60} / l_{10} \qquad \text{or:} \quad 823 / 1000 = 0.823$$

However, the probability that a woman aged 10-14 years will survive to age 40-45 would be:

$$_{30}p_{10} = l_{30} / l_{10} \qquad \text{or:} \quad 930 / 1000 = 0.930$$

A feature of these calculations is that there is often a lower probability of surviving into old age if the woman is young than if she has already reached an older age. For example, the probability that a cohort woman aged 10-14 survives until she is 65-69 ($_{55}p_{10}$) is 0.757. However, if a woman is 60-64 she has a probability of surviving to age 65-69 ($_{5}p_{60}$) of 0.919. This is a typical feature of many populations. Whilst the mortality risk during each 5 year period of the adult years is relatively low compared to the mortality risks of the very young and very old, the cumulative risk of dying as an adult (between ages 15 and 60) is large.

3.22 Results and interpretation of the cohort life tables

As discussed earlier, the mortality of the thesis sample can also be described using cohort life tables, where birth cohorts are treated separately. Instead of considering the mortality of women by their age at death, the data can be disaggregated and the age specific mortality of women dependent on their birth years. This method specifically allows identification of any important secular changes in mortality determinants that may have occurred. For example, it is hypothesised that in a developing country such as Bangladesh the mortality experience of women born in earlier years will be heavier at all ages, than women born more recently, given that the younger women may have had exposure to 'improved' determinants of mortality. There may be greater availability of health care services and essential drugs or they may be subject to a more relaxed "*purdah*".[2]

[2] Defined by Cain et al (1979) *as "a system of secluding women and enforcing high standards of female modesty,"* operating within families and in society generally.

A cohort life table can be constructed for each birth cohort group, Table 3.5 presents a summarised life table, using an adjusted follow-up period to the end of 1994, Figure 3.4 shows a graph of the ${}_nq_x$ values for each 5 year period of follow-up by birth cohort.

Table 3.5

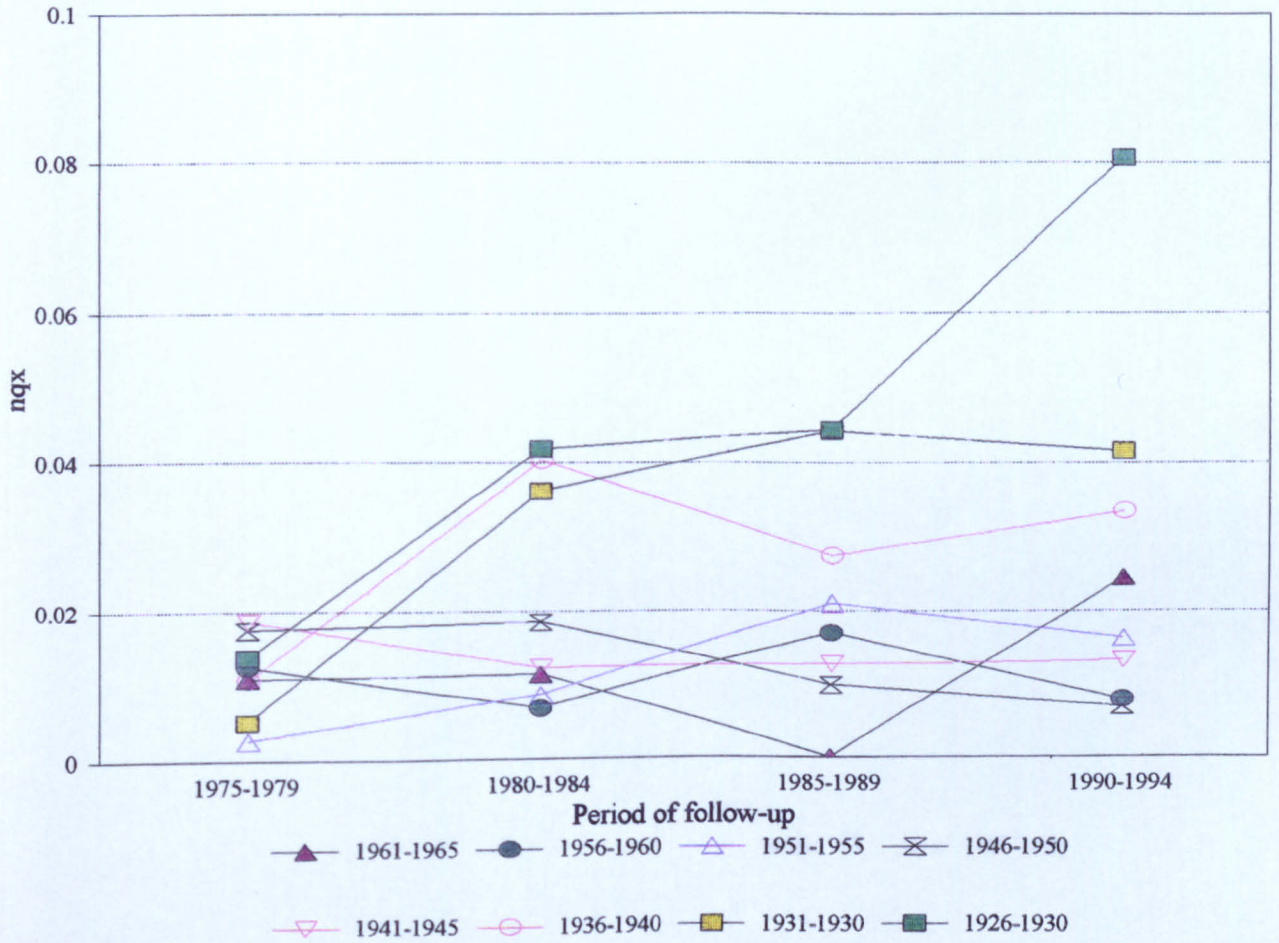
${}_nq_x$ probabilities of dying for each birth cohort, by 5 year follow-up period

	Birth cohorts (${}_nq_x$)							
Follow-up period	1961-1965	1956-1960	1951-1955	1946-1950	1941-1945	1936-1940	1931-1935	1926-1930
1975-1979	0.010869	0.0125	0.002663	0.017467	0.018669	0.010714	0.005037	0.013698
1980-1984	0.011299	0.006734	0.008498	0.018489	0.012224	0.040366	0.036269	0.041958
1985-1989	0	0.016969	0.021021	0.009836	0.012690	0.027504	0.044321	0.044444
1990-1994	0.024539	0.007822	0.016181	0.006849	0.013175	0.033472	0.041543	0.080645

It is more difficult to interpret cohort life tables than period life tables, particularly where there are several birth cohorts and small numbers. The concept here is that each ${}_nq_x$ line represents the level of mortality of a 5 year birth cohort. Each point on the x axis is the 5 year period of follow-up in which the mortality occurred, and each subsequent 5 year period means that surviving women are 5 years older.

Figure 3.4 shows some slight suggestion that the older birth cohorts have a higher probability of dying than the younger birth cohorts at any period of the follow-up. However the patterns are erratic and not convincing. In the last period the mortality rates are sequentially higher over the oldest three birth cohorts but the pattern is not sequential for the younger birth cohorts. The largest changes over the four periods in mortality probability are in the oldest birth cohort (1926-1930) which also shows a markedly higher mortality than other birth cohorts in the last period. The youngest birth cohort, 1961-1965, has a markedly erratic ${}_nq_x$ pattern. Possibly this is a result of the small number of women, 93, in this birth cohort. The confused pattern of the ${}_nq_x$ values in the first period of follow-up (1975-1979) is probably due to artefacts resulting from the staggered point of entry into the cohort, leading to an underestimate in the mortality over the first period.

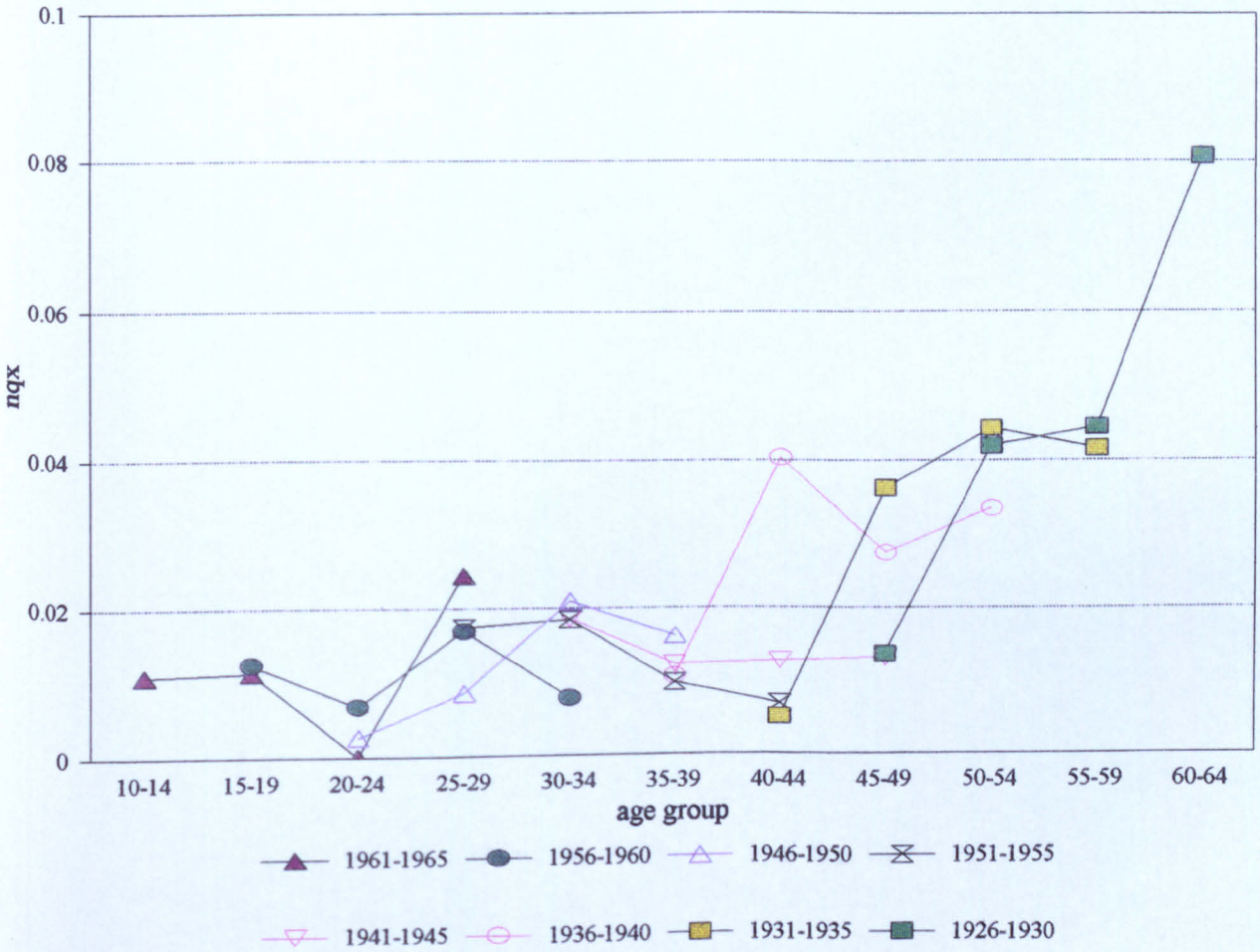
Figure 3.4 Probability of dying ($_nq_x$) for birth cohorts: 1975-1994



To examine the mortality of different birth cohorts at a given age the presentation of the life table data needs to be altered. In Figure 3.5, the ${}_nq_x$ values for each birth cohort are presented by age, rather than, as in Figure 3.4, the period of follow-up in which the mortality rates are calculated. This allows the probability of a woman dying at age x, given that she was born within a specified 5 year period to be estimated.

Again there is no evidence of a trend towards lower mortality in the younger birth cohorts. The pattern is erratic. For oldest birth cohort there is an increase in mortality with increasing age. This is not clear for younger birth cohorts. Comparing the birth cohorts by period of follow-up shows that unexpectedly the oldest birth cohort of 1926-1930 has the lowest probability of dying. This is possibly another artefact due to under-estimation of mortality in the first period of follow-up due to the staggered enrolment.

Figure 3.5 Probability of dying ($_nq_x$): 1930-1965 birth cohorts



SECTION 3.3 Comparing the mortality of the cohort with population based mortality data from the DSS.

It is rare in developing countries to have a long sequence of vital registration data. However the Matlab DSS provides population based data from the same area of Bangladesh with which to compare the age-specific mortality of the cohort over the period of follow-up. All the comparative data used in this section are those of females from the comparison area, unless noted. Life tables from the DSS are available annually from 1979 to 1994.

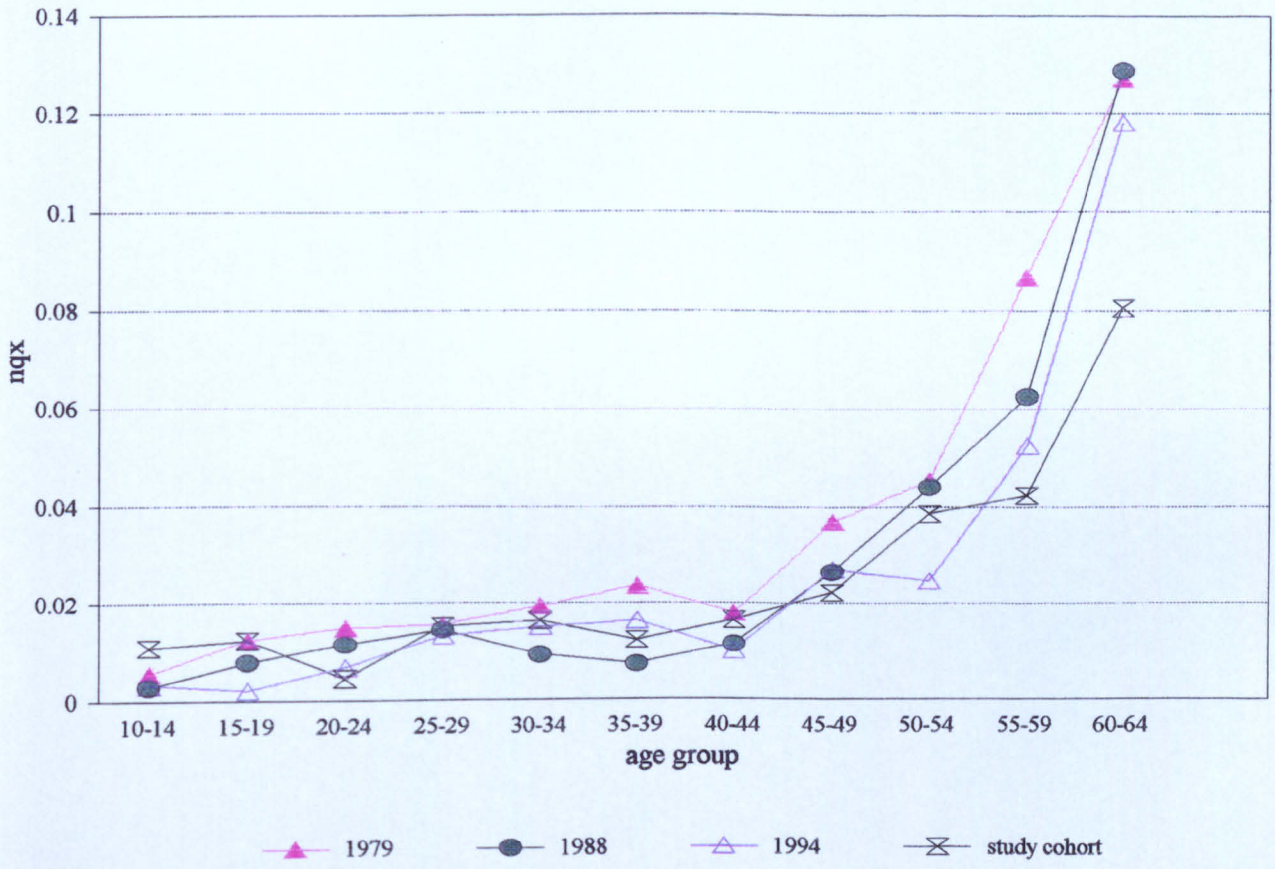
The simplest method of comparing mortality patterns between two populations is to examine differences in the survival probabilities by age group. Figure 3.6 presents the period ${}_nq_x$ values for the study cohort, compared with those extracted for the same age women from the DSS life tables of 1979, 1988, 1994.

Generally, the ${}_nq_x$ values from the comparison area in 1979 are slightly higher than those in 1979 and 1988, perhaps tentatively suggesting that the levels of female adult mortality may have declined for all age groups in the DSS area. However the levels are not markedly different. In many age groups the ${}_nq_x$ values overlap. Age group 55-59 show the largest differentials in mortality between the years.

The study cohort mortality lies at a level in the middle of the various years of DSS data. This is expected because in these period calculations mortality is the product of the years 1975-1995 rather than any single year. Therefore it reflects the accumulated exposure to changes in mortality risks.

The oldest age groups, 55-59 and 60-64, showed the greatest departure from the levels of the DSS. The low mortality in the last period for the 60-64 age group may be a function of the method chosen to model the expected mortality in the years 1993 and 1994, which may have underestimated mortality more significantly in the older age group. Only 2 deaths were added to the oldest age group, based on the ${}_nq_x$ in the previous 3 years.

Figure 3.6 Women's period ($_nq_x$): 1975-1994 compared with DSS ($_nq_x$): 1979,1988 and 1994



Possible explanations for lower mortality in the higher age groups, could include:

1) Selection bias. Women aged 60-65 in the last period would have been approximately 40-45 years at enrolment and would have had a birth in the last 5 years. There may be selection bias for women who have children at older ages which results in lower mortality risk.
2) Non-random misclassification of ages of women on entry into the DNFS.
3) Sampling variability due to small numbers in the oldest age group.
4) Period effects, for example, if older women in the cohort have benefited proportionately more from improvements in mortality determinants.

It is possible to disaggregate the data and examine whether any birth cohorts differ with that found in the DSS for the same aged women during the same period. i.e. to compare the thesis women's life table probabilities of dying shown in Table 3.5, with age specific probabilities from DSS annual period life tables. The DSS age specific probabilities are shown in Table 3.6. The DSS data selected are for females in the comparison area. In each 5 year follow-up period, a single year DSS report was selected from the middle of the period, with the exception of period 1975-1979 where no DSS life tables are available before 1979.

Table 3.6

Period ${}_nq_x$ rates by age group from the published DSS reports, comparison area

Age group	${}_nq_x$ 1979	${}_nq_x$ 1983	${}_nq_x$ 1988	${}_nq_x$ 1992
10-14	0.0055	0.0065	0.0027	0.0042
15-19	0.0124	0.0124	0.0077	0.0052
20-24	0.0149	0.0139	0.0114	0.0056
25-29	0.0154	0.0109	0.0143	0.0120
30-34	0.0193	0.0114	0.0092	0.0073
35-39	0.0237	0.0188	0.0072	0.0125
40-44	0.0179	0.0188	0.0114	0.0204
45-49	0.0369	0.0262	0.0265	0.0140
50-54	0.0455	0.0412	0.0440	0.0558
55-59	0.0868	0.1453	0.0625	0.0697
60-64	0.1269	0.1590	0.1287	0.1069

Comparing the mortality experience of women from the thesis cohort with those of other women in the comparison area, who were of a similar age during the same years,

effectively allows mortality to be compared by birth cohort. The comparative data is shown in Table 3.7 and was derived from Tables 3.5 and 3.6. For the period 1975-1979, DSS ${}_nq_x$ rates from (1979) are compared, for 1980-84 (DSS - 1983), 1985-1989 (DSS - 1988), 1990-1994 (DSS - 1992). The DSS ${}_nq_x$ rates selected from Table 3.6 are on the diagonal since in each 5 year period a cohort ages by 5 years.

Figure 3.7 presents the results of subtracting the probabilities of dying, ${}_nq_x$, for each study birth cohort from the ${}_nq_x$ of the same aged women taken from the DSS period tables of each follow-up period. The calculation for each age group:
DSS ${}_nq_x$ - Cohort ${}_nq_x$ (calculation repeated for each period of follow-up)
A negative difference would mean that the probability of dying in that age group is higher in the cohort than was experienced by women in the DSS comparison area during similar years.

The graph shows that the oldest birth cohorts experienced a greater difference in mortality from the DSS than any younger birth cohorts. They are markedly lower than the DSS in the periods 1975-1979, 1985-1989 and 1990-1994. Age group 30-34 showed the closest comparison to the DSS mortality. There is no clear pattern of the cohort mortality being consistently lighter or heavier than the DSS, except in the first period, where as has been discussed earlier, the staggered period of follow-up probably underestimates mortality in the cohort. From Figure 3.6, most birth cohorts have a lower mortality than that of the DSS in 1979.

An estimate of the difference between the cohort and DSS population mortality, which could be accounted for by sampling variation, can be estimated by calculating the standard errors of each probability. A difference of -0.003, as found between the ${}_nq_x$ of the 10-14 study cohort age group and the 10-14 DSS age group during the first period of follow-up, would mean that in an artificial cohort of 1000 women, if the probability of dying was the same as that of the study cohort, 992 women would survive the 5 years, or 995 would survive if the probability of dying was that of the DSS in 1979. This is a difference of only 3 deaths. The large differences shown by the oldest cohort age group would result in 893 women surviving into the next period if the study cohort mortality were applied, or 769 women surviving with the 1992 DSS mortality. This is a larger difference of 124 deaths.

Figure 3.7 Differences between cohort and DSS ($_nq_x$): 1975-1994

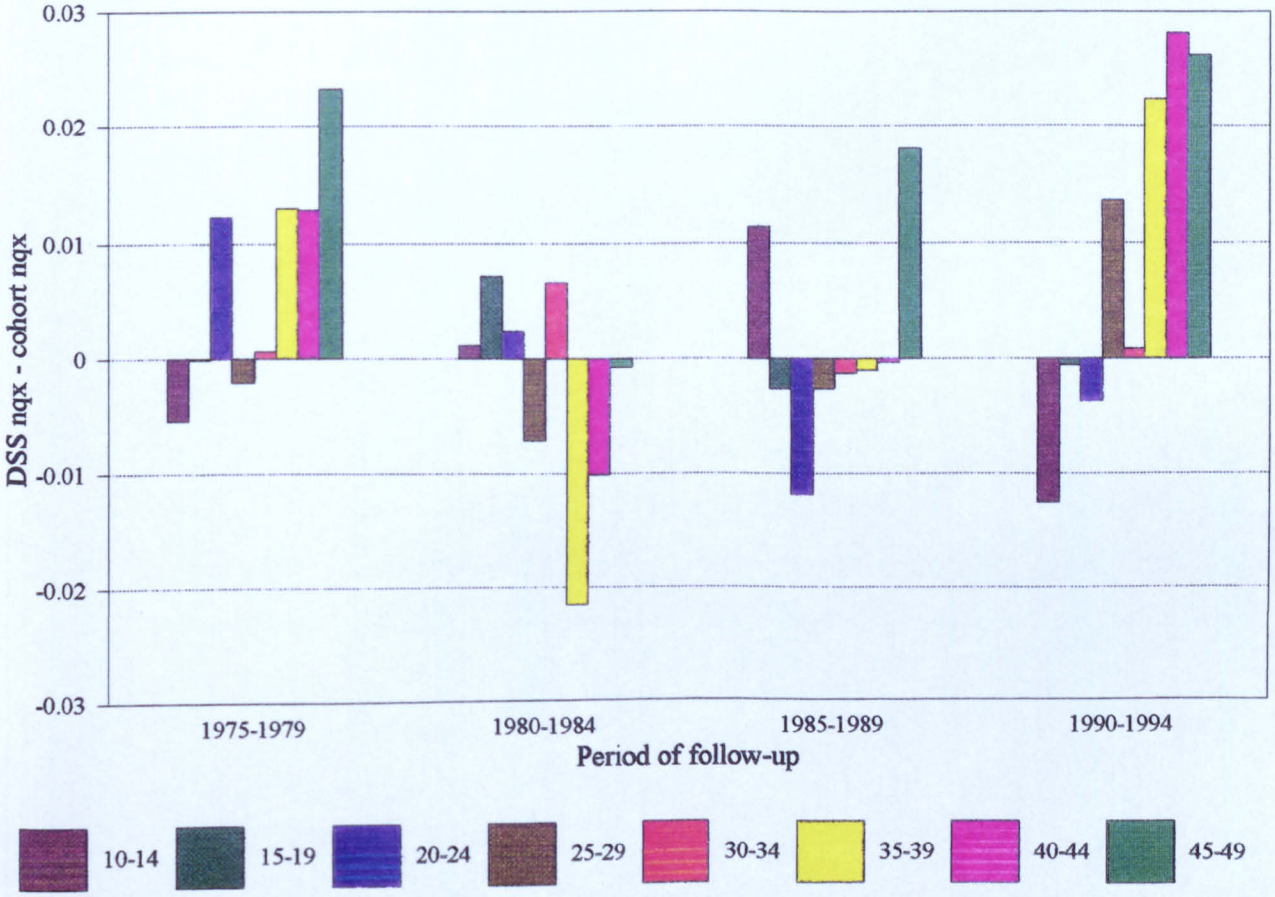


Table 3.7

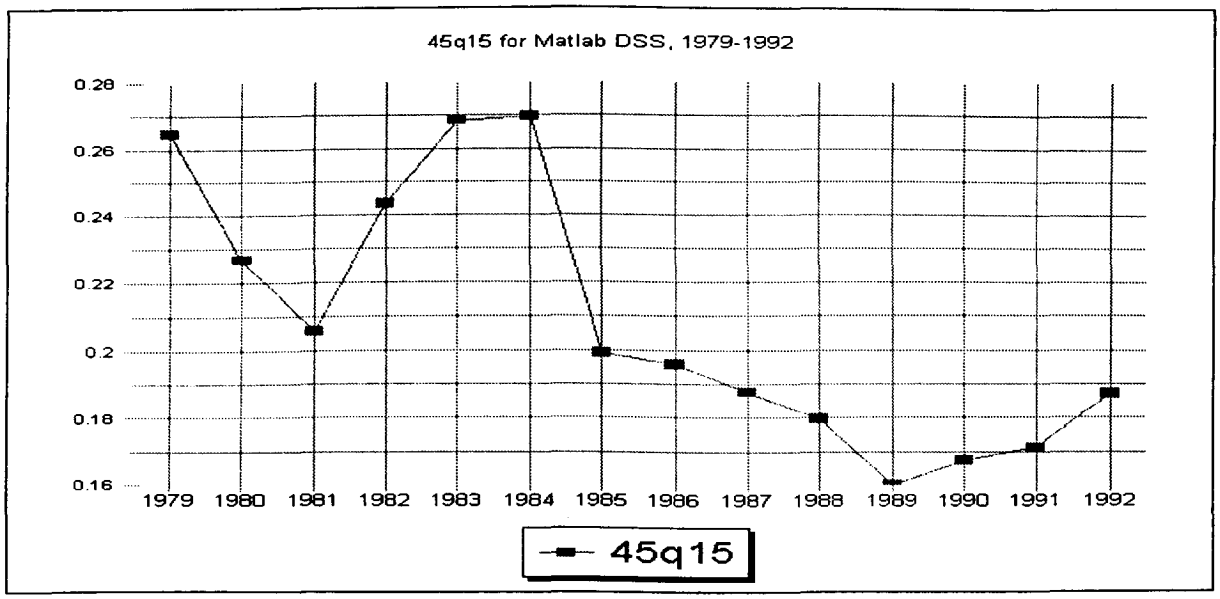
Cohort $_nq_x$ rates compared between the study cohort and equivalent years of DSS population based data

	Birth cohort															
	1961-1965		1956-1960		1956-1960		1951-1955		1946-1950		1941-1945		1931-1935		1926-1930	
Age	Cohort nqx	DSS nqx	Cohort nqx	DSS nqx	Cohort nqx	DSS nqx	Cohort nqx	DSS nqx	Cohort nqx	DSS nqx	Cohort nqx	DSS nqx	Cohort nqx	DSS nqx	Cohort nqx	DSS nqx
10-14	0.0108	**0.0055**														
15-19	0.0112	**0.0124**	0.0125	**0.0124**												
20-24	0	**0.0114**	0.0067	**0.0139**	0.0026	**0.0149**										
25-29	0.0245	**0.012**	0.0169	**0.0143**	0.0084	**0.0109**	0.0174	**0.0154**								
30-34			0.0078	**0.0073**	0.0210	**0.0092**	0.0184	**0.0114**	0.0186	**0.0193**						
35-39					0.0161	**0.0125**	0.0098	**0.0072**	0.0122	**0.0188**	0.0107	**0.0237**				
40-44							0.0068	**0.0204**	0.0126	**0.0114**	0.0403	**0.0188**	0.0050	**0.0179**		
45-49									0.0131	**0.014**	0.0275	**0.0265**	0.0362	**0.0262**	0.0136	**0.0369**
50-54											0.0334	**0.0558**	0.0443	**0.044**	0.0419	**0.0412**
55-59													0.0415	**0.0697**	0.0444	**0.0625**
60-64															0.0806	**0.1069**

To observe whether there is evidence of marked changes in mortality determinants in the years of the follow-up period, the DSS data can be summarised by individual years. Figure 3.8 presents the $_{45}q_{15}$ annual values from the DSS published reports. This is the probability of women dying between the ages 15 and age 60.

Figure 3.8.

Probability of dying between 15 and 60 years of age ($_{45}q_{15}$), for females in the DSS comparison area, by year (1979-1992)



There appears to be a general downward trend in adult mortality with the exception of 1982, 1983 and 1984. No published discussion of causes of higher mortality in these years was found, reflecting the paucity of adult mortality studies in Bangladesh. In 1990, 1991 and 1992 there was also smaller increase in adult mortality. Bennish and Ronsmans (1992) discuss the effect on national health of the economic and food disruption of the Gulf war, the cyclone and tidal wave. Both these occurred in 1991 but it is not possible to relate these events to mortality in the cohort.

In the following section 3.4, the cause of death data for the cohort is described and compared with similar data from the DSS in Matlab.

SECTION 3.4 Causes of death within the thesis cohort

One of the hypotheses proposed at the start of this study was that the anthropometry of adult women is associated with specific causes of death. The small number of deaths, 131 in the cohort over the follow-up period, is insufficient to allow meaningful sub-analyses of individual causes of deaths. In this section the causes of death are described principally to examine the representiveness of the cohort of the causes of death in the Matlab comparison area over the follow-up period.

3.41 Profile of causes of death within the cohort

Cause of death information for women in the study was available from 1975 to mid-1993, from two principle sources, 1) ICDDR,B DSS and, 2) a validation study of adult female causes of deaths in the Matlab area. The sources of data and the methodology used to classify causes of death in this study have been described in Chapter 2. Table 3.8 presents the causes of death for the 126 women in the cohort who died.

The two largest single causes of deaths were liver, ulcer and hepatitis complications (13.5%), and respiratory tract infection, including TB (13.5%). Deaths that were impossible to specify was the third largest category (10.3%). This is expected because many women did not seek professional medical assistance prior to death, and their symptoms were generalised and/or poorly reported by their relatives.

Narrow categories of maternal death causes were available due to the focus of the reclassification study. The first eight causes of death can be grouped together as causes of direct obstetric mortality. 21 (16.7%) of the women died of direct obstetric causes of death, a ratio of maternal mortality of approximately 1 in 5 deaths. As described in chapter 2, the validation study of female deaths in Matlab made a separate classification of maternal death based on a definition of time of death rather than the direct cause of death. The definition coded women as maternal death if they died whilst pregnant or within 90 days of pregnancy outcome. If all maternal deaths from the DSS are combined with the reclassification deaths classified as maternal death on the time of death definition, the number of maternal deaths in the cohort increases to 33 (25%), a ratio of 1 in 4.

In populations where communicable diseases decline, the relative proportion of deaths from causes such as homicides, suicides and accidents increase (Feachem et al,1992; Murray et al,1992). In this cohort eight women (6.4%) died from these causes, though there should be some reservation in accepting the exact cause of death, since homicide and suicides are often misclassified.

Table 3.8

Frequency table of causes of death (n=126)

Cause of death	Code	Number of deaths	% of all deaths†
Spontaneous abortion	1	2	1.6
Induced abortion	2	5	4.0
Toxaemia, eclampsia	3	3	2.4
Obstructed labour	4	2	1.6
Haemorrhage	5	3	2.4
Infection puerperium	6	2	1.6
Post-partum tetanus	7	1	0.8
Complications post-partum	8	3	2.4
Diarrhoea, dysentery, cholera	9	11	8.7
Liver, ulcer, hepatitis	10	17	13.5
Respiratory tract infection, including T.B	11	17	13.5
Cardio-vascular disease, stroke	12	8	6.4
Suicide	13	3	2.4
Homicide	14	2	1.6
Accident, snake-bite, burns	15	3	2.4
Fever	16	10	7.9
Rheumatism	17	4	3.2
Tumours	18	3	2.4
Paralysis and malnutrition	19	3	2.4
Diabetes	20	1	0.8
Impossible to specify	21	13	10.3
Oedema and ascitis	22	7	5.6
Complications of health care providers	23	3	2.4

†May not equal 100% due to rounding Total=126

The mean age at death for the different causes shows an expected pattern. Mean ages were highest for those women dying due to fever (48.6 years); respiratory tract infections, including T.B (45.7 years); liver, ulcer, and hepatitis (41.9 years) and cardio-vascular (41.63 years). Those dying of direct obstetric causes had a mean age of 30.5 years (minimum 16 years, maximum 42 years). The youngest mean age at death was in women dying from injury, suicide and homicide (26.1 years), see discussion below. Cause specific mortality in all populations show age specific differences. The pattern of mortality in a population is not a simple function of the overall level of mortality or life expectancy (Graham,1991; Murray et al,1992; Gribble and Preston,1993). Non-communicable diseases, such as cardio-

vascular problems and cancers, have an increasing incidence with age. Maternal mortality is specific to the reproductive ages. Therefore the mean age is lower than for all other causes of death, except for accidental and intentional injuries.

3.42 Comparing the causes of death in the thesis cohort with Matlab DSS population-based data

Table 3.9 presents broad categories of causes of death in the thesis cohort and their approximate comparative data from the DSS in 1992. The DSS reports cause of death data for women in the surveillance system (both areas) by age. However the cause of death categories are slightly different than those used in this study.

Table 3.9

Broad comparison between causes of death in the thesis cohort and the 1992 DSS (both areas, women aged 10-64)

Broad classification of causes of death	**Thesis cohort (%)**	**1992 DSS (%)**
Respiratory disease and T.B.	13.5	11.2
Liver, ulcer, hepatitis	13.5	8.2†
Injuries, violence, suicide	6.4	18.0
Impossible to specify or unknown	10.3	15.0
Direct obstetric complications	16.7	5.0

† DSS includes only infectious and gastro-intestinal diseases

For women aged 10-64 in 1992 the leading causes of death were unknown causes (15%) and cardio-vascular (10.8%). Combining DSS deaths of respiratory disease and T.B (11.2%) is comparable with the 13.5% mortality in the cohort. Combining the DSS deaths of other infectious and gastro-intestinal (8.2%) is lower than the (13.49%) classified as liver, ulcer and hepatitis complications. This is probably due to the DSS summarising some deaths due to these causes in a category called 'not elsewhere classified'. The largest disparity between the thesis mortality and the DSS are for injuries, violence and suicide, and direct obstetric complications. These are discussed below however the small numbers of deaths in the cohort suggest that the differences in the percentages should be interpreted with caution.

Fauveau and Blanchet (1988), using the data from the reclassification study, examined the deaths from injuries and induced abortion among reproductive aged women, 15-44, in Matlab between 1976 and 1986. They estimated that 18% of women were victims of unintentional injuries or violence. In the cohort, 13 women (10.3%) died from these causes. The lower proportion of these causes of death in the cohort will be largely due to their marital status. Fauveau and Blanchet found that unmarried women suffered a higher proportion of deaths from injury and violence (36%) than married women (15%). In addition, the risk of injury and violence declined markedly with age more than 19 years, 31% of 15-19 year old women died from these causes compared with 10% of women aged 35-44. In this cohort, the majority of women were older than 19 years at the start of the study. The mean age of death for injury, suicide and homicide in this cohort was 26.1 years, lower than the mean age of death for any other causes.

16.7% of women in our study were classified as dying of a direct obstetric death, compared with (5%) in the 1992 DSS report. Four explanations may be suggested for the comparatively higher rate of maternal mortality in the study cohort:

1) Using the reclassification study causes of death for 56% of the cohort increases the numbers of maternal deaths from those coded by the DSS. The reclassification study which classified 43.8% of the 73 study deaths as maternal deaths compared with the DSS who only coded 15% of the 73 deaths to be maternal deaths. The higher proportion of deaths coded as maternal deaths in the reclassification study than in the DSS has been suggested to be due to the failure of the DSS to record information about pregnancy or post-partum status at the time of death; and in addition a different protocol for assigning maternal deaths (Fauveau et al (1988). Also it may be that the focus of the reclassification study on maternal deaths may have led to over-reporting of maternal deaths. Given the relatively small number of deaths in the cohort, over-reporting of maternal deaths may raise the proportion significantly.
2) The 1992 DSS statistics are based on all women in the surveillance system, including the MCH-FP area. However, all women in the cohort were living in the comparison area which does not receive extra MCH-FP services and consequently may have a higher risk of maternal mortality.
3) Deaths within the cohort were recorded between 1975 and mid-1993. Consequently the determinants of maternal mortality experienced by cohort may have been less

advantageous and therefore the proportion of deaths is higher than that of women observed in later years.

4) The data from the DSS is for women aged 10-64. Therefore it includes a proportion of women who would not be at risk of dying of direct obstetric complications due to their age. In the thesis cohort all women were of reproductive age for a proportion of the years of the follow-up.

Comparing the cohort death causes with the findings of the reclassification study, a lower proportion of maternal deaths based on the time of death definition of maternal death is observed. In papers published by Fauveau et al (1988) during the period 1976-1985, 1 in 3 deaths in Matlab would be considered to have been maternal deaths. The maternal death ratio is 1 in 5 among the cohort. The lower proportion of maternal deaths observed in the cohort could be due either to classification differences or the age profile of the cohort. The older birth cohorts followed were not at risk of dying of a maternal death during the last periods of follow-up. Women, aged 45-49 in the first 5 years of the study follow-up period, were not in the reproductive ages for the next 15 years of follow-up. Only 73 (55%) of the deaths were subject to investigation by the reclassification study., No time definition for maternal deaths is available for the remaining 53 deaths.

The majority of studies which have examined the relationship between anthropometry and all cause mortality have been conducted in developed countries (see Chapter 1). One constraint in comparing mortality studies between the cohort and other studies is the difference in both the level and causes of mortality. In Table 3.10 the percentages of adult mortality (males and females) for England and Wales are presented by causes of death and age group 15-74 years in 1994 (OPCS,1996).

Table 3.10

Causes of death in different age groups in England and Wales, 1994 (OPCS,1996)

	Age group (years)		
Cause	**15-44**	**45-64**	**65-74**
Total deaths (000s)	6.3	27.7	53.9
% of deaths attributable to:			
Circulatory system	13.6	25.8	40.9
Neoplasms	39.4	52.5	36.2
Respiratory disease	4.7	6.7	11.3
Injury and poisoning	20.9	3.2	1.3
Others	21.4	11.8	10.3

The largest single group of causes of death for ages 15-44 are circulatory diseases, 39.4%. The proportion of heart disease and cancer are markedly higher than found in the Bangladeshi cohort. Proportionate mortality due to heart disease and cancer increases in older ages, whilst mortality due to injury and poisoning declines significantly. The striking comparative difference with the thesis cohort is the small number of deaths due to infectious diseases and direct obstetric deaths, which in the table above would be included in the category 'other'.

Chapter summary

The results of the period and cohort life tables presented in section 3 suggest that there is an increasing risk of mortality for all age groups with duration of follow-up period. There is some tentative suggestion that older birth cohorts have tended to experience a higher probability of mortality at all ages than younger women. However the cohort life tables show very erratic mortality patterns by birth cohort, possibly a result of the small number of deaths in each birth cohort. Also period mortality rates from the DSS in 1979, 1983, 1988 and 1992 do not indicate any evidence for important changes in mortality determinants in the Matlab area, with the possible exception of mortality risk in older women age 50-64.

In Chapter 2 it is suggested that very young married women are relatively disadvantaged socially and economically. The mortality patterns for women aged 10-14 in the cohort were very erratic, being elevated and suppressed relative to older women However, it is not possible to distinguish whether there was a plausibly higher risk due to risk experiences, or a statistical artefact of small numbers.

From the comparative data presented in Section 3.3, the period and birth cohort mortality of the thesis sample generally corresponds to the population based data from the comparison area of Matlab. The cohort can be considered as representative of the mortality experience for similar aged women during the period 1975-1994; with the possible exception of the 60-64 age group where mortality is markedly lower than similarly aged women in the DSS.

The cause of deaths recorded for the cohort are comparable with that obtained for adult women from the DSS and the reclassification study. 126 deaths to women in the cohort is too small to meaningfully examine changing patterns of mortality over time. However knowledge of the principal causes of death in the cohort can inform the discussion of later survival analyses.

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Chapter 4 Anthropometric data - methods of data preparation and the cohort profile

Chapter introduction

This chapter is a summary of the anthropometric data of the cohort. It bridges the previous chapters which described the socio-economic, demographic and mortality experience of the cohort and the following chapter, which analyses the relationship between nutritional status and mortality. The repeated measurements of weight and arm circumference available from the DNFS require preparation before they can be used in the proportional hazards models described in Chapter 5. Proportional hazards models analyse the comparative risk of mortality associated with different levels of anthropometric indicators, adjusted for potential socio-economic and demographic confounding factors. For reasons discussed in Section 4.1, it seemed preferable to calculate a single, representative level of each indicator for each woman. This would be treated as her anthropometry measure at the start of follow-up.

A summary measure is a value that captures the general weight or arm circumference level of a woman during the 2½ years of repeated nutritional measurements and perhaps, more importantly, is appropriate in placing an individual women's nutritional status relative to others in the cohort. Because weight and arm-circumference fluctuate in response to a myriad of determinants, for example, food intake, energy expenditure, water retention or loss, the summary measure should represent a level from which the woman may often depart, but one that describes her 'typical' weight.

In calculating a summary measure, the probability that data may contain measurement errors must be considered. A protocol was created to identify and remove measurement errors which would reduce confidence in the representativeness of the summary measure. Inherent to this protocol is an attempt to distinguish biological plausible changes in an anthropometric measure over time from measurement errors. A single observation value may be plausible on its own, i.e. lie in a range similar to that of other women, but be implausible when compared to other values of the indicator recorded for a woman during the follow-up. Making this distinction is difficult in cases where women have few

anthropometric measures or where there is a long interval between measures. In addition, although height was only measured once for each woman, a protocol was also developed to identify possible measurement errors.

The first section of this chapter considers the important issues underlying the testing of the hypothesis, and the approaches to summarising the repeated measurements. This is illustrated with a hypothetical example. The second section examines the relative importance of these requirements within the cohort data, the variability of the weight and arm circumference repeated measurements, and presents the algorithm for detecting measurement errors. The third section presents the summary statistics for the cohort, which are compared with anthropometry profiles from other studies and surveys in Bangladesh.

SECTION 4.1 Methodological issues in using historical, longitudinal anthropometric data

As a requirement for entry into the thesis cohort each woman had to have at least one non-pregnant measure of either height, weight or arm circumference. Table 4.1 shows the number of women for whom each anthropometric indicator was available. Note that validation results presented in Tables 4.1 to 4.5 was conducted on a sample of 2,344 women.[3] All summary statistics refer to the cohort of 2,314 women.

Table 4.1

DNFS anthropometric data available for cohort women

Anthropometric data	**Number of women**
Height, weight and arm circumference	1,885
Height	2,203
Weight	1,999
Arm circumference	2,076

[3] 30 women were included in the validation but not in the cohort discussed in other chapters because they were not identified in the vital events DSS database (see Section 2.12).

There are potentially 27 repeated measures of weight and 28 for arm circumference during the period of 1975-1978. Height was only measured once on entry into the study.

4.11 Rationale for choosing the summary method for repeated measurements

The following questions were considered important in selecting a summary method for the repeated measurements:

1. Why is a summary measure for weight and arm circumference for each woman needed?
2. What does this summary measure represent?
3. What are the methods available for summarising repeated measurements? What is important in choosing a robust summary measure?
4. In calculating a summary measure from repeated measurements is it necessary and possible to distinguish between biologically plausible measurement changes and measurement errors?

1. Why summarise the repeated measurements?

In analysing the mortality risk associated with nutritional status indicators in a longitudinal cohort study, the analysis of choice would be a form of survival analysis such as the Cox's proportional hazards model which would allow multivariate analysis. In these models the effect of each nutritional status indicator is related to the probability of dying over the follow-up period. It is possible to examine the effect on survival of updated covariates, as discussed by Altman and De Stavola (1994). This approach is typically used in oncology studies, where a change in a covariate, such as drug dosage, needs to be considered with respect to survival and also can be clearly quantified. However nutritional measurements were not made over the whole period of follow-up in which survival was observed. There were no updated measurements in the last 14 years of the study follow-up period selected. In addition, the principal research question is whether the general level of women's nutritional status is related to their survival, rather than whether changes in nutritional status over time relates to changing probabilities of survival.

This is not to dismiss the potential role of nutritional changes as a mechanism through which mortality risk is determined. In several studies from developed countries weight loss in low BMI adults increased their risk of dying (Blair,1993; Hamm et al,1989). In this study however, the absence of information about changes in weight over the total follow-up period does not permit weight change to be meaningfully examined as a risk factor for mortality in this study. Consequently a single summary measure for each nutritional indicator covariate is appropriate, because the principle objective of this chapter is to differentiate women into levels of nutritional status.

2. What does this summary measure represent?

Both weight and arm circumference are functions of a wide range of determinants, including skeletal mass, calorific intake, energy expenditure and water retention. In repeatedly measuring women to an accuracy of 0.1kg or 0.1cm over successive months, at different times of the day, it is expected that the values would not be identical, but would have a range of different values. The variability in weight and arm circumference indicators among women in the DNFS has been reported by other authors, particularly in relation to seasonal influences on food availability. Huffman et al (1985) describe changes in the average non-pregnant weight of the cohort over the changing months. Weight peaks in the months of June and July, declining between July and October, with the lowest weights recorded in October and November. They observed an average range of increase and decrease of 0.5kg over the year.

A summary measure for each nutritional indicator needs to represent the general level of a woman's nutritional status, i.e. a 'typical' weight or arm circumference, from which value she may depart over time. The summary measure also needs to be discriminating in placing women relative to each other with respect to the nutritional indicator, since the hypothesis being tested is that women of low nutritional status have a higher risk of mortality than women of higher nutritional status. The summary measure should place women along the spectrum of nutritional status present in the cohort.

3. What methods are available for summarising repeated measurements? What is important in choosing a robust summary method?

The most common methods for summarising repeated quantitative measurements are to calculate either the mean or the median. Where data are symmetrical and unimodal the

mean and the median are on average equal. The median is a more useful descriptive measure if there are occasional extreme values in the data set or the data is not normally distributed. If the mean value was used, there is the potential for extreme high or low values (due to measurement error or perhaps a severe illness), to misrepresent the women's general level for a nutritional indicator. This is illustrated in Section 4.1.5.[2] The median is more robust as a description of the central tendency of a series of measurements and is therefore suggested as an appropriate summary measure for this study.

4. In calculating a summary measure from repeated measurements is it necessary and possible to distinguish between biologically plausible measurement changes and measurement errors?

The need to remove all potential errors in measurement is to a certain extent governed by both the choice of summary method and the patterns of weight change experienced by individuals. In using secondary data there is conflict between wanting to ensure maximum validity of data and being able to make a rational protocol for identifying measurement errors. Systematic errors made over all measurements, for example, poor calibration of weight scales or interviewer technique, cannot be detected in this secondary data analysis.

By using the median, there is a need to only remove measurements that are clearly implausible relative to other consecutive measures, and which markedly alter the summary measure. The more measures there are, the less likely extreme values are to alter the median. The central issue is whether 'biologically plausible' changes in anthropometric measurements over the period of one month or more are known. The seasonal influences on weight and arm circumference are observed in most women in the cohort, but they produce regular perturbations in the pattern of weight rather than extreme oscillations. This does not necessarily mean that large differences between consecutive measures are errors. For example, severe diarrhoeal episodes may cause rapid weight loss. Measurement errors may also be hidden because they lie in a range that seems plausible and therefore would be impossible to detect. The validation of an individual's anthropometric data is also constrained by the substantial number of missing measures and the exclusion of measurements obtained from women who were pregnant or in the post-partum period. These issues are illustrated in the following hypothetical example of a woman's changes in weight.

4.12 A hypothetical example of repeated weight measures illustrating features of summary statistics in the cohort

Figure 4.1 presents a fictitious series of 23 weight measurements for an individual taken over a period of 27 months. Four features of the data can be highlighted:

i) Most of the weight measurements follow a systematic trend, increasing or decreasing by less than 1kg from the previous month, with the increasing trend towards the months of June, July and August in 1976 and 1977.

ii) The measurements in **December** 1976 and July 1977 are noticeably different to their consecutive monthly measurements.

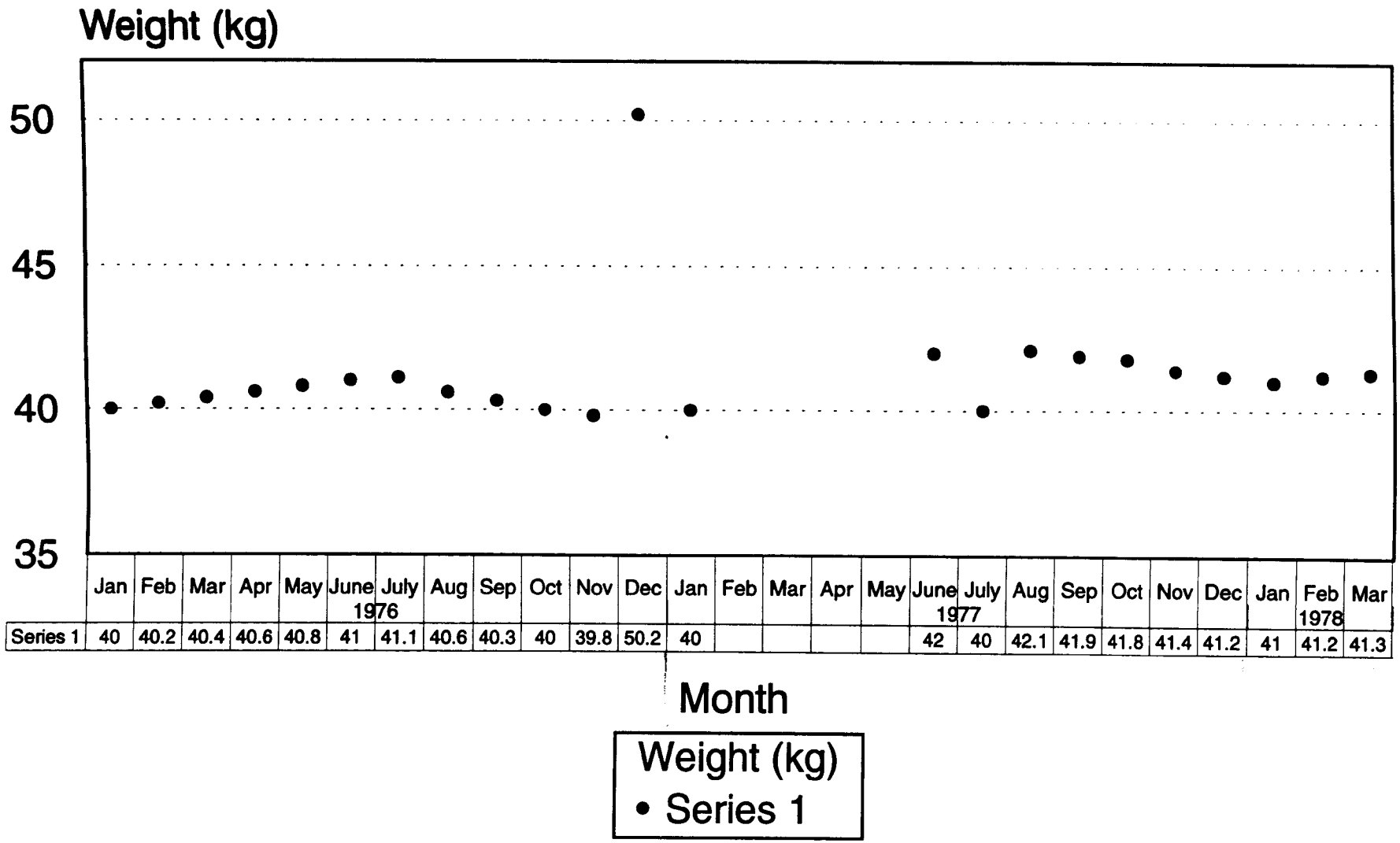
iii) Measurements are missing for four months in early 1977.

iv) Generally the level of weight measurements in 1977 is slightly higher than that of the corresponding months in 1976.

The mean weight of these measurements is 41.3kg the median measurement is 41kg. The mean being slightly elevated by the high value of 50.2kg. Biologically the gain and then loss of over 10 kg in two months seems implausible and suggests that the measure of December 1976 is a measurement error. If this value was excluded, the median remains the same at 41kg and the mean value is decreased to 40.9kg. This is closer to the median value. It suggests that if the median is used as a summary measure, single measurement errors, regardless of magnitude, will not affect the ability of the median to reflect the general level of the woman's nutritional indicator, unless there are very few measurements. In contrast, the decrease of 2kg from June to July 1977 is more plausible. This is possibly due to an episode of illness, where weight is regained over the following month.

The presence of missing data means that weight has been increased by 2kg from January to June 1977. Where consecutive measures were missing for reasons other than pregnancy, the possibility of modelling the missing values was explored. This approach is used in other studies, for example in child growth studies where the pattern of growth has been used. Several different methods were tested to predict weights in the missing 4 months but none resulted in a significantly different estimate for women's median measures.

Figure 4.1
Hypothetical example of repeated weight measurements



	Jan	Feb	Mar	Apr	May	June 1976	July	Aug	Sep	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	June 1977	July	Aug	Sep	Oct	Nov	Dec	Jan	Feb 1978	Mar
Series 1	40	40.2	40.4	40.6	40.8	41	41.1	40.6	40.3	40	39.8	50.2	40					42	40	42.1	41.9	41.8	41.4	41.2	41	41.2	41.3

SECTION 4.2 Preparing and validating the cohort anthropometric data

The previous sections have outlined some of the issues involved in summarising the repeated anthropometric measurements. In this section the importance of these issues are considered with respect to the anthropometric data available for the cohort data.

4.21 Missing rounds of weight and arm circumference measurements

The staggered entry into the study, the exclusion of weights during pregnancy and the post-partum period, as well as absences at study visits, result in many women having less than the potential maximum of 27 weight or arm circumference measurements. Table 4.2 presents the number of non-pregnancy anthropometric measures available for weight and arm circumference.

Table 4.2

Number of weight and arm circumference measurements recorded

Weight		**Arm circumference**	
No of measures	No of women (%) n=2,022	No of measures	No of women (%) n=2,076
1	133 (6.6)	1	128 (6.2)
2-5	478 (23.6)	2-5	502 (24.2)
6-10	427 (21.1)	6-10	434 (20.9)
11-27	984 (48.7)	11-28	1012 (48.7)

A high proportion of women had 6 or more measurements: 69.8% and 69.65% for weight and arm circumference respectively. This suggests that summarising the repeated measures by using the median, will be representative of the general nutritional experience of women over a significant period between 1975-1978. There is less confidence in the representativeness of a summary measurement for the women with only 2-5 measurements. This is especially relevant in summarising weight, given the seasonal variation in weight observed in the DNFS cohort (Huffman et al,1985). Women with 2-5 measurements were consequently selected for further investigation, as discussed in Section 5.24. The simplest approach (for women with only one measurement), is to

exclude values outside a plausible range, for example, a weight of 5kg. However, the range of values for women with single measurements was 32.5kg-50.0kg for weight, and 18.5cm-24.7cm for arm circumference, all levels of anthropometry which are biologically plausible. Other methods for validating these single measures, for example, predicting the weight from the height, were impractical because height itself only collected once. Therefore, for the 6.6% of women who had one weight measure and the 6.2% of women with one arm circumference measure, the data were used without correction.

4.22 Inter-individual variability of weight and arm circumference measurements

The first step used in understanding the patterns of weight and arm circumference was a visual examination of graphs where anthropometric measures were plotted for each individual. The majority of women experienced regular oscillations in weight over several months consistent with the seasonal patterns described in Section 4.12. The lack of scatter between consecutive measures suggests that there was consistency in the measuring techniques and interviewers used by the DNFS.

To quantify the degree of variability the range of weight measurement values, i.e. the difference between the minimum value and the maximum value was calculated for each woman. Table 4.3 shows the ranges of weight values of women.

Table 4.3

The difference between the maximum and minimum weight values.

Difference between maximum and minimum weight values by woman	**Number of women (%) n=2,022**
0	143 (7.1)
0.1-1kg	144 (7.1)
1.1-2kg	231 (11.4)
2.1-5kg	1062 (52.5)
5.1-10kg	412 (20.4)
10.1+kg (max=35kg)	30 (1.5)

78% of women had a difference between their minimum and their maximum values of weight of <=5kg. This combined with the result that 69.8% of women had 6 or more measures, further suggests that for the majority of women there is confidence about the appropriateness of using the median as a summary measure. There were a few women with very erratic patterns and the methodology used to identify them is described below.

Note that this exploratory approach was less rigorous for arm circumference measurements because in the data all women with an arm circumference measurement > 24.7cm were coded as 24.7cm. However, as with weight, for the majority of women the approach of summarising using the median value was used.

4.23 Creating a protocol to identify women with high anthropometric variability

Since most women experience changes in their arm circumference and weight over time, it is necessary, and to some extent only possible to identify a small proportion of women for whom there is doubt about the ability of a summary measure to be representative of their nutritional status. Only the most extreme erroneous values needed to be identified given the objectives of this study. The protocol used is described below. Note that identification does not automatically imply an erroneous measure, merely that the anthropometric data for an individual was screened more carefully.

In order to identify marked changes between one month and the next, the difference between each measure and it's previous and consecutive measure could be calculated for example, as:

weight round(wr) **x** - (wr **x** - **1**) ; wr **x** - (wr **x+1**) .....

However, the missing weight rounds present a significant problem in interpreting the results. Where a woman was absent or pregnant/post-partum, several months of follow-up data may be missing, during which time her weight may have plausibly altered by several kilograms.

In the absence of statistical convention in handling this type of data, the systematic protocol used to identify suspicious measurements focused on women with very few

measurements, and/or measures that differ markedly from the median value for each woman. On calculating the difference from the median of all measurements, 95% of measurements lay within ±4.8kg and 1.85cm of the median value of each women for weight and arm circumference respectively. The measurements in the upper 5% level are referred to as outlying values. Table 4.4 shows how many women have measurements which are identified by this 95% centile cut-off.

Table 4.4

The number of women with weight or arm circumference measurements that are greater than the 95% centile cut-off

Weight		**Arm circumference**	
	No of women n=2,022 (%)		No of women n=2,076 (%)
0 observation >4.8kg from the median	1,921 (95.0)	0 observation >1.85cm from the median	1,971 (94.9)
1 observation >4.8kg from the median	69 (3.4)	1 observation >1.85cm from the median	73 (3.5)
2+ observations >4.8kg from the median	32 (1.6)	2+ observations >1.85cm from the median	32 (1.6)

There are 101 women with one or more measure of weight ±4.8kg from their median, and 105 women with one or more measures of arm circumference ±1.85cm from their median. There are very small numbers of women with several measurements that differ from the median. As discussed above, a single observation that differs significantly from the median does not alter the median markedly providing that there are several repeated measurements for that individual. Therefore, the protocol selected women with a single outlying observation only if they had less than 6 repeated observations and all women with more than one outlying observation. Table 4.5 presents the number of women selected using this algorithm.

Table 4.5

Number of women selected by the weight/height algorithm†

	Weight	Arm circumference
More than one outlying value	32	31
One outlying value but less than 6 repeated measurements	4	6
Total	36	37

†Algorithm: more than one outlying value or one outlying value but less than 6 repeated measurements

The weight and/or arm circumference data of the women identified by the protocol were then examined further.

4.24 Guidelines for recoding potentially erroneous values of weight and arm circumference

There are no standard conventions on recoding erroneous values. The following procedure was used given the small numbers of women identified by the algorithm. The repeated measures of the nutritional indicator were graphed for all selected women. These graphs were examined to assess the plausibility of the value given its relative level compared with the nearest monthly observations and, if those were missing, to the overall pattern of indicator change for the woman. Appendix 4.1 presents the graphs for the women selected for potential erroneous weights and Appendix 4.2 presents the graphs for arm circumference. Most women selected had decreasing or increasing trends in the nutritional indicator and these were not recoded. In total 5 and 3 women had measurements set to missing for weight and arm circumference respectively. The values which were reset to missing are highlighted in the relevant Appendix. Note that the median values were altered slightly for women selected for weight observations, little change resulted for the median values of arm circumference.

4.25 Validation of height measurements

The single height measurements for the cohort were in the range of 1.087m-1.654m. The two lowest heights, 1.087m and 1.195m, were significantly lower than those of other women. Their respective median weights were 39.2kg and 30.0kg. The plausibility of the former seems doubtful and her height was recoded to missing. All other heights were accepted.

SECTION 4.3 Anthropometry summary statistics for the cohort

4.31 Description of cohort anthropometry, and comparison with other data presented on the DNFS cohort

Table 4.6 presents the summary statistics for each anthropometric indicator based on the methodology described in the previous sections. In the thesis cohort, the sample size varied by indicator presented. Also included in the table are the summary statistics from another sub-sample of women from the DNFS cohort. Huffman et al (1985) examined relationships between nutritional status and fertility in 2,161 non-pregnant DNFS women.

Table 4.6

Anthropometric summary statistics for the thesis cohort and the Huffman et al (1985) study, which also used a sub-sample of non-pregnant women from the DNFS

Anthropometric indicator	**No. of women in thesis cohort**	**Thesis cohort Mean (sd) [minimum, maximum]**	**Huffman et al (1985) (n=2,161) Mean (sd)**
Height (cm)	2202	147.9 (5.2) [119.5, 165.4]	149.9 (5.2)
Weight (kg)	1999	40.5 (4.6) [22.2, 61.2]	40.4 (4.6)
BMI	1888	18.5 (1.8) [10.5, 25.6]	18.5†
Arm circumference (mm)	2050	21.9 (1.5) [16.6, 24.7]	21.8 (1.7)

†No standard deviation was reported.

The mean and standard deviations of the anthropometric summary measures of the cohort selected for this study compare very closely to those of the Huffman study of the DNFS sample. This suggests that both the criteria used for selecting this sub-sample, and the summary measure protocol used for repeated measurements results in a similar level of nutritional status to that of the Huffman et al (1985) study of non-pregnant women in the

DNFS. There are a wide range of anthropometric levels in the cohort, weight for example ranging from 22.2kg to 61.2kg. In the context of the determinants of nutritional status this might be expected since, as described in Chapter 1, genetics, disease, childhood nutritional experiences, and household food allocation practices, may influence adult anthropometric levels, independent of adult socio-economic status.

The frequency distribution of each anthropometric indicator are presented in Appendix 4.3; and regression analyses suggesting that BMI is independent of height are presented in Appendix 4.4.

4.32 Comparison of cohort anthropometry with other data from Bangladesh

Comparing nutritional status indicators between populations has considerable interpretational difficulties. The average anthropometric levels in the cohort are much lower than those of women, for example in the US. The NCHS reference for women's height is 163.7cm (6.0) compared with the cohort average height of 147.9cm (5.3) (Krasovec and Anderson,1991). Such comparisons are not of primar y importance in this study, where the focus of interest is the relative risk of mortality associated with the different levels of anthropometric indicators present in this cohort. It is important to examine the representativeness of the cohort with respect to anthropometric data reported for other groups of women in Bangladesh. Table 4.7 presents summary statistics from other studies of women in Bangladesh.

There are differences with respect to weight and height between these studies. The average height of women in the thesis cohort is lower than that observed in the USS or the MCH-FP project. The average height of women in the Fauveau (1994) study is nearly 2cm higher than that of the thesis cohort. The taller cohorts were observed around 9-16 years after the DNFS and the age profiles of women in the thesis and the USS studies are similar. This may tentatively suggest that heights may be increasing in younger birth cohorts in Bangladesh. The urban USS study has a lower average weight than the rural MC project; but it is similar to that found in this study. The higher BMI and MUAC of the USS compared with the thesis cohort is surprising since it was conducted in an area which included many slum dwellers, whose adult socio-economic status may be generally more disadvantaged than women in the rural Matlab area.

Comparability is complicated by the pregnancy status of women in the MC project who were on average women who were 6.8 months pregnant, and would be expected to have gained weight. Average BMI was highest among women eligible for BRAC assistance. These are considered on the basis of their land ownership and selling of manual labour to be the poorest households in the Matlab area. The BMI levels and standard deviations of measures were similar in the other studies.

Table 4.7

Bangladesh studies which present data on adult women's anthropometry

Source	Year of study	Age (yrs)	Area	Height (cm) mean (sd)	Weight (kg) mean (sd)	BMI mean (sd)	MUAC (mm) mean (sd)
Thesis cohort n=2,314	1975-1978	mean 27.5	Matlab, rural area	147.9 (5.2)	40.5 (4.6)	18.5 (1.8)	21.9 (1.5)
Urban Surveillance System n=2,417[1]	1991		Dhaka district, urban slums	148.82 (5.42)	41.82(6.58)	18.81 (2.66)	23.3 (2.5)
Maternity Care Project n=1,633[2]	1987-1989	-[5]	Matlab, rural, MCH-FP area	149.7(5.5)	45.8(5.2)	-	21.7 (1.8)
BRAC-ICDDR,B baseline survey n=987[3]	1992	<50	Matlab, rural, both DSS areas eligible for BRAC	-	-	18.87 (2.0)	-
BRAC-ICDDR,B baseline survey n=669[4]	1992	<50	Matlab, rural, both DSS areas non-eligible for BRAC	-	-	19.3 (2.2)	-

[1] (Baqui et al,1993) non-pregnant mothers with at least one child under 5 years and who were participants in the ICDDR,B Urban Surveillance System. 1991.

[2] (Faveau,1994) Cross-sectional measurements recorded for 1,633 pregnant women in the MCH-FP area examined between the third and nine months of pregnancy.

[3] (BRAC-ICDDR,B,1994) 987 female respondents from 24 villages in the DSS area. These were from house-holds eligible for assistance from the Bangladesh Rural Advancement Committee (BRAC). All were currently married women <50 years of age.

[4] (BRAC-ICDDR,B,1994) 669 female respondents from 24 villages in the DSS area. These were from house-holds non-eligible for assistance from the Bangladesh Rural Advancement Committee (BRAC). All were currently married women <50 years of age.

[5] Matlab MCH-FP data on ages not presented, however would be of reproductive ages

Chapter summary

The DNFS source of the anthropometric data, was a study designed to examine fertility status with relation to anthropometric levels and changes. On the basis of discussions with ICDDR,B researchers, the data can be expected to be of high quality. The selection of only 8 women who may have important measurement errors affecting the summary value tentatively supports this. However if there were systematic errors, for example if the weight scales were calibrated incorrectly for all measurement rounds, the procedure used for validation is unable to detect this.

A wide range of approaches for summarising repeated measurements are presented in the literature. Unfortunately many papers do not describe their methodology for preparing nutritional data. The absence of 'rules' means that the protocol needs to reflect the requirements which the testing of the study hypothesis places on the data preparation and how the data meets those requirements. The approaches used in this study balance the need to describe the general nutritional status of an individual relative to others in the cohort, with the intrinsic biological variability of these nutritional indicators.

The median value with adjustment for extreme outlying values is an adequate summary measure, since in the survival analyses presented in Chapter 5, women are entered into the models based on categories of each nutritional indicator. The categories are calculated on the basis of quartiles (25%) cut-offs, and 10%,90% cut-offs. The latter grouping chosen to discriminate women with anthropometry at the extremes of the population profile. If mortality is associated with anthropometry, there should be confidence in the survival models being able to detect the risk associated with different categories of an indicator.

The summary statistics when compared with similar data for adult women in Bangladesh suggest that the cohort may be considered representative of similarly aged women in the Matlab area of Bangladesh in the period 1975-1978. The generalisability of the results of this study to women in other areas of Bangladesh, both rural and urban, is less clear. However, data from studies conducted in the late 1980's and early 1990's suggest very similar levels and variability in women's anthropometry.

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Chapter 5 The association between anthropometric indicators and mortality

Chapter introduction

This chapter presents the results of the Cox's proportional hazards analyses used to test the hypothesis that:

> *Women of low nutritional status, as measured by anthropometry, are at a higher risk of mortality during the period of follow-up than women with higher nutritional status, after adjusting confounding.*

Although the focus of the thesis is the hypothesised association between anthropometry indicators and mortality, many of the determinants of both survival and nutritional status indicators are socio-economic. For example, poverty affects both the ability to purchase food, and to access medical treatment. Consequently, any analysis of the association between survival and nutritional status should consider the potentially confounding effect of socio-economic factors. The proportional hazards model allows the testing of the association between anthropometric indicators and mortality, with adjustment for confounding variables.

Section 5.11 describes the underlying principles of Cox's proportional hazards models and the likelihood ratio test. Several texts were used as principal sources of statistical methodology (Parmar and Machin, 1995; Breslow and Day,1987; Khan and Sempos,1989; Collet,1994). Section 5.12 presents univariate analyses of the different anthropometric indicators. The approaches used to minimise bias due to confounding are described in Section 5.13. Section 5.2 describes the results of the models testing the association between anthropometric indicators and mortality risk after adjusting for socio-economic confounding variables. In Section 5.3 additional analyses are presented, in 5.31 an approach to modelling missing anthropometric data based on other available indicators, and in 5.32, adjusting models for early mortality to minimise bias due to existing illness at time of measurement.

SECTION 5.1 Survival analysis - methodological issues

5.11 Proportional hazards models - their use and interpretation

Cox's proportional hazards models regress the instantaneous risk of death (or hazard), λ, at time t on a vector of subjects' covariate values, z. The models are used to explain the underlying determinants of survival, or to adjust estimates of the effect of one variable for the potentially confounding effect of others. The model assumes that at any time, t, the hazard of a subject with a set of covariate values, zi is a multiplicative function of an underlying baseline hazard dependent only on time, and a function exp $(\beta^T zi)$ for some linear function $\beta^T zi$ giving the model:

$$\lambda (t : z) = \lambda_0 (t) \exp (\beta^T zi)$$ at time t for an individual i.

Time t is entered into the model as partial years, and is the time from entry into the study until she dies or is censored. For women surviving until the end of the follow-up period, censoring is made on 31st July 1993. The average years of follow-up in the cohort (n=2,314) is 15.8 years with a range of 0.10 to 18.46 years.

The models are used to calculate several important estimates:

i. The coefficient is estimated for each level of the covariate against its baseline level. The log of the coefficient is the hazard ratio (HR), which describes the risk of instantaneous death in the category, compared to the baseline category where the HR is set to 1.00. A HR of 2.00 in category (1) against the baseline category (0) indicates that women in category (1) are at twice the risk of instantaneous death at any time during the follow-up period, than women in category(0). Phrased another way, the immediate risk of death for women in category (1) is 100% higher than women in category (0).

ii. The 95% confidence intervals of the HR are calculated from the standard error of the coefficient: *exp [coefficient* $\pm$ *(1.96 x standard error)].*

iii. In addition, a test of difference between the HR of each category compared to that of the baseline is calculated. The p value reflects the level of significance with which the null hypothesis - that there the hazard rates are the same between the two categories of a variable- is accepted or rejected.

The likelihood ratio (LR) test, is the probability of the observed data being 'explained' by the given model. In other words for the models shown in this chapter it assesses the level of statistical association between one or more covariates and mortality. Consequently, the LR test allows the testing of a model (l_o) against another model (l_v) which contains more variables, providing that the same individuals are included in both models. The null hypothesis is that there is no difference in the two models, i.e. the inclusion of the extra variable(s) in (l_v) does not help to explain the survival any more satisfactorily than the model (l_o). The LR statistic obtained is a function of the difference between the log likelihood values in each model. This statistic has a χ^2 distribution and the results can be tested with their degrees of freedom to produce the degree of significance between the two models. A significant result suggests that the model (lv) explains a greater amount of variation than the model (l_o). In this chapter the null model is used to describe the (l_o) model for mortality risk containing no covariates.

Another important consideration is the form in which variables are entered into the model. Some variables, for example, height, could be entered as a continuous or a grouped variable. Where variables were available as continuous variables, all except age were categorised. This is because the model constrains continuous variables to have a linear association with the survival function. Categorising allows greater flexibility in the nature of the explanatory-survival relationship (Parmar and Machin, 1995). Age was entered as a continuous variable since its relationship with mortality is expected to be exponential. In the absence of standard groupings in the literature, the choice of categories for continuous variables, not pre-coded on collection, was defined by centiles. For the anthropometric indicators, the categories (g1) were based on the 10%,90% centiles, and (g2) on the 25%,50%,75% centiles (quartiles).

In the following Section 5.12, the nutritional indicators are assessed independently by means of univariate (bi-variate) models. These models are later repeated in Section 5.21 with adjustments for potential confounding covariates.

5.12 Univariate analyses of nutritional status and mortality

The preliminary analyses examine the univariate relationship between each anthropometric indicator and mortality. These crude results are an important basis with which to compare later multivariate models. In Table 5.1 the results of entering each anthropometric indicator independently (both percentile groupings shown) into a proportional hazards model are

shown. In columns 5 and 6 are the mortality hazard of each anthropometric indicators, and in columns 8 and 9 are the age adjusted results. When a covariate is placed in the model, women missing data on this covariate cannot be included. Therefore, the results presented in Table 5.1 are not always the same subset of women. Included in the table are the LR results for the individual covariates compared with the model containing no other covariate variables (column 7) and with a model containing age (column 9). The effect of age as a potential confounder is described before socio-economic variables, because as described in Chapter 1, 3 and 4 age is strongly associated with both mortality and anthropometry.

In Table 5.1 the LR statistics from the univariate analyses (column 7), suggest that the anthropometric indicators weight, BMI and arm circumference are statistically associated with mortality at the 10% confidence level. These nutritional measures were associated with mortality when entered as either (g1) or (g2) with the exception of arm circumference, where only (g1) was significantly associated. Of all the anthropometric measures, BMI (g1) shows the strongest statistical association with mortality in the univariate analysis (LR=16.63,p=0.0002). Including height as either grouping did not statistically improve the model in explaining the mortality experience of women. Both LR results were not significant at the <10% level.

Table 5.1

Univariate proportional hazards model results for nutritional status indicators

Variable	No. who died	No. who survived	No. censored	Hazard ratio	(SE) p value	LR statistic§ (p value)	Hazard ratio Adjusted for age	(SE) p value	LR statistic§ (p value)
Height (g1)									
< 1.415	19	177	26	1.00			1.00		
1.416 - 1.549	89	1414	258	0.60	(0.15) p = 0.042**		0.67	(0.10) p = 0.114	
>=1.545	11	178	30	0.58	(0.23) p = 0.154	3.79 (0.1505)	0.67	(0.25) p = 0.284	2.34 (0.31)
Height (g2)									
< 1.440	35	385	70	1.00			1.00		
1.441 - 1.479	31	484	101	0.72	(0.18) p = 0.191		0.78	(0.10) p = 0.324	
1.480 - 1.512	24	461	73	0.60	(0.16) p = 0.051**		0.64	(0.17) p = 0.096*	
>=1.513	29	439	70	0.75	(0.19) p = 0.254	4.01 (0.261)	0.82	0.82 (0.21) p = 0.442	2.88 (0.411)
Weight (g1)									
< 34.7	24	154	22	1.00			1.00		
34.71 - 46.4	76	1305	219	0.39	(0.09) p= 0.000***		0.48	(0.12) p = 0.002***	
>=46.41	9	158	32	0.38	(0.15) p = 0.013***	13.83 (0.0010)***	0.54	(0.22) p = 0.123	8.22 (0.0164)**
Weight (g2)									
< 37.49	42	396	66	1.00			1.00		
37.5 - 40.49	23	406	70	0.55	(0.14) p = 0.02**		0.61	(0.16) p = 0.061*	
40.5 - 43.59	18	420	68	0.41	(0.12) p = 0.002***		0.49	(0.14) p = 0.013***	
>=43.6	26	395	69	0.64	(0.16) p = 0.071*	11.78 (0.0082)***	0.84	(0.22) p = 0.502	7.99 (0.0462)**

* p <0.1 ** p <0.05 *** p <0.01

‡ (g1) are the 10%,90% percentiles; g(2) are the quartiles, 25%,50%,75%

§ LR test statistic for β=0 or HR=1.00

Table 5.1 (continued)

Variable	No. who died	No. who survived	No. censored	Hazard ratio	(SE) p value	LR statistic§ (p value)	Hazard ratio Adjusted for age	(SE) p value	LR statistic§ (p value)
BMI (g1)									
<16.39	24	142	23	1.00			1.00		
16.39 - 20.71	69	1234	207	0.35	(0.82) p = 0.000***		0.44	(0.11) p = 0.000***	
>20.71	9	149	31	0.37	(0.14) p = 0.011***	16.63 (0.0002)***	0.55	(0.22) p = 0.137	9.83 (0.0074)***
BMI (g2)									
< 17.30	38	376	58	1.00			1.00		
17.30 - 18.41	27	385	63	0.70	(0.18) p = 0.153		0.80	(0.20) p = 0.377	
18.42 - 19.61	14	397	60	0.36	(0.11) p = 0.001***		0.44	(0.14) p = 0.009***	
>19.61	23	367	80	0.61	(0.16) p = 0.061*	12.17 (0.0068)***	0.83	(0.23) p = 0.482	7.69 (0.0528)**
Ac φ(g1)									
< 20.0	22	162	27	1.00			1.00		
20.0 - 24.1	83	1337	221	0.48	(0.11) p = 0.002***		0.56	(0.14) p = 0.018***	
>24.1	7	163	28	0.34	(0.16) p = 0.012***	9.79 (0.0075)***	0.41	(0.18) p = 0.043**	6.19 (0.0452)**
Ac (g2)									
< 21.0	40	463	61	1.00			1.00		
21.0 - 22.0	30	412	72	0.84	(0.20) p = 0.457		0.90	(0.22) p = 0.665	
22.1 - 23.0	19	380	77	0.57	(0.16) p = 0.046**		0.66	(0.18) p = 0.137	
>23.0	23	407	66	0.66	(0.17) p = 0.108	5.14 (0.162)	0.76	(0.20) p = 0.30	2.68 (0.44)

* p <0.1 ** p <0.05 *** p <0.01

φ Ac = arm circumference

‡ (g1) are the 10%,90% percentiles; g(2) are the quartiles, 25%,50%,75% § LR test statistic for β=0 or HR=1.00

The LR results remain similar after adjusting for age (column 10). The exact level of the LR was reduced in all models for both (g1) and (g2). Age adjusted weight (g1) (g2), BMI (g1) (g2), and arm circumference (g1), are associated with mortality at the 5%-10% level. BMI (g1) showed the strongest statistical association with mortality (LR=9.83, p=0.0074). Height, after adjusting for age, does not show any significant relationship with mortality.

The hazard ratios obtained from the univariate analyses unadjusted for age, show that all anthropometric indicators, including height, had at least one category which had a significantly lower mortality hazard than the lowest centile. Figure 5.1 graphically represents the hazard ratios for each indicator with significance to baseline category. The general shape of mortality risk associated with anthropometric quartiles is a backwards J-shape, where the lowest mortality risk is observed in the 3rd quartile. For anthropometric categories (g1), the lowest mortality risk is in the 11-89% and 90% percentile. All non-baseline hazard ratios are less than 1.00, suggesting that the general pattern of mortality risk may be lower for women in the upper centiles of each anthropometric indicator. For weight (g1,g2), BMI (g1) and arm circumference (g1), all categories showed a significantly lower risk of mortality than the lowest centile.

On adjusting for age, the HR values of all non-baseline categories become closer to 1.00, and the statistical significance associated in the mortality risk between the different centiles is reduced. For height (g1,g2) and arm circumference (g2) the LR were not significant (p>0.1) between the hazard ratios of the categories. For weight (g1,g2), BMI (g1,g2), and arm circumference (1), the upper centile does not show significant difference (p>0.1) to the baseline category after adjustment for age.

The reduction in the LR results, after controlling for age, suggests that age is an important confounding factor in the observed association between nutritional status indicators and mortality. In the cohort older women have significantly lower levels of anthropometry (see Section 5.133). Since older women have an increased risk of mortality, failure to adjust for age in a model may overestimate the association between lower levels of nutritional status and mortality. In addition, for younger women not adjusting for age may overestimate the protective effect of higher nutritional status levels. This suggestion is supported by the reduction in the statistical level of difference between the highest centile and the baseline category once age was included in model. The issue of confounding is discussed further in Sections 5.13.

Figure 5.1 Hazard ratios for each anthropometric indicator

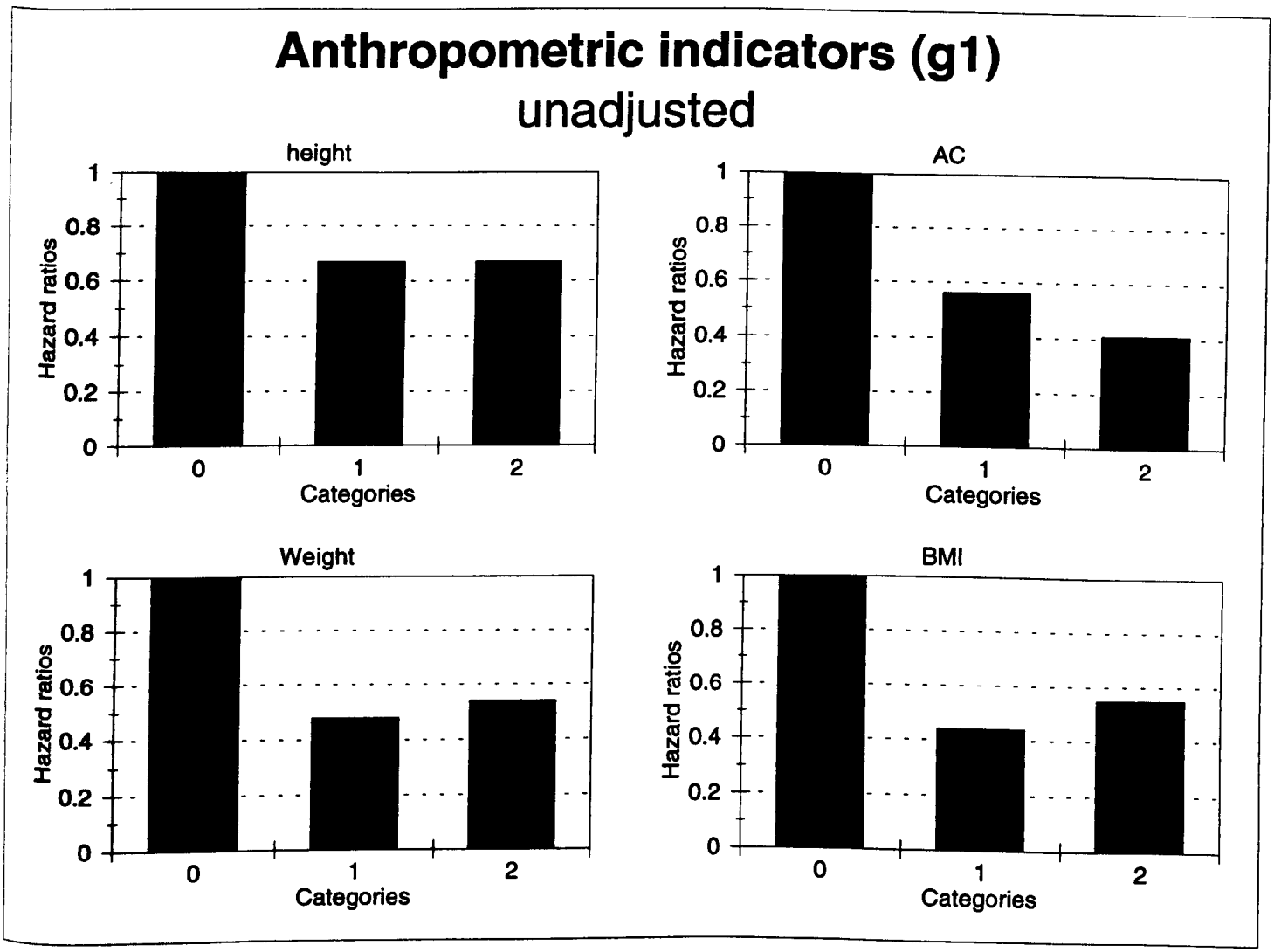
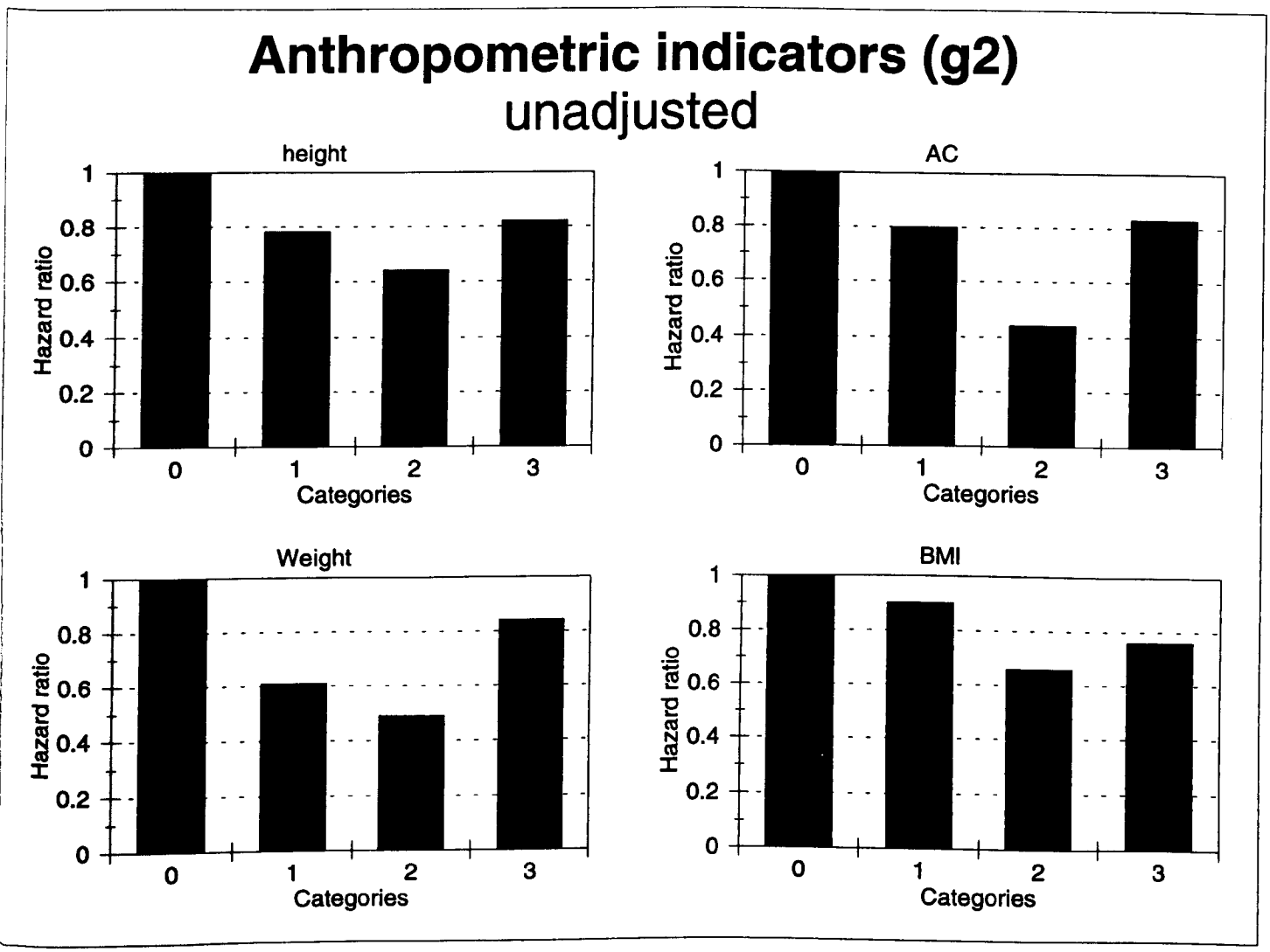


Figure 5.1 (continued) Hazard ratios for each anthropometric indicator (g2)



5.13 Identification and control of possible confounding factors in the association between nutritional status and mortality

The crude univariate analyses presented in the previous section are not satisfactory evidence for the association between anthropometry and mortality, since no account has been made for the effect of other factors, i.e. socio-economic or demographic factors, which may be confounding the mortality hazard associated with each anthropometric indicator. A hypothetical example would be that of dietary intake and mortality. A direct association between dietary intake and mortality could be overestimated or underestimated, if for example, family income was not included in the hazard model. Higher family income may be associated both with higher dietary intake, and with lower mortality due to improved access to medical care. Consequently, a crude model would overestimate the effect of dietary intake in mortality risk.

Rothman (1986) describes a confounding variable as a variable which is: a) a risk factor for the disease; b) associated with the exposure under study in the population from which the cases derive; c) not an intermediate step in the causal path between the exposure and the disease.

5.131 Rationale for choosing the methods of identifying potential confounding variables

There are numerous approaches to the identification, reporting and control of confounding in epidemiological studies (Hennekens and Buring,1987; Rothman,1986). In this thesis, the approach used to identify and select possible confounding variables, which would be adjusted for in later models, was based on:

1. the study objectives;
2. the availability of data on confounding factors;
3. current epidemiological understanding regarding the control of confounding in survival analyses;
4. the size of the study sample.

1. Study objectives

The objective of this chapter is to control for important confounding, in order that the models can be considered to be a plausible statistical test of the relationship between the levels of nutritional status indicators in 1975-1978 and subsequent mortality. Consequently, a detailed examination of the inter-relationships between the different demographic, socio-economic and nutritional variables is not pertinent.

2. Information on potential confounders

In similar cohort studies reviewed in Chapter 1, a variety of variables were controlled for in analyses. For example, in most recent studies examining the relationship between BMI and mortality, adjustment was made for age and smoking behaviour. Blood pressure, alcohol use, marital status, educational level, race, exercise level, coffee intake, dietary pattern, estrogen use, region, and waist-hip ratio were also included in some papers (See Table 1.3). In historical cohorts the information available is typically limited by the study objectives of the original researchers. Although several authors noted that their results may be biased due to residual confounding, they rarely state which other confounders might be important (Hoffmans et al,1988; Wienpahl,1990; Kushner,1993).

For this cohort information on 15 possible socio-economic or demographic confounding variables are available from the DNFS or from the 1982 census, as described in Chapters 2 and 3. These variables could plausibly have associations with both mortality and nutritional status, operating at a family or individual level. Therefore, all variables were initially considered as potential confounders, and examined for their separate univariate association with mortality and anthropometry. Parity showed very strong colinearity with age and was therefore, not included in the models presented.

3. Current approaches to identifying and controlling for confounding variables

Various methods are suggested in the literature for identifying important confounders in survival models. Parmar and Machin (1995) describe two approaches to identifying important confounders using Cox's hazard models. These are generally described as step-up selection, using statistical significance in the LR statistic to include or exclude variables. However, selecting potential confounders using a reliance on statistical significance levels has been criticised by several authors as potentially misleading (Rothman,1986). It is possible that

individual variables do not exhibit strong confounding, but a group of variables used together may produce considerable confounding (Rothman,1986; Arason VA,1996)

In the light of the issues raised above, the approach used to identify potential confounders was a crude and age adjusted examination of the association of each socio-economic and demographic variable with hazard mortality risk. The results identified seven variables which were strongly associated with mortality, the rest showed no evidence of an association (See Section 5.132). The association of each of the seven potential confounding variables with anthropometry was then examined and five were significantly associated. Given that all seven variables were plausible confounding variables, and the proportional hazards model remained robust, for the reasons described above, it was decided to retain all seven variables, which were used in the subsequent analyses. In this study the objective is not to precisely explain all variability in mortality, but rather to assess the importance of the available anthropometric indicators.

4. The sample size and the number of deaths during the follow-up period

There are no definitive rules for the number of covariates which can be included in a proportional hazards model. Simon and Altman (1994), and Parmar and Machin (1995), discuss the issues surrounding the 'rules of thumb' for the number of covariates. A very precise 'rule' suggests that no more than one fourth root of the number of events available should be included. After individuals with missing data were excluded, there were 100 deaths, therefore, $100^{1/4} = 3.16$, so three to four covariates could be included in the model. However, another approach suggested by Parmer and Machin (1995) is that there should be a reasonable number of subjects in each sub-category of interest before constructing the regression model. Although "reasonable" is not clearly defined, the inclusion of eight covariates, i.e. one anthropometric variable and seven potential confounders, was considered justified. Where small numbers of deaths were present in covariate strata, the number of deaths in some categories were very small, for example, among those who were educated.

The relationship between potential confounding variables and mortality, and anthropometric indicators are discussed in the following sections.

5.132 Potential confounding variables, the relationship between demographic and socio-economic variables, and mortality risk

The results of hazard models to examine the association between demographic and socio-economic variables and mortality risk, in order to identify potential confounding factors, are presented in Table 5.2. The crude HR results are shown in columns 5 and 6 and the age adjusted results in columns 8 and 9. There are seven socio-economic covariates which are strongly associated with the instantaneous risk of death ($p<0.05$): age, education, religion, dimension of dwelling, distance to water, items owned, husband's occupation. They remain strongly associated with mortality after adjusting for age. The other seven variables do not show evidence of an association with mortality, ($p>0.2$).

The strongest association with survival is age (LR=20.86, p=0.000), with a hazard ratio of 1.31 for each 5 year increase in age. This indicates that a woman who is 5 years older than another, has a 31% higher relative risk of death. The relationship between survival and education was also strong, and when categorised by years of schooling, showed a protective trend. The HR for mortality for uneducated women, was 2.11 ($p = 0.005$) suggesting that women who were uneducated have a risk 111% higher than that of educated women. Even when age is adjusted for, the hazard remains 82% higher.

Hindus have a higher risk of mortality (HR=1.70, p=0.03) than Muslims. Women whose husbands' have low status occupations are at greater risk of dying (HR=1.80, p=0.003) than those of high status occupations. Those women whose family own very few items are more likely to die (HR=1.47, p=0.05) than families with many household items. Those women living in larger houses are less likely to die (HR=0.59, p=0.008) than those living in a smaller house or homeless and those whose houses are located 15 yards or more from a source of drinking water are more likely to die (HR=1.48, p= 0.05) than other women whose houses are in close proximity to a water source.

Consequently, seven demographic and socio-economic variables are identified as predictors of mortality, and therefore, possible confounders in the association between anthropometry and mortality. These seven variables are discussed below with respect to their relationship with height, weight, BMI and arm circumference.

Table 5.2

Univariate proportional hazards model results for socio-economic variables

Variable	No. who died	No. who survived	No. censored	Hazard ratio	(SE) p value	LR statistic§ (p value)	Hazard ratio†	(SE) p value	LR statistic§ (p value)
Age	126	1861	327	1.05	(0.01) p = 0.000***	20.86 (0.000)***	-	-	-
Muslim **Hindu**	106 20	1710 151	236 91	1.00 1.70	(0.41) p = 0.030**	4.18 (0.0409)**	1.00 1.73	(0.42) p = 0.024**	4.46 (0.0347)**
Education **No education**	16 110	443 1418	88 239	1.00 2.11	(0.56) p = 0.005***	9.34 (0.0022)***	1.00 1.82	(0.49) p = 0.026**	5.69 (0.0171)**
0 years of education **1 - 5 years of education**	110 15	1418 388	239 68	1.00 0.51	(0.14) p = 0.014***		1.00 0.59	(0.01) p = 0.054**	
6 + years of education	1	55	20	0.23	(0.23) p = 0.145*	10.08 (0.0065)***	0.28	(0.28) p = 0.207	6.32 (0.0425)**
No land owned **1-4 ha owned**	34 38	470 488	86 74	1.00 1.11	(0.26) p= 0.665		1.00 1.08	(0.25) p = 0.759	
5-12 ha owned	27	398	68	0.95	(0.24) p = 0.831		0.94	(0.24) p = 0.814	
13+ ha owned	26	467	91	0.78	(0.20) p = 0.34	2.01 (0.57)	0.76	(0.30) p = 0.295	2.02 (0.568)
No cows owned **1-2 cows owned**	79 29	1173 358	180 76	1.00 1.18	(0.26) p = 0.452		1.00 1.19	(0.26) p = 0.430	
3+ cows owned	17	293	63	0.85	(0.23) p = 0.538	1.21 (0.54)	0.82	(0.22) p = 0.461	1.50 (0.47)

* p <0.1 ** p <0.05 *** p <0.01

† model is adjusted for age, entered as a continuous variable

§ LR test statistic for β=0 or HR=1.00

Table 5.2 (continued)

Variable	No. who died	No. who survived	No. censored	Hazard ratio	(SE) p value	LR statistic§ (p value)	Hazard ratio†	(SE) p value	LR statistic§ (p value)
No boats owned	32	619	146	1.00			1.00		
1 boat owned	82	1043	143	1.56	(0.33) p = 0.032**		1.42	(0.30) p = 0.095*	
2+ boats owned	12	199	38	1.17	(0.40) p = 0.628	5.10 (0.078)*	1.17	(0.39) p = 0.649	3.00 (0.229)
1-4 family members	32	550	102	1.00			1.00		
5-6 family members	46	535	84	1.47	(0.34) p = 0.092*		1.45	(0.33) p = 0.109*	
7 + family members	47	739	133	1.10	(0.25) p = 0.687	3.30 (0.19)	1.05	(0.82) p = 0.825	3.29 (0.19)
Dimension of house 0-187 sq. ft	58	606	109	1.00			1.00		
188+ max sq. ft	67	1215	210	0.59	(0.12) p = 0.008***	6.75 (0.0094)***	0.61	(0.12) p = 0.0012***	6.11 (0.014)**
Dimension of house 0-187 sq. ft	58	606	109	1.00			1.00		
188 - 294 sq. ft	36	599	110	0.63	(0.13) p = 0.028**		0.63	(0.13) p = 0.03**	
295 - max. sq. ft	31	616	100	0.54	(0.12) p = 0.006***	9.13 (0.0104)**	0.54	(0.12) p = 0.005***	9.27 (0.0097)***
Other source of drinking water	86	1164	189	1.00			1.00		
River or tank	39	659	130	0.79	(0.15) p = 0.226	1.50 (0.22)	0.79	(0.15) p = 0.227	1.50 (0.2204)
Water <15 yards	34	642	125	1.00			1.00		
Water 15+ yards	90	1156	148	1.48	(0.30) p = 0.057**	3.81 (0.05)**	1.48	(0.3) p = 0.052**	3.99 (0.0457)**
Wall material tin	101	1550	262	1.00			1.00		
Wall material other	22	249	53	1.30	(0.31) p = 0.258	1.21 (0.27)	1.30	(0.31) p = 0.263	1.19 (0.276)
Husband's occupation higher	34	736	165	1.00			1.00		
Husband's occupation lowest	92	1120	161	1.80	(0.36) p = 0.003***	9.32 (0.0023)***	1.63	(0.33) p = 0.016***	6.18 (0.0129)**
Items owned 11 - max	33	627	123	1.00			1.00		
Items owned 0 - 10	92	1196	196	1.47	(0.30) p = 0.058**	3.80 (0.0514)**	1.47	(0.30) p = 0.059**	3.77 (0.052)**

* p <0.1 ** p <0.05 *** p <0.01

† model is adjusted for age, entered as a continuous variable

§ LR test statistic for β=0 or HR=1.00

5.133 Potential confounding variables, the relationship between demographic and socio-economic variables, and anthropometric indicators

Table 5.3 presents the results of t-tests for differences in the mean value of each anthropometric indicator by category of the seven demographic and socio-economic variables. In addition, another t-test was conducted to test whether the mean value of the nutritional indicator was different between the women for whom each socio-economic variable was missing compared with those for whom the variable was available. This allows us to test whether the exclusion of women with missing socio-economic data would potentially bias the nutritional profile present in the model. No women were missing data on age, education or religion.

The data in Table 5.3 suggests there are significant associations between socio-economic variables and anthropometry. There were significant differences in the mean level of anthropometric indicators between categories of age, religion, education and husband's occupation. Age was strongly associated with all indicators. Women older than the mean age of 29 years, had on average, significantly lower anthropometric levels than younger women. Given the absence of information regarding prior nutritional experiences of this cohort, or even more generally about the historical nutritional trends in rural adult Bangladeshis, it is <u>not</u> possible to ascribe these elevated levels of anthropometry in the younger women to i) improvements in childhood and adult nutritional experiences affecting the younger birth cohorts; and/or ii) age-related biological or behavioural changes, which would result in declining anthropometric levels in the older women.

Table 5.3

T-test results for demographic and socio-economic variables differentials in anthropometry

Demographic and socio-economic variable	No. x̄ Height (sd)	t test	No. x̄ Weight (sd)	t test	No. x̄ BMI (sd)	t test	No. x̄ ac (sd)	t test
Age < = 29 years	1232 1.481 (0.051)		1117 41.45 (4.46)		1024 18.87 (1.75)		1144 22.18 (1.44)	
Age > 29 years	970 1.474 (0.053)	t = 3.41 p = 0.0007***	882 39.36 (4.66)	t = 10.22 p = 0.0000***	864 18.07 (1.74)	t = 9.85 p = 0.0000***	906 21.70 (1.56)	t = 7.34 p = 0.00000***
Muslim	1943 1.481 (0.052)		1769 40.70 (4.66)		1661 18.52 (1.79)		1811 21.98 (1.49)	
Hindu	259 1.461 (0.055)	t = 5.75 p = .0000***	230 39.18 (4.51)	t = 4.68 p = 0.0000***	227 18.40 (1.81)	t = 0.97 p = 0.3333	239 21.85 (1.67)	t = 1.31 p = 0.19
1 + years of education	509 1.481 (0.049)		476 41.20 (4.73)		438 18.73 (1.88)		484 22.19 (1.51)	
No education	1693 1.478 (0.053)	t = 1.18 p = 0.24	1523 40.31 (4.62)	t = 3.63 p = 0.0003***	1450 18.44 (1.76)	t = 3.03 p = 0.0024***	1566 21.90 (1.51)	t = 3.73 p = 0.0002***
Dimension of the house 0-187 sq ft	728 1.479 (0.055)		674 40. 40 (4.60)		630 18.49 (1.72)		639 21.96 (1.51)	
>188 sq ft	1429 1.478 (0.051)	t = 0.04 p = 0.97	1281 40.56 (4.69)	t = -0.75 0.45	1281 18.51 (1.83)	t = -0.25 p = 0.80	1316 21.97 (1.52)	t = -0.22 p = 0.83
missing	*45* *1.485 (0.053)*	*t = -0.90* *p = 0.078**	*44* *41.44 (4.80)*	*t = -1.34* *p = 0.18*	*40* *18.72 (1.77)*	*t = -0.77* *p = 0.44*	*45* *22.08 (1.37)*	*t = -0.50* *p = 0.61*
Distance to water <15 yds	765 1.478 (0.053)		697 40.39 (4.70)		661 18.41 (1.77)		714 21.93 (1.52)	
Distance to water 15+ yds	1365 1.478 (0.052)	t = -0.11 p = 0.91	1232 40.57 (4.65)	t = -0.82 p = 0.413	1163 18.56 (1.80)	t = -1.69 p = 0.0917*	1266 21.99 (1.52)	t = 1.78 p = 0.0749*
missing	*72* *1.489 (0.050)*	*t = -1.77* *p = 0.077**	*70* *41.14 (4.54)*	*t = -1.13* *p= 0.26*	*64* *18.50 (1.82)*	*t = 0* *p = 1.00*	*70* *22.06 (1.44)*	*t = -0.54* *p = 0.59*
Husband's occupation higher	884 1.48 (0.053)		819 40.97 (4.84)		768 18.60 (1.85)		840 22.04 (1.50)	
Husband's occupation lower	1313 1.4	t = -0.16 p = 0.098*	1175 40.20 (4.51)	t = 3.62 p = 0.0003***	1116 18.43 (1.74)	t = 2.07 p = 0.0384**	1205 21.92 (1.52)	t = 1.78 p = 0.075*
missing	*5* *1.445 (0.036)*	*t = 2197* *p = 0.13*	*5* *44.42 (3.94)*	*t = -1.87* *p = 0.061**	*4* *21.32 (2.09)*	*t = -3.16* *p = 0.0016****	*5* *23.23 (2.21)*	*t = -1.88* *p = 0.061**
Items owned 11 - max	749 1.481 (0.052)		683 40.42 (4.75)		649 18.41 (1.85)		698 21.97 (1.55)	
Items owned 0 - 10	1410 1.477 (0.052)	t = 1.50 p = 0.13	1273 40.55 (4.61)	t = -0.61 p = 0.544	1200 18.55 (1.75)	t = -1.53 p = 0.1266	1309 21.97 (1.50)	t = -0.01 p = 0.9904
missing	*43* *1.483 (0.053)*	*t = -0.62* *p = 0.53*	*43* *41.45 (4.86)*	*t = -1.32* *p = 0.19*	*39* *18.71 (1.79)*	*t = -0.73* *p = 0.47*	*43* *22.05 (1.36)*	*t = -0.36* *p = 0.72*

* p <0.1 ** p <0.05 *** p <0.01

Age and religion were the only variables which showed a significant difference in mean height. Hindu women were on average shorter than Muslims (p=0.0000). If one considers that height is determined by childhood nutritional experiences, then, since a religious affiliation in Matlab rarely changes, Hindus may be generally disadvantaged during childhood. Religion was not associated with BMI which may suggest that nutritional status due to religious affiliation could be of greatest importance during childhood. In contrast, education and husband's occupation, show an association with BMI and arm circumference which might suggest their role as, or proxies for, determinants of nutritional experience during adulthood.

The associations between missing socio-economic data and anthropometric levels only identified significant associations ($p<0.05$) between husband's occupation and BMI. However, only four women had missing data on husband's occupation, suggesting that excluding women for missing socio-economic data may not be an important source of bias.

These analyses suggest that there are potentially important confounding effects of the variables age, education, religion and husband's occupation, which need to be adjusted for in a proportional hazards model testing the association between nutritional status and the risk of death. Proximity to water, dimension of the house and the number of items owned although not showing a strong univariate association with any of the anthropometric measurements. However, they are included in all subsequent analyses. As discussed in Section 5.131, given our poor understanding of the complex inter-relationships between social and biological factors, identification of confounding factors should not be merely based on statistical significance.

5.134 An examination of how the confounders operate when included together in a proportional hazards model

Table 5.4 presents the results of univariate and multivariate proportional hazards models which included the seven demographic and socio-economic confounding variables. Column 3 presents the results of unadjusted, univariate analyses separately for each anthropometric indicator. The crude, unadjusted hazard ratios previously described in Table 5.1 are presented for comparison in column 2. In columns **4** and **5**, the results of a single hazard model are shown, where all seven variables were included together. The number of women included in the model has been reduced

to 1,816, due to the exclusion of women for whom there was incomplete data on any of the seven selected confounders, and/or the absence of a summary measure of weight, height and arm circumference. This approach was used to ensure consistency in sub-sample numbers.

Ideally variables with strong associations and potentially important confounding roles, would be divided into several categories to adjust the model fully for their effect. This was not done in this study since in the higher years (6+) of education category there is only one death, and in the dimension of housing the upper two categories have no significant difference in their hazard rates. Other authors analysing DNFS data have used different cut-offs for categories of years of education (Huffman et al, 1985). These categories were not used in this study since they have no relationship with the educational system in Bangladesh, and the consensus of statistical theory is to avoid manipulation of categories merely to produce significance (Parmer and Machin, 1995).

Table 5.4

Proportional hazards model results for selected confounders (n=1,816)

Variable	Crude HR From Table 5.2Φ	Crude HR Covariates inc. separately	HR All covariates included	(95% CI) All covariates included
Age (years)	1.05***	1.06***	1.05***	(1.03, 1.07)
Education				
1+ year of schooling				
no schooling	2.11***	5.72***	4.18***	(1.69, 10.36)
Religion				
Muslim				
Hindu	1.70**	1.93***	1.56*	(0.94, 2.61)
Husband's Occupation				
High				
Low	1.80***	1.54**	1.15	(0.74, 1.78)
Distance to water				
<15 yards				
> = 15 yards	1.48**	1.69**	1.46	(0.92, 2.31)
Dimension of house				
0-187 sq ft				
188 - max sq ft	0.61***	0.59***	0.73	(0.48, 1.10)
Items owned				
11 - max items				
0 - 10 items	1.47**	1.46*	1.15	(0.73, 1.82)

* p <0.1 ** p <0.05 *** p <0.01

Φ The samples used to calculate the crude HRs in Table 5.2 were calculated using different sized samples.

Comparing the two sets of crude univariate analyses, with each variable entered in a separate model, the hazard ratios have the same direction in the smaller, fixed sample of 1,816 women, as

those obtained from the larger samples of women. However, the magnitude of the HR has changed slightly for religion, occupation and distance to water, and markedly for education.

When the seven socio-economic variables are included together in the model, Hindus and non-educated women have significantly higher risks of dying, HR=4.18, 1.56 respectively, compared to their baseline categories. Age also remains an important predictor of mortality, an increase in mortality risk of 5% with each 5 year increase in age. By contrast, occupation, distance to water, dimension of dwelling and items owned, no longer show significant differences in the mortality risk between their respective categories.

The hazard ratio for women having had no education compared with those with any years of education was raised markedly from the level of HR=2.11, SE=0.56 in the univariate model unadjusted for age, to HR=4.2, SE=1.94 in the multivariate model. The very large standard error of the education category in the multiple covariate model suggests that there was a need to further examine the education variable.

The crude HR for women with no education was also high in the reduced cohort of 1,816 women (column 3) HR=5.72, (SE=0.46). This suggests that the elevated HR is not a result of co-linearity with other socio-economic variables. However, a strongly significant relationship was observed between women missing height measurements and their educational status (not presented). Women without education were less likely (relative risk = 0.97) to be missing a height measurement than women with education ($\chi^2 = 6.90$, p=0.0086). Of the women missing height, 4 educated women, and 3 uneducated women died. Consequently, only including women for whom height, weight and arm circumference were available, both reduces the number of educated women in the model by 23.04%, compared with a decrease of 21.06% of uneducated women. It also disproportionately reduces the number of deaths experienced by educated women. An attempt to model the missing values of the nutritional measurements resulted in a reduced hazard ratio for education. This is described in Section 5.31.

Tests were conducted to check for plausible interactions between socio-economic variables. Education and husband's occupation were tested for interaction with all other socio-economic variables. For example, it may be hypothesised that a reduction in mortality risk associated with being educated may be relatively higher for Hindus than for Muslims, if educated Hindus, independent of economic status, have more confidence in accessing health care outside the Hindu

community despite the fear of discrimination. There was however, no evidence of interaction between variables tested.

SECTION 5.2 Relationship between anthropometric indicators and mortality, controlling for confounding factors

5.21 Results of the proportional hazards models adjusted for demographic and socio-economic confounders

Table 5.5 presents the crude and adjusted Cox's hazard ratios for height, weight, BMI and arm circumference in relation to mortality (n=1,816). Each anthropometric indicator is entered into the model independently. The LR results presented in column 5, represent the statistical significance of the difference in the 'improved' model, i.e. that which additionally contains the anthropometric indicator, compared to a model containing only the seven adjustment variables.

After adjusting for the demographic and socio-economic covariates, the protective effect of the higher percentiles of all anthropometric indicators is reduced. The adjusted HR are closer to 1.00 than their crude equivalents. The LR results suggest that weight (g1) and BMI (g1) ($p<0.05$), and BMI (g2) ($p=0.07$) are significantly associated with mortality. However, height, arm circumference, and weight (g1) are not significantly associated with mortality ($p>0.1$).

Table 5.5

Proportional hazards model results of anthropometric indicators adjusted for socio-economic variables (n=1,816)

Variable	Crude HR, no adjustment	Hazard Ratio adjusted†	(95% CI) adjusted†	LR result§
Height (g1)‡				
< 1.415	1.00	1.00		LR= 0.20
1.416 - 1.549	0.72	0.90	(0.51, 1.60)	p= 0.9031
>=1.545	0.61	0.83	(0.34, 1.99)	
Height (g2)				
< 1.440	1.00	1.00		LR= 0.46
1.441 - 1.479	0.82	0.93	(0.54, 1.60)	p= 0.9278
1.480 - 1.512	0.76	0.85	(0.49, 1.50)	
>=1.513	0.84	1.01	(0.58,1.77)	
Weight (g1)				
< 34.7	1.00	1.00		LR= 7.14
34.71 - 46.4	0.39***	0.50***	(0.31, 0.81)***	p= 0.0282**
>=46.41	0.34*	0.54	(0.23, 1.29)	
Weight (g2)				
< 37.49	1.00	1.00		LR= 5.69
37.5 - 40.49	0.57**	0.67	(0.40, 1.14)	p= 0.1275
40.5 - 43.59	0.44***	0.54***	(0.30, 0.96)***	
>=43.6	0.60**	0.90	(0.52, 1.55)	
BMI (g1)				
< 16.39	1.00	1.00		LR= 9.44
16.39 - 20.71	0.34***	0.45***	(0.27, 0.73)***	p= 0.0089***
>20.71	0.38***	0.55	(0.25, 1.22)	
BMI (g2)				
< 17.3	1.00	1.00		LR= 6.86
17.3 - 18.41	0.74	0.82	(0.50, 1.37)	p = 0.0765*
18.42 - 19.61	0.38***	0.46***	(0.25, 0.87)***	
>19.61	0.65*	0.89	(0.52, 1.52)	
Ac (g1)				
< 20.0	1.00	1.00		
20.0 - 24.1	0.51***	0.62*	(0.37, 1.04)*	LR = 4.19
>24.1	0.36**	0.44*	(0.17, 1.10)*	p = 0.123
Ac (g2)				
< 21.0	1.00	1.00		
21.0 - 22.0	0.88	0.96	(0.58, 1.59)	LR = 1.37
22.1 - 23.0	0.66	0.79	(0.45, 1.39)	p = 0.7116
>23.0	0.64*	0.76	(0.43, 1.33)	

* p <0.1 ** p <0.05 *** p <0.01

† Adjusted for covariates: age, education, religion and husband's occupation, proximity to water, dimension of the house, and the number of items owned. Categories as described in Table 5.4

‡ (g1) are the 10%,90% percentiles; g(2) are the quartiles,25%,50%,75%.

§ LR test statistic for β=0 or HR=1.00

Figure 5.2 graphically shows the adjusted HR and their p values for each anthropometric indicator grouping, adjusted for the socio-economic covariates. For weight (g1) and BMI (g1); and the quartile (g2) of height, weight and BMI, the adjusted HR maintain the backwards J-shape observed in the crude univariate analysis. Adjustment has resulted in the upper quartile, and the

Figure 5.2 Adjusted hazard ratios for each anthropometric indicator

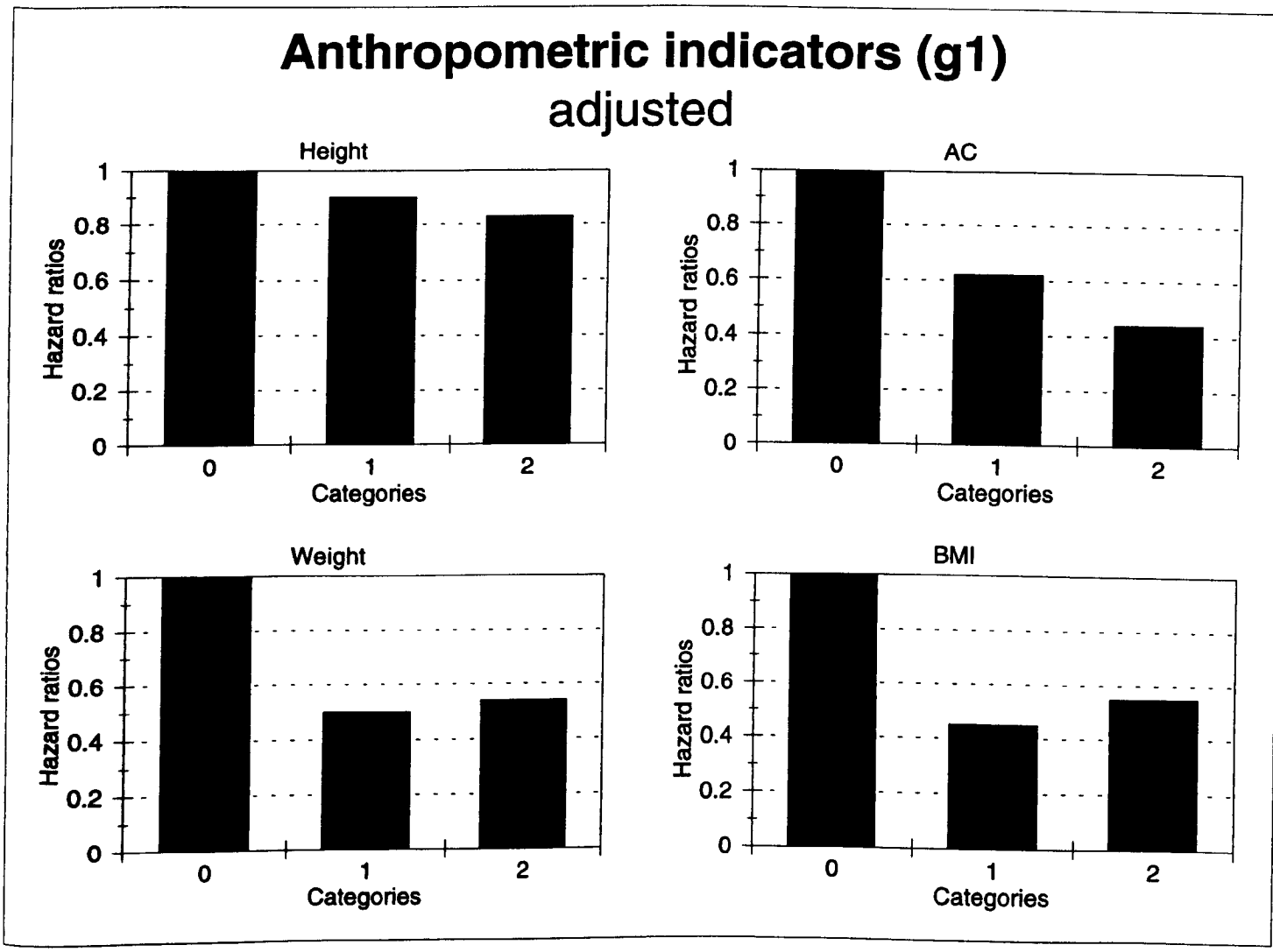
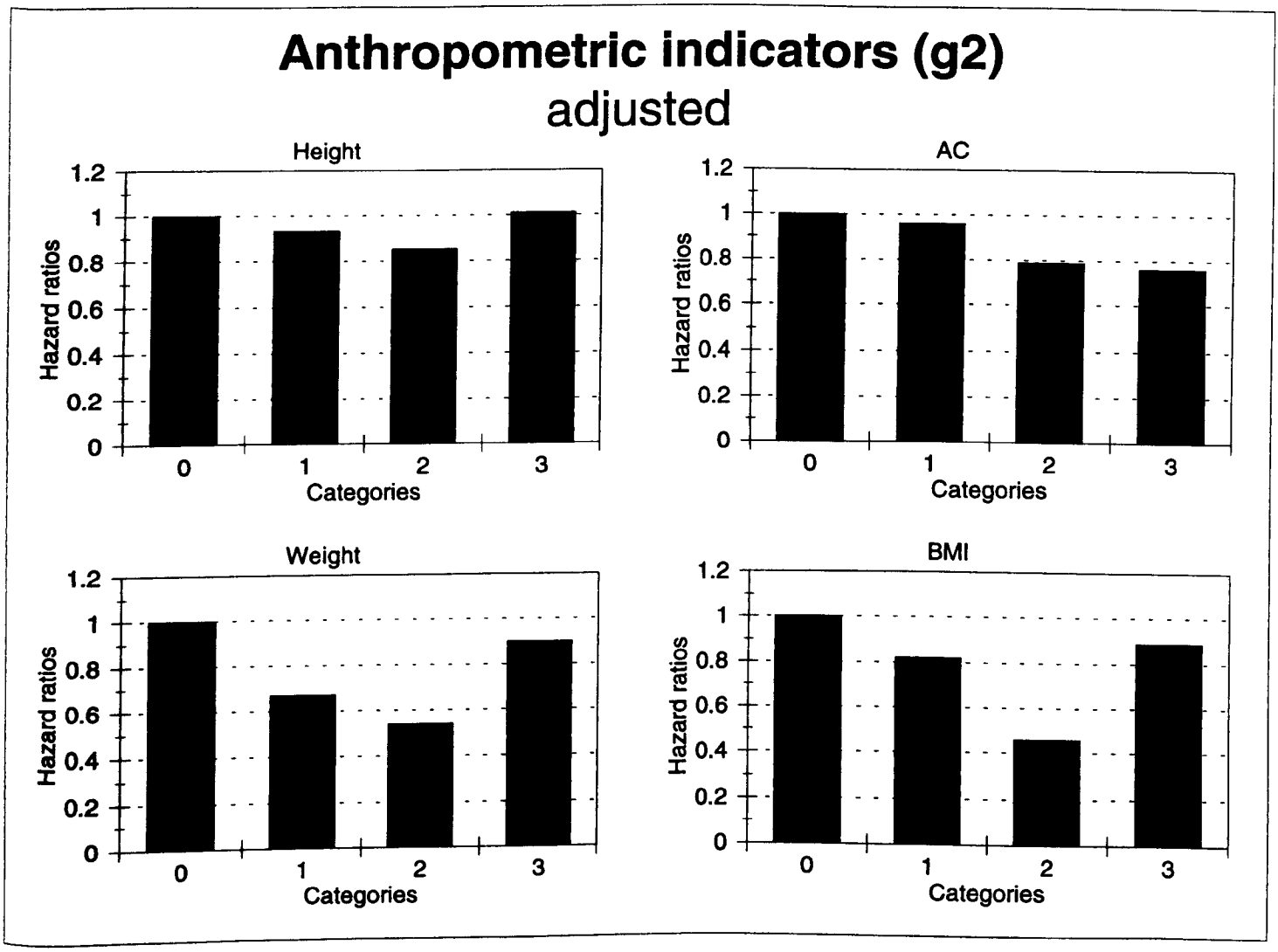


Figure 5.2 (continued) Adjusted hazard ratios for each anthropometric indicator (g2)



90% percentile losing significant difference to the lowest category. Arm circumference (g1,g2) and height (g1) show a direct inverse relationship with mortality risk, the risk decreasing with each increasing percentile of the indicator. The proportionate reduction in mortality risk between categories is largest for arm circumference (g1).

Mortality risk was significantly lower for women in the 11-89% percentile of BMI 16.39-20.71, compared with those in the lowest 10% percentile, BMI <16.39 (HR= 0.45, p = 0.001). This can be also expressed as women with BMI in the lowest 10% centile, have a hazard ratio of 2.24 (95% CI [1.38, 3.65]) compared with women in the 11-89% centile. For women with BMI in the highest 10% of the distribution, their hazard ratio was also protective (HR= 0.55), suggesting they have half the mortality risk of those in the lowest 10%. However, this difference was not statistically significant after adjustment for confounding. This loss of statistical significance may be attributed to the statistical constraints of adjusting small numbers of deaths for multiple confounding variables, thus leading to low power for investigating the association.

When BMI is included in model as quartiles of BMI (g2), the highest mortality risk is observed in the lowest quartile (BMI <17.3), with the lowest mortality risk in the 3rd quartile, (BMI 18.42-19.61). The second and fourth quartiles also have a lower mortality risk when compared with the first quartile, but the difference is not statistically significantly different.

From the LR result, weight (g1) was also associated with mortality (p=0.02), but not when it is entered as quartiles (g2). The lowest mortality risk was associated with weights of 34.71-46.3kg for (g1) and 40.5-43.59kg for (g2). Both these weight ranges were significantly different to the lowest weight percentile. The LR results suggest that only when weight is categorised as (g1) can it predict mortality at the 5% level. Since weight is strongly correlated with height, which in this cohort was not associated with mortality, it suggests that weight only predicts mortality in individuals of very low weight (<34.7kg). At such low weight levels, these individuals would be expected to have low BMI levels regardless of their heights. Weight is less strongly associated with mortality than BMI and is difficult to interpret. The correlation between weight and height was the principal rationale in the development of body mass indexes, such as the Quetelet index (weight(kg)/height(m)^2) used in this study.

Height showed the least differences in hazards between categories. In height (g2) quartiles, the lowest mortality risk was observed in the 3rd quartile (1.480-1.512m), but it has a HR of only

0.85. The mortality relationship is more U-shaped than that of BMI or weight, the upper quartile having a HR of 1.01. The 11-89% and 90% percentiles of height (g1) have hazard ratios of 0.72 and 0.61 respectively. This suggests that those women in the lowest 10% (<1.415m) may be at higher risk compared with taller women, but none of the differences were statistically significant, which is confirmed by the LR results for height and mortality.

Arm circumference showed a direct, inverse relationship with mortality risk when entered as either (g1) or quartiles (g2). When entered as (g1) categories, there were significant differences between the highest percentiles and the lowest 10%. There was no significance in the differences between quartile categories after adjustment for confounding. The 3rd and the 4th quartiles had very similar HR, 0.79 and 0.76 respectively. This may be an artefact of the DNFS data coding, where all arm circumferences >24.7cm were coded as 24.7. Consequently, the upper range of arm circumference values is truncated.

Comparing the LR results for all anthropometric indicators, suggests that BMI (g1) is best able to predict mortality risk after adjustment for possible confounding factors. This is reflected in the markedly elevated risk of dying for women with BMI in the lowest 10% of the population. BMI (g2) is also weakly associated with mortality. This suggests that women with the very lowest BMI levels have a higher risk of mortality compared with women of higher BMI, independent of other demographic and socio-economic factors. The extent to which the observed association between low BMI levels and mortality could be biased due to confounding cannot be assessed, and is discussed further in Chapter 6.

5.22 Examining the independent effects of BMI and arm circumference using the hazard model

At a workshop of the Dietary Energy Consultancy Group (IDECG) held in 1992, discussants considered the use of BMI to identify individuals with chronic energy deficiency (CED) (Norgan,1994). One of the discussants, Shetty, suggested that the BMI cut offs could be strengthened with an additional anthropometric indicator, possibly arm circumference. This was countered by another speaker, Naidu, who argued that arm circumference would not be useful since *'..the variability is very high and the sensitivity is no better than BMI'*.

Given this interest in the possible uses of combining arm circumference and BMI measures, both were entered together into the Cox's model to test whether arm circumference has an independent association with mortality after adjusting for BMI. The results are presented in Table 5.6. One reservation was the extent to which the BMI and arm circumference data are correlated, and therefore, would show co-linearity in their categories, or might cause interaction.

Table 5.6

LR results comparing adjusted models containing BMI alone, with models with both BMI and arm circumference (n=1,816)

Model†	LR statistic§
BMI (g1)‡	9.44 p = 0.0089***
BMI (g2)	6.86 p = 0.0765*
BMI (g1) + Ac (g1)	10.81 p = 0.0288 **
BMI (g2) + Ac (g2)	7.62 p = 0.2670
BMI (g1) + Ac (g2)	10.47 p = 0.0630*
BMI (g2) + Ac (g1)	10.05 p = 0.0737

* p <0.1 ** p <0.05 *** p <0.01

† Adjusted for covariates: age, education, religion and husband's occupation, proximity to water, dimension of the house, and the number of items owned. Categories as described in Table 5.4.

‡ (g1) based on the 10%,90% percentiles; g(2) are the quartiles,25%,50%,75%.

§ LR test statistic for β=0 or HR=1.00

The addition of arm circumference (g1) or (g2) to the adjusted model containing BMI increases the LR statistic result. This is most marked where arm circumference (g1) is included with BMI (g2). However, none of the arm circumference/BMI combinations showed more significance than models which included only BMI. Initially, the LR results might appear to suggest that a suitable adjusted model to explain mortality among the cohort, is one that includes BMI (g1) + Ac (g1) (LR=10.81, p=0.0288). However, this needs careful consideration. This model has a lower significant rejection of the likelihood ratio null hypothesis, than BMI (g1) alone (LR= 9.44, p = 0.0089). In addition, when we tested for interaction between BMI (g1) and arm circumference (g1) there was total co-linearity between the highest categories of BMI and arm circumference. This means that all women with BMI in the highest 10% also have values in the highest 10% centile for arm circumference. This level of correlation between BMI and arm circumference precludes their inclusion together in a proportional hazards model. When both nutritional

indicators are included together, the standard errors are highly elevated to levels suggesting little confidence in the stability of the model. BMI has significant explanatory power with mortality, not shown by arm circumference. If the null model contains BMI and socio-economic factors the addition of arm circumference is not significant (LR=1.37, p=0.50), however, in reverse, the addition of BMI significantly improves the explanatory power of the model (LR=6.64, p=0.036).

5.23 Additional analyses focusing on the relationship between BMI and mortality

From the previous analyses, BMI (g1) has the strongest predictive discrimination of mortality risk after adjustment for potential confounders. To assess the relative importance of BMI, demographic and socio-economic variables as predictors of mortality risk, the statistical results for all covariates are shown in Table 5.7.

Table 5.7

The proportional hazards model results for BMI (g1) and socio-economic variables (n=1,816)

Variable	Categories	Hazard Ratio	(95% CI)
Age	**years**	1.04***	(1.02, 1.06)***
Education	**1+ year of schooling** **no schooling**	4.20***	(1.68, 10.30)***
Religion	**Muslim** **Hindu**	1.52	(0.91, 2.53)
Occupation	**High** **Low**	1.21	(0.78, 1.87)
Distance to water	**<15 yards** **> = 15 yards**	1.45	(0.92, 2.30)
Dimension of house	**0-187 sq ft** **188 - max sq ft**	0.72	(0.48, 1.09)
Items owned	**11 - max items** **0 - 10 items**	1.15	(0.72, 1.82)
BMI (g1)‡	**< 16.389** **16.39 - 20.719** **20.72 - max**	0.45** 0.55	(0.27, 0.73)** (0.25, 1.22)

* p <0.1 ** p <0.05 *** p <0.01 value compared to the baseline category
‡ (g1) based on the 10%,90% percentiles

In the model the variables which have statistical significance between categories at the 5% level, are age, education and BMI. The 95% confidence intervals of all other co-variate HR include 1.00. The inclusion of BMI has not markedly altered the individual HR values of the demographic and socio-economic variables from their univariate level presented in Table 5.4.

Education is the covariate with the strongest evidence of association with mortality. The hazard ratio for women with no education is 4.20, compared to educated women (p = 0.002, 95% CI = [1.68, 10.30]). This suggests that women who have never attended school are 320% more likely to die at any time of the follow-up than women who have attended school. This is a very large hazard ratio, and due to the standard error, has a wide set of confidence intervals. The large standard error is a feature of the small numbers of women, (n=438), who were educated, and consequently, the small numbers of deaths among these women. Referring back to Section 5.134, the large hazard ratio may be a result of bias in the women excluded for missing height measurements, and not from co-linearity between education and other covariates in the model. The wide confidence intervals indicate that emphasis should not be placed on the exact hazard ratio obtained. The result should be interpreted only as suggesting that education is a significantly protective risk factor for mortality.

SECTION 5.3 Additional approaches to analysing the relationship between anthropometry and mortality.

In this last section two additional analyses are described, 5.31) the treatment of missing anthropometric measurements using modelled data and, 5.32) adjustment for existing illness by excluding mortality early in the follow-up period.

5.31 Modelling the missing nutritional status data and re-fitting the proportional hazards model

The mortality hazard ratio associated with education was elevated from 2.11 in the sample of 2,314 women (Table 5.2), to 5.72 after the sample was reduced to 1,813 women (Table 5.4), and 4.72 after adjusting for BMI and confounding covariates (Table 5.7). This may be a result of bias due to the exclusion of women who were missing a height measurement. To verify this hypothesis and explore the plausibility of modelling missing nutritional status data, predicted values of the missing nutritional status based on regression models were calculated. For example, where height was missing, the regression of all heights and weights available was fitted, and the missing height replaced with a value of height that would be predicted based on her weight, and

vice versa. Where no arm circumference measure was available, the regression of arm circumference and weight was used to replace the missing measurements. Huffman et al (1985) found a high correlation (r=0.80, p<0.0001) between mean arm circumference and mean weight in non-pregnant women in the DNFS cohort. In this sub-sample this prediction was only necessary for 5 women. Table 5.8 presents proportional hazards model results using the same covariates as those presented in Table 5.7, the model has an increased sample size of 2,202 women due to the modelled data.

Table 5.8

Proportional hazards model results using modelled missing anthropometric data, BMI (g1) and socio-economic variables (n=2,202)

Variable	Categories	Hazard Ratio	(95% CI) p value
Age	**years**	1.03***	(1.01, 1.05)
Education	**1+ year of schooling** **no schooling**	1.49	(0.87, 2.55)
Religion	**Muslim** **Hindu**	1.35	(0.83, 2.19)
Occupation	**High** **Low**	1.57**	(1.04, 2.35)
Distance to water	**<15 yards** **> = 15 yards**	1.30	(0.87, 1.95)
Dimension of house	**0-187 sq ft** **188 - max sq ft**	0.68**	(0.47, 0.98)
Items owned	**11 - max items** **0 - 10 items**	1.22	(0.81, 1.85)
BMI (g1)‡	**< 16.389** **16.39 - 20.719** **>=20.72**	0.46*** 0.57	(0.29, 0.74) (0.27, 1.21)

* p <0.1 ** p <0.05 *** p <0.01

‡ (g1) based on the 10%,90% percentiles

The direction of the hazard ratios for all variables is the same as that for the model presented in Table 5.7. The use of the modelled data results in a reduction in the coefficient and standard error of education in the model, supporting the suggestion about bias in the role of education due to missing height. Note that for education in this model, education no longer exhibits a significant protection against the risk of dying compared with uneducated women. In contrast, both smaller dimension of the dwelling and lower status husband's occupation are now significantly associated with a higher risk of death. BMI maintains its significant association, with women in the middle percentile (11%-89%) having a lower risk of death than women in the lowest 10% centile (HR=0.46, p=0.001).

There are many assumptions in generating predicted values of BMI from regression models. Although the approach has been widely used in children, it is uncommon in adult nutritional studies. The regression correlations for this cohort, although significant, had a large degree of variation, and suggests that the use of exact values of BMI from the cohort, is a more valid model to assess the importance of BMI as a determinant of mortality than the model with fitted measurements.

5.32 Adjustment for early mortality

In Chapter 1 sources of bias in longitudinal cohort studies examining nutritional status and mortality are discussed (Kushner, 1993;Manson et al,1987;Simopoulus and Van Itallie,1984). One potential bias with relevance for this study is the possibility that weight, BMI and arm circumference measurements collected during the DNFS might have been low in individuals due to the presence of existing chronic illnesses which would have increased their risk of subsequent mortality. If this bias is present, the mortality risk in the lowest centiles may be over-estimated. In the absence of clinical assessments for health status on entry into the DNFS, adjustment for existing illness was made by excluding mortality occurring early in the follow-up period. This has been the approach of other prospective studies in developed countries (Rissanen,1991; Rissanen,1989; Stevens,1992; Hubert et al,1983;Rhoads and Kagan,1983;Waaler,1984). The period of early follow-up excluded by studies ranges from 4-7 years. Manson et al (1987) state in their review:

> *" A reasonable approach to obviate the artifactually high mortality associated with lower body weight is to disregard mortality within the first few years of follow-up, based on the assumption that such deaths are largely due to disease present at entry."*

Unfortunately there is little relevant context specific literature available to suggest how many 'first few years' are appropriate given the causes of morbidity and mortality experienced by a rural Bangladeshi population. The largest single cause of death (13.5%) among the 2,314 women in the cohort was T.B and respiratory diseases. It may be hypothesised for example, that very underweight women, who have experienced weight loss due to acute pulmonary T.B, probably in the absence of appropriate and maintained medical treatment; would have a poor prognosis and a short survival time. Simopoulus and Van Itallie (1984) suggest that extra mortality associated

with morbid conditions would be generally highest in the period immediately after measurement, declining with increasing length of follow-up.

In the analyses which follow the first four years of follow-up for all women are excluded. This resulted in 76 women being excluded completely, 57 who had outmigrated, and 19 who died. The years of follow-up were reduced by four years, and the period of follow-up is now treated as starting between 1979 and 1982, with the end of follow-up remaining as June 1993. Table 5.9 presents the proportional hazards models for each anthropometric indicator entered into the model adjusting for confounding covariates. This table has the same format as Table 5.5 with the crude HR for each variable entered without the socio-economic variables presented in column 1. The LR results compare the addition of each anthropometric measure to the model containing the demographic and socio-economic covariates.

After adjusting for both early mortality and potential confounders, the LR results suggest that BMI (g1) may show only weak association with mortality ($p=0.06$). This indicates that early mortality among women with low BMI levels due to pre-existing illness may have considerably over-estimated the mortality risk associated with the lower levels of BMI. For BMI (g2) the adjustment results in the 2nd quartile having a hazard ratio of 1.22 compared to the lowest quartile, however, this is not statistically significant. The lowest mortality risk continues to be associated with the 3rd quartile of BMI (18.41-19.61); or if entered as (g1) percentiles, the lowest mortality risk is in the 11-89% of BMI (16.39-20.71).

Weight (g1) remains associated with mortality ($p=0.02$). For weight (g2) the significance level for the difference between the 3rd and the 1st quartile of weight (g2) is reduced from ($p<0.01$) to ($p<0.1$). The mortality risk associated with categories of height and arm circumference are not altered on adjusting for early mortality.

It is not possible to assess whether the approach used above is sufficient to remove all mortality in the lowest percentiles due to pre-existing illness.

Table 5.9

Proportional hazards models for all anthropometric indicators adjusted for early mortality (n=1,737)

Variable	Hazard Ratio†	Hazard Ratio†	HR (95% CI) adjusted †	LR statistic§
	Complete follow-up	> 4 years	> 4 years	> 4 years
Height (g1)‡				
< 1.415	1.00	1.00		0.58
1.416 - 1.549	0.90	0.81	(0.43, 1.51)	p = 0.75
>=1.545	0.83	0.96	(0.39, 2.38)	
Height (g2)				
< 1.440	1.00	1.00		0.99
1.441 - 1.479	0.93	0.82	(0.45, 1.43)	p = 0.80
1.480 - 1.512	0.85	0.77	(0.41, 1.44)	
>=1.513	1.01	0.98	(0.53,1.80)	
Weight (g1)				
< 34.7	1.00	1.00		7.66
34.71 - 46.4	0.50***	0.46***	(0.27, 0.77)	0.022**
>=46.41	0.54	0.57	(0.22, 1.45)	
Weight (g2)				
< 37.49	1.00	1.00		5.38
37.5 - 40.49	0.67	0.77	(0.43, 1.38)	p = 0.33
40.5 - 43.59	0.54***	0.59*	(0.31, 1.12)	
>=43.6	0.90	1.01	(0.55, 1.85)	
BMI (g1)				
< 16.39	1.00	1.00		5.38
16.39 - 20.71	0.45***	0.51***	(0.29, 0.89)	p=0.068*
>20.71	0.55	0.70	(0.30, 1.66)	
BMI (g2)				
< 17.3	1.00	1.00		6.74
17.3 - 18.41	0.82	1.22	(0.74, 1.99)	p = 0.081
18.42 - 19.61	0.46***	0.46**	(0.22, 0.96)	
>19.61	0.89	1.03	(0.56, 1.89)	
Ac (g1)				
< 20.0	1.00	1.00		3.72
20.1 - 24.1	0.62*	0.60*	(0.34, 1.06)	p = 0.15
>24.1	0.44*	0.44	(0.16, 1.20)	
Ac (g2)				
< 21.0	1.00	1.00		0.68
21.0 - 22.0	0.96	0.81	(0.45, 1.45)	p = 0.88
22.1 - 23.0	0.79	0.83	(0.45, 1.53)	
>23.0	0.76	0.83	(0.46, 1.52)	

* p <0.1 ** p <0.05 *** p <0.01

† Adjusted for covariates: age, education, religion and husband's occupation, proximity to water, dimension of the house, and the number of items owned. Categories as described in Table 5.4.

‡ (g1) = grouping (1) are the 10%,90% percentiles; g(2) = grouping (2) are the quartiles,25%,50%,75%.

§ LR test statistic for β=0 or HR=1.00

Computer print out for the adjusted models are presented in Appendix 5.1.

5.33 Adjustment for young women aged 10-14 years

61 women aged 10-14 years were included in the sample (n=1,816) used in the proportional hazards models presented in Section 5.21. These women were included because they were married, but some had not yet reached menarche. From growth studies in many countries menarche is a marker of the time of last growth in height before adult height is attained (WHO,1995). If these young women were still growing, they may have been assigned to a category which was lower than their adult height (Riley et al,1989). The effect their inclusion in the models would have on the results, would depend on how many grew to levels which would have placed them in a different category; and, whether their mortality risk was higher or lower than other women in the category they were assigned to. In Chapter 2 and 3, very young married women were suggested to be a vulnerable sub-group within the cohort with respect to socio-economic status, but comparison of their mortality was difficult due to small numbers. Inclusion of adolescents in other cohorts would usually be expected to under-estimate mortality in the lower height categories.

Of the 61 women aged 10-14 years old only one died during the follow-up period. To examine the effect including these very young women has on the proportionate hazards models, the analyses was repeated after excluding 10-14 year olds. The sample was reduced to 1,755 women with 99 deaths. The results of the proportional hazards models are shown in Table 5.10, in columns 2 and 3 the HR and the 95% CI obtained from the larger sample in which the youngest age group were included (Table 5.5) are presented for comparison. Although the main concern is the issue of bias in height, all anthropometric indicators were tested after adjusting for socio-economic variables.

From Table 5.10 the estimates obtained on excluding the youngest age group of women, 10-14 years, did not alter the level of the HR or the statistical significance to the baseline category. The LR results for all anthropometric indicators remained the same, however BMI (g2) showed a reduction in its association with mortality risk, p= 0.07 to p=0.1. Therefore there is no evidence that the inclusion of a small number of pre-menarchic

women has significantly biased the estimates of association between anthropometry and mortality.

Table 5.10

Proportional hazards models for all anthropometric indicators adjusted for socio-economic variables, women aged 10-14 years old are excluded (n=1,755)

Variable	Hazard Ratio including 10-14 year olds adjusted†	(95% CI) adjusted†	Hazard Ratio excluding 10-14 year olds adjusted†	(95% CI) adjusted†	LR result§
Height (g1)‡					
< 1.415	1.00		1.00		LR= 0.23
1.416 - 1.549	0.90	(0.51, 1.60)	0.88	(0.49,1.57)	p= 0.89
>=1.545	0.83	(0.34, 1.99)	0.83	(0.34,1.99)	
Height (g2)					
< 1.440	1.00		1.00		LR= 0.53
1.441 - 1.479	0.93	(0.54, 1.60)	0.89	(0.52,1.54)	p= 0.91
1.480 - 1.512	0.85	(0.49, 1.50)	0.85	(0.49,1.49)	
>=1.513	1.01	(0.58,1.77)	1.01	(0.58,1.77)	
Weight (g1)					
< 34.7	1.00		1.00		LR= 7.37
34.71 - 46.4	0.50***	(0.31, 0.81)***	0.49	(0.30,0.80)	p= 0.025**
>=46.41	0.54	(0.23, 1.29)	0.55	(0.23,1.31)	
Weight (g2)					
< 37.49	1.00		1.00		LR= 5.31
37.5 - 40.49	0.67	(0.40, 1.14)	0.69	(0.41,1.17)	p= 0.151
40.5 - 43.59	0.54***	(0.30, 0.96)***	0.55	(0.31,0.99)	
>=43.6	0.90	(0.52, 1.55)	0.93	(0.54,1.61)	
BMI (g1)					
< 16.39	1.00		1.00		LR= 9.44
16.39 - 20.71	0.45***	(0.27, 0.73)***	0.44	(0.27,0.72)	p=
>20.71	0.55	(0.25, 1.22)	0.55	(0.25,1.22)	0.0089***
BMI (g2)					
< 17.3	1.00		1.00		LR= 6.25
17.3 - 18.41	0.82	(0.50, 1.37)	0.84	(0.51,1.40)	p = 0.10
18.42 - 19.61	0.46***	(0.25, 0.87)***	0.48	(0.26,0.90)	
>19.61	0.89	(0.52, 1.52)	0.91	(0.53,1.57)	
Ac (g1)					
< 20.0	1.00		1.00		
20.0 - 24.1	0.62*	(0.37, 1.04)*	0.61	(0.37,1.02)	LR = 4.34
>24.1	0.44*	(0.17, 1.10)*	0.43	(0.17, 1.09)	p = 0.11
Ac (g2)					
< 21.0	1.00		1.00		
21.0 - 22.0	0.96	(0.58, 1.59)	0.92	(0.55, 1.53)	LR = 1.30
22.1 - 23.0	0.79	(0.45, 1.39)	0.79	(0.45, 1.39)	p = 0.73
>23.0	0.76	(0.43, 1.33)	0.75	(0.43, 1.32)	

* p <0.1 ** p <0.05 *** p <0.01

† Adjusted for covariates: age, education, religion and husband's occupation, proximity to water, dimension of the house, and the number of items owned. Categories as described in Table 5.4.

‡ (g1) = grouping (1) are the 10%,90% percentiles; g(2) = grouping (2) are the quartiles,25%,50%,75%.

§ LR test statistic for $\beta=0$ or HR=1.00

Chapter summary

In summary, the BMI and weight showed a significant association with the instantaneous risk of mortality, after adjustment for confounding. Height and arm circumference showed no evidence of an association with mortality. The relationship between BMI and ~~weight~~ is a backward J-shaped pattern, for height and arm circumference the pattern was inverse and direct.

BMI categories based on the 10%, 90% percentiles (g1) is the strongest predictor of mortality, suggesting that women whose BMI levels are in the lowest 10% of the population (<17.3) have twice the risk of dying than women whose BMI lie above this level. The nadir mortality risk obtained from the BMI (g1) category is between 16.38 and 20.71, and for BMI (g2) between18.42 and 19.61. The upper category for all indicators had a protective hazard ratio compared to the lowest centiles, however, for no indicator was this statistically significant after adjusting for confounding. Caution is suggested in interpreting the upper levels of BMI due to the small numbers of deaths, which results in a low level of power to identify associations between BMI and mortality among women in this category.

There appeared to be no independent effect of arm circumference in the relationship observed between BMI and mortality, however, the data shows strong co-linearity. Models using fitted data to replace missing measurements were calculated, but their plausibility are constrained by biological variability in the correlation between anthropometric indicators, and the presence of missing data due to pregnancy or post-partum period.

The frequently cited possibility of bias in the association between low BMI and mortality due to pre-existing illness at the time of measurement was supported by the results. After adjustment for mortality in the first four years, the association between BMI and mortality was considerable reduced (p=0.06). Weight continued to be associated with mortality after adjustment for early mortality but only when the categories discriminate the very lowest weight individuals. In Chapter 6 the findings from this chapter are discussed further with reference to those of earlier chapters.

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Chapter 6 Discussion of results and recommendations

Chapter introduction

This discussion is divided into three sections. The first section (6.1) gives a brief overview of the results of Chapters 2,3,4 and 5. The principal findings are summarised and the cohorts representativeness is discussed. The results of the survival analyses are then discussed for each anthropometric indicator separately. This is necessary since no single study has previously considered the relationship between all of the four anthropometric indicators and mortality. The results are discussed with respect to the current understandings of anthropometry and its functional and social consequences, the context of rural Bangladesh and the associations found by other similar cohort studies. In the second section (6.2) the limitations of the design, data and methodology of the study are discussed. Finally (6.3) recommendations for policy makers and researchers based on the findings of this study are presented.

SECTION 6.1 Discussion of results

6.11 Summary of principal findings

The 1,813 women used in the final proportional hazards models had summary measures for all four anthropometric indicators tested which were validated and/or generated using uniform protocols. The mortality risks associated with each indicator was calculated for the same group of women. Height, weight and arm-circumference measures were collected over similar months during the same years. The follow-up period started for all women within approximately one and a half years. Therefore, all women would have had similar exposures to external factors, including health service provision in the area, and the general availability of food. In addition, vital events were identified through the same system throughout the entire follow-up period. The sample size was sufficient to support proportional hazards models for each anthropometric indicator adjusted for potential confounders.

The socio-economic, mortality and anthropometric characteristics were described and discussed in Chapters 2,3 and 4. All were shown to be representative of women in the Matlab area for the years of the follow-up. The socio-economic characteristics of the women describe a cohort drawn from a rural Muslim society with predominantly agricultural or low salaried employment, where communal arrangements between families are important, and where women and men have little or no education and few material assets.

The mortality of the cohort was described in Chapter 3. Life tables were used to consider age-specific period and cohort mortality rates. The small number of deaths constrain the interpretations which can be made from the results and this is discussed in Section 6.2. Mortality was observed to be higher amongst the oldest age groups in all periods, notably for the oldest ages 55-64. No evidence was found of a marked change in the overall level of mortality during the period of follow-up. This matches evidence from population based data for the Matlab area, although small reductions in mortality levels for all age groups is observed.

The methodology used to prepare and validate the anthropometric data was described in Chapter 4. A median measure was calculated for the repeated measures of weight and arm circumference, with an adjustment in a few cases for extreme outlying values. The presence of missing measurements had implications for robustness of the summary measure and for the sample size in the survival analyses and this is discussed in Section 6.2. Comparison of the anthropometric profile of the cohort, with that found in other studies in Bangladesh, suggests that the cohort is representative of women of similar ages during the period of the follow-up.
However, it is not possible to assess the representativeness of the results for other areas of the Indian sub-continent where anthropometric levels are similar, such as rural India and Nepal.

There are many factors which may have important influences in the relationship observed between anthropometry and mortality. For example, the strong seasonal fluctuations in weight observed among adult women in Matlab, the levels and causes of mortality, and context specific social and behavioural determinants of nutritional status and mortality.

In Chapter 5, the relationship between four anthropometric indicators, height, weight, arm circumference and BMI, and subsequent mortality during a 19 year period, 1975-1993 was analysed using the Cox's proportional hazards survival technique. In bi-variate analyses weight, BMI and arm circumference showed a significant association with mortality. Of the four indicators, only weight and BMI were found to be associated with mortality, after adjusting for demographic and socio-economic confounders. Height and arm circumference were not associated with mortality. The pattern of mortality associated with weight and BMI was a backward J-shape, with the highest mortality risk being associated with the lowest centiles of the anthropometric indicator. Although not significantly associated, height and arm circumference had a shallow direct and inverse mortality risk pattern, with decreasing mortality risk with increasing centiles of the anthropometric indicator. On adjustment for early mortality in the first four years of follow-up, the statistical association between BMI and mortality was reduced to p=0.06 whilst the weight association remained significant at p=0.02. The grouping of weight and BMI which showed the strongest association with mortality was that of groupings, 10%,90%. The results of the LR test for association are summarised in Table 6.1.

Table 6.1

Summary of the LR results estimated from the Cox's proportional hazards models used to test for associations between anthropometric indicators and mortality

Anthropometric indicator	Crude	Adjusted for demographic and socio-economic variables	Adjusted for demographic, socio-economic and early mortality
QUARTILES			
Height	No	No	No
Weight	Yes	No	No
BMI (Quertelet's index)	Yes	Slight (p=0.07)	No
Arm circumference	No	No	No
GROUPINGS (10%,90%)			
Height	No	No	No
Weight	Yes	Yes (p=0.02)	Yes (p=0.02)
BMI (Quertelet's index)	Yes	Yes (p=0.008)	Slight (p=0.06)
Arm circumference	Yes	No	No

In the survival models seven demographic and socio-economic variables were found to have a strong association with both mortality, of which five were also strongly associated with anthropometry. These were age, education, religion, husband's occupation,

dimension of house, distance to water, and the number of items owned. On adjusting for all seven confounders together, age and education were strongly associated with mortality.

In the following sub-sections specific issues relating to each indicator are discussed in sections 6.12 (BMI), 6.13 (height) and 6.14 (arm circumference), and comparisons are made with other similar cohort studies.

6.12 The relationship between BMI and mortality

This section discusses several features of the association between BMI levels and mortality risk in this cohort.

Supporting evidence for an association between low levels of BMI and higher mortality risk

Women in the lowest 10% of BMI (<16.4) had a significantly higher hazard ratio ($p<0.001$) than women with BMI 11-80%, after adjusting for socio-economic status and early mortality. Although there are no comparable data from other studies, the biological and social consequences of low BMI have been discussed by many other authors. Some of this work offers possible explanations for association found between the lowest BMI and mortality.

Much of the literature considering the consequences of low BMI, do so in the framework of BMI as a possible indicator of chronic energy deficiency (CED). CED is defined as

> *'... a steady state where an individual is in energy balance, i.e. the energy intake equals the energy expenditure, despite the low body weight and low body stores.'* (Shetty and James,1994).

CED describes individuals with low calorie intake, who may also be more likely to be deficient in other nutritional requirements, e.g. vitamins, minerals and proteins. In the sample of 1,816 subjects included in the hazards models, 52% (943) of subjects had BMI which would be classified as CED, using the definition of <18.5 proposed by James et al (1988). Of these, 12.7% (120) were type 3 CED (<16.0), 24.6% (232) were

type 2 (16.0-16.99) and 62.7% (591) were type 1 (17.0-18.45). Shetty and James (1994) suggest that among individuals with pre-existing CED, acute energy deficiency (AED)[4] may episodically occur, for example, during seasonal food shortages. Huffman et al (1985) have described marked seasonal variations in the DNFS cohort. All women in the lowest 10% of BMI distribution in the thesis cohort would be classified as either grade II or III CED, suggesting that they may also be vulnerable to episodes of AED. However, the use of a summary measure for weight and arm circumference in the analyses presented in this thesis, does not allow mortality risk to be related to patterns of seasonal variation (see Section 6.5, point 5).

The development of marker levels of BMI to distinguish individuals with CED, and the functional consequences of CED is an area of intense research with many unresolved questions (James,1994). The data from this thesis does not inform the clinical understanding of CED, since there are no measures of physical activity, basal metabolic rates, or dietary intake. All of which are considered important features of CED.

The biological and social consequences of low body weight in developing countries are discussed in the literature despite limited supporting evidence. The role of low BMI in determining work capacity, illness, and mortality are clearly complex and potentially inter-related. With respect to mortality, many of the possible consequences of low BMI described below could also be suggested to operate as pathways through which low BMI may influence mortality risk.

Low BMI has been shown to be associated with lower physical activity levels (Durnin,1994, Shetty and James,1994). Durnin concludes that BMI <17.0 would result in a significant reduction in physical work capacity.[5] In addition, as work capacity decreases, physical activity may be altered, with individuals either avoiding tasks or taking longer to complete the same work (Satayanarayana et al,1977; Desai et al,1984). The reduction in work capacity, and physical activity has been related to a degree of moderate or severe 'stress' imposed by physical tasks on people with low BMI (Durnin,1994).

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[4] They define AED as '.. *a state of negative energy balance, i.e. energy intake is less than the energy expenditure so that, despite changes in metabolic efficiency or physical activity patterns, there is a progressive loss of body weight and body energy stores.*'

[5] Work capacity being measured and expressed as: a) maximal oxygen consumption (VO_2) during graded tests of increasingly severe physical intensity, or b) per kilogram of body weight.

It has been hypothesised that immune competence may be compromised in low BMI individuals, resulting in an increased susceptibility to disease (WHO,1995). However, there appears to be little detailed evidence with respect to the specific causes of infectious morbidity (and mortality) found in developing countries. This is partly a reflection of the difficulty in collecting morbidity data, and several studies have used days of illness or work loss, as a proxy (Pryer,1993; Strickland and Ulijaszek,1994; deVasconcellos,1994). These were reviewed in Section 1.214, and all found significantly higher levels of 'morbidity' in subjects with BMI <17-18. Unfortunately these studies give no insight into the aetiology of illness with respect to specific features of CED or low BMI. There are two important difficulties in interpreting relationships between morbidity, mortality and BMI. i) reverse causality, and ii) the confounding role of socio-economic and behavioural determinants.

i) Reverse causality may exist between illness and BMI. This has been discussed in other studies and reviews described in Chapter 1 (Manson et al,1987; Kushner,1993). BMI may decrease in response to disease or illness, for example malabsorption following chronic diarrhoea, or loss of appetite due to cancer. Consequently, BMI will be low at death. In this cohort the adjustment for early mortality reduced the statistical significance of the association between low BMI, when the extreme 10% of BMI (g1) were considered, and mortality. This suggests that many of the women with very low BMI levels may have been already ill at the time of measurement, however the sample sizes are small. There is no information about their previous BMI, and consequently, it is not known whether these women had experienced marked and sustained weight loss due to illness. Involuntary weight loss has been found to elevate mortality risk in low BMI individuals in developed country cohorts (Rhoads and Kagan,1983; Harris et al,1988).

ii) There are many other factors which might confound an association between morbidity, mortality and low BMI, including socio-economic status and behaviour. Socio-economic status indicators, husband's occupation and religion were found to have a strong association with both BMI and mortality. Lower socio-economic status is associated in most societies with poorer access to medical care, a situation which is very marked in rural Bangladesh. Individuals with low BMI, independent of pre-existing illness, would be expected to live and work in environments with high exposure to infectious diseases, for

example, poor sanitary and water conditions, and therefore, have a higher incidence of diarrhoeal disease. They might be expected to have less use of preventive health care, for example, hepatitis vaccination, and when ill or injured, have less access to timely and appropriate medical care. Therefore, among individuals with the lowest BMI it might be argued that the higher mortality risk is largely determined by their lower socio-economic status.

Other consequences of low BMI are described as social consequences, many of which may further increase the risk of morbidity and mortality (Shetty and James,1994). Where physical activity is reduced in response to declines in work capacity, there may be economic and social consequences (Shetty and James,1994). Days of paid work lost may cause immediate financial hardship for individuals and their dependants. In communal production, such as agriculture and domestic tasks, declines in the work productivity of one individual would place extra workloads on others and/or result in lower productivity and economic gain. It is possible that in some families, these individuals may become marginalised, further reducing their access to food or medical care, which would result in both lower BMI levels, and an increased risk of morbidity and mortality. The adverse effects of loss of manual work capacity due to low BMI may be greater in lower socio-economic status families.

The limited availability of socio-economic variables; the absence of indicators of behavioural aspects of food access, work productivity, health seeking behaviour; and the poor understanding of the relationships between biological and social aspects of adults in Matlab, with respect to their nutritional status; constrain the interpretations which can be drawn from the study results. In this study no information was available on the dietary intake and energy expenditure, or social and behavioural factors, which could inform the discussion, or be used in the analysis as potential confounders.

As discussed elsewhere in this thesis, no cohort studies were identified from developing countries which considered the risk of mortality with respect to low BMI levels (WHO,1995).

The plausibility of the absence of significant differences in mortality risk between women in the highest and lowest percentiles

In Chapter 5 the decrease in the statistical significance of the protective hazard ratio in the upper 10% or highest quartile (75%) was discussed. Can these results be used to support Kushner's (1993) suggestion that a cross country recommendation would be a BMI either below or above the standard range is associated with a worsened health and increased mortality?

Although not statistically significant, the hazard ratio for the upper centiles were less than 1.00, suggesting that the level was protective. For the highest quartile (19.6-25.7) was HR=0.89 and the highest 10% (>20.7) was HR=0.55. If the absolute values of BMI in the upper centiles are examined, the upper quartile only included 3 women who would be defined as grade 1 overweight (>24.99) (James et al,1988). None were in the higher levels, grades 2 or 3. The remainder had BMI in the normal range, BMI 18.50-24.99. No other study has observed that BMI levels in this range would place women at increased risk, rather that they would be protective.

It may be argued that the lack of statistical difference is a result of the small numbers of deaths in these upper centiles. Only nine deaths were observed in women in the highest 10% of BMI, and large standard errors were observed. Therefore, it would not be appropriate to suggest that the higher levels of BMI observed in the cohort were not protective, nor would Kushner's cross country recommendation be supported in this cohort.

The plausibility of the observed pattern of mortality risk associated with BMI, comparisons with patterns observed in other cohort studies

Although the association between BMI and mortality is not strong after adjusting for early mortality, it is interesting to compare the pattern of mortality relationship with the studies from developed countries reviewed in Chapter 1. Two differentials are observed, i) mortality risk associated with BMI levels is a backward J-shaped curve in this cohort, whereas, recent reviews suggest the pattern in developed countries is J-shaped or direct

(WHO,1995); ii) minimum mortality, regardless of shape, occurs at a lower BMI levels than found in some other prospective studies.

The mortality risk in the cohort suggest that mortality risk is lower in individuals whose BMI lie in the average to higher range, quartiles 3 and 4. The backward J-shape is observed and the nadir of mortality risk for this cohort lies between a BMI of 18.4-19.6, the upper limit is tentative due to the reservations about the existence of a true up-turn in mortality risk associated with the higher BMI levels. Whereas the nadir of the curve, estimated by combining several actuarial and prospective studies in developed countries, lies between 19.0 and 27.0kg (Kushner,1993).

In comparing the BMI mortality shape and the level of BMI associated with nadir mortality, with the female cohort studies reviewed in Chapter 1, the striking feature is the very different profiles of BMI found in this study, this is illustrated in Table 6.2. In the thesis cohort, the first, second and third quartiles of BMI corresponds to the lowest quartile in other studies.

Table 6.2

Average BMI levels for the thesis cohort and US studies for BMI by age group 30-39, 40-49. (Adapted from Simopoulos and Van Itallie, 1994).

Study	**Women's mean BMI (SD)**
All ages	
Thesis cohort	18.5 (1.8)
Charleston Heart Study	
White women	24.5
Black women	27.4
Age group, 30-39	
Thesis cohort	18.2 (1.7)
NHANES I	24.7 (5.7)
NHANES II	24.9 (5.8)
Framingham Heart Study	24.2 (4.3)
Build Study 1979	23.6
Age group 40-49	
Thesis cohort	17.7 (1.8)
NHANES I	25.7 (5.6)
NHANES II	25.7 (6.1)
Framingham Heart Study	25.7 (4.6)
Build Study 1979	23.6

For other studies the strong increase in mortality for women above the average BMI, was attributed to the effect of high BMI (overweight) in determining mortality, primarily on the risk of cardiovascular disease. In the Charleston Heart Study, 46% of black women, and 25% of white women were obese, BMI>27.2 (Stevens et al,1992). CED was not highlighted as a concern in other cohorts since few individuals were at low BMI levels and the effect of very low BMI was considered by many authors to be strongly confounded by pre-existing illness or residual confounding for smoking behaviour (Sidney et al,1987). In the thesis cohort, pre-existing illness was also found to be an important source of overestimating the differences in risk between women with the lowest BMI and those of higher BMI levels. However, in rural Bangladesh smoking among women has not been reported to be a common practice.

The extent to which the thesis cohort lies so much lower than other cohorts, combined with the very different causes of mortality, offers possible explanations for the observed differences in the BMI level associated with nadir mortality. Few women had BMI comparable with the average BMI in developed countries, therefore, it is not possible to know whether raising the average level of BMI in this cohort would result in the nadir mortality risk being extended to that found in developed countries. In addition, it is not possible to predict how BMI would relate to mortality if the causes of mortality were predominantly chronic diseases, rather than infectious disease and obstetric.

These speculative issues have interested many researchers. Popkin (1994) has suggested that many developing countries are undergoing, or will begin to undergo, a nutritional transition with increased food security, higher calorific intake, and less nutrient deficiency, with consequent increases in anthropometry. However, this study is unable to provide reliable information on generational changes in either anthropometry or cause-specific mortality.

<u>The loss of significant association between BMI, but not weight, after adjusting for early mortality</u>

The LR results for the models where BMI and weight were entered as group (g1) and adjusted for socio-economic confounders, suggested that BMI had a stronger association with mortality (LR=9.44, p=0.008) than weight (LR=7.14, p=0.02). However, when the first four years of follow-up are excluded to remove subjects who might have severe pre-

existing illnesses at the time of measurement and died within four years, the association between BMI and mortality was considerably reduced to LR=5.38, (p=0.06), whilst weight maintained its significance level with respect to mortality (LR=7.14, p=0.02). What might result in this difference between weight and BMI in the effect of adjusting for early mortality?

Weight is a difficult indicator to interpret because of its relationship with height. BMI is used to adjust weight for height, and as a measure of leanness may be considered a better indicator of current nutritional status than weight alone. Women with similarly low weights at enrolment could have been in very different states of energy balance. For example, a tall subject who had experienced weight loss due to illness at the time of measurement, might have a similar weight to a very short women whose weight was 'normal'. Consequently, low weight would not appear to be a sensitive discriminator of women with weight loss due to illness, in contrast to BMI. After adjusting for early mortality, women of mid- and upper heights whose weight-for-height was low (i.e. low BMI) were disproportionately excluded compared to women who were both very short with low weight. This suggests that pre-existing illness was an important contributing factor in the relationship between BMI and mortality risk. That weight continues to discriminate women at different risks of mortality, after adjustment for early mortality, may be due to the high mortality risk in women in the lowest 10% of weight, who are also very short and likely to be a particularly vulnerable sub-group of women.

6.13 The relationship between height and mortality

Height was not found to be associated with mortality in the thesis cohort, and no significant difference was observed between the different categories of height, all HR lay close to 1.00. The average height and variability between women in the thesis cohort, was very similar to those found in other studies of rural and urban Bangladeshi women, with a mean of 147.9cm (sd=5.2) (Bacqui et al,1993; BRAC-ICDDR,B,1994;Fauveau,1994).

The levels of height in the thesis cohort were considerably lower than cohorts observed in developed countries. All quartiles in the thesis cohort overlapped with the lowest tercile of women in a study of Swedish women conducted by Peck and Vågerö (1989). Although

the studies reviewed in Chapter 1, do not present information on variations in cohort heights, the US NCHS reference standard is 163.7cm (sd=6.0). For the thesis cohort the mean height was 147.9cm (sd=5.2), therefore does not give support to the tentative suggestion that Bangladeshi women might show less variation in their heights than women in developed countries.

As described in Chapter 1, the results of cohort studies in developed countries have not convincingly demonstrated an association between height and all cause mortality (Waaler, 1984; Peck and Vågerö,1989; Leon et al,1995). The association was reduced or eliminated by adjusting for adult socio-economic status. This was consistent with results from several studies which found a strong association between adult height and adult socio-economic status (Davey Smith et al,1990; Riley,1994).

Many authors consider height to be a useful proxy for fetal, infant, childhood and adolescent circumstances due to their role in determining growth (Notkola et al,1985). Often this is generally phrased as height is an indicator of past nutritional experiences. In some studies its relationship with mortality has been used to test whether there is a relationship between childhood socio-economic status and adult mortality (Ben-Schlomo and Davey Smith,1991; Leon et al,1995). There are many conceptual constraints. In particular the strong relationship between adult socio-economic status and both height and mortality. In the absence of information on social and behavioural determinants of mortality and the lack of longitudinal data which can measure age-related changes, a clear understanding of the mechanism through which adult height relates to mortality risk is difficult. Most studies found adult socio-economic status was a stronger predictor of adult mortality than childhood socio-economic status (Notkola et al,1985;Ben-Schlomo and Davey Smith,1991).

In the thesis cohort variable reflecting current socio-economic status variables were not found to be strongly associated with height, with the exception of religion. This contrasts with the strong association between socio-economic variables and weight, BMI and arm circumference, all anthropometric indicators considered to be indicators of current nutritional experiences. No information was available on childhood socio-economic status in this cohort, and no papers were found which discuss social mobility in rural Bangladesh. However, it might be hypothesised that social mobility is limited in Matlab,

where over 90% of marriages are arranged, and the level of socio-economic status of a woman's family and that of her in-laws are generally similar.

From the results height would not appear to be strongly associated with adult socio-economic status. Its association with childhood socio-economic status may also be hypothesised to be weak. Therefore, height would be expected to show no strong association with mortality in this cohort. This is in contrast to other studies, where height showed some crude association with mortality, by acting as a marker of socio-economic status (Waaler,1984).

The inclusion of very young women was a possible source of bias in the hazards analyses. 93 women aged 10-14 were included in the cohort (n=2,314) since they were married, but some had not yet reached menarche. Growth studies in many countries suggest that some of these women may not have attained adult height and their inclusion may have biased the estimate of mortality risk associated with height (Riley et al,1989;WHO,1995). The issue of bias was examined in Chapter 5, Section 5.33, the estimates obtained from proportional hazards models after women aged 10-14 years were excluded were not significantly different to those models where they were included.

In Chapter 1, changes in anthropometry in older ages observed in developed country cohorts were described (Noppa et al,1980,WHO,1995a). In Swedish women, height was found to decline from the end of the fourth decade, but the rate of height loss was small, 1mm/y until older ages, >70 years. No longitudinal data are available on ageing effects on anthropometry in women of developing countries. All women included in the DNFS were pre-menopausal. Therefore, it may be suggested that at the time of their measurement, they had not experienced marked declines in height which would affect the plausibility of the results, particularly since height was entered as a categorical variable.

6.14 The relationship between arm circumference and mortality

After adjustment for age and socio-economic status, no significant relationship was observed between arm circumference, entered as either categorisation, and mortality. The unadjusted hazard ratios, showed significant protection for the upper categories, 20.0-

24.1cm and >24.1 compared with women <20.1cm. Adjustment eliminated the significant difference in mortality risk between the categories.

Arm circumference in adults is the least documented indicator with respect to its functional significance, correlations with other anthropometric measurements, or associations with mortality. The data available are primarily from studies assessing its use as an antenatal screening tool. The current screening cut-off levels suggested are 23cm for 'undernutrition' and 20.7 for 'more severe 'undernutrition', however, there are no papers which present data relating arm circumference with maternal or adult mortality. Krasovec and Anderson (1990) consider that the major feature of arm circumference is that unlike weight, it is a very stable indicator, since fat deposited or lost will predominantly occur around the abdomen and gluteal region, little in the arms.

The results of this study suggest that the relationship between arm circumference and mortality in this cohort is not an independent relationship, but one due to its higher levels among young women and those of higher socio-economic status. Therefore, arm circumference would not be advised as a useful predictor of mortality in this cohort. This interpretation may support the statement by Durnin (1989):

> *"Anyone with wide experience in measuring healthy individuals of varying age would instinctively distrust such a simplistic way of assessing such a complex situation as 'nutritional status'. As far as upper-arm circumference is concerned, the very large variability in healthy individuals makes it difficult for such a measurement to be of more than peripheral help in the assessment of abnormality."*

SECTION 6.2 Limitations of the study

The descriptive statistics and the results of the survival analyses need to be considered with respect to the methods of data collection, preparation and analysis. Cohort studies, particularly retrospective studies which use secondary sources, have methodological constraints imposed by the type of data available, the size of the cohort, and the length of the follow-up period. The principal limitations are discussed below with consideration of their potential effects on the interpretation of the observed results.

1. Validation of secondary data

The use of data from historical secondary sources, principally the DNFS and the DSS, imposes constraints on validation. For example, anthropometric data could not be cross-checked with other sources, or re-measured. The approaches used to validate anthropometric data and vital status were described in Chapters 2 and 5.

The DSS validation of its own vital statistics database was often delayed and, therefore inconsistencies in vital statistics in the computer database needed to be checked manually against the field record books which are updated every two months. The ability of the DSS to record young adult female deaths in Matlab would be expected to be high, given that it is a relatively rare event (Fauveau,1994). However, the exact date of death or outmigration may be less well recorded. Vital status data was missing from the DSS for 30 of the original 2,446 DNFS women. Whether this was due to incorrect identification numbers recorded by the DNFS, or failures in DSS system is unknown.

Migration events present a particular challenge to the Matlab DSS, where women may often temporarily move to their parental or siblings houses (Maloney et al,1981). The in- and out- migration records obtained from the DSS were often inconsistent, and where this occurred, the last known date of outmigration was used. The possible impact of poor outmigration data, which was the only loss to follow-up in this study, is discussed in point 3.

Validation of the demographic and socio-economic data from the DNFS and the DSS 1982 census was also limited. The 1982 census had collected information at the

household level rather than from the women themselves, had been conducted several years after the DNFS, and used a different set of variables and coding system. Consequently, where possible the more complete data collected by the DNFS was used. Confidence in the accuracy of the demographic and socio-economic data varies. For example, cause of death data in Matlab has been shown to be poorly classified (Zimicki et al,1985; Fauveau et al,1989).

Age may be suggested to be one of the most unreliable demographic variables. Most women in Matlab have no official date of birth. For most women in the cohort, their ages were estimated during the first population census of the DSS in 1966.

The effect of poor quality demographic and socio-economic data in the survival models is difficult to estimate. There is no information to suggest the extent of any misreporting or whether it was systematic or random. Misreporting of variables which resulted in an artefactually strong or weak association with mortality, would have affected the choice of variables used to adjust survival models. It might be suggested that very young married women might overestimate their ages given the legal marriage age. The implications for the survival analyses results would depend on whether the youngest age group had a higher or lower mortality rate or anthropometric level than the age group to which they may have been incorrectly assigned.

2. Sample size

2,314 women were included in the thesis cohort. The end point of follow-up, mid-1993, was selected to maximise the length of follow-up, given the validity of the ICDDR,B computerised database in 1994. The mortality observed, 126 deaths, was at the lower end of the predicted range of numbers of deaths estimated from DSS age-specific mortality rates, whilst the number of outmigrants (n=327) was higher than expected.

The need to exclude women with missing covariate data before estimating the survival models, further reduced the sample to 1,813 with only 100 deaths. This resulted in small numbers of deaths, in some categories of covariates. For example, in the highest 10% centile of arm circumference, only 7 deaths were observed. Consequently, the hazard ratios, particularly in the upper centiles of anthropometric indicators have large confidence intervals limiting the confidence in the true estimate of risk. The limitation of

small numbers of deaths in reducing confidence in the plausibility of the results is further illustrated by artefacts due to missing data discussed in point 4.

In Chapter 3, the mortality experience of the cohort was described, and compared with population based DSS mortality data over the same period. The small numbers of deaths resulted in the survival probabilities calculated for each age group, and/or period, being very erratic. This presented difficulties in describing the mortality patterns, and in comparing the cohort mortality with population based data. The small numbers of deaths among the cohort argued against conducting analyses to test the relationship between anthropometry and cause-specific mortality. The cause-specific mortality collected, was used to compare the causes of death among the cohort with DSS population based data to assess the representativeness of the cohort's mortality experiences.

3. Loss to follow-up

All loss to follow-up which occurred in the thesis cohort was due to their out-migration from the DSS. It was not possible to obtain information on the survival status of outmigrants due to the absence of information on their subsequent address, and the length of time which had elapsed since their outmigration. The effect of the losses to follow-up would be an important source of bias if out-migrants were not representative of the remainder of the cohort. Examination of all covariates showed no significant differences in their anthropometric, demographic or socio-economic status, compared with women who died or remained registered in the DSS. This suggests that loss to follow-up in this study is not be an important source of bias. However, loss to follow-up might be expected to have underestimated the proportion of deaths in the cohort.

4. Missing anthropometric, demographic and socio-economic data

The criteria for selecting the thesis sub-sample from the DNFS cohort required at least one non-pregnant measurement for any of the four anthropometric indicators tested to be available. Missing data in cohort studies is most problematic when it is non-random. From the DNFS reports, the most likely reason for persistent absence during follow-up would be women leaving their in-laws to deliver in their family home. Since pregnancy measures of weight and arm circumference would not have been included in the summary

measure used in this thesis, this would not affect the summary measure calculated. There is no indication why some women were missing height information but have repeated measures of weight or arm circumference. In the absence of any suggestion of systematic bias in missing anthropometric data, the most important implication of the missing data was to reduce the sample size used in the survival analyses.

The missing demographic and socio-economic information for the seven adjustment variables selected also reduced the sample size of adjusted models. The DNFS data had almost complete data, but the 1982 census was absent where a woman and her family had outmigrated from the DSS area before 1982. From the results presented in Chapter 5, there was no evidence of significant differences in the level of anthropometry between women with and without the seven demographic and socio-economic adjustment variables.

Although there is no evidence of a systematic bias in missing anthropometric data, the small number of deaths in the educated category led to a potentially biased estimate of the hazard risk associated with education after women missing a height were excluded. Among women excluded for missing height those with education were more likely to die than educated women included in the model. This disproportionately elevated the protective effect of education compared with non-educated women. The use of modelled data verified that the levels of the hazard ratios for other variables were not biased due to excluded data.

5. The limited data on prior or subsequent changes in anthropometric or socio-economic status

In common with many other similar retrospective cohort studies, anthropometry is historical and limited to the period of the previous DNFS study. In this study, the anthropometric information on the cohort refers only to the period of their inclusion in the DNFS between 1975-1978. No information on the women's anthropometry was available for the remainder of the follow-up period, 1979-1993. Current nutritional status as measured by weight, BMI and arm circumference, is subject to change. As discussed in Section 6.12 other studies have shown that weight loss among those of low BMI was strongly associated with increased mortality risk. Categories of BMI measured at the start

of the follow-up period may not discriminate possibly higher risk for some individuals within each category due to subsequent weight loss.

The models therefore, describe the relationship of a women's anthropometry level during some part of 1975-1978, and their subsequent mortality risk in the period 1975-1993. In addition, the information on socio-economic status was only available at two points during the follow-up period, at enrolment between 1975-1978, and in 1982. Socio-economic status is also subject to change, and in the absence of updated information for the whole period it is not possible to be sure that the adjustment for confounding in the survival models was an accurate representation of a woman's status later in the follow-up period. It is not possible to suggest what effect such changes might have on the results. It might be argued that residual confounding may have been a more important source of potential bias.

The lack of updated measures of height also limits the evaluation of possible bias due to the inclusion of very young women who may have continued to grow after enrolment. This was discussed in Section 6.13. Longitudinal anthropometric data would have also provided additional information on age-related changes which could aid interpretation of the observed significantly higher anthropometry in younger women compared with older women. From this study it is not possible to interpret this as evidence of a secular change in anthropometry, since older women may have experienced weight and height loss caused by the ageing process. As discussed in Section 6.13, there is no information on the age-related changes in anthropometry in developing countries.

It is was also not possible to consider the correlation between infant and childhood weights and growth, and adult weight and BMI levels, to explore the relative contributions of both in determining adult mortality. There are scant data in developing countries which can be used to assess the hypothesised relationship between fetal and childhood determinants of adult disease and mortality.

6. Selection criteria for the DNFS cohort

Another potential limitation of studies using historical cohorts is the criteria used in the original sample selection. For the DNFS the criteria sought to select a sample of women

Where cohorts are not the same age at recruitment, selection bias may result in differences in the mean level of anthropometry in different age groups. This may be a consequence of differential mortality among women of a particular level of anthropometry. Older subjects may be generally 'healthier' independent of their anthropometric level. In these data older women 29+ years were shown to have significantly lower mean levels of height, weight, BMI and arm circumference than women <29 years. One of the objectives of grouping anthropometric indicators is to classify women relative to each other. Quartiles calculated on the basis of the distribution of an anthropometric indicator in the whole sample may result in older women being disproportionately assigned to lower centiles. Women may have levels in the lowest 25% centiles compared with other similarly aged women, but would be placed in a different quartile if classified on quartiles based on the whole cohort.

An example of addressing this issue is from Rissanen et al (1991), who in their study of weight and mortality in Finnish women adjusted the groupings of BMI by age, calculating quintiles of BMI by 10-year age groups. They observed a mean BMI of 23.3 (sd=3.5) and 26.5 (sd=4.5) respectively in the age groups 25-34 years and 65-74 years. In addition, they adjusted the mortality risk in the Cox's survival analysis for confounding by age by including age as a continuous variable. One disadvantage of calculating age-specific centiles is in comparing exact levels of an indicator with those found in other studies.

From the data used in this study the effect that this selection bias might have cannot be determined but would be an interesting challenge to address in further research.

with a high probability of having at least one pregnancy during the follow-up period. The exclusion of women who although of reproductive age and non-contracepting, had not had a birth in the previous five years, may have two different bias effects. It may have minimised the number of women with severe illness in the cohort by excluding women who were unmarried and nulliparous due to poor health status. Using national UK longitudinal data, Green et al (1988) found higher mortality among nulliparous women than parous women. Alternatively, the large proportion of the cohort (60%) with parity 3 or more, may have increased the mortality risk profile of the cohort. Using the same data, Green et al (1988) observed that mortality from all circulatory diseases, hypertensive disease, ischemic heart disease and subarachnoid haemorrhage increased with parity. No information was recorded on the health status of women who were not included in the study.

From the results presented in Chapters 2, 3, and 4 the cohort is suggested to be representative of similarly aged women in the Matlab area, with the possible exception of the very young married women. Therefore, the DNFS selection criteria does not appear to bias the results obtained in the survival analyses.

Another aspect of selection bias may influence the appropriateness of quartiles based on the distribution of anthropometric levels for the whole cohort (see facing page).

7. Availability of information on possible confounders

The epidemiological concept of confounding is a fundamental concern in all studies examining risk factors for mortality. The underlying principle behind adjusting for confounding factors is to estimate the independent association of anthropometry and mortality. The limited ability of many of the retrospective cohorts studies reviewed in Chapter 1 to adjust fully for confounding, i.e. the presence of residual confounding, was discussed by their authors. In studies in developed countries the principal concern was residual confounding in the association between obesity and mortality, and failure to adjust appropriately for smoking.

In this study we were able to consider the possibility of confounding by 15 demographic and socio-economic factors collected by the DSS and the DSS census, selecting 7 for inclusion in the final model. Although they are variables which may operate at the individual, family, and environmental level, it may be suggested that they are not able to reflect the range of determinants which may confound the observed association between

BMI and mortality. Unfortunately there is a dearth of detailed qualitative and quantitative studies which have described the determinants, inter-relationships and age and period changes in the many features of socio-economic status.

In the thesis cohort, the adjustment of models would be improved by additional information on behavioural factors, for example, ability and motivation in seeking preventive and curative medical care, food access and dietary patterns; or economic indicators which might be able to discriminate individuals more accurately than husband's occupation. These might include measures of wealth, e.g. income from land and houses, inheritance and remittances from other family members. The strong association between education and mortality argues that there are important behavioural and social features which are pathways through which education reduces mortality risk.

None of the socio-economic factors included are direct measures of childhood socio-economic status. Ben-Schlomo and Davey Smith (1991), in their review 'Deprivation in infancy or in adult life: which is more important for mortality risk?' state:

> *"Since the measurement of socio-economic status is crude, it is reasonable to assume that even after adjustment a large degree of residual confounding will remain. If we wish to advance our understanding of the relative importance of risk factors acting throughout life, it is important that cohort studies should obtain information on both early childhood factors and factors in later life."*

There are no papers which discuss social mobility in the Matlab context, and therefore, as discussed in Section 6.2, there are limitations in the interpretation and further analyses which can be given to the associations between anthropometry and mortality in this study, in particular with respect to height.

8. Adjustment for pre-existing illness

In other cohort studies reviewed in Chapter 1, the sample sizes ranged from 279 to 1.8 million, and follow-up periods from four to 26 years. Sjöström (1992) examined the relationship of sample size, follow-up duration and sample ages with associations observed between mortality and BMI in developed countries. In the Build Study (1979) underweight individuals mortality ratios declined over an observation period of 20 years, in contrast the mortality ratio of overweight subjects increased during the period of follow-up. Consequently, Sjöström (1992) suggests that longer durations of follow-up are

recommended for cohorts with high proportions of relatively underweight individuals to allow for the elevated mortality risk in the early years of follow-up.

The decision to eliminate the first four years of follow-up to retrospectively adjust the model for pre-existing illness was comparable with the adjustment used in other studies. A longer follow-up period was not considered appropriate in this study. The causes of mortality observed in the cohort, suggested that for women who may have experienced significant weight loss due to the disease, e.g. TB and hepatitis, that they would later die from, this interval would be unlikely to be longer than four years. The reduction in the association between BMI (grouped as 10%,90%) and mortality after adjusting for early mortality suggests that if pre-existing illness may have biased the unadjusted estimates. It is not possible to state that all biases due to the effects of weight loss caused by chronic illnesses are removed by the approach used in this study.

Concluding remarks

BMI and arm circumference are indicators of the nutritional status of an individual at the time of measurement, height reflects previous determinants of growth, including fetal and childhood nutritional experiences and genetic inheritance. The thesis findings suggest that mortality risk is slightly higher in women who have very low BMI levels, reflecting their current inadequate nutritional status. There is no evidence that height is independently associated with mortality in this cohort. The strongest predictors of mortality among the cohort were age and socio-economic characteristics which indicate low personal or familial economic status, (for example husband's occupation), and social determinants of mortality (for example religious affiliation and education).

SECTION 6.3 Recommendations

For health programmes in Matlab, the findings do not support the use of height, weight, arm circumference or BMI as screening tools to identify women at higher risk of all cause mortality. BMI showed a slightly significant independent relationship with mortality. The large reduction in the strength of the association after adjusting for early mortality, suggests that the association was overestimated by low BMI due to illness.

The small numbers of deaths in this study did not allow cause specific mortality to be examined, and it is therefore, not possible to make any assessment about the use of anthropometry in screening women for cause specific mortality for example the value of using height to predict the risk of obstructed labour.

After adjustment for early mortality, there is some evidence that women with very low BMI might benefit from supplementation which increases their BMI to that of the average level in the population. However, the programme costs should be carefully considered, since the attributable mortality due to low BMI is small in comparison to that resulting from older age and low socio-economic status.

The principle limitation of this study is its small sample size. Other larger studies are required to validate the findings, and these studies should also attempt to examine the relationship with cause-specific mortality and the role of weight loss in individuals with low BMI.

This study has highlighted many areas where knowledge of the determinants of mortality are poorly understood. Many of the interpretations made in this study are constrained by the lack of published information about biological, social and behavioural relationships in

rural Bangladesh. In Matlab where a large number of studies have been conducted since 1966, it is surprising that socio-economic, cultural and behavioural contextual information is scarce. This dearth of information about adult lifestyles and health is evidence that this is a neglected area of research. Bangladesh may be undergoing changes in economic and social determinants of nutrition and mortality. Without more information about the current situation, the ability of studies to evaluate health status and contribute to the improvements of health programmes will continue to be limited.

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Appendices

Appendix 2.1 : Facsimile of Death Registration Form used in the Matlab DSS prior to the 1986 changes (sample).

DEATH REGISTRATION FORM

ICDDR,B/MATLAB

Serial No. 16 (1–3) Village Code : V56 (4–6) Date of death : 30 11 83 (DD MM YY) (7–12)

Name of deceased : Zazzaburnessa Date of birth : 0 0 53 (DD MM YY) (13–18) Sex : [1] M [X] F [3] UNK (19)

Current id No. V56 - 217 - 2 (20–29) Registration No. 1 V56 - 174 - 2 (30–39)

Mother's current id No. — (40–49) Registration No. — (50–59)

Marital Status at the time of death :

Never Married [1] Married [X] Widowed [3] Separated [4] Divorced [5] [ ] (60)

Education at death : M 12 (61–62) Occupation at death : H/W 17 (63–64)

Events and symptoms leading up to death :

Measles	02	Diarrhoea : Acute	07	Chronic	08
Tetanus	03	Dysentery : Acute	09	Chronic	10
Drowning	04	Childbirth	11		
Murder	05	Jaundice	12		
Suicide	06	Other not covered above	X	Fever	

Symptoms leading up to death : Had been suffering from fever for 2 months as a result she could not take meals regularly (65–66)

Place of death : Village Pailgasa P. S. Matlab Dist. Comilla Code V56 (67)

Type of Doctor Consulted :

Licenced Allopath [1] Allopath quack [2] Homeopath [3] Kabiraj [4] Other [5] Doctor not consulted [X] (68)

Reported by : Hossain Entered by : 6/7/12

Date : 19/12/83 Date :

Date Entered : Field Vol. 19/12/83 Matlab Vol. 21/12/83

Remarks : P.T.O.

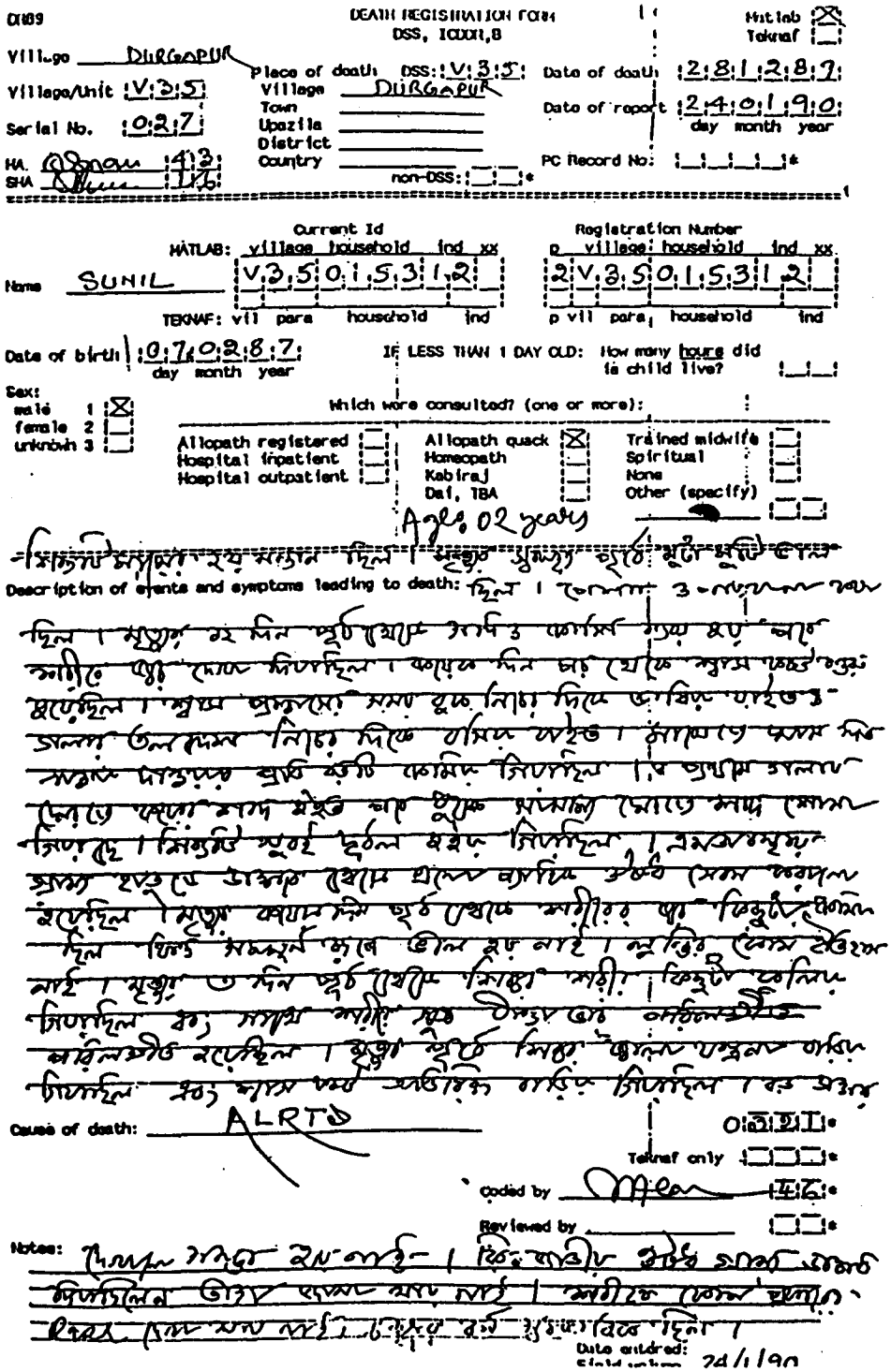
[Source: Fauveau, ed (1994)]

Appendix 2.2 Facsimile of classification and code list of causes of death used prior to 1986, Matlab DSS [Source: Fauveau, ed (1994)]

International Centre for Diarrhoeal Disease Research, Bangladesh
Demographic Surveillance System (DSS), Codes for cause of Death
(prior to 1986)

CATEGORY	CODE
smallpox	01
measles	02
tetanus *(takuria,* evil spirit)	03
drowning	04
murder	05
suicide	06
diarrhoea (acute)	09
dysentery (chronic)	10
childbirth	11
jaundice	12
disease of G.I. (other than cholera) stomach pain	14
respiratory disease (cold fever, cough, TB, asthma)	15
heart disease	16
liver disease	17
venereal disease	18
skin disease	19
E.N.T. disease	20
cholera (proved)	21
dropsy22	
rheumatism	23
accident	24
old age complication	25
fever (all forms)	26
diabetes	27
cancer27	
cancer29	
appendicitis	30
other 91	
unknown	99

Appendix 2.3 : Facsimile of Death Registration Form used in the Matlab DSS after the 1986 changes (sample).



[Source: Fauveau, ed (1994)]

Appendix 2.4

: Facsimile of Classification and code list of causes of death used from 1986, Matlab DSS.

International Centre for Diarrhoeal Disease Research, Bangladesh
Matlab Demographic Surveillance System: Classification of cause of death, from Jan 1986 and correspondance between new codes and ICD codes

Notes :
- Not all codes have one correspondance
- * : causes for which hospital, laboratory, X-Ray, or specialist's confirmation is required.
- R : rare cause, does not need to be included in tabulations.
- # : all possible decimals for the four-digits codes are acceptable

CAUSE OF DEATH	DB-CODE FROM 1986	WHO-ICD
DIARRHOEAL DISEASES		
cholera (proven) (*)	001	001#
acute watery diarrhoea (any other)	010	001-3#, 008-9#
shigellosis (proven) (*)	012	004#
acute dysentery, with blood in stools	013	004#, 006#, 008#-9#
acute non-watery diarrhoea, with mucus and NO blood in stools	014	004-9#
persistent or recurrent dysentery (>14 days) with blood, and NO obvious malnutrition	016	002-4, 006, 008-9
persistent or recurrent diarrhoea (>14 days) w/NO blood and NO obvious malnutrition	017	004-9#
persistent or recurrent diarrhoea or dysentery (>14 days), with severe and/or recent malnutrition (wasting)	191	002-9#,263.8
diarrhoea (any kind) with pneumonia		
INFECTIOUS DISEASES		
pulmonary tuberculosis (*)	020	010-12,018#
diptheria (*,R)	033	032#
whooping cough (pertussis)	034	033#
meningitis (*)	036	036#, 320-2#
tetanus (EXCEPT neo-natal and post-partum)	037	037
septicemia (*)	038	038#
acute polio (*,R)	040	045-9#
measles (death within rash period)	642	055.0,055.8,055.9
post measles pneumonia (within 6 weeks of rash)	742	055.1, 484.0
post measles dysentery (within 6 weeks of rash)	842	055.7
viral hepatitis acute (death during initial jaundice period)	046	070#
rabies (R)	047	071
malaria (R,*)	052	084#
infection of skin or sub-cutaneous tissues	420	680-6#
NEOPLASMS		
malignant neoplasm of digestive organs (*)	099	140-59#
malignant neoplasm of respiratory organs (*)	109	160-5#
malignant neoplasm of female breast (*)	113	174#
malignant neoplasm of uterus (*)	122	179-84#
malignant neoplasm of other sites (*)	139	170-5#, 185-99#
leukemia (*)	141	200-8#
NUTRITIONAL		
diabetes mellitus (*)	181	250#
marasmus-kwashiorkor (nutritional oedema)	190	260-62#
severe and/or recent malnutrition associated with chronic/persistent diarrhoea	191	263.8
marasmus due to lack of breast feeding	192	783.3
marasmus due to insufficient food intake	193	783.3
marasmus due to other chronic infection (non-diarrhoeal, non-respiratory)	194	269.8
death associated with nutritional. blindness (R)	195	264#
MENTAL DISEASES		
psychoses	213	290-9#
physiological malnutrition arising from mental factor	217	306
CARDIO-VASCULAR DISEASES		
rheumatic heart disease, complication of (*,R)	251	390-1#
acute myocardial infarction (*)	270	410-4#
acute but ill-defined C.V. diseases (stroke)	293	342,430,448
late complication of the above	294	
RESPIRATORY DISEASES		
pneumonia, ALRI	321	464-6#, 480-7#
chronic respiratory disease, bronchitis, emphysema, asthma (complication of)	323	490-4
pneumonia with diarrhoea	325	
pneumonia, ALRI associated with, or as a complication of severe malnutrition	328	-
GASTRO-INTESTINAL DISEASES		
peptic ulcer (complication of)	341	531-5#
appendicitis, peritonitis (*,R)	342	540-3,567
intestinal obstruction	344	550-2, 560
chronic liver disease or cirrhosis (>1 month after initial jaundice)	347	571-2#
GENITO-URINARY DISEASES		
nephritis, nephrotic syndrome, renal failure (*,R)	350	580-6#
genito-urinary tract infection (unrelated to pregnancy), salpingitis (*,R)	371	614-5#
DIRECT OBSTETRIC COMPLICATIONS		
spontaneous abortion, miscarriage	380	634
induced abortion	382	635-8
haemorrhage of pregnancy	390	640-1
toxaemia, eclampsia	390	642#
infections in pregnancy (G.U. Tract)	392	646.6
obstructed labour, prolonged labour	393	652-3, 660-2#
other obstetric complications of pregnancy (ectopic preg, ruptured uterus, unknown)	394	643-8#
post-partum haemorrhage (within 12 hours after birth)	395	666#
post-partum haemorrhage (delayed, >12 hours after birth)	396	666#
post-partum sepsis	397	670, 672
post-partum tetanus	398	670
other obstetric complications of post-partum	399	671, 673-4#
NEONATAL PROBLEMS (WITHIN 30 DAYS)		
any congenital anomaly, obvious malformation	449	740-59#
sudden infant death (suffocation during sleep)	450	798.0
birth trauma	451	760-3#
dysmaturity, small-for-date (including due to twin birth)	452	764#
cord haemorrhage	453	767#
hypoxia, birth asphysxia	454	768-70#
neonatal tetanus	456	771.3
other neonatal infections	457	771#
prematurity (direct complication of)	458	765#
unspecified neonatal death	459	772-9#
MISCELLANEOUS		
fever of unknown origin	460	780.6
oedema + ascitis of unspecified origin	461	782.3, 789.5
anaphylaxis, allergic shock	468	995.0, 995.3
IATROGENIC CAUSES		
complication of medical care by official health practitioners	540	996-9, E870-9
complication of medical care by tradition health practitioners	541	" "
complication of surgical care by official health practitioners	542	" "
complication of surgical care by traditional health practitioners	543	" "
ACCIDENTS, INJURIES		
transport accident	550	E800-38
other accidental injuries	551	E850-69, E880-8, E916-28
burns, complications of extended burns	552	948-9 E890-99
drowning	553	E910
snake bite	554	E905.0
homicide by violence	555	E965-9
suicide by poisoning	557	E950
other violent deaths, accidental suffocation accidental poisoning, lightning, electric shock	559	{799.0, 798.1 {994.7, 798.2 E911-28, E907 {994.8, 994.0
accidental falls due to epilepsy	560	- E928
OTHER AND UNSPECIFIED		
other cause (specified but rare)	990	-
senility (old age death without specified disease)	991	979
impossible to specify	998	799
unreported or unknown	999	-

Appendix 2.5 : **Facsimile of Questionnaire for interview of relatives and attendants in cases of an adult female death between 15 and 44 years of age, Matlab DSS, 1986.**

DECEASED WOMAN CID : ..... RID ..... DOD: ...../...../AGE: .....Y CHW CODE NUMBER..... NAME.....

1. DID YOU KNOW THAT WOMEN ? | YES | NO |

2. WHAT WAS HER MENSTRUAL STATUS (2-3 MONTHS BEFORE HER DEATH) ? | P | N. | A. | PPA. | MENOPAUSE | UNKNOWN | PREMENSES |

3. WHAT WAS HER CONTRACEPTIVE STATUS (2-3 MONTHS BEFORE HER DEATH) ? | P. | L. | I. | S. | C. | OTHER | NONE | UNKNOWN | NA |

4. DO YOU REMEMBER THE CIRCUMSTANCES OF HER DEATH ? | YES | NO | IF YES DESCRIBE.....
(use back of this form)

5. WAS SHE KNOWN BY YOU AS PREGNANT BEFORE DEATH ? | YES | NO | NOT SURE | IF YES: 1. HOW LONG ? ..... MONTH 2. WAS IT HER FIRST PREGNANCY ? | YES | NO | UNKNOWN |

3. WAS THE PREGNANCY: | UNWANTED | ACCEPTED | ILLEGITIMATE | DESIRED |

4. STOP HERE

6. DO YOU REMEMBER OF ANY ABNORMAL SYMPTOMS OR COMPLAINS IN THE WEEK(S) PRIOR TO DEATH, RELATED TO PREGNANCY OR ANY OTHER CONDITION ? (LIKE ABNORMAL BLEEDING, HAEMORRHAGE, NAUSEA, VOMITING, DIZZINESS, FEVER, CHILLS, FAINTS, CONVULSIONS, PELVIC PAINS, ACCIDENT, BEATEN) | YES | NO | IF YES, DESCRIBE .....

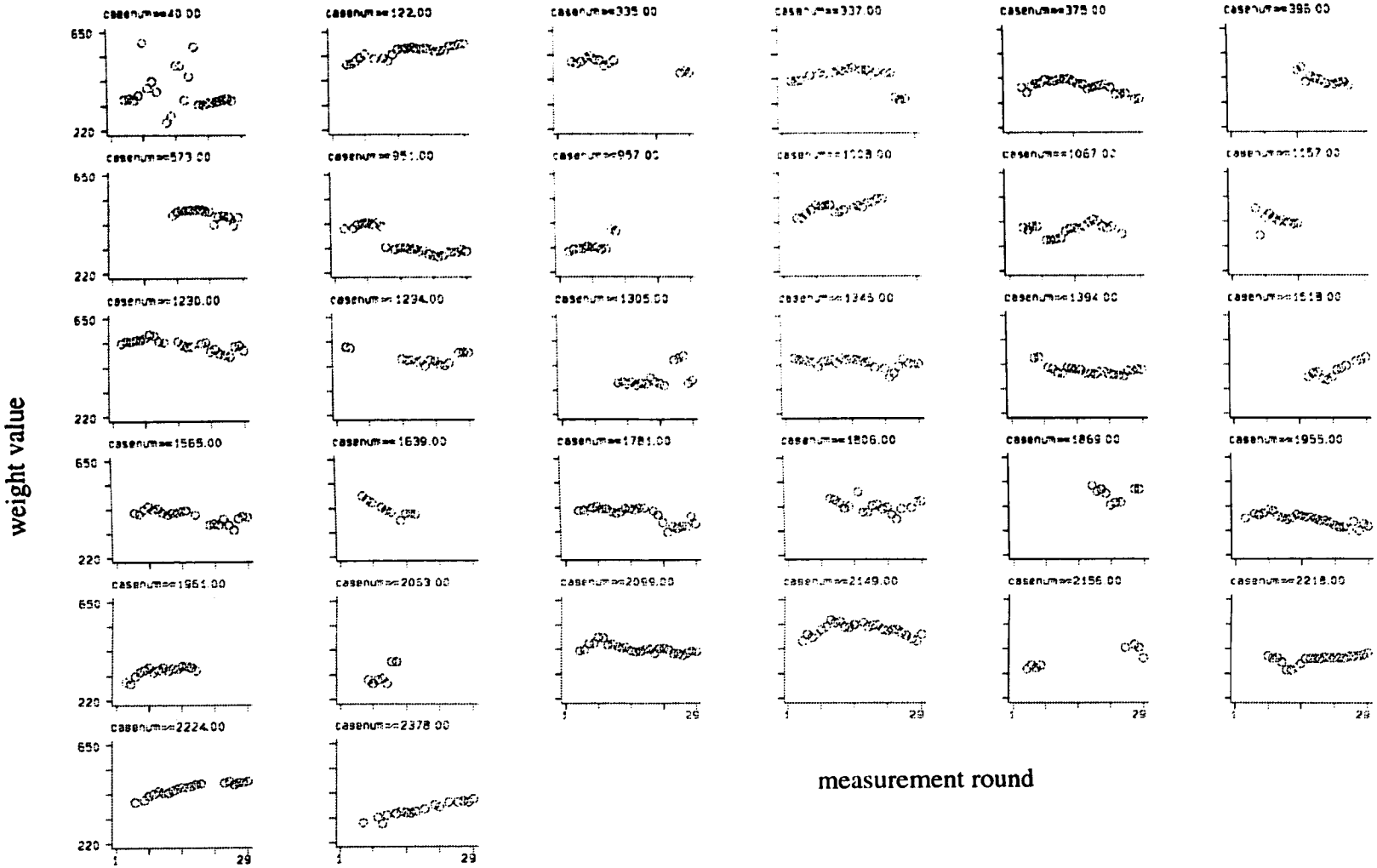
7. DID YOU KNOW OF ANY SOCIAL/FAMILIAL/MARITAL PROBLEM OF THE WOMAN IN THE MONTH(S) BEFORE DEATH ? | YES | NO | IF YES, DESCRIBE THE PROBLEM .....

8. WAS THAT WOMAN KNOW AS HAVING A PERMANENT (LONG TIME) PHYSICAL PROBLEM ? (LIKE MALFORMATION OF BONES, EPILEPSY, CHRONIC DEPRESSION, CHRONIC PELVIC INFECTION, TB,. SURGICAL OPERATION OF ABDOMEN.....) | YES | NO | IF YES, DESCRIBE .....

9. OTHER RELEVANT INFORMATION

10. CONCLUSION: | PREGNANT | NOT PREGNANT | SUSPECTED / UNLIKELY |

[Source: Fauveau, ed (1994)]

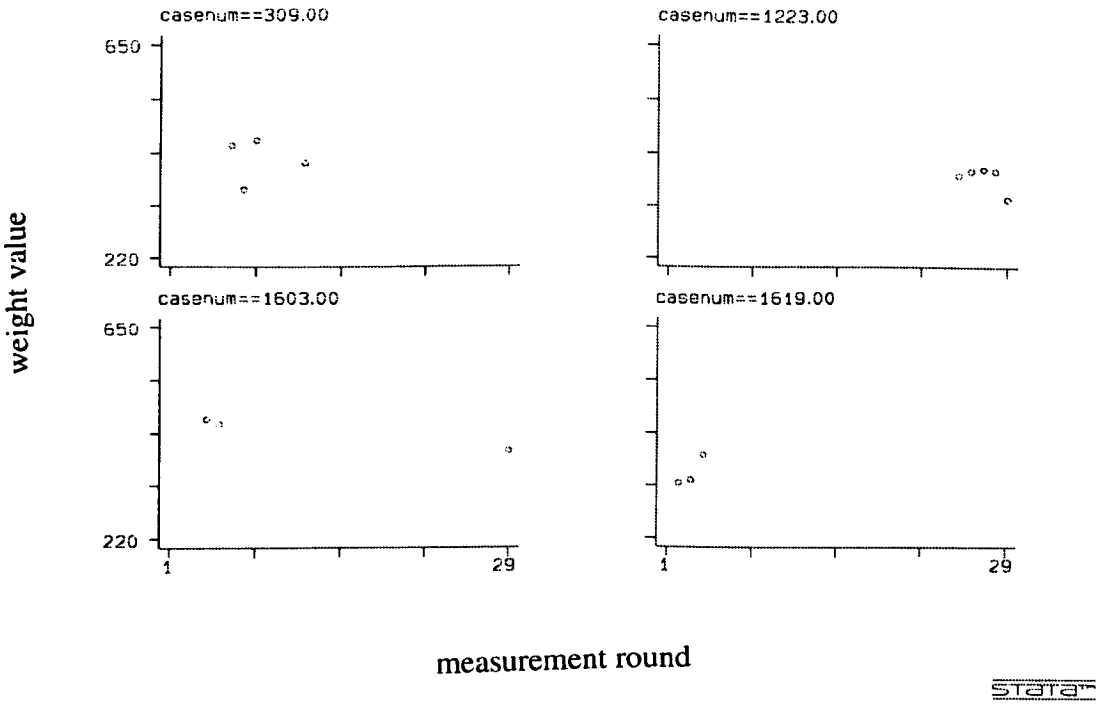


Appendix 4.1a

Women selected by weight algorithm for further validation

More than one measure of weight ± 4.8kg from their median

Subject no.	Pre-recoded median	New median after recode
40	35.5	35.3
1157	43.75	44.0
1305	36.15	36.0
1806	43.55	43.3

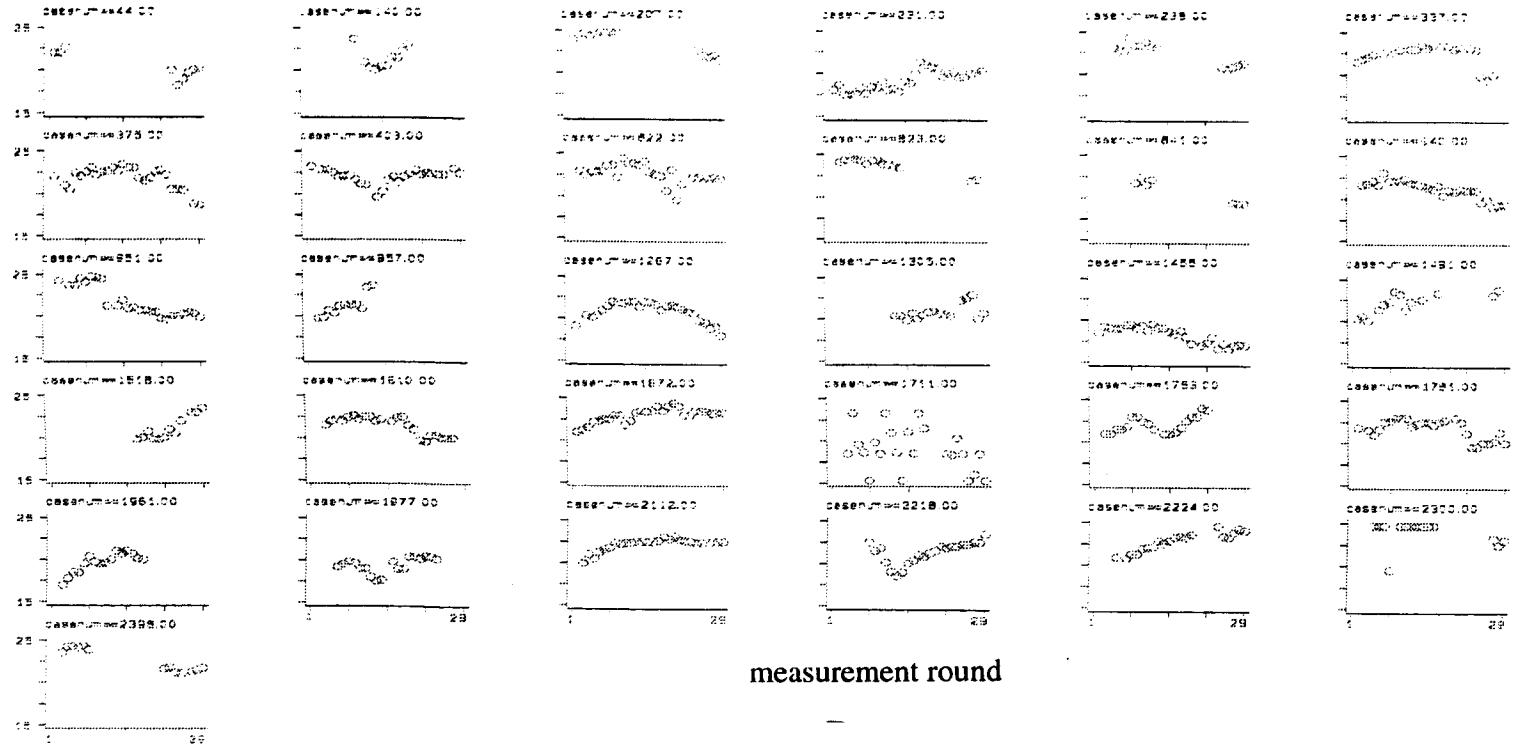


Subject no.	Pre-recoded median	New median after recode
309	43.2	45.0

Appendix 4.1b

Women selected by weight algorithm for further validation

Only one measure of weight ±4.8kg from their median but less than 6 repeated measurements

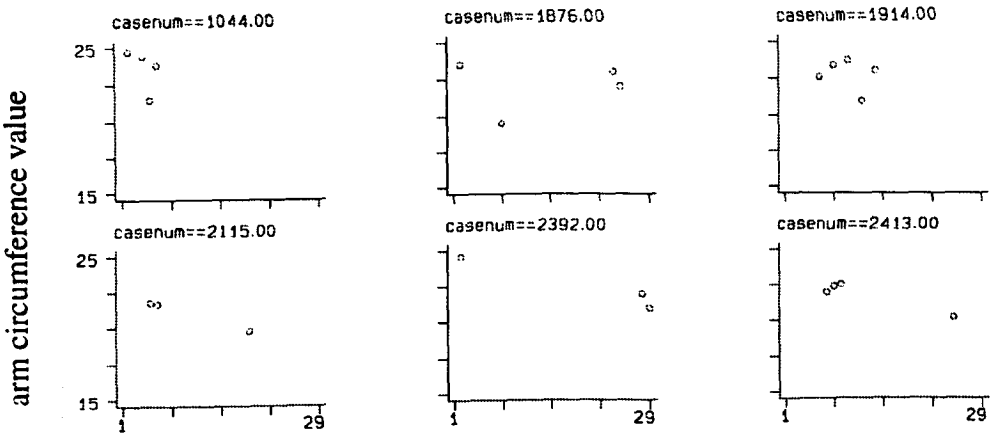


Appendix 4.2a

Women selected by arm circumference algorithm for further validation

More than one measure of arm circumference ±1.85cm from their median

Subject no.	Pre-recoded median	New median after recode
822	22.6	22.7
2300	24.7	24.7



Subject no.	Pre-recoded median	New median after recode
1044	24.1	24.4

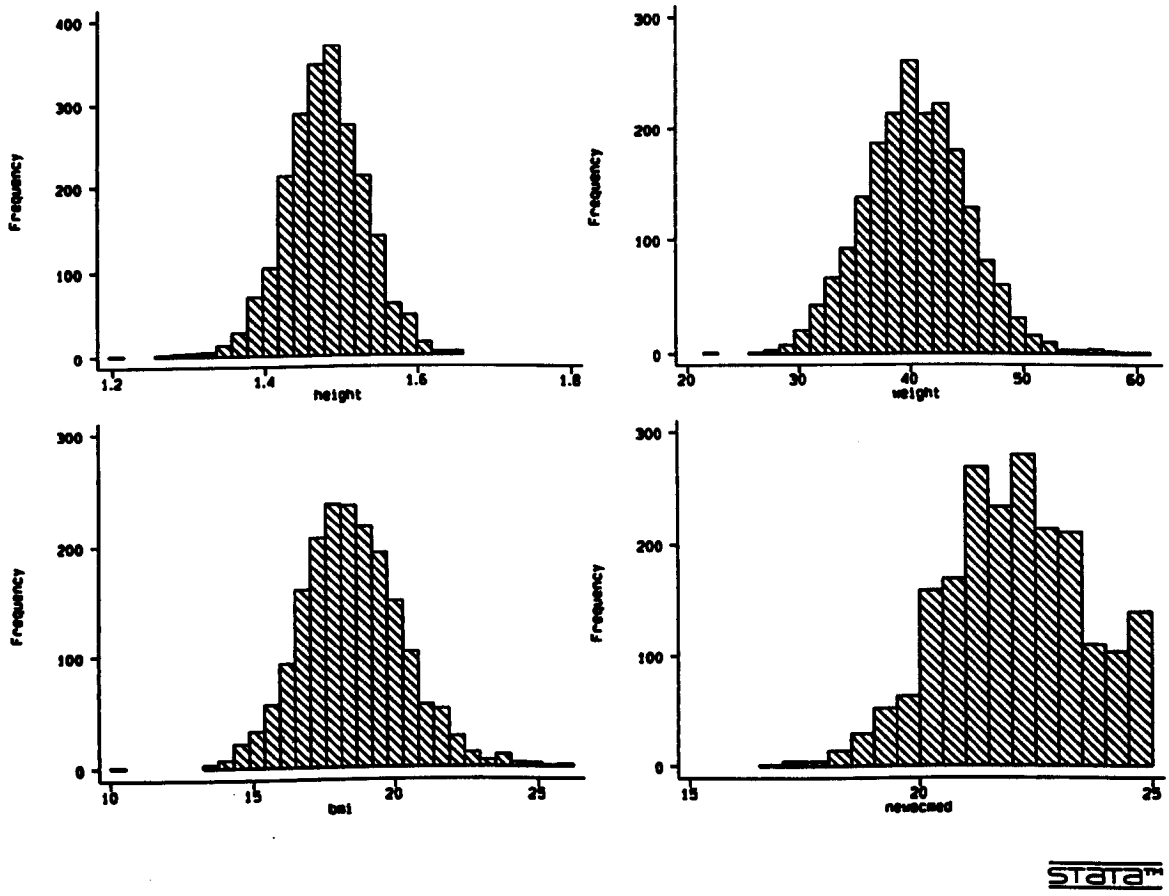
Appendix 4.2b

Women selected by arm circumference algorithm for further validation

Only one measure of arm circumference ±4.8kg from their median but less than 6 repeated measurements

APPENDIX 4.3

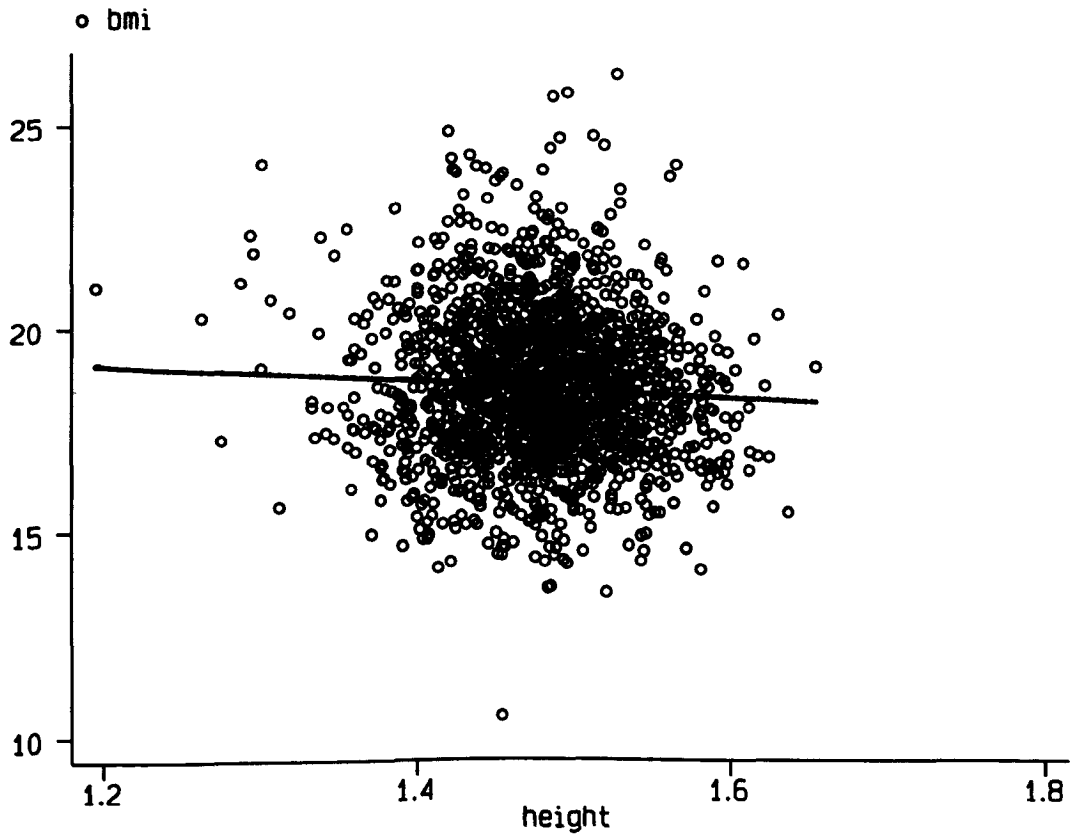
Frequency distributions for height (m), weight (kg), BMI and arm circumference (cm) for women in the thesis cohort (n=2,314)*.



* some women are missing measures of an anthropometric indicator, the number of women included in the graphs are: height (n=2,202), weight (n=1,999), BMI (n=1,888) and arm circumference (n=2,050).

APPENDIX 4.4

Graph of BMI and height, with the predicted regression line (n=1,816).



BMI (weight [kg]/height[m]^2) is an index where an individual's weight is adjusted for height. Although a significant correlation (p=0.01) is observed between BMI and height calculated for the same subjects, the correlation is extremely weak (R^2= 0.0034). In addition the slope of the line (ß=-2.03) suggests that the calculations of BMI in this cohort are not influenced by the level of height and are, therefore, independent of height. For every 25cm change in height there will be a change of around 0.5 BMI units. The regression statistics calculated by STATA Release for Windows are shown below:

```
regress bmi height

  Source |       SS       df       MS              Number of obs =    1888
---------+------------------------------           F(  1,  1886) =    6.51
   Model |  20.8165237     1  20.8165237           Prob > F      =  0.0108
Residual |  6028.42046  1886  3.19640533           R-squared     =  0.0034
---------+------------------------------           Adj R-squared =  0.0029
   Total |  6049.23698  1887  3.20574297           Root MSE      =  1.7878

------------------------------------------------------------------------------
         bmi |      Coef.   Std. Err.      t    P>|t|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
      height |  -2.026297   .7940173    -2.552   0.011    -3.583542   -.4690525
       _cons |   21.50007   1.174538    18.305   0.000     19.19654    23.80361
------------------------------------------------------------------------------
```

APPENDIX 5.1

The computer print out presented below were calculated using STATA Release for Windows. The four Cox's proportionate hazards models were calculated entering each anthropometric indicator separately and repeating the model for the different groupings of the variable (g1) or (g2).

Years is the number of follow-up years for each subject included in the model, **d(death)** identifies the outcome of interest as death, where a subject who dies at time, t, and is assigned the value 1.

In the following models socio-economic and demographic variables are coded as follows:

Variable name in model	Variable	Reference category (set at 1.00)	Comparison category for which HR is calculated
age	age in years	continuous	n/a
educ2	education	1+ year of schooling	no schooling
rel2	religion	Muslim	Hindu
dim4	dimension of house	0-187 sq ft	188+ sq ft
dist2	distance to water	<15 yards	15+ yards
items2	items owned	11+ items	0-10 items
occup3	husband's occupation	high status	low status

The reference categories for all anthropometric variables irrespective of which centile groupings were used, were the lowest centile.

Models A1 and A2 present the coefficient and the hazard ratio calculations for the model where only the socio-economic covariates are included.

Model A1 - no anthropometric indicator included, coefficient

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3, d(death)

Cox Regression -- entry time 0                  Number of obs =   1816
                                                chi2(8)       =  55.94
                                                Prob > chi2   = 0.0000
Log Likelihood = -711.89318                     Pseudo R2     = 0.0378

------------------------------------------------------------------------------
       years |
       death |      Coef.   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
         age |   .0483607   .0112717     4.290  0.000     .0262687    .0704528
    Ieduc2_1 |   1.427428   .4633569     3.081  0.002     .5192646    2.33559
     Irel2_1 |   .4426675   .2619611     1.690  0.091    -.0707668    .9561017
     Idim4_1 |  -.2828012    .237588    -1.190  0.234     -.748465    .1828627
     Idim4_2 |  -.3783706   .2716892    -1.393  0.164    -.9108717    .1541305
    Idist2_1 |   .3683045   .2357858     1.562  0.118    -.0938271    .8304361
    Iitems_1 |   .1236527   .2390723     0.517  0.605    -.3449203    .5922258
    Ioccup_1 |   .1447185    .222969     0.649  0.516    -.2922926    .5817296
------------------------------------------------------------------------------
```

Model A2 - no anthropometric indicator included, hazard ratios

```
. xi: cox years age i.educ2 i.rel2 i.dim5 i.dist2 i.items2 i.occup3, d(death) h
> r nolog

Cox Regression -- entry time 0                  Number of obs =   1816
                                                chi2(7)       =  55.83
                                                Prob > chi2   = 0.0000
Log Likelihood = -711.95074                     Pseudo R2     = 0.0377

------------------------------------------------------------------------------
       years |
       death | Haz. Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
         age |    1.04961   .0118352     4.294  0.000     1.026668    1.073065
    Ieduc2_1 |    4.17999   1.936481     3.087  0.002     1.685915    10.3637
     Irel2_1 |    1.562678   .409268     1.704  0.088     .9352733    2.610963
     Idim5_1 |   .7253079   .152908    -1.523  0.128      .479815    1.096405
    Idist2_1 |   1.457167   .3419889     1.604  0.109     .9198927    2.308243
    Iitems_1 |   1.150232   .2697611     0.597  0.551     .7263656    1.821444
    Ioccup_1 |   1.153057   .2570047     0.639  0.523     .7449488    1.78474
------------------------------------------------------------------------------
```

Model B1 - height (g1), coefficient

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.htgp1, d(
> death)

Cox Regression -- entry time 0                    Number of obs =   1816
                                                  chi2(10)      =  56.15
                                                  Prob > chi2   = 0.0000
Log Likelihood = -711.78845                       Pseudo R2     = 0.0379

------------------------------------------------------------------------------
  years |
  death |      Coef.   Std. Err.      z    P>|z|     [95% Conf. Interval]
--------+---------------------------------------------------------------------
    age |   .0481481   .0113014     4.260  0.000     .0259978    .0702984
 ieduc2_1 |  1.424078   .4634737     3.073  0.002     .5156863    2.33247
  irel2_1 |  .4274822   .2644161     1.617  0.106    -.0907639    .9457282
  idim4_1 | -.2796506     .23772    -1.176  0.239    -.7455732    .186272
  idim4_2 | -.3778387   .2729688    -1.384  0.166    -.9128476    .1571703
 idist2_1 |  .3701864   .2358631     1.569  0.117    -.0920968    .8324695
 iitems_1 |  .1225575   .2396203     0.511  0.609    -.3470897    .5922047
 ioccup_1 |  .1402969    .223614     0.627  0.530    -.2979785    .5785723
 ihtgp1_1 | -.1027723   .2939183    -0.350  0.727    -.6788417    .473297
 ihtgp1_2 | -.1979683   .4481107    -0.442  0.659    -1.076249    .6803126
------------------------------------------------------------------------------
```

Model B2 - height (g1), hazard ratios

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.htgp1, d(
> death) hr nolog

Cox Regression -- entry time 0                    Number of obs =   1816
                                                  chi2(10)      =  56.15
                                                  Prob > chi2   = 0.0000
Log Likelihood = -711.78845                       Pseudo R2     = 0.0379

------------------------------------------------------------------------------
  years |
  death | Haz. Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
--------+---------------------------------------------------------------------
    age |   1.049326   .0118588     4.260  0.000     1.026339    1.072828
 ieduc2_1 |  4.154026   1.925282     3.073  0.002     1.674787    10.30336
  irel2_1 |  1.533392   .4054535     1.617  0.106     .9132333    2.574688
  idim4_1 |  .7560478   .1797277    -1.176  0.239     .4744622    1.20475
  idim4_2 |  .6853411   .1870767    -1.384  0.166     .4013796    1.170195
 idist2_1 |  1.448004   .3415308     1.569  0.117     .9120168    2.298989
 iitems_1 |  1.130384    .270863     0.511  0.609     .7067419    1.80797
 ioccup_1 |  1.150615   .2572937     0.627  0.530     .7423173    1.78349
 ihtgp1_1 |  .9023324    .265212    -0.350  0.727     .5072042    1.605278
 ihtgp1_2 |  .8203959   .3676282    -0.442  0.659     .3408717    1.974495
------------------------------------------------------------------------------
```

Model C1 - height (g2), coefficient

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.htgp2, d(
> death)

Cox Regression -- entry time 0                    Number of obs =   1816
                                                  chi2(11)      =  56.40
                                                  Prob > chi2   = 0.0000
Log Likelihood = -711.66567                       Pseudo R2     = 0.0381

------------------------------------------------------------------------------
  years |
  death |      Coef.   Std. Err.      z    P>|z|     [95% Conf. Interval]
--------+---------------------------------------------------------------------
    age |   .0481798    .011289     4.268  0.000     .0260538    .0703058
 ieduc2_1 |  1.43538   .4634165     3.097  0.002     .5271006    2.34366
  irel2_1 |  .4334297   .2654371     1.633  0.102    -.0868174    .9536769
  idim4_1 | -.2771872   .2379916    -1.165  0.244    -.7436422    .1892679
  idim4_2 | -.3714359    .272321    -1.364  0.173    -.9051753    .1623034
 idist2_1 |  .3662334   .2358987     1.553  0.121    -.0961195    .8285863
 iitems_1 |  .1263951   .2392407     0.528  0.597    -.3425081    .5952983
 ioccup_1 |  .1519221   .2230894     0.681  0.496    -.2853251    .5891693
 ihtgp2_1 | -.0727829   .2773069    -0.262  0.793    -.6162945    .4707286
 ihtgp2_2 | -.1612555   .2866628    -0.563  0.574    -.7231043    .4005933
 ihtgp2_3 |  .0106724   .2845797     0.038  0.970    -.5470935    .5684383
------------------------------------------------------------------------------
```

Model C2 - height (g2), hazard ratios

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.htgp2, d(
> death) hr nolog

Cox Regression -- entry time 0                    Number of obs =   1816
                                                  chi2(11)      =  56.40
                                                  Prob > chi2   = 0.0000
Log Likelihood = -711.66567                       Pseudo R2     = 0.0381

------------------------------------------------------------------------------
  years |
  death | Haz. Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
--------+---------------------------------------------------------------------
    age |   1.049359   .0118462     4.268  0.000     1.026396    1.072836
 ieduc2_1 |  4.201242   1.946925     3.097  0.002     1.694013    10.4193
  irel2_1 |  1.542539    .409447     1.633  0.102     .9168445    2.595234
  idim4_1 |  .7579126   .1803769    -1.165  0.244     .4753793    1.208365
  idim4_2 |  .6897432   .1878316    -1.364  0.173     .404471     1.176217
 idist2_1 |  1.442292   .3402347     1.553  0.121     .9083555    2.290079
 iitems_1 |  1.13473    .2714737     0.528  0.597     .7099874    1.813572
 ioccup_1 |  1.16407    .2596916     0.681  0.496     .7517698    1.802491
 ihtgp2_1 |  .9298026   .2578407    -0.262  0.793     .5399415    1.60116
 ihtgp2_2 |  .8510746   .2439715    -0.563  0.574     .4852436    1.49271
 ihtgp2_3 |  1.01073    .2876331     0.038  0.970     .5786291    1.765508
------------------------------------------------------------------------------
```

Model D1 - weight (g1), coefficient

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.wtgp1, d(
> death)

Cox Regression -- entry time 0                    Number of obs =   1816
                                                  chi2(10)      =  63.08
                                                  Prob > chi2   = 0.0000
Log Likelihood = -708.32618                       Pseudo R2     = 0.0426

------------------------------------------------------------------------------
  years |
  death |      Coef.   Std. Err.      z    P>|z|     [95% Conf. Interval]
--------+---------------------------------------------------------------------
    age |   .0427695   .0114536     3.734  0.000     .0203208    .0652181
 ieduc2_1 |  1.42024   .4635969     3.064  0.002     .5116071    2.328873
  irel2_1 |  .3875547   .2629535     1.474  0.141    -.1278247    .9029342
  idim4_1 | -.2981307   .2374563    -1.256  0.209    -.7635366    .1672751
  idim4_2 | -.3922205   .2710785    -1.447  0.148    -.9235245    .1390835
 idist2_1 |  .3741804   .2354063     1.590  0.112    -.0872074    .8355682
 iitems_1 |  .1413466   .2391016     0.591  0.554    -.327284     .6099772
 ioccup_1 |  .1735388   .2224772     0.780  0.435    -.2625085    .6095861
 iwtgp1_1 | -.6970971   .2464201    -2.829  0.005    -1.180072    -.2141226
 iwtgp1_2 | -.611153    .4417361    -1.384  0.167    -1.47694     .2546337
------------------------------------------------------------------------------
```

Model D2 - weight (g1), hazard ratios

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.wtgp1, d(
> death) hr nolog

Cox Regression -- entry time 0                    Number of obs =   1816
                                                  chi2(10)      =  63.08
                                                  Prob > chi2   = 0.0000
Log Likelihood = -708.32618                       Pseudo R2     = 0.0426

------------------------------------------------------------------------------
  years |
  death | Haz. Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
--------+---------------------------------------------------------------------
    age |   1.043697   .0119541     3.734  0.000     1.020529    1.067392
 ieduc2_1 |  4.138115   1.918417     3.064  0.002     1.66797     10.26637
  irel2_1 |  1.473374   .3874288     1.474  0.141     .8800077    2.466831
  idim4_1 |  .7422043   .1762411    -1.256  0.209     .4660154    1.182079
  idim4_2 |  .6755551   .1831284    -1.447  0.148     .3971169    1.14922
 idist2_1 |  1.453799   .3422335     1.590  0.112     .916487     2.306124
 iitems_1 |  1.151824    .275403     0.591  0.554     .720879     1.84039
 ioccup_1 |  1.189507   .2646381     0.780  0.435     .7691198    1.83967
 iwtgp1_1 |  .498029    .1227243    -2.829  0.005     .3072568    .8072494
 iwtgp1_2 |  .5427247   .2397411    -1.384  0.167     .2283354    1.289989
------------------------------------------------------------------------------
```

Model E1 - weight (g2), coefficient

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.wtgp2, d(
> death)

Cox Regression -- entry time 0                  Number of obs =   1816
                                                chi2(11)      =  61.66
                                                Prob > chi2   = 0.0000
Log Likelihood = -709.03367                     Pseudo R2     = 0.0417

------------------------------------------------------------------------------
      years |
      death |      Coef.   Std. Err.      z    P>|z|     [95% Conf. Interval]
------------+-----------------------------------------------------------------
        age |   .0461898   .0116219     3.974  0.000     .0234114    .0689682
   educ2_1 |   1.444772   .4630686     3.120  0.002     .5371737    2.352369
   rel2_1 |   .4244183   .2647343     1.603  0.109    -.0944514    .943288
   dim4_1 |  -.2942557   .2378391    -1.237  0.216    -.7604119    .1719004
   dim4_2 |  -.3999764    .270683    -1.478  0.139    -.9305053    .1305525
  dist2_1 |   .3529785   .2353304     1.500  0.134    -.1082606    .8142177
  items_1 |    .143746   .2384953     0.603  0.547    -.3236962    .6111881
  occup_1 |   .1536791   .2228703     0.690  0.490    -.2831387    .5904968
  wtgp2_1 |  -.3940706   .2687592    -1.466  0.143     -.920829    .1326878
  wtgp2_2 |  -.6187571   .2940939    -2.104  0.035    -1.195171   -.0423436
  wtgp2_3 |  -.1061599   .2786358    -0.381  0.703     -.652276    .4399562
------------------------------------------------------------------------------
```

Model E2 - weight (g2), hazard ratio

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.wtgp2, d(
> death) hr nolog

Cox Regression -- entry time 0                  Number of obs =   1816
                                                chi2(11)      =  61.66
                                                Prob > chi2   = 0.0000
Log Likelihood = -709.03367                     Pseudo R2     = 0.0417

------------------------------------------------------------------------------
      years |
      death | Haz. Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
------------+-----------------------------------------------------------------
        age |   1.047273   .0121713     3.974  0.000     1.023688    1.071402
   educ2_1 |   4.240883    1.96382     3.120  0.002     1.711164    10.51044
   rel2_1 |   1.528701   .4046996     1.603  0.109     .9098719    2.568413
   dim4_1 |   .7450859   .1772106    -1.237  0.216     .4674739     1.18756
   dim4_2 |   .6703359   .1814485    -1.478  0.139     .3943544    1.139458
  dist2_1 |   1.423301   .3349459     1.500  0.134     .8973937    2.257409
  items_1 |   1.154591   .2753645     0.603  0.547      .72347    1.842619
  occup_1 |   1.166117   .2598927     0.690  0.490     .7534153    1.804885
  wtgp2_1 |   .6743064   .1812261    -1.466  0.143     .3981888    1.141893
  wtgp2_2 |   .5386135   .1584029    -2.104  0.035     .3026523    .9585404
  wtgp2_3 |   .8992808   .2505718    -0.381  0.703     .5208589    1.552639
------------------------------------------------------------------------------
```

Model F1 - BMI (g1), coefficient

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.bmigp1, d
> (death)

Cox Regression -- entry time 0                  Number of obs =   1816
                                                chi2(10)      =  65.39
                                                Prob > chi2   = 0.0000
Log Likelihood = -707.17086                     Pseudo R2     = 0.0442

------------------------------------------------------------------------------
      years |
      death |      Coef.   Std. Err.      z    P>|z|     [95% Conf. Interval]
------------+-----------------------------------------------------------------
        age |   .0396732   .0116358     3.410  0.001     .0168676    .0624789
   educ2_1 |   1.431971    .463008     3.093  0.002     .5244916     2.33945
   rel2_1 |   .4126898   .2609949     1.581  0.114    -.0988507    .9242304
   dim4_1 |  -.2877614   .2378946    -1.210  0.226    -.7540262    .1785034
   dim4_2 |   -.383755   .2709826    -1.416  0.157    -.9148712    .1473612
  dist2_1 |   .3655561   .2358319     1.550  0.121    -.0966659    .8277781
  items_1 |   .1230065   .2387181     0.515  0.606    -.3448723    .5908853
  occup_1 |   .1914467   .2229946     0.859  0.391    -.2456147    .628508
  bmigp_1 |  -.8084672    .248727    -3.250  0.001    -1.295963   -.3209712
  bmigp_2 |  -.5914415   .4032241    -1.467  0.142    -1.381746    .1988632
------------------------------------------------------------------------------
```

Model F2 - BMI (g2), hazard ratio

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.bmigp1, d
> (death) hr nolog

Cox Regression -- entry time 0                  Number of obs =   1816
                                                chi2(10)      =  65.39
                                                Prob > chi2   = 0.0000
Log Likelihood = -707.17086                     Pseudo R2     = 0.0442

------------------------------------------------------------------------------
      years |
      death | Haz. Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
------------+-----------------------------------------------------------------
        age |   1.040471   .0121067     3.410  0.001     1.017011    1.064472
   educ2_1 |   4.186942   1.938587     3.093  0.002      1.6896    10.37552
   rel2_1 |   1.510876    .394331     1.581  0.114     .9058779    2.519928
   dim4_1 |   .7499405   .1784068    -1.210  0.226     .4704685    1.195427
   dim4_2 |   .6812983    .18462    -1.416  0.157     .4005682    1.158772
  dist2_1 |   1.441315   .3399081     1.550  0.121     .9078593    2.288229
  items_1 |   1.130892   .2699643     0.515  0.606     .7083108    1.805586
  occup_1 |    1.211    .2700465     0.859  0.391     .7822236    1.874811
  bmigp_1 |   .4455405    .110818    -3.250  0.001     .2736342    .7254441
  bmigp_2 |   .5535288   .2231961    -1.467  0.142     .2511396    1.220015
------------------------------------------------------------------------------
```

Model G1 - BMI (g2), coefficient

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.bmigp2, d
> (death)

Cox Regression -- entry time 0                  Number of obs =   1816
                                                chi2(11)      =  62.84
                                                Prob > chi2   = 0.0000
Log Likelihood =  -708.4479                     Pseudo R2     = 0.0425

------------------------------------------------------------------------------
      years |
      death |      Coef.   Std. Err.      z    P>|z|     [95% Conf. Interval]
------------+-----------------------------------------------------------------
        age |   .0444293   .0116707     3.807  0.000     .0215552    .0673034
   educ2_1 |   1.441163   .4636761     3.108  0.002     .5323748    2.349952
   rel2_1 |   .4200997   .2610956     1.609  0.108    -.0916382    .9318377
   dim4_1 |  -.2893988   .2374908    -1.219  0.223    -.7548722    .1760746
   dim4_2 |  -.3972103   .2711318    -1.465  0.143    -.9286188    .1341982
  dist2_1 |   .3502692   .2358125     1.485  0.137    -.1119148    .8124532
  items_1 |   .1495757   .2386502     0.627  0.531    -.3181701    .6173216
  occup_1 |   .1457261   .2226547     0.654  0.513    -.290669     .5821213
  bmigp_1 |  -.1923979   .2573283    -0.748  0.455    -.6967521    .3119563
  bmigp_2 |  -.7728665   .3198789    -2.416  0.016    -1.399818   -.1459154
  bmigp_3 |  -.1223104   .2753469    -0.444  0.657    -.6619804    .4173595
------------------------------------------------------------------------------
```

Model G2 - BMI (g2), hazard ratio

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.bmigp2, d
> (death) hr nolog

Cox Regression -- entry time 0                  Number of obs =   1816
                                                chi2(11)      =  62.84
                                                Prob > chi2   = 0.0000
Log Likelihood =  -708.4479                     Pseudo R2     = 0.0425

------------------------------------------------------------------------------
      years |
      death | Haz. Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
------------+-----------------------------------------------------------------
        age |   1.045431   .0122009     3.807  0.000     1.021789    1.06962
   educ2_1 |   4.225608   1.959313     3.108  0.002     1.702972    10.48506
   rel2_1 |   1.522113   .3974171     1.609  0.108     .9124352    2.539171
   dim4_1 |   .7487136   .1778126    -1.219  0.223     .4700707    1.192527
   dim4_2 |   .6721927   .1822528    -1.465  0.143     .3950991    1.143619
  dist2_1 |    1.41945    .334724     1.485  0.137     .8941204    2.253429
  items_1 |   1.161341   .2771544     0.627  0.531     .7274791    1.853956
  occup_1 |   1.156879   .2575846     0.654  0.513     .7477631    1.789831
  bmigp_1 |   .8249786   .2122903    -0.748  0.455     .4982008    1.366095
  bmigp_2 |   .4616878   .1476842    -2.416  0.016      .246642    .8642308
  bmigp_3 |   .8848736   .2436472    -0.444  0.657     .5158288    1.517948
------------------------------------------------------------------------------
```

Model H1 - arm circumference (g1), coefficient

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.acgp1, d(
> death)

Cox Regression -- entry time 0                    Number of obs =   1816
                                                  chi2(10)      =  60.14
                                                  Prob > chi2   = 0.0000
Log Likelihood = -709.79785                       Pseudo R2     = 0.0406

------------------------------------------------------------------------------
  years |
  death |      Coef.   Std. Err.      z    P>|z|     [95% Conf. Interval]
--------+---------------------------------------------------------------------
    age |   .0452195   .0113622     3.980  0.000     .0229499    .0674891
leduc2_1|   1.417266   .4632795     3.059  0.002     .5092553    2.325278
 lrel2_1|   .4154757   .2625164     1.583  0.113    -.099047     .9299984
 ldim4_1|  -.2936011   .2379158    -1.234  0.217    -.7599074    .1727053
 ldim4_2|  -.3963212   .2716811    -1.459  0.145    -.9288064    .1361639
ldist2_1|   .3781403   .2357482     1.604  0.109    -.0839176    .8401983
litems_1|   .1287623   .2394124     0.538  0.591    -.3404773    .598002
loccup_1|   .1471756   .2226502     0.661  0.509    -.2892107    .583562
lacgp1_1|  -.4720608   .2619339    -1.802  0.072    -.9854418    .0413203
lacgp1_2|  -.8288386   .470521     -1.762  0.078    -1.751043    .0933656
------------------------------------------------------------------------------
```

Model H2 - arm circumference (g1), hazard ratio

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.acgp1, d(
> death) hr nolog

Cox Regression -- entry time 0                    Number of obs =   1816
                                                  chi2(10)      =  60.14
                                                  Prob > chi2   = 0.0000
Log Likelihood = -709.79785                       Pseudo R2     = 0.0406

------------------------------------------------------------------------------
  years |
  death | Haz. Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
--------+---------------------------------------------------------------------
    age |   1.046257   .0118878     3.980  0.000     1.023215    1.069819
leduc2_1|   4.125827   1.911411     3.059  0.002     1.664052    10.22952
 lrel2_1|   1.515091   .3977364     1.583  0.113     .9057001    2.534505
 ldim4_1|   .7455739   .1773838    -1.234  0.217     .4677097    1.188516
 ldim4_2|   .6727905   .1827845    -1.459  0.145     .3950249    1.14587
ldist2_1|   1.459568   .3440904     1.604  0.109      .919507    2.316826
litems_1|    1.13742   .2723124     0.538  0.591     .7114306    1.818482
loccup_1|   1.158557   .257953      0.661  0.509     .7488544    1.792412
lacgp1_1|   .6237156   .1633723    -1.802  0.072     .3732743    1.042186
lacgp1_2|   .436556    .2054088    -1.762  0.078     .1735928    1.097863
------------------------------------------------------------------------------
```

Model I1 - arm circumference (g2), coefficient

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.acgp2, d(
> death)

Cox Regression -- entry time 0                    Number of obs =   1816
                                                  chi2(11)      =  57.32
                                                  Prob > chi2   = 0.0000
Log Likelihood = -711.20607                       Pseudo R2     = 0.0387

------------------------------------------------------------------------------
  years |
  death |      Coef.   Std. Err.      z    P>|z|     [95% Conf. Interval]
--------+---------------------------------------------------------------------
    age |   .0468444   .0113457     4.129  0.000     .0246071    .0690816
leduc2_1|    1.41399   .4634749     3.051  0.002     .5055963    2.322385
 lrel2_1|   .4481113   .2619508     1.711  0.087    -.0653028    .9615254
 ldim4_1|  -.2807437   .2375201    -1.182  0.237    -.7462745    .1847871
 ldim4_2|  -.3768828   .2712715    -1.389  0.165    -.9085651    .1547995
ldist2_1|   .3664916   .2358462     1.554  0.120    -.0957585    .8287418
litems_1|   .1248864   .2388825     0.523  0.601    -.3433146    .5930875
loccup_1|   .1535149   .2231021     0.688  0.491    -.2837573    .590787
lacgp2_1|  -.0410884   .2573506    -0.160  0.873    -.5454862    .4633095
lacgp2_2|  -.2370285   .2872827    -0.825  0.409    -.8000923    .3260353
lacgp2_3|  -.2746436   .2861772    -0.960  0.337    -.8355406    .2862535
------------------------------------------------------------------------------
```

Model I2 - arm circumference (g2), hazard ratios

```
. xi: cox years age i.educ2 i.rel2 i.dim4 i.dist2 i.items2 i.occup3 i.acgp2, d(
> death) hr nolog

Cox Regression -- entry time 0                    Number of obs =   1816
                                                  chi2(11)      =  57.32
                                                  Prob > chi2   = 0.0000
Log Likelihood = -711.20607                       Pseudo R2     = 0.0387

------------------------------------------------------------------------------
  years |
  death | Haz. Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
--------+---------------------------------------------------------------------
    age |   1.047959   .0118899     4.129  0.000     1.024912    1.071524
leduc2_1|   4.112333   1.905963     3.051  0.002     1.657974    10.19997
 lrel2_1|   1.565353   .4100454     1.711  0.087     .9367837    2.615683
 ldim4_1|   .7552219   .1793804    -1.182  0.237     .4741296    1.202962
 ldim4_2|   .6859965   .1860913    -1.389  0.165     .4031022    1.167424
ldist2_1|   1.442664   .340247     1.554  0.120     .9086834    2.290435
litems_1|    1.13302   .2706586     0.523  0.601      .709415    1.809567
loccup_1|   1.165925   .2601204     0.688  0.491     .7529494    1.805409
lacgp2_1|   .9597443   .2469908    -0.160  0.873     .5795599    1.589325
lacgp2_2|   .7889688   .2266571    -0.825  0.409     .4492875    1.385464
lacgp2_3|   .7598429   .2174497    -0.960  0.337      .43364     1.33143
------------------------------------------------------------------------------
```