Anthropometry, morbidity and mortality in rural Sarawak.

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ABSTRACT

This thesis is concerned with the use of anthropometric measurements and indices as tools to assess current adult nutritional status. The practical characteristics, distribution and determinants of the anthropometric variables and also their relationships to morbidity and mortality were examined in a rural population in Sarawak, East Malaysia.

The study made use of baseline anthropometric data obtained on 1047 adults in 1990. 94% of the survivors since 1990 were re-measured and re-interviewed in 1996. Verbal autopsies were applied to the closest relatives of the non-survivors since 1990.

The body mass index (kg/m^2) and mid-upper arm circumference better fulfilled the characteristics required for an ideal index of nutritional status in adults than any other anthropometric measurements or indices. Both the body mass index and mid-upper arm circumference were highly correlated with body energy stores and were relatively independent of height and shape. Negative associations of the body mass index and mid-upper arm circumference with mortality were observed when overweight ($>=25kg/m^2$) individuals were excluded from the analyses. Age altered the strength of these associations. Low mid-upper arm circumference and body mass index were associated with increased risks of self-reporting episodes of fever and epigastric or respiratory problems not associated with hypertension in a cross-sectional analysis of the non-obese ($<=30kg/m^2$) population. Amongst the non-obese section of the population who reported being healthy at the baseline, low mid-upper arm circumference was also associated with increased risks of self-reporting subsequent episodes of fever and epigastric or respiratory problems not associated with hypertension.

Logistic regression found that the body mass index cut-off point of $18.5kg/m^2$ was associated with increased risks of mortality when overweight individuals were excluded and early mortality was discounted. Amongst the non-obese section of the population who reported being healthy at the baseline, the body mass index cut-off point of $18.5kg/m^2$ was associated with increased risks of self-reporting subsequent episodes of fever and epigastric or respiratory problems not associated with hypertension. Thus this study provides support for the hypothesis that the body mass index may be useful in the diagnosis of chronic energy deficiency. However, the results suggest that a scheme which assesses current nutritional status in adults using anthropometric indices should consider employing distinct cut-offs for older adults.

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ABBREVIATIONS AND CONVENTIONS

AED	Acute energy deficiency
AMBA	Arm and muscle bone area
BMR	Basal metabolic rate
BMI	Body mass index
CED	Chronic energy deficiency
CI	Cormic Index
FM	Fat mass
FFM	Fat-free mass
Hb	Haemoglobin
HH	Household
HR	Hazard ratio
HT	Hypertension
MUAC	Mid-upper arm circumference
OR	Odds ratio
PAL	Physical activity level
RR	Relative risk
RW	Relative weight
SES	Socio-economic status
TB	Tuberculosis
p	
chi	
anova	

CHAPTER ONE

INTRODUCTION

This project is concerned with adult energy nutrition. Thus where the description "well-nourished" is employed it means well-nourished in terms of energy. The health of a well nourished individual will not be compromised by insufficient energy stores; a healthy individual is defined as being free of disease and able to perform his social roles.

Conversely, an individual who is suffering from energy deficiency will not fulfil all the criteria of health. Such an individual could be suffering either from acute energy deficiency (AED) or chronic energy deficiency (CED). AED is regarded, a priori, as a state of negative energy balance, i.e. energy intake is less than energy expenditure so that, despite changes in metabolic efficiency or physical activity patterns, there are progressive losses of body weight and body energy stores (Shetty & James, 1994). CED is defined as a steady state where energy intake equals energy output, however, body energy stores are deemed to be below the acceptable range of normality and an impairment of health or function may be the result. This project is especially concerned with CED in adults in the developing world.

1.1 Ideal Measurements of Nutritional Status

When nutritional status is assessed in a clinical situation a battery of indices are used. This is probably the "gold standard" technique of assessing the nutritional status of an individual. However, this project does not aim to assess indices of CED in a clinical setting, rather the aim is to assess measurements which can be used to evaluate CED in a population in the field. Measuring a battery of indices (especially biochemical ones) may not be practical under such circumstances as it is time-consuming and expensive to obtain and interpret all the measurements required. Moreover, this study aims to evaluate measurements of CED which can be found by looking at a population only once. Repeated measurements would probably be useful to assess a steady state of energy balance, but would not always be easy to obtain in field settings (Immink et al, 1992).

Ideally, a single, non-biochemical, index could be used to assess CED. Such an index would have to meet the following requirements -

1. It must correlate well with body energy stores, preferably measured by the "gold standard" of underwater weighing.

2. As CED is defined as a low level of nutrition at which health or function may be impaired, thus any index used to measure CED must be correlated with health or functional outcomes.
3. It must be simple to obtain and interpret in the field. It must also be accurate (close to the true value), valid (represent what it is thought to represent) and precise (repeatable).

An ideal index of nutrition would fulfil all the requirements described above in any population, irrespective of the individuals' age, sex and ethnicity. This project aims to evaluate adult indices of nutrition in terms of these requirements.

1.2 Historical measurements of nutritional status

Historically, attempts to assess the nutritional status of a population have been made by measuring food intake and determining its adequacy in comparison to assumed or measured energy requirements (Shetty & James, 1994). This method may be unsatisfactory for two reasons. Firstly, energy requirements are complicated to estimate as they vary not only according to an individual's sex, age and body size, but also according to the amount of physical work or exercise undertaken. Secondly, habitual energy intake is difficult to measure. The presence of a researcher is thought to influence the subjects' diet and, furthermore, it is time consuming and expensive to obtain accurate dietary intake data on a large number of people.

In 1992 (Ferro-Luzzi et al) it was proposed that anthropometric indices alone could be used to diagnose CED in adults. Anthropometric indices are used as proxy measurements to rate availability of energy for growth and function. The most commonly used anthropometric indices - height, weight, limb circumferences and skinfold measurements - are relatively simple and inexpensive to obtain and do not use invasive techniques.

Anthropometric indices underlie various schemes currently being used to quantify child under-nutrition at population level (e.g. the Waterlow classification). Recent prospective studies confirm their value in predicting childhood mortality (Pelletier, 1991). However, children have an extra source of energy output - growth (the formation of new tissue). Thus although anthropometric indices represent the sum of energy intake and output the indices must be interpreted differently in adults and children.

1.3 An Ideal Adult Anthropometric Index of Nutrition

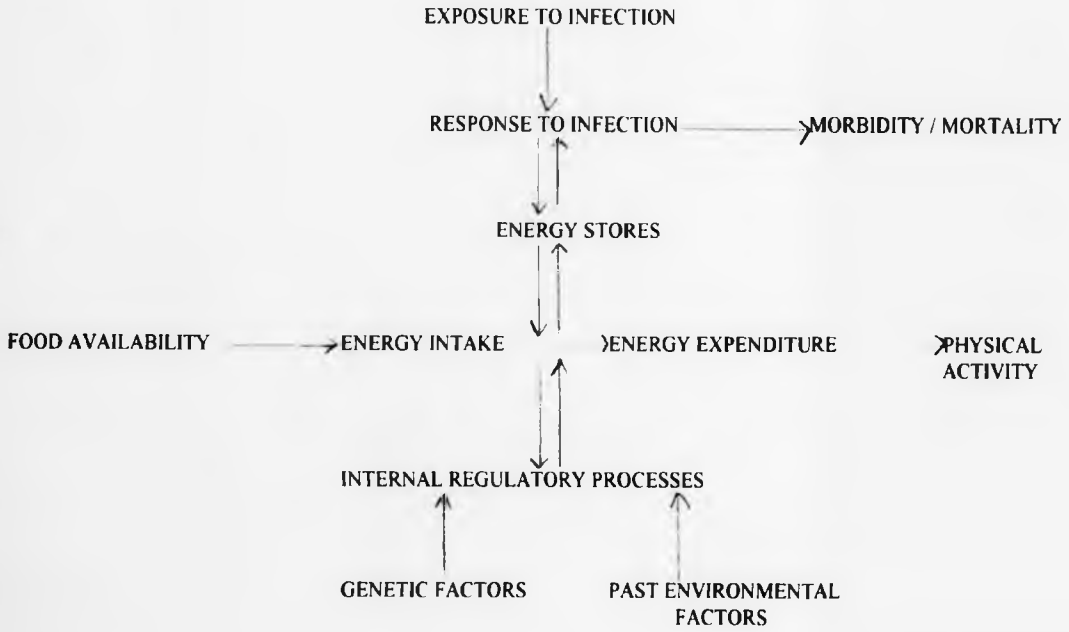
Anthropometric indices represent the sum of both internal and external influences on energy intake and energy output. Internal factors include the regulation of energy intake and the regulation of energy metabolism; external factors include food available for consumption by the individual, mandatory physical activity, socially desired physical activity, infection as a cause of anorexia, and infection (fever) as a cause of increased energy expenditure (Beaton et al, 1990). See Figure 1.1.

Anthropometric measurements are influenced by genetic factors as well as both past and current environmental factors. It is important to try to distinguish whether distinct populations have different mean values of anthropometric measurements because of past environmental or genetic factors; or if the differences are due to current environmental factors. The arguments and discussions which arise from these considerations are similar to those discussed when attempts were made to assess whether all children, given similar environmental conditions, would grow at the same rate (Habicht et al, 1982; Goldstein & Tanner, 1980; Van Loon et al, 1986; Waterlow, 1992).

As adult height is largely determined by an individual's genotype and childhood nutritional experience (Eveleth and Tanner, 1976; Jelliffe & Jelliffe, 1989), it follows that if an index is to reflect current nutritional status in adults it must be independent of height.

The term "body energy stores" generally refers to body fat stores, however fat-free mass (FFM) can also be oxidised to provide energy. Loss of body weight is normally accompanied by a reduction in both fat mass (FM) and FFM (Norgan, 1994b, Shetty, 1995; Keys et al, 1950). However, the changes in body composition (i.e. proportion of FM and FFM) do not bear a simple relation to the changes in body mass (Shetty, 1995). In general, more FM is lost than FFM and hence the percentage of body weight made up of fat will decrease more than the percentage of FFM will decrease. The size of the losses of the two components depends both on the initial levels of body fat and the amount of weight lost (Elia, 1992). In this project an attempt will be made to discuss the relationship between the various indices of nutrition and energy stores in terms of both FM and FFM.

Figure 1.1 *A portrayal of the association between energy stores (anthropometrical measurements) and the processes underlying energy balance in adults (after Beaton et al. 1990)*



To summarise, if an anthropometric index is to be used to assess current adult nutritional status it must ideally be:-

(i) highly correlated with body energy stores
(ii) independent of body height
(iii) independent of sex, age and ethnic origin
(iv) highly correlated with health and/or functional outcomes
(v) fulfil all the practical characteristics (i.e.: be simple to measure and interpret, accurate, valid and precise)

This study will assess the suitability of various anthropometric measures and indices. These include height, weight and skinfold thicknesses. Skinfold thicknesses can be transformed to proxy measures of FM and FFM using various equations (Durnin & Wormersley, 1974). However, the project focuses on two anthropometric indices in particular - the Body Mass Index (BMI) and mid-upper arm circumference (MUAC). The BMI is a weight-for-height index and is calculated as weight divided by height squared (weight/height^2); it was first introduced by Quetelet (1870) in order to eliminate the confounding effects of height on weight. In normal adults, the ratio of weight to the square of height is roughly constant, and a person with a low BMI is under weight-for-height (Quetlet, 1870). The MUAC is a measure of the circumference of the midpoint of the upper left arm. The arm contains subcutaneous fat and muscle; a decrease in mid-upper arm circumference may therefore reflect either a reduction in muscle mass, a reduction in subcutaneous tissue, or both.

1.4 Anthropometry and Chronic Energy Deficiency in adults

1.4.1 CED defined by the BMI

In 1992 Ferro-Luzzi et al proposed a simplified approach to the diagnosis of CED. It was suggested that CED in adults may be diagnosed using the BMI alone. Specific cut-off points of the BMI have been proposed below which an individual is deemed to be of a certain CED grade. The expectation is that at a BMI<18.5 kg/m^2 an individual's health or function will be impaired and will become progressively more impaired as his BMI decreases further. Table 1.1 shows the classification of adult CED and overweight categories by BMI status.

Table 1.1: Classification of adult CED and overweight by BMI Status

BMI (kg/m^2)	Classification
=<15.9	CED grade III
16.0-16.9	CED grade II
17.0-18.4	CED grade I
18.5-24.9	Normal
25.0-29.9	Overweight
30.0-39.9	Obese
40.0>=	Severe obesity

Sources: Ferro-Luzzi et al. 1992 and Garrow, 1988

There is no empirically defensible single BMI cut-off point which may be used to diagnose CED (James et al. 1988) and the cut-off points suggested above must be seen as the outcome of a series of compromises (James & Fransois, 1994a). The following factors were taken into consideration - (a) the distribution of the BMI in a healthy population; (b) the cut-off point for the upper limit of an acceptable BMI range above which risk of mortality increases; (c) the level of BMI below which risks of morbidity start to increase or maternal reproductive success appears to be compromised; and (d) the level of BMI below which work capacity may be reduced.

The authors of the BMI cut-off scheme have chosen 18.5 kg/m^2 as a universal reference point for CED. The choice of the same BMI cut-off values for men and women is a notable feature of this scheme which merits discussion. Given that women naturally have more body fat than men, a classification of CED based exclusively on body fat would mean that women can have a lower BMI than men to obtain the same proportion of energy reserves per unit height (Shetty & James, 1994). However, women require additional energy to sustain pregnancy, lactation and menstruation and many women in the developing world are required to engage in demanding agricultural work as well as child raising and household care. An additional advantage is the simplicity of having one criterion for all adults for nutritional surveillance and monitoring purposes.

1.4.2 CED diagnosed by the MUAC

It has recently been proposed that MUAC measurements could play a role in diagnosing CED in adults (James et al. 1994b). James et al have proposed that MUAC could be used in addition to

the BMI in order to differentiate further between adults who are healthy and have a low BMI and those who are unhealthy with a low BMI. The MUAC has been added to account for peripheral wasting in adults with a marginally low BMI. It is hoped that use of both the BMI and MUAC will prevent thin but healthy individuals form being diagnosed as CED.

The cut-off points for defining low MUAC, which are sex specific, were selected after a study examining the relationship of MUAC to the BMI in selected regions of the developing world (James et al. 1994b). In this scheme adults with a BMI of 17.0-18.4 and a MUAC above the cut-off point may be considered normal in the developed world, but possibly 'at risk' in the less developed world. Table 1.2 shows the classification of CED based on both BMI and MUAC (adapted from James et al. 1994b).

Table 1.2. The classification of CED based on both BMI and MUAC

MUAC (cm)	BMI (kg/m^2)			
	<16	16-16.9	17.0-18.4	>18.5
<23.0 (men) <22.0 (women)	III	II	I	? Normal
>23.0 (men) >22.0 (women)	II	I	Normal, but vulnerable *	Normal

* In third world conditions only

In this study a further definition of CED diagnosed by use of the MUAC alone will also be assessed. A male adult will be defined as CED if his MUAC is less than 23.0cm and a female adult will be defined as CED if her MUAC is less than 22.0cm. Recently, several authors (Collins, 1996; Ferro-Luzzi and James, 1996) have suggested that the MUAC alone may be suitable as an index of current adult nutritional status in an emergency situation. Alemu and Lindtjorn (1997) compared the frequency of "protein-energy malnutrition" diagnosed by either the BMI (<18.5kg/m^2) or MUAC (<25.1cm in men and <22.1cm in women) in two Ethiopian communities.

1.5 CED and mortality

In AED is there is a short term progressive loss of body weight and body stores. As this weight loss and energy imbalance continues health and body function will be impaired over a period of time eventually leading to death (Shetty & James, 1994). The effects on mortality or morbidity of CED, where the subject is in energy balance but has low anthropometric measures of fat reserves, are not well understood. This project aims to evaluate these effects.

1.5.1 Mortality studies in the developing world

The relationships between adult anthropometry and mortality have been extensively researched in the developed world. The research, which was partially initiated and funded by life-insurance companies, has centred predominantly on the risks of chronic diseases which are common in affluent societies. In adults obesity is a risk factor for a whole range of conditions, including cardiovascular diseases, arthritis, gall bladder diseases, some forms of cancer, failure of respiratory function and late onset non-insulin dependent diabetes mellitus and its sequelae. Life-insurance companies may include measures of obesity in the actuarial calculation of insurance premiums in adults.

Such research is only of limited usefulness to this project. The key problem is that these studies have not usually studied the risks of mortality at low values of anthropometric indices. They have tended to concentrate on the risks of obesity. Measures of fatness or obesity are typically split into quintiles or relative weight (RW) groupings for a given cohort in order to construct mortality risk ratios. As the cohorts are from the developed world, this results in cut-off points of low weight-for-height indices which are usually well above a BMI of 18.5 kg/m^2. Another problem is that mortality risk curves for affluent populations are unlikely to apply to the developing world, where disease patterns, physical activity levels and medical facilities are different. No studies on the relationship between adult MUAC and mortality in the developed world have been undertaken, or at least published.

Many of the studies also suffer from one or more of the following failures - (i) failure to control for smoking which is a significant risk factor for early mortality and is strongly associated with low body weight; (ii) failure to control for alcohol consumption which may be associated with mortality risks; (iii) inappropriate control of physiological and metabolic effects of obesity which are intermediate in steps in the causal pathway between obesity and death, e.g., hypertension and (iv) failure to eliminate early mortality from the analysis - resulting in the effects of disease on weight being evaluated in addition to the effect of weight on the development of diseases (Manson et al, 1987). Furthermore, almost all the studies have examined the relationship between mortality and anthropometric status only in men and women of European origin - the Kaiser foundation study, the National Health Interview Survey (NHIS), the NHANES-I epidemiological follow-up study (NHEFS) and the Honolulu Heart Foundation study being the exceptions.

Despite these problems, the findings from the work on the relationship between anthropometric indices and mortality in the developed world will be briefly described below. This work is important as no longitudinal studies have been conducted on mortality in the developing world. Note that there are many ways of describing the associations seen between mortality and anthropometric status. A distribution may be described as "U" shaped - both low and high values of the BMI lead to an increased risk of mortality and the minimum mortality risk lies in the centre of the BMI distribution. Alternatively, the relationship may be "J" shaped when mortality risks are more highly skewed to the right. Other studies show a direct association between the BMI and mortality, i.e. as BMI increases so does the risk of mortality. These descriptions, particularly the "U" and "J" shaped types are not necessarily exclusive and may depend on what range of BMIs were studied.

Table 1.3 shows a summary of the results and characteristics of some of the most influential studies conducted on the relationship between weight-for-height indices and the relative risk (RR) of mortality in the developed world. The list of studies is not exhaustive.

Table 1.3: The characteristics and results of studies of the association between weight-for-height indices and mortality in the developed world

Study	No. & sex of subjects	Length of study (Y)	W/H index (lowest cut-off employed)	Appropriate control for			Shape of association(is association affected by age?)
				Smoking or alcohol	Intermediate factors	Early mortality	
Build & blood pressure 1959[1]	4 900 000 (M&F)	19	RW		Yes	Yes	Direct association
Build Study 1979[2]	4 200 000 (M&F)	22	RW		Yes	Yes	J-shaped (age)
American Cancer Society[3]	750 000 (M&F)	12	RW	Smoking	Yes	No	J-shaped (age)
Norway[4]	1 715 515 (M&F)	16	BMI (19kg/m^2)		Yes	Yes	U-shaped (age)
Whitehall[5]	18 403 (M)	10	BMI (22.4kg/m^2)	Smoking	No	Yes	J-shaped (age)
Harvard[6]	19 297 (M)	27	BMI (22.5kg/m^2)	Smoking	Yes	Yes	Direct association
Finland[7]	40 154 (M&F)	12	BMI (22.0kg/m^2)	Smoking	Yes	Yes	U-shaped (age)

Sources: 1. Chicago society of actuaries. 2. Boston society of actuaries (1980). 3. Lew & Garfinkel (1985). 4. Waaler (1984). 5. Jarrett et al (1982). 6. Lee et al (1993). 7. Rissanen et al (1989 & 1991).

Table 1.3 (cont.) *The characteristics and results of studies of the association between weight-for-height indices and mortality in the developed world*

Study	No. & sex of subjects	Length of study (Y)	W/H index (lowest cut-off employed)	Appropriate control for			Shape of association (is association affected by age?)
				Smoking or alcohol	Intermediate factors	Early mortality	
E. Finland[8]	7 906 (M&F)	9	BMI (22.9kg/m^2)	Smoking	Yes	Yes	J-shaped for men, no relationship women
Finland[9]	12 000 (F)	29	BMI (21kg/m^2)	Smoking	Yes	Yes	U-shaped, but not significant for "lean" v "normal"
Seventh day Adventist men[10]	8 828 (M)	26	BMI (22.3kg/m^2)	Smoking Alcohol	Yes	Yes	Direct association (age)
Seventh day Adventist women[11]	12 576 (F)	26	BMI (21.3kg/m^2)	Smoking Alcohol	Yes	Yes	J-shaped or direct association, dependent on age
Kaiser foundation[12]	2 453 (M)	15	BMI (17-24kg/m^2)	Smoking Alcohol	Yes	Yes	J-shaped
NHIS & NHEFS [13]	128 196 (M & F)	5-10	BMI (19kg/m^2)	Smoking	Yes	(excluded chronically ill)	U-shaped

Sources: 8. Tuomilehto et al (1987) 9. Laara & Rantakallio (1996) 10. Linsted et al (1991) 11. Linsted & Singh (1997) 12. Wienpahl & Ragland (1990) 13. Durazo-Arvizu et al (1997)

Table 1.3 (cont.) *The characteristics and results of studies of the association between weight-for-height indices and mortality in the developed world*

Study	No. & sex of subjects	Length of study (Y)	W/H index (lowest cut-off employed)	Appropriate control for			Shape of association (is association affected by age?)
				Smoking or alcohol	Intermediate factors	Early mortality	
Honolulu Heart Programme[14]	8 000 (M)	19	BMI (21.2kg/m^2)	Smoking Alcohol	Yes	Yes	J-shaped
Copenhagen City Heart Study[15]	13 285 (M&F)	12	BMI (20-25kg/m^2)	Smoking Alcohol	Yes	Yes	No association if alcohol taken into account
Gothenborg[16]	792 (M)	13	BMI	Smoking	No	No	No association (age)

Sources 14 Chyou et al (1997) 15 Gronback et al (1994) 16 Larsson et al (1984)

Table 1.3 shows that the relationship between the BMI and mortality is not identical in all population groups. Such findings make brief summaries difficult. However, a few general comments:-

(i) Except in studies 12-14, the lowest cut-off points of the weight-for-height indices employed by the studies do not approach the values found where CED is present.

(ii) It seems clear that a high BMI or RW value leads to an increased RR of mortality, although the Only two studies did not report this finding. The Copenhagen city heart study (Gronback et al. 1994) found that the BMI had a negligible (and non-interacting) effect on mortality if alcohol intake was taken into account. The Gothenburg study (Larsson et al. 1984) did not find an association between mortality and BMI.

(iii) Approximately half the studies that found that the shape of the association between the BMI or RW and mortality may be altered by age (see section 1.7).

(iv) In all the studies which accounted for smoking, mortality was raised in smokers (except for the study in Copenhagen, where smoking was found to have a negligible effect compared to alcohol). The mortality risk from smoking is often found to be greater than that from the BMI (Kushner, 1993).

(v) It can be seen that the majority of the studies found a J-shaped association between BMI and mortality rate. This indicates an increased risk of mortality at the lower and upper levels of BMI. However, this result was not universal. Several important studies found a direct association and others noted a U-shaped association. Two studies found no association between the BMI and mortality risk.

From the comments above it could be concluded that, after controlling appropriately for smoking and other factors, a J-shaped relationship between mortality and weight-for-height indices (especially the BMI) is the most commonly observed i.e. that, at the population level, individuals with relatively low and relatively high BMIs have higher mortality rates than individuals with more middling values of the BMI. However, Linsted et al's (1991) study on Seventh-day Adventist men showed a direct relationship between BMI and mortality. As Adventists are prohibited from using tobacco and alcoholic drinks, and are discouraged from consuming meats and caffeine-containing beverages, they make a good population for epidemiological investigation (Kushner, 1993). No increase in mortality was seen in the lean group. The authors suggest that leanness is a surrogate for healthy lifestyle practices and is not in itself responsible for increased mortality.

The relationship between anthropometric indices other than weight-for-height indices and mortality have only rarely been studied. Some of the cohorts in these studies are large and it would be impractical to take other, more complex measurements (impossible, where the measures were self-reported). Waaler (1984) looked at the risk of mortality associated with height alone. He found a decreased RR of mortality with increasing height in all age groups and both sexes, with the exception of the very tall. Waaler concluded that, as adult height is a reflection of nutritional history, the excess mortality may be associated with past socio-economic history. Barker et al (1990) also reported that mortality from various causes across the counties of England was associated with the average height of the residents in the counties. He concluded that this provided support for the influence of childhood growth rates on later illnesses.

1.5.2 Mortality in the developing world

There is only one published study which assesses the risks of mortality for CED (diagnosed by the BMI alone) groups compared to non-CED groups in adults in the developing world. Data on the relationship between the BMI and mortality rates are available for severe famine situations (Collins, 1995), but the BMI levels are much lower than those used to define CED. Table 1.4 shows data from Satyanarayana et al's (1991) study on Indian men in the rural area surrounding Hyderabad.

Table 1.4: Mortality rates according to BMI level in India

BMI (kg/m^2)	Mortality rate (deaths/1000/year)
< 16.0	32.5
16.0-16.9	18.9
17.0-18.4	13.2
>18.5	12.1

Source: Shetty & James (1994)

There is an inverse relationship between the subjects' BMI and mortality rates. However, it is difficult to infer much more from this study, as no age or socio-economic data were provided. Moreover, it is difficult to state categorically whether the low BMI values observed were the result of the disease process leading to death, or whether the BMI state preceded and increased the men's proneness to illness (Shetty & James, 1994).

1.6 CED and morbidity

1.6.1 Morbidity in the developed world

The associations between anthropometric indices and morbidity have not been studied as frequently as those with mortality. However, many studies of mortality have considered the underlying cause of death and hence inferences may be made about the relationship between various anthropometric indices and morbidity.

It appears that high weight-for-height and low weight-for-height measurements bring with them risks for different types of diseases. Low weight-for-height is typically characterised by tuberculosis (Waaler, 1984), lung cancer (Waaler, 1984; Kabat & Wynder, 1992; Kushner, 1993) and obstructive lung diseases. Whereas high weight-for-height is characterised by cerebrovascular diseases (Waaler, 1984, Kushner, 1993; Rexrode et al, 1997) cardiovascular diseases (Waaler, 1984, Fitzgerald & Jarrett, 1992; Kushner,1993; Tuomilehto et al, 1987, Rabkin et al, 1997, Tavani et al, 1997), non-insulin dependent diabetes mellitus (Rabkin, 1997), cancer of the colon (Waaler, 1984, Kushner, 1993), prostate cancer (Cerhan et al, 1997) and gastric cancer (Hansson et al, 1994).

1.6.2 Morbidity in the developing world

There has not yet been a systematic prospective longitudinal study of the relationship between adult anthropometric indices and morbidity in the developing world. Hence the long term effects of low values of adult anthropometric indices on morbidity experience in the developing world are not yet known.

The studies which have been conducted to date have been either cross-sectional (Pryer, 1990 & 1994, Strickland & Ulijaszek, 1993a, Francois, 1990; de Vasconcellos, 1994; Ulijaszek, 1997 - in press) or retrospective (Campbell & Ulijaszek, 1994) or a mixture of methods (Garcia & Kennedy, 1994). In general, cross-sectional studies which have results consistent with plausible biological mechanisms provide some, although not sufficient, support for causality (Susser, 1991; Rothman & Greenland, 1998). However, the relationship between low BMI or MUAC and morbidity is circular. If a person is ill they may lose weight, on the other hand, if a person is thin they may be more likely to become ill. If low BMI or MUAC is found to be associated with excess morbidity in a cross-sectional study either case may apply. Thus there are two

biologically credible hypotheses which may account for the associations seen in a cross-sectional study of this nature. An analysis into the relationships of the BMI or MUAC with morbidity must be based on subjects who are fundamentally healthy i.e. have no chronic illness. None of the studies above control for pre-existing illness and hence no causal inferences about the association between anthropometrical status and morbidity can be drawn.

Given the problems described above, it is still worth looking at the studies listed above in some detail as they are the only ones which have considered the relationship between the BMI and morbidity in the developing world.

Pryer (1990 & 1994) examined the relationship between BMI and "number of complete working days missed in the month prior to interview due to incapacitating illness" in men living in an urban slum in Bangladesh. She found a significant inverse association between BMI and work-disabling morbidity. Below a BMI of 16.0 kg/m^2 (Grade III CED) 55% of the men lost one or more working day. This proportion dropped to 35% among those with a BMI of 16.0-17.0 kg/m^2 (Grade II). Above a threshold BMI of 17.0 kg/m^2 the percentage of men incapacitated from work was similar in each BMI category. The association held true when loss of working days due to work-related accidents was excluded. Table 1.5 shows the relative risks of morbidity at various BMI grades.

Table 1.5 The relative risks of morbidity at various BMI grades in Pakistani men.*

BMI (kg/m^2)	RR of morbidity*
< 16.0	5.9
16.1-17.0	3.8
17.1-18.5	1.8
>18.5	1

* Relative risk of being incapacitated from wage work for one or more days in month prior to interview

De Vasconcellos (1994) performed a similar study to that of Pryer's in Brazil. Adults were asked how many days they had spent in bed during the two weeks before they were surveyed. A U-shaped association was seen between this proxy measure for morbidity and the BMI. The largest increase in "morbidity" rate occurred in the BMI range of 16.0 - 17.0 kg/m^2, the rate was much lower in BMIs above this range. Francois (1990) has also shown that at BMI<17.0 kg/m^2 Rwandan women have a greater likelihood of illness and are more frequently confined to bed (James, 1994a).

Strickland and Ulijaszek (1993) studied the relationship between anthropometric indices and reported morbidity on the day of interview in the same population as the current study. They found that the BMI and AMBA (arm muscle and bone area) were sensitive to reported single symptoms of any kind in men over 40 years, significantly so for respiratory conditions. In women the BMI alone was sensitive to reports of two concurrent symptoms in women under 40 years and to respiratory complaints. Morbidity was significantly related to the BMI and AMBA independently of age in men. In older women, reported morbidity showed no effects on BMI or AMBA, while age effects were more pronounced than in men. The authors suggested that this is consistent with the evidence that female lean tissue is more resilient to the insults of malnutrition and disease than that of males (Stini, 1968; Henry, 1990).

Ulijaszek (1997, in press) examined the association between anthropometric indices and blood pressure in a small sample of adults in Papua New Guinea. Blood pressure was not associated with height or weight. Rather it was associated with fat patterning, with increasing trunkal fatness being associated with greater systolic blood pressure for both males and females.

Campbell and Ulijaszek (1994) studied the relationship between current anthropometry and retrospective morbidity in very poor men who attended the Middleton Row Street clinic in Calcutta. Using discriminant analysis techniques (after Mascie-Taylor, 1994) it was found that the BMI was a better discriminator of overall retrospective morbidity in men <65 years than weight, height, age, arm circumference, arm fat area and body fat percent. However, in certain disease categories (e.g. tuberculosis) calf circumference , height, weight and age were more significant discriminators. Moreover, in men above 65 years the BMI was much less useful as a discriminatory tool.

The study of Garcia and Kennedy (1994) is difficult to discuss. They have attempted to assess the linkage between BMI and morbidity in adults in four developing countries. They conclude that, although their results are mixed, the probability of being sick does not vary substantially between those with low BMI and those with normal BMI. Furthermore, they state that the 18.5 kg/m^2 proposed CED cut-off point is not generally consistent with the threshold at which morbidity begins to rise. This study is difficult to interpret as the methods employed both in the collection and analysis of the data are unclear.

It is important to state, again, that these studies only show an association between low BMI and morbidity. Causality cannot be inferred from cross-sectional data of this nature. Campbell and Ulijaszek's (1994) study was retrospective, but it made no allowance for the element of

circularity between the BMI and morbidity. The paucity of studies examining the relationships between the BMI or MUAC and mortality or morbidity measured in the developing world provided the main impetus for this study.

1.7 Weakness of the anthropometrical method in diagnosing CED

Anthropometric indices are used as proxy measurements of nutritional status. They are simple and cheap to use. However, they are far from perfect measures of CED. Three of the major problems associated with the use of anthropometry in diagnosing CED are briefly outlined below -

(i) The lower limits of BMI or MUAC may have a different significance in the developed and developing world in terms of their effects on functional capacity. Thin and tall adults with a marginally low BMI may be fit and healthy in the developed world, but certain conditions in the developing world may result in an individual with a marginally low BMI being significantly disadvantaged in terms of health or functional capacity compared to individuals with a higher BMI. Such conditions include periods when an individual has access only to limited food supplies, or when their physical work demands are high, or when they are ill. It is probable that the functional significance of the BMI or MUAC is also different in distinct sub-groups of the developing world.

(ii) Ageing presents a serious problem to the present classification of CED. As the BMI and MUAC both change significantly with age it may be necessary to diagnose CED differently in distinct age groups. Further, de Vasconcellos (1994) has shown that the BMI changes associated with increasing age are distinct in different SES groups.

(iii) Mean BMI values may differ between the sexes. Thus the cut-offs employed may need to be sex-specific.

Given that this study examines the properties of the BMI and MUAC in a single ethnic group in a small area problem (i) is less important for this project than it might be in studies covering several countries. However, the effects of sex or ageing may present a serious challenge to the currently recommended methods of diagnosing CED and will be closely examined in this study. An outline of the problems faced when assessing the significance of low values of anthropometric variables in the elderly is given below.

At the population level, ageing is the overall process of progressive, generalised impairment of the functions of organs and tissues which results in an increasing age-specific death rate. A

survivorship pattern known as the Gompertzian curve is seen. Ageing at the individual level is harder to describe as the process varies enormously between individuals. Korenchevsky (1961) has stated that old age is an abnormal, pathological syndrome, in which physiological processes of ageing are complicated by the degenerative diseases of old age. This definition has highlighted one of the major problems of research into ageing, that it is difficult to make distinctions between "normal" and "pathological" changes associated with old age. Borkan (1986) believes that from a practical standpoint the pathological / physiological argument is moot - all the changes seen with ageing are measures of functional status in late adulthood and are indications of the relative viability of the individual. The ultimate purpose of the research into ageing is to improve the health of the elderly and therefore it does not matter whether the changes are associated with physiological or pathological processes.

However ageing is defined, it is generally agreed that most tissue functions decrease during adult life (Munro & Danford, 1989). Immunological function is no exception to the rule. There is abundant evidence confirming a decline in immunological responsiveness with increasing age for both cellular and humoral immunity (although the molecular mechanisms underlying these changes remain to be elucidated). Furthermore, body composition changes with ageing (Young et al, 1963; Novak, 1972; Forbes, 1976; Borkan & Norris, 1977; Noppa et al, 1980; Bowman & Rosenberg, 1982; Munro & Danford, 1989; Shephard, 1991).

What then is the link between ageing, anthropometry and immunological function and hence morbidity and mortality? Can anthropometric measurements in older adults be interpreted, in terms of body composition, in the same manner as for younger adults? Are the BMI cut-off points suggested as appropriate for the elderly as they are for young adults i.e. does a BMI < 18.5kg/m^2 have more or equal or less significance with respect to functional capacity for older adults as compared to younger adults?

There has been a limited number of studies on the relationship between anthropometry, mortality and ageing in the developed world. Table 1.6 shows a summary of the results and characteristics of some of the most influential of these studies. It can be seen that the majority of these studies have been described in section 1.5.1. The additional studies have focused entirely on the elderly and hence were not introduced earlier. An attempt has been made to avoid the studies which have looked at hospitalised subjects as it was felt that this would lead to bias with respect to underlying illness. However, it should be noted that these studies are subject to the same flaws as those described above.

Table 1.6 The characteristics and results of the studies of the association between weight-for-height indices and mortality in elderly subjects in the developed world

Study	No. & sex of subjects	Definition of elderly	Length of study (Y)	W-H index (lowest cut-off employed)	Appropriate control for			Shape of the association in the non-elderly	Shape of association in the elderly and influence of age
					Smoking or alcohol	Intermediate factors	Early mortality		
Build Study 1979[1]	4 200 000 (M & F)	60+	22	RW		Yes	Yes	J-shaped	Lowest RR mortality occurs at progressively higher BMI with increasing age
American Cancer Society	750 000 (M & F)	60+	12	RW	Smoking	Yes	No	J-shaped	RR mortality in overweights decreases with increasing age
Norway[3]	1 715 515 (M & F)		16	BMI (19kg/m^2)		Yes	Yes	U-shaped	RR mortality in overweights decreases with increasing age. U-shaped curve flattens
Whitehall[2]	18 403 (M)		10	BMI (22.4kg/m^2)	Smoking	No	Yes	J-shaped	Found direct association aged 40-49y, 50+ found J-shaped association
Finland[5]	40 154 (M & F)	75 M 65 F	12	BMI (22.0kg/m^2)	Smoking	Yes	Yes	U-shaped	Minimum RR found in "overweight" elderly

Sources: 1. Boston Society of Actuaries 2. Lew & Garfinkel (1979) 3. Waaler (1984) 4. Jarrett et al (1982) 5. Rissanen et al (1989, 1991).

Table 1.6 (cont.) The characteristics and results of the studies of the association between weight-for-height indices and mortality in elderly subjects in the developed world

Study	No. & sex of subjects	Definition of elderly	Length of study (Y)	W-H index (lowest cut-off employed)	Appropriate control for			Shape of the association in the non-elderly	Shape of association in the elderly and influence of age
					Smoking or alcohol	Intermediate factors	Early mortality		
Seventh day Adventists[6]	8828 (M & F)		26	BMI ($20kg/m^2$)	Smoking Alcohol	Yes	Yes	Direct association	Protective effect of low BMI decreases with increasing age, non-existent after 90+.
Framingham[7]	597 (M) 1126 (F)	65	23	BMI M ($23kg/m^2$) F ($24kg/m^2$)	Smoking	Yes	Yes		J-shaped
Iowa[8]	41 837 (F)	55+	5	BMI (22.9kg/m2)	Smoking Alcohol	Yes	Yes (short)		BMI J-shaped
Tampere[9]	181 (M) 541 (F)	84+	3	BMI ($22kg/m^2$)	Smoking	Yes	Yes (short)		L-shaped, only significant in men
New Zealand[10]	295 (M) 463 (F)	70+	3.5	BMI		No	No		L-shaped
NHIS[11]	24 612 (F)	65+	5	BMI		Yes	No		L-shaped

Sources: 6. Linsted et al (1991) 7. Harris et al (1988) 8. Folsom et al (1993) 9. Rajala et al (1990) 10. Campbell et al (1990) 12. Brill et al (1997)

The important points to note from table 1.6 are:

(i) The definition of elderly varies from study to study. The length of the follow-up (inevitably) decreases with increasing age at the baseline.

(ii) In all the studies where the RR of mortality in the elderly is compared with that in younger adults it can be seen that the RR of mortality associated with overweight decreases with increasing age. This will result in a "flattening" of the association between obesity and mortality.

(iii)In some studies the minimum RR of mortality was actually found in "overweight" persons and hence an L-shaped association was seen between BMI and mortality.

It seems that the excess mortality among overweight adults is highest in middle age and tends to diminish with advancing age (Lew & Garfinkel, 1979). Indeed, some of these results suggest that a higher BMI may be protective in old age. Obesity may not be such a health risk for the elderly. Andres et al (1981) have commented that the undoubted relation of obesity to specific, important diseases which should, but do not, increase overall impact of obesity on mortality (in the elderly) suggests that there are counterbalancing benefits to the obese state which have not received the attention of epidemiologists or of clinical investigators.

Thus the available evidence suggests that moderate degrees of fatness are perhaps of no medical or physical disadvantage to the elderly of either sex and may constitute a benefit. Potter (1988) has suggested that the common tendency to gain weight late in middle life (in the developed world) is natural and perhaps this extra margin of nutritional reserve is beneficial during the stress of acute illness. Alternatively, Durnin (1989) has suggested that marked leanness may be not in itself indicative of undernutrition, but it may imply a reduced capability to respond to a nutritional emergency. This may be especially relevant to the elderly living in the developing world where the ability to withstand food shortages or bouts of infectious disease is especially important.

The authors of the BMI-cut off scheme have decided to ignore the effect of ageing on the BMI and the nature of the relationship of the BMI with mortality and morbidity at different ages. CED grades are diagnosed identically for each adult age group. This may be a simplistic approach, for the balance of the data described above suggests that low BMI becomes an increasingly important risk factor with increasing age. This study will pay close attention to the problems associated with the definition of CED by the BMI or MUAC in different age groups.

1.8 Conclusions

The purpose of this study is to examine the utility of anthropometric measurements or indices as tools used to assess current adult nutritional status. This will involve examining the distribution and determinants of the anthropometric variables and also their relationships to morbidity and mortality.

Weight, height, BMI, MUAC and skinfold thicknesses will be assessed in terms of their characteristics of an ideal anthropometric index of current adult nutritional status, so that they should be:-

(i) highly correlated with body energy stores
(ii) independent of body height
(iii) independent of sex and age
(iv) highly correlated with health and/or functional outcomes
(v) fulfil all the practical characteristics (i.e. be simple to measure and interpret, accurate, valid and precise)

Characteristic (v) is assessed in chapter two, characteristics (i) and (ii) are assessed in chapters three and four and characteristic (iv) is assessed in chapters in five and six

CHAPTER TWO

METHODS

2.1 Study Design

In 1990 Strickland and Ulijaszek spent approximately three months in the Song and Kanowit Districts of Sarawak, East Malaysia. During this period they collected data on age, self-reported morbidity, socio-economic status (SES) and anthropometric measurements of individuals in 24 randomly selected longhouse-villages. Anthropometric measurements were obtained from 1047 adults and 816 children. This thesis presents a follow-up study of the adult population seen in 1990. In 1996 the author returned to Sarawak in order to trace, re-interview and measure as many of the adult survivors of the 1990 study as possible. The survivors were then asked to participate in further studies which were conducted in 1996.

The research conducted in 1996 can be split up into two distinct sections in terms of design and analysis - (i) the follow-up study - concerned with mortality and anthropometrical change during the period 1990-96 and (ii) a longitudinal study - concerning morbidity over a six month period in 1996 and anthropometric change during this period.

2.1.1 Follow-up study

The inclusion criteria for the subjects in this study was that the individual had been aged 18y or above and was measured by Strickland or Ulijaszek in 1990. Data collected from the survivors included demographic data, current anthropometrical measurements and information on SES. In addition, date and cause of death was established for non-survivors by verbal autopsy (VA).

The follow-up period was six years (1990-1996). Generally, studies involving mortality rates have employed a longer follow-up period than this (see sections 1.5.1 and 1.7), the length of follow-up is usually determined by expected mortality rates and sample size. It is evident that a longer follow-up would result in more deaths which would make the statistical analysis more powerful. Also, some studies in the West have found that excess mortality in the underweight may occur early in the follow-up period, while that of overweight may be delayed (Manson et al, 1987). It has been suggested that this may be due to confounding between low measures of anthropometrical status and chronic illness at the time of measurement and that a "wash-out period", ignoring the first few years of mortality, should be employed during the analysis to allow

for this. In the developed world, where causes of deaths are centrally recorded in a systematic manner (e.g. in Sweden or the USA) it may be possible to undertake near ideal mortality studies with large sample sizes and long follow-up periods. However, in the developing world death registration systems are rarely complete and hence such 'ideal' studies are more difficult to undertake.

In a survey completed during the pilot phase of this project it was estimated that only 40% of deaths since 1990 were registered by the rural Iban in the Kanowit area. Moreover, only 15% of the registered deaths stated a cause of death. Thus VAs had to be applied to the closest living relatives of the deceased in order to elucidate the dates and causes of deaths for the non-survivors. It has been shown that the usefulness of VAs decreases as time-after-death increases (Garenne & Fontaine, 1990). After testing of the VA method in the pilot study, it was felt that a follow-up period of more than six years would be too long to obtain accurate responses to the VA's questions.

A further reason for the short length of the follow-up was that it was important that as many of the survivors from the 1990 study as possible be traced in order for the study to be complete. Relatively high levels of migration were reported for the Iban. Research conducted in the pilot study found that approximately 15% of the adults migrate from their longhouse over a five year period. This figure corresponds closely to the data produced by the 1990 Malaysian census (Department of Statistics Malaysia, 1995) and a separate study by Padoch (1982). The census estimated that 11% of the Sarawaki population migrated within the state over the period 1986-1991. Padoch found that 13% of Iban individuals may move from a previous residence over five years. If the follow-up period had been extended much longer than six years it would have become difficult to trace all the migrants.

2.1.2 Longitudinal Study

The inclusion criteria for this section of the project were that the individual was still alive in 1996 and resided in one of the study longhouses on a full-time basis. The author visited each longhouse-village at least twice during the year. The first visit (A) was between February and May and the second between August and October (visit B). Migrants were mainly measured at the end of each round i.e. June and November.

Data on morbidity were collected at both visits as were anthropometric measurements. Ideally, morbidity data should be studied over a one year period as seasonal variation in morbidity may

exist (Adams, 1995). However insufficient funds prevented this study from collecting more than six months of morbidity data.

Figure 2.1 shows how the morbidity data collection was organised. It can be seen that the data collection took place at different times of the year in each longhouse. Thus, if seasonality of morbidity does exist in Sarawak, the data may have been collected at different levels of morbidity in different longhouses. Provided that the adults with a low anthropometrical status were randomly distributed between the longhouses, and not concentrated in certain longhouses or areas this protocol should not have biased the relationship between low anthropometrical status and morbidity.

Figure 2.1 Timing and organisation of morbidity data collection in 1996

Longhouse	1996									
	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV
1	*				M		*			M
2	*				M		*			M
3	*				M		*			M
4	*				M		*			M
5	*				M		*			M
6	*				M		*			M
7	*				M		*			M
8	*				M		*			M
9	*				M		*			M
10	*				M		*			M
11	*				M		*			M
12	*				M		*			M
13	*				M		*			M
14	*				M		*			M
15	*				M		*			M
16	*				M		*			M
17	*				M		*			M
18	*				M		*			M
19	*				M		*			M
20	*				M		*			M
21	*				M		*			M
22	*				M		*			M
23	*				M		*			M
24	*				M		*			M

* - author visits longhouse. M - author traces migrants

2.2 Study area and population

The study took place in a rural Iban population in Sarawak, East Malaysia. Sarawak, located in the North-western part of Borneo, has a land area of 124,499 km^2. Its 1990 population was 1,718,400, of which 62.4% lived in the rural areas (Department of Statistics Malaysia, 1995). The indigenous peoples number the largest group in the state. In particular, the Iban account for approximately 30% (506,528 individuals) of the Sarawaki population.

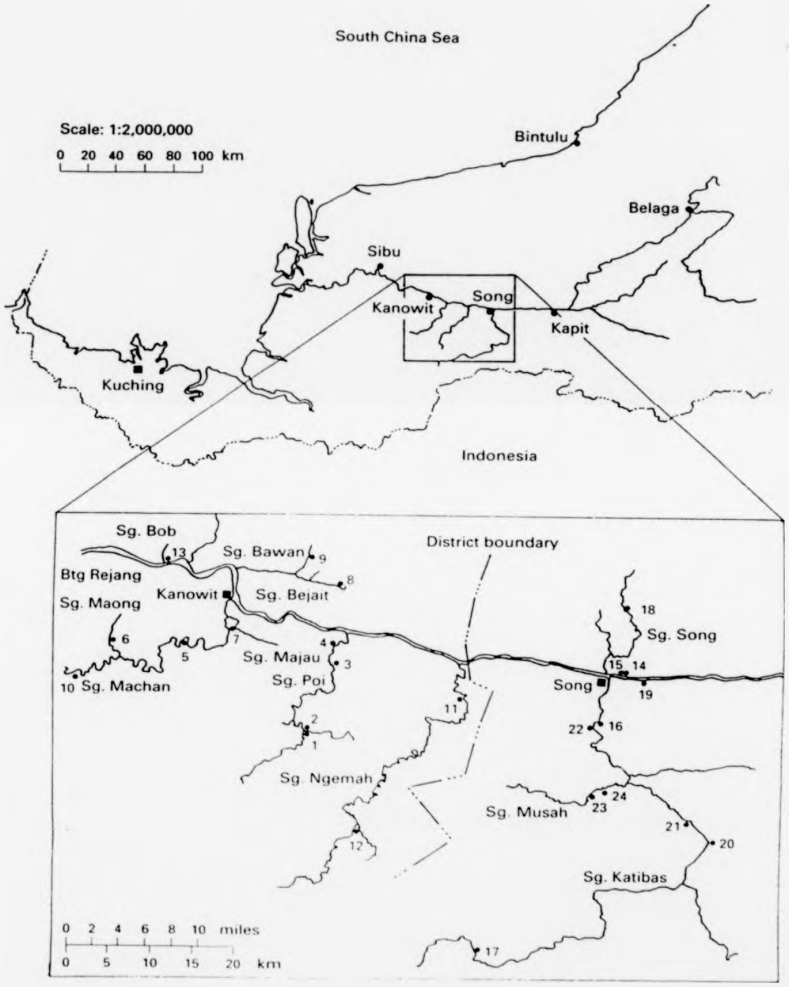
Administratively, the state of Sarawak is divided into seven Divisions. The study concentrated on the populations found in the Song (Division seven) and Kanowit (Division three) Districts. The Song and Kanowit Districts are adjacent and are located on the banks of the Rejang river, both are well established market towns serving the interior population.

Both the Song and Kanowit areas are predominantly made up of Iban communities. The population of the Song District is 17,576 of which 88% are Iban. The population of Kanowit is 25,380 of which 82% are Iban (Department of Statistics Malaysia, 1995). Both towns have schools, government offices and medical facilities. Kanowit has a large district hospital with four full time doctors. Song has only a small clinic with a single doctor.

In 1990 a selection of longhouse villages was randomly made from lists of the Song and Kanowit Districts obtained through the State Planning Unit. The aim was to be able to measure approximately 1,000 individuals of all ages in each of the two Districts. Thus, villages were selected until this figure appeared likely to be obtainable after making allowances for absenteeism (Strickland and Uhjaszek, 1990).

Most of the Iban studied in this project live in the rural areas in these two Districts. They have established communities along the banks of the Rejang and its tributaries. Each community lives in a longhouse (see figure 2.2). A longhouse is made up of a series of independently owned family apartments which are longitudinally joined one onto another to produce a single connected structure. The apartments are supported on hard-wood posts so that they are raised above the ground (Freeman, 1992).

Figure 2.2: Location of the survey area in Sarawak and the 24 longhouse villages covered



The fundamental unit of organisation of the Iban social structure is the bilek. Each family has its own apartment, or bilek, within the longhouse. The members of a bilek-family are intimately related by ties of kinship and affinity. Its members constitute a single household, food being prepared and cooked for the *bilek*-family as a whole. The *bilek*-family possesses both land and property in its own right. It is likewise an independent entity economically, cultivating its own padi etc. (Freeman, 1992).

A high degree of inter-relatedness exists between the *bilek*-families in a given longhouse. However, under normal circumstances, not all members of the longhouse are related. It is important to realise that the longhouse holds virtually no property under communal ownership, nor is there any collective ownership of land. There is also an absence of economic activity as a corporate group within the longhouse. Thus the socio-economic questionnaires used each *bilek* as the unit of analysis and not the longhouse as a whole. However, general responsibility for the longhouse's ritual well-being ensures that the community is linked via ritual ties. Furthermore, the inhabitants of a longhouse must elect a *tuai rumah* (head man) who is responsible for the safeguarding and administration of the customary law.

The subsistence economy of the Iban is based on the cultivation of hill rice. The subsistence method they employ is that of shifting cultivation. Traditionally, the whole way of Iban life is described as being "based upon the cultivation of rice" (Freeman, 1992; Hong, 1987). All adult members of a bilek are usually employed in working on their farms, although men and women may undertake different tasks. However, development has meant change and although rice cultivation is still of paramount importance to the Iban - it is their staple diet - some of the younger generation may earn their living in a different manner. Many young men, for example, work as labourers in the large cities or in the logging camps scattered across Sarawak, Sabah and Brunei.

The Iban are an excellent population to work with on follow-up studies because of their social organisation. Firstly, it was reasonably easy to find out where the migrants had moved to by asking other members of their *bilek* or longhouse for an address. It was common for several members of a longhouse to work for the same company or in the same area and hence a meeting with all the migrants of a particular longhouse could often be arranged. Many of the migrants were interviewed during the *Gawai* festival which was celebrated in June. At this time of year nearly all the Iban return to their longhouses to celebrate the harvest. Furthermore, it is normal for those who are terminally ill to return to their longhouses to die. Hence the VAs could be applied, in nearly all cases, to individuals from the same *bilek* as the deceased which meant that

the author did not have to travel to various hospitals to trace clinical records concerning the deceased.

2.3 Demographic data

2.3.1 Method of demographic data collection

A HH questionnaire was applied to every *bilek* which had at least one individual who was interviewed in 1990. The questionnaire (see Appendix A1) was designed to obtain information on each of the *bilek* members' age, sex, geographical location, migrant status and relationship to the head of the household. Demographic data were recorded at both visits to each *bilek* as some individuals' migrant status changed between visits.

Demographic data collected in 1990 were recorded on to each *bilek's* HH questionnaire prior to the author's first visit in 1996. On arriving at a *bilek* the author was thus able to start the protocol by asking questions about those recorded in 1990. Later, further questions were posed about any other *bilek* members (either those who had joined the *bilek* since 1990 or those who had not been recorded in 1990). A *bilek* member was defined as an individual who returned to the *bilek* for the *Gawai* celebrations i.e. an individual who regarded the *bilek* as their "home". Thus both *de facto* and *de jure* members of the *bilek* were recorded on the form. Any adult who had been measured in 1990 and hence was part of the follow-up study and who had migrated to a new *bilek* since 1990 was recorded on a new HH questionnaire. Data about the members of his new *bilek* were recorded on this new form.

Information on adult age was collected in the first instance by reference to identity cards which all Malaysian adults must carry according to the law. The ages on the cards were then cross-referenced by referring to subjects of local historical importance (e.g. the Japanese invasion of Borneo or the great fire in Kanowit or the appointment of a certain government official etc.). If the exact day of birth was not known an assumption that the individual was born on 1st July was made. The age of children under eighteen is generally well documented as the maternal and child health service has been effective in registering the majority of births for some time (Medical and Health Services Department Sarawak, 1990).

Data on migrant status assessed whether or not the individual was a permanent migrant from the *bilek* (e.g. had married away, divorced or left the *bilek* for another reason) or a temporary migrant i.e. was away from the *bilek* for educational or employment purposes. Inevitably, at times it was difficult to make this distinction and some overlap may exist. Within these

permanent / non-permanent categories of migrants an attempt was also made to describe the migrants in terms of the geography of their move. Some of the permanent migrants had married into a nearby longhouse whereas others had moved permanently to a nearby city, or town, or to peninsular Malaysia. This geographical distinction was made as it was felt that those migrants who had moved to a nearby rural environment would probably be experiencing similar environmental conditions to the rest of the study population; however those migrants who had moved to the cities would probably not have comparable environments.

2.3.2 Demographic characteristics of the study population

In total some 3486 individuals with information on their dates of birth and sex were recorded in 1996, compared to 2337 in 1990. Not all of the extra individuals were newcomers to the *bilek* or children born since 1990. Unfortunately, in 1990 the system of recording HH members was incomplete, so that in some cases temporary migrants were recorded and in others they were not. Thus any changes in the population's demography between 1990 and 1996 cannot be rigorously assessed.

In 1996, 3254 individuals were recorded in *bileks* which had at least one participant in the follow-up study. The population pyramids in figure 2.3 and table 2.1 show, by age and sex, the proportion of the population which was either in their *bilek*, at school or migrant at visit A.

An almost equal number of men (n=1636) and women (n=1618) were recorded. As expected, the pyramid is wider at the base than the top indicating a larger number of young people than old. It is interesting to note that roughly similar numbers of individuals are found in the three youngest age groups - implying that the population fertility rate may be decreasing. There is no significant difference in the proportion of boys or girls attending school. However, significantly more men aged between 20 and 60 are temporary migrants from the *bilek* than women (χ^2=49.4, p<0.001). Similarly, significantly more young people (<40y) are temporary migrants than old (>60y). Hence proportionally fewer young males will be found in the longhouse than any other group.

Figure 2.3 Population pyramid at visit A

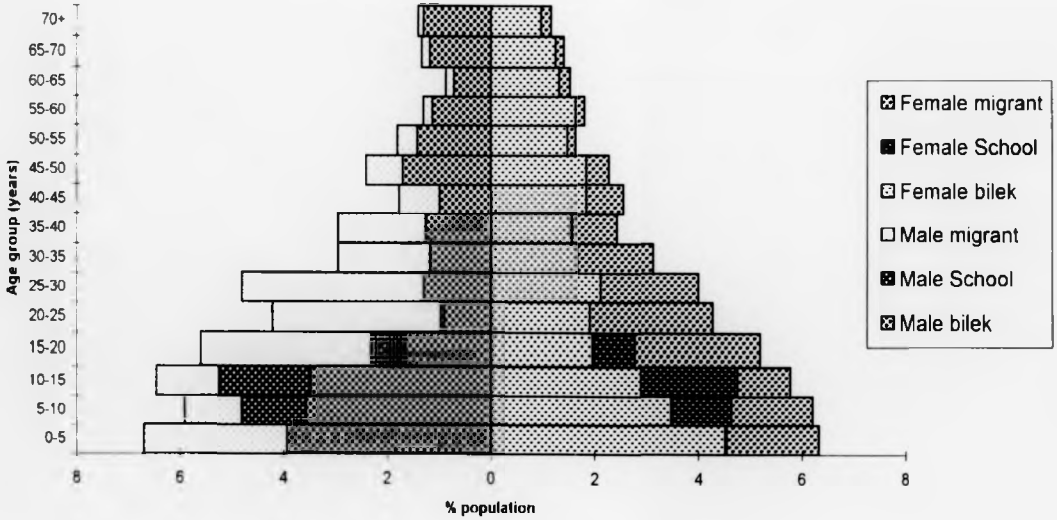


Table 2.1: Migrant status by sex and age group at visit A (% population)

Age group	MALE			FEMALE		
	In the *bilek*	School	Temporary Migrant	In the *bilek*	School	Temporary Migrant
0-4.9	3.9	0.0	2.8	4.5	0.0	1.8
5.0-9.9	3.6	1.2	1.1	3.5	1.2	1.6
10.0-14.9	3.5	1.8	1.2	2.9	1.9	1.0
15.0-19.9	1.6	0.7	3.3	2.0	0.8	2.4
20.0-24.9	0.9	0.1	3.3	1.9	0.0	2.4
25.0-29.9	1.3	0.0	3.5	2.1	0.0	1.9
30.0-34.9	1.2	0.0	1.8	1.7	0.0	1.5
35.0-39.9	1.3	0.0	1.7	1.6	0.0	0.9
40.0-44.9	1.0	0.0	0.8	1.8	0.0	0.7
45.0-49.9	1.7	0.0	0.7	1.8	0.0	0.4
50.0-54.9	1.4	0.0	0.4	1.5	0.0	0.2
55.0-59.9	1.1	0.0	0.2	1.6	0.0	0.2
60.0-64.9	0.7	0.0	0.2	1.3	0.0	0.2
65.0-69.9	1.2	0.0	0.2	1.3	0.0	0.2
70.0 +	1.3	0.0	0.1	1.0	0.0	0.2
All ages	25.6	3.7	21.1	30.5	3.9	15.4

Of the original 1047 adults measured in 1990, 962 were alive at visit A in 1996. 11.6% of these individuals had permanently migrated from their original 1990 bileks. Table 2.2 below shows the reasons for these permanent migrations. It can be seen that most of the "permanent" migrants had moved into a new *bilek* in the same longhouse, although others had moved to a new longhouse after marrying.

Table 2.2: Reasons for permanently leaving the 1990 bilek

Type of migrancy	Male (n=49)	Female (n=63)	All (n=112)
New *bilek* in the same longhouse	73.5	74.6	74.1
Married away from longhouse	16.3	20.6	18.8
Divorced and left longhouse	10.2	4.8	7.1

2.3.3 Household composition data

A total of 442 *bileks* which had at least one participant in the follow-up study were recorded. The mean HH size was 7.36 (SD=3.21) and the median was 7 (interquartile range=5-9). The mean

number of individuals present in the *bilek* at visit A was 4.12 (SD=2.66) and the median was 4 (interquartile range=2-6).

HH composition data was analysed by examining the ratios of dependant to independent individuals. A dependant individual was defined as one who needed to be fed or cared for by the rest of the household. As no information was collected concerning an individual's general economic "usefulness" it was decided to classify dependency by age. Cut-off points of below 10 years and more than 70 years were used to define dependants. These figures were adopted after discussion with key informants and reflected the general activity pattern of the population e.g. few children under 10 helped with farm work but many aged 10-15 did.

The household mean ratio of dependants to independents was 0.41 (SD=0.36). Amongst those present at visit A (i.e. the *de facto* population) the mean ratio was 0.53 (SD=0.56). These figures are related to the data above and show that many of the independent individuals leave the *bilek* for employment purposes, leaving a few other independent members (usually women) to care for the dependant members of the HH.

2.4 Socio-economic and "lifestyle" data

2.4.1 SES questionnaire

SES data were collected at the HH level during the first visit to each *bilek*. A questionnaire (see Appendix A2) was applied to the head of the household, although frequently other members of the family joined in and helped to answer the questions.

There is no standard SES questionnaire which can be applied to all populations. The variables which were included in this questionnaire were selected after reference to the literature (Sizaret, 1994; Nutritional CRSP, 1993) and consultation with Sarawaki informants during the pilot visit. It is widely reported that respondents of studies in which SES data are required are often reluctant, or unable, to provide an account of their economic situation in terms of cash income. Moreover, a complex (and lengthy) questionnaire may be required to obtain detailed information about cash income. The majority of the topics in the questionnaire did not concern cash income; instead most of the questionnaire focused on the economic resources available to the respondents other than cash income.

The questionnaire contained questions on the *bilek's* (a) construction characteristics, toilet facilities and sources of water, (b) material possessions, (c) type of land farmed, (d) cash cropping, (e) savings and (f) members' employment outside the longhouse.

Although the Iban live "communally" in longhouses and hence share a certain amount of living space (e.g. the veranda area), the families within each longhouse may not have identical living conditions. Each *bilek*-family is responsible for building their own *bileks* onto the longhouse. The construction materials used to build a *bilek* may therefore vary in quality, depending on the wealth of the *bilek*-family concerned. Moreover, some families may add a separate toilet facility to their *bilek* and / or piped water directly into it, whereas others may not be able to afford these conveniences. In most cases, the piped water and the toilets are added by the government (Medical and Health Services Department Sarawak, 1990). Similarly, the government may supply a longhouse with electricity or a generator or separate *bileks* may have to provide for themselves. Hence questions about the quality of the materials used to construct the walls, floors, roofs and windows of each *bilek* were posed. Similarly, questions about electricity, toilet facilities and water sources were included.

A material possessions scale was employed. This involved questioning the head of household about the ownership of certain possessions - a chainsaw, a cassette player, a television and either an outboard motor for a boat or a motorcycle or a car. During the analysis an index of material possessions from 0-4 was created, each item which was owned scoring a point on the scale. In later analyses this index was converted to a categorical variable with the division centring on the median score of 3. Those who scored >=3 were classified as scoring highly on the material possessions scale.

Land ownership and tenure are complex matters in Iban society. Some *bilek*-families rent or borrow land from each other (Sutlive, 1992) and thus to pose questions such as "how much land do you own?", or even, "do you own any land ?" was difficult. It was considered more important and easier to ask about the quality of land a *bilek*-family was currently farming. For those who farm hill padi it is possible to describe accurately the quality of a farm by asking how long the land lay fallow before its current use (or what size trees had to be cleared from it in order to plant the rice). The Iban occasionally supplement their income by growing and selling cash crops such as cocoa, rubber, fruits and illipe nuts. Questions concerning these activities were included in the questionnaire.

Although it is stated above that respondents of studies in which SES data is required are often reluctant, or unable, to provide an account of their economic situation in terms of cash income a question on savings was posed in this study. This idea was first proposed by the key informants in the pilot study. They suggested that the possession of savings was the best way to differentiate between rich and poor *bilek*-families, as those with savings could survive an emergency, (e.g., a bad harvest or illness in the family) whereas those without might be more seriously affected by such an event. Questions concerning the amount of savings (if any) held by each *bilek*-family were posed during the pilot. The respondents did not appear to be offended by the questions and thus similar questions were posed in the main phase (three HH declined to give an answer to the question about savings).

As stated earlier, the subsistence economy of the Iban is based on the cultivation of hill rice, indeed, most of its population is involved in farming the *padi*. However, employment outside the longhouse is becoming more frequent, especially among young men. A section on employment outside the longhouse was included in the questionnaire. Each *bilek*-family was asked if any of their members were employed outside the longhouse and, if so, broadly what category this employment fell into. They were also asked if they received remittances from this person. In addition widow's are eligible for remittances from the government and where applicable these were recorded. No questions concerning the size of remittances were posed.

It was assumed that the SES of temporary migrants was the same as that of the other members of their *bilek*. However, this assumption was not made for permanent migrants - their SES was assessed independently at their new residence. It was assumed that the non-survivors since 1990 had a similar SES to the members of their *bilek* who were still alive.

Some of the variables described above are inter-related but most do represent different aspects of SES. The variables concerning electricity, water source and building-materials represent the physical conditions of the *bilek* and they also provide information about its hygienic conditions. *Bileks* with glass windows, lineoleum floors, piped water and electricity may be easier to keep clean than those with uneven wooden floors, no windows or running water and no toilet facilities or electricity. As some of these factors may have been paid for by the government these variables are not a direct measure of the HH's wealth. The material possessions scale and land quality variable represent the past wealth of the *bilek*, e.g., at one stage they were able to afford a television etc. even if they could not now. The savings variable may represent the amount of cushioning a family would have against a future disaster - economic or physical. Some families

will choose to save money and others to spend it on material goods. thus both types of variables are important measurements of wealth

2.4.2 SES data handling

All the SES data described above are treated as categorical variables in the analysis. Appendix A3 gives an explanation of their transformation to categorical variables (where necessary). Table 2.3 shows the percentage distribution of the SES variables by *bilek*.

Table 2.3: The % distribution of SES variables by bilek (n=423)

	Yes (=1)	No (=0)
Savings	73.9	26.1
Material Possessions scale	60.8	39.2
Paid work	76.7	23.3
Remittances	74.1	25.9
Sold crops	81.2	18.8
Electricity	66.5	33.5
Toilet	84.3	15.7
Piped water	68.9	31.1
Glass windows	78.4	21.6
Lincolcum floors	86.2	13.8

Many of the SES variables are inter-related. For example. 66.9% of the HH's which have savings score more highly on the material possession score compared to 44.5% of those which do not possess savings (χ^2=17.0, p<0.001), i.e. those who have savings also tend to score highly on the material possessions scale. Table 2.4 shows the associations seen between the various SES factors described above.

Table 2.4. The associations seen between the various HH SES factors. χ^2 tests used, only significant associations reported (N = 423)

	Mat. Pos. Scale	Savings	Paid work	Remittances	Sold crops	Electricity	Water source	Toilet	Window material	Floor material
Mat. Pos. Scale										
Savings	+									
Paid work	+	+								
Remittances	+	+	+							
Sold crops		+								
Electricity	+	+	+	+						
Water source					+					
Toilet	+				+	+	+			
Window material	+				+	+		+		
Floor material	+	+	+	+	+	+		+	+	

+ a significant positive association is seen between the two variables, e.g.: those who score highly on the material possessions scale are more likely to have savings and vice versa.

2.4.3 Validation of the SES data collection method

An attempt was made to validate the socio-economic questionnaires during the pilot visit. One longhouse, which did not take part in the 1990 study but was located within the study region, was chosen as the validation test-case. The socio-economic questionnaire was applied to the nineteen heads of *bileks* within the longhouse. Two methods of validation were employed - (i) the questionnaire was applied by two separate interviewers on different days in six *bileks* and the responses were compared, and (ii) three inhabitants of the longhouse were asked to rank all the *bileks* by wealth from 1 (very wealthy) to 5 (very poor) independently. The rankings given by these informants were averaged and the average ranking of each *bilek* was compared to that obtained by the questionnaire method using the index of material possessions and savings described above.

The results of the validation tests were satisfactory. It was found that all the respondents from the six *bileks* gave identical responses to the two different interviewers. Using the indices of material possessions and savings described above, the *bilek*-families were ranked according to their capital wealth from 1 to 5. When this ranking was compared to that suggested by the informants it was found that 47% of the rankings were identical and that a further 47% of the rankings differed only by one position.

2.4.4 Educational levels

Each subject was asked about his education and literacy levels at visit A. Data on the educational level of the non-survivors was obtained as part of the VA procedure.

Table 2.5 shows the distribution of school attendance amongst the study population (including non-survivors). More men than women attended school and could read (χ^2=44.6, $p<0.001$). A higher proportion of the younger groups were literate and had attended school than the older groups (χ^2=265.7, $p<0.001$).

Table 2.5: The % frequency of school attendance by age and sex grouping

	Men (n=412)	Women (n=568)	Both (n=980)
18-24.9	80.4	65.9	71.6
25.0-29.9	86.5	50.0	64.5
30.0-34.9	77.8	49.2	59.4
35.0-39.9	82.3	35.6	52.7
40.0-44.9	51.7	15.5	33.6
45.0-49.9	45.9	4.8	20.2
50.0-54.9	10.8	0	5.1
55.0-59.9	9.1	0	3.3
60.0-64.9	6.7	0	3.0
65.0-69.9	6.2	0	3.8
70.0	9.1	0	4.2
All	46.8	26.2	34.9

2.4.5 Physical activity level

A questionnaire (see Appendix A4) was used to establish the general activity pattern of adults by recall of activity for the twenty-four hours prior to each interview. The data obtained was qualitative and did not attempt to quantify how much energy had been expended in the 24 hour recall period. Some months into the study, the author decided to add a further question concerning an individual's physical activity level (PAL) - "had the individual worked on the farm (or any other physically demanding job) at all that year?" This question was added in order to assess whether an individual had actually undertaken any real physical work during the previous six months as opposed to being mainly sedentary in the longhouse. 84.8 % of the population who were asked this question (some of those who were not seen at visit B could not be asked the question) reported having worked physically hard in the previous six months.

During analysis some reorganisation of the activity categories was undertaken. House work was split into three categories - essential, food collection and non-essential. The essential work comprised washing, cooking, child care and the collection of wood for the fire - these jobs had to be carried out every day even if work on the farm was very hard. Food collection included fishing, hunting and gardening. These jobs were useful in terms of increasing the amount and choice of food available and also (possibly) for supplementing any income received but were not absolutely necessary. Finally, the non-essential work included handiwork (weaving or basket making) and re-building the *bilek* or a boat - these jobs were only undertaken if there was

"nothing else to do" and served to either supplement an HH's income or improve their living environment. Farm work was also split up into three categories - physically hard work (harvesting, clearing and planting), easier work (threshing, weeding and inspecting the farm) and extra-income work (tapping rubber).

Figure 2.4-2.6 show the distribution of activity patterns by sex. At visit A less men do housework than women (χ^2=113.9, p<0.001), but more men undertake farm work (χ^2=5.3, p=0.02) and more men work outside the longhouse (χ^2=45.8, p<0.001). At visit B significantly more women than men do the housework and less women still work outside the longhouse, however approximately equal proportions of men and women work on the farm (figure 2.4).

When examining the type of work undertaken by individuals who were present in the longhouse at both visit B and visit A it can be seen that more women undertake the essential household tasks than men (χ^2=492.3, p<0.001) and that an almost equal number of women perform these tasks at both times of the year (2.5). Conversely, the men collect significantly more food than women at both times of the year. At visit A there was no significant difference between the proportions of men and women doing non-essential housework, however at visit B more women undertook this type of work than men (χ^2=13.9, p<0.001).

Proportionally, both men and women undertook the same amounts of hard farm work at visits A and B, although both sexes undertook much more hard farm work at B (figure 2.6). More men worked on easy jobs on the farm than women at both visits (χ^2=6.1, p=0.02). Significantly more men also tapped rubber than women at visit A although at visit B few men or women undertook this job.

Thus a general picture of activity levels and patterns was that (a) more women than men undertook housework, particularly essential housework at all times, (b) the men were more likely to go to the farm than the women when light farming or rubber tapping was required but the sexes shared the harder farming tasks and (c) both sexes undertook more physically demanding farm work at visit B than at visit A.

Figure 2.4: Type of activity or work by sex and visit

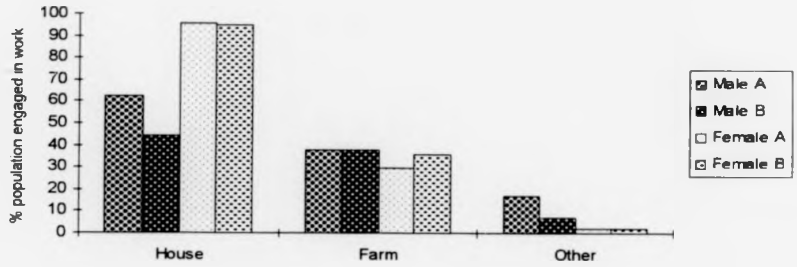


Figure 2.5: Type of activity in the longhouse by sex and visit

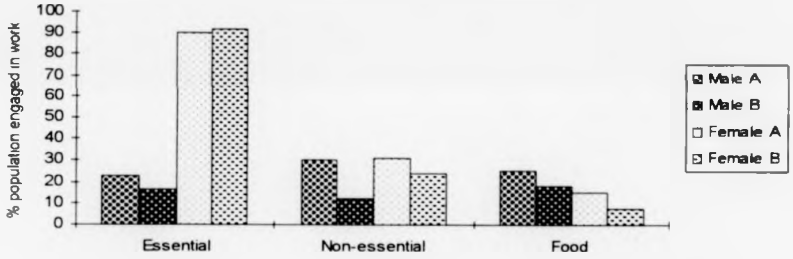
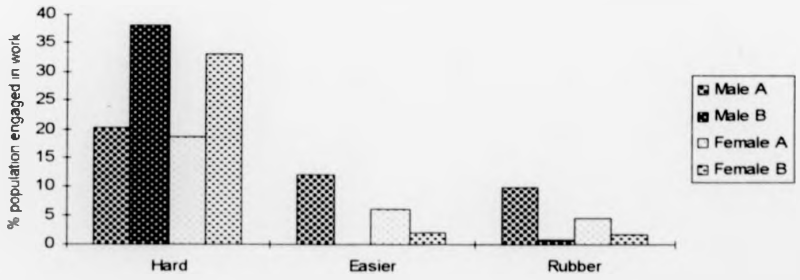


Figure 2.6: Type of activity in the farm by sex and visit



2.5 Anthropometry

Anthropometrical measurements were made on all consenting adults who were traced during 1996. Measurements were made both at visit A and B. The original 1990 measurements were made between February and April. The order in which the longhouses were visited in 1996 was almost identical to that of the 1990 study. Thus the first round of measurements in this study were taken at roughly the same time of year as those in the 1990 study, and any comparisons between the two sets of data should exclude seasonal effects.

2.5.1 Anthropometric data collection

The anthropometric measurements taken were weight, height, sitting height, mid-upper arm circumference, half-span and skinfolds at the biceps, triceps, subscapular and suprailiac sites. Standard techniques were employed (Jelliffe & Jelliffe, 1989).

Weight was measured using Soehnle scale to the nearest 0.1 kg. Test weights up to a total of 50kg were used at the beginning and end of the study when only a maximum error of 100g was shown. Subjects were asked to remove their shoes and any bulky clothing.

Standing height was measured using standard methods to the nearest 0.1 cm with a portable, rigid CMS anthropometer. Sitting height was measured by placing the anthropometer on a low table with the subject sitting at the base of it with their buttocks and shoulder blades in contact with the vertical surface of the anthropometer.

Limb circumferences were measured using standard techniques to the nearest 0.1 cm with fibre glass measuring tapes. Half-span was measured using standard techniques with a steel measuring tape to the nearest 0.1 cm. Skinfold thicknesses were estimated to the nearest 0.1 mm with Holtain calipers. Each of the skinfold measurements was taken three times and an average was recorded.

2.5.2 Indices of adult body composition

The percentage of body fat in adults was calculated from the sum of four skinfolds (biceps, triceps, subscapular and suprailiac) using the equations of Durnin and Wormersley (1974) appropriate for the age and sex of the subjects and the Siri equation (Siri, 1956). FM and FFM were then estimated.

The BMI was usually calculated as the BMI derived from stature (weight/height^2). However, a BMI derived from half-span measurements was also calculated for the older groups (>=50 years). Arm-span measurements are the preferred index of stature in the elderly where height is unreliable because of age-related loss or distortion (Smith et al, 1995; Bassey, 1986). Regressions relating stature to half-span for men and women under aged 40y, i.e. before loss of stature begins, were derived (see table 2.6). The BMI derived from half-span was then calculated as weight/(stature derived from halfspan)^2

Table 2.6: Equations derived to relate stature and halfspan in population aged 24.0-39.9 years.

	N	Equation
Male	118	Stature = 1.373(half-span) + 44.95
Female	189	Stature = 1.236(half-span) + 54.30

A Benn's weight-for-height index was also calculated for each sex (table 2.7). A Benn index is completely independent of height (Benn, 1971) and is population specific. The index (W/H^b) is calculated as -

$\sqrt{-}$ = b (mean height/mean weight).

where b = regression between weight and height

Table 2.7: Calculation of Benn's index for men and women

	Men	Women	All
Mean height	157.05	147.01	151.96
Mean weight	51.10	45.02	48.01
Gradient in regression	0.787	0.913	0.731
Benn's index	2.42	2.98	2.31

The Cormic index (CI) is a measure of the relative length of the trunks or legs, and was calculated as -

C.I. = Sitting height/Standing height

2.5.3 Difficulties with anthropometric data collection

As reported by Chumlea et al (1984, 1989a, 1989b) the author found that accurate measurements were harder to obtain from the elderly than the young. This problem applied to all the measurements. In particular, height was difficult to measure in the elderly. Even those individuals who did not have advanced spinal curvature found it difficult to stand up "straight". In this age group, precise half-span measurements were easier to obtain than measurements of stature.

Greater and / or poor compressibility of skinfolds in the elderly (Bowman & Rosenberg, 1982) also made these measurements and the MUAC unreliable in some cases. Skinfold measurements were made more problematic in the elderly as the separation of subcutaneous adipose tissue from the underlying fascia and muscle is not as clearly discernible as it is in young adults.

In general the author found obtaining accurate and precise skinfold thickness measurements to be quite difficult. Obtaining the true values of skinfolds in the fatter individuals was generally harder (Garrow, 1988) as the fat was so densely packed. Accurate skinfold thickness measurements can only be made up to 40mm with Holtain calipers. Thus all individuals with at least one skinfold measurement of more than 39.9mm were excluded from the analyses involving body composition variables (115 women and 26 men). Naturally this exclusion criterion resulted in removing more fat people than thin from the analyses. Hence a bias in the body composition data is seen - more low FM and fat percent individuals are included in these analyses. This is important when considering analyses of the body energy stores in chapters 3 and 4.

2.5.4 Data Cleaning

All anthropometric data were entered into a database programme by the author and a data entry clerk. Thus in the first instance the two sets of data were compared and field records were checked in order to determine any data entry errors. Outliers were identified by plotting variables measured in different seasons against each other and referring back to 1990 data if necessary. Field records were checked in order to determine whether the outliers were due to measurement error or recording error. Measurement errors were deleted and other errors were corrected where possible.

Table 2.8 below shows the number of individuals for whom accurate anthropometric data are available after cleaning at visit A and visit B. A large drop in the number of accurate body composition measurements can be seen for women, this is because the women were generally

fatter than the men (see chapter 3) and hence (a) they had more skinfolds >=40mm and (b) their skinfolds were harder to measure.

Table 2.8: The number of individuals for whom accurate anthropometric data are available after cleaning at visit A and visit B

	Visit A		Visit B	
	Men	Women	Men	Women
Total measured (inc. pregnant)	369	528	257	406
Height	364	521	256	402
Sitting height	359	508	257	403
Half-span	368	526	257	406
Weight	367	505	257	385
MUAC	367	504	257	386
BMI	363	500	256	382
Fat percent	331	360	246	354
Fat mass	330	360	246	354
Fat-free mass	330	360	246	354

Note the numbers seen in table 2.8 may drop further after individuals with no SES data are discarded from the analyses. For example, three HH (eight subjects) refused to give information on their savings and hence the sample size for analyses where the savings variable is used is reduced compared to those in table 2.8.

2.5.5 Measurement error

In longitudinal studies of anthropometry there is a need for reliable and precise measurements (Roche, 1992). An error in the consistency of measurement can either create spurious changes in stature or body composition or attenuate a legitimate change (Bailey, 1991). Both inter- and intra-observer error measurements were made during and after the study. The equation below was used to calculate the technical error of measurement (TEM).

$$(TEM)^2 = \frac{\Sigma D^2}{2N}$$

where, D is the difference between measurements
N is the number of individuals measured

(Ulijaszek & Lourie, 1994)

This equation can be used to calculate either intra-observer TEM or inter-observer TEM involving two measurers. If the TEM values approach a reference value and if there are no biases (i.e. systematic inaccuracies) in measurement, then the measurements can be considered accurate (Frisancho, 1990). Guidelines for reference values of TEMs for different age groups and measurements have been developed by Ulijaszek and Lourie (1994).

A TEM value has the same units as the anthropometric measurement it is assessing (e.g. cm for height), but a "coefficient of reliability" can be created in order to compare TEMs from different measurements by the following equation:

$$R = 1 - \frac{(TEM)^2}{(S)^2}$$

where S is the inter-subject standard deviation.

(Ulijaszek & Lourie, 1994)

The coefficient of reliability ranges from 0 to 1 and reveals what proportion of the between-subject variation is free from measurement error (Ulijaszek & Lourie, 1994). Some authors suggest that this coefficient may be used to compare the reliability of different measurements (Mueller and Martorell, 1988). However, different measurements have different dependability (sensitivity and specificity) and thus this coefficient may not really be useful in attempts to find the anthropometric measurement most sensitive to change but which is robust with respect to consistency and accuracy (Bailey, 1991).

Given that both Strickland (SSS) and Ulijaszek (SJU) measured subjects in 1990 and Duffield (AD) measured the subjects in 1996, it was necessary to calculate inter-observer errors between AD-SSS and AD-SJU. AD and SSS measured 15 Iban adults aged 20-70 years in the field during 1996. However, SJU did not visit Sarawak in 1996 and hence AD and SJU measured 15 students (aged 20-25 years) from Cambridge University in 1997. The inter-observer measurement errors (TEM, R, mean and percentage bias) found between AD and SS and AD and SJU can be found in tables 2.9 and 2.10. It was assumed that measurements made by SSS and SJU were more accurate than those made by AD since the former were more experienced. Thus bias was calculated as (SSS-AD) and (SJU-AD) and percentage bias as ((SSS-AD)*100%/AD) and ((SJU-AD)*100%/AD). Intra-observer measurements for AD are shown in table 2.11.

Table 2.9: Inter-observer measurement error between AD and SSS

	TEM	Coefficient of Reliability	Mean bias	t	p
Height (cm)	0.14	1.00	0.03 (0.02%)	0.63	
Sitting height (cm)	0.30	0.99	0.29 (0.36%)	3.54	!
MUAC (cm)	0.23	0.99	-0.17 (-0.63%)	-2.33	+
Biceps sf (mm)	0.64	0.89	-0.45 (-7.53%)	-2.14	
Triceps sf (mm)	1.87	0.88	-1.82 (-16.08%)	-3.55	!
Subscapular sf (mm)	2.99	0.86	2.87 (13.78%)	3.45	!
Suprailliac sf (mm)	3.08	0.87	-2.58 (-17.34%)	-2.74	+
BMI			-0.01 (-0.04%)	-0.54	
% fat			-0.45 (-4.68%)	1.18	
Fat mass (kg)			-0.23 (-4.68%)	-1.10	
Fat-free mass (kg)			0.23 (0.62%)	1.10	

NB no values were calculated for the errors for weight as different scales were used in 1990 and 1996

Table 2.10: Inter-observer measurement error between AD and SJU

	TEM	Coefficient of Reliability	Mean bias	t	p
Height (cm)	0.21	0.99	0.02 (0.01%)	0.25	
Sitting height (cm)	0.40	0.99	-0.31 (-0.34%)	-2.45	+
MUAC (cm)	0.60	0.97	-0.49 (-0.49%)	2.54	+
Biceps sf (mm)	0.80	0.91	0.25 (0.36%)	-0.81	
Triceps sf (mm)	0.84	0.97	-0.04 (-1.22%)	0.11	
Subscapular sf (mm)	2.30	0.49	-2.37 (-18.54%)	3.85	!
Suprailliac sf (mm)	2.30	0.89	-0.99 (-6.72%)	1.16	
BMI			-0.01 (-0.02%)	0.29	
% fat			-0.87 (-3.62%)	2.63	+
Fat mass (kg)			-0.57 (-3.62%)	2.60	+
Fat-free mass (kg)			0.57 (1.17%)	-2.60	+

NB no values were calculated for the errors for weight as different scales were used in 1990 and 1996

Table 2.11: Intra-observer measurement error for AD

	TEM	Coefficient of Reliability
Height (cm)	0.58	0.99
Sitting height (cm)	0.54	0.97
MUAC (cm)	0.35	0.99
Biceps sf (mm)	0.42	0.95
Triceps sf (mm)	1.63	0.91
Subscapular sf (mm)	1.96	0.94
Suprailliac sf (mm)	1.61	0.96

The intra-observer TEM values for AD are all lower than or equal to the reference values suggested by Ulijaszek and Lourie (1994). Furthermore, all the coefficient of reliability values are high. Thus the intra-observer errors are deemed to be acceptable.

The TEM values for stature, sitting height and the MUAC between AD and SSS or SJU are all also lower than or equal to the reference values. Similarly, all the R values are very high. However, a small significant difference between the mean values of the sitting height and the MUAC are observed between both AD-SSS and AD-SJU. No significant differences are observed for the mean values of BMI measured by AD, SSS or SJU.

The TEM values for the subscapular skinfolds are higher than those recommended by Ulijaszek and Lourie (1994) at a reliability value of 0.95. The AD-SSS coefficients of reliability for the skinfolds range from 0.86-0.89 and those for AD-SJU from 0.49-0.97. The R for the subscapular is particularly low, the percentage bias for this measurement is 13.8% for AD-SSS and 18.5% for AD-SJU. The t-tests for the subscapular skinfold are also found to be significantly different. The body composition variables are not significantly different for AD-SSS as the mean biases cancel each other out. However, the body composition variables are significantly different when measured by AD compared to SJU.

These results and those in section 2.5.3 suggest that MUAC, weight or height probably best fulfil the practical characteristics required for an anthropometric index of nutritional status. Accurate and precise skinfold thickness measurements are difficult to obtain and hence are not suitable as anthropometric indices.

2.5.6 Correction for bias

The results discussed above suggest that, for the skinfold measurements at least, AD's measurement techniques resulted in different measurements to those of SSS and SJU. It also appears that the TEM between SSS and SJU may be unacceptably high in at least one measurement (suprailliac). Given the need to make comparisons between the 1990 and 1996 data, the author decided to adjust the 1990 measurements for bias. The measurements made by SSS and SJU were corrected to become "AD measurements".

Regressions between the AD-SSS and AD-SJU measurements were run for each variable and the 1990 measurements were then altered accordingly. The equations for these transformations can be seen in Appendix A5.

2.6 Mortality data

As stated above, a VA was used to ascertain the causes of death from data obtained from the relatives or associates of the deceased. This technique was necessary as the death registration system in Sarawak is incomplete and certification of the cause of death by a physician was lacking in the vast majority of cases.

2.6.1 The verbal autopsy

The VA employed in this study was developed by Chandramohan et al (1994). It was originally developed for use in sub-Saharan Africa by obtaining frequency distributions of the causes of adult admissions and deaths recorded at hospitals in the region. Despite the fact that the VA was not specifically developed for use in Asia, local physicians in Sarawak found it to be appropriate for use in this study.

The VA has a combined open / closed format. The open section allows the interviewer to record the respondent's verbatim account of the illness and, in order to facilitate this, a table, to list the reported symptoms, their duration and severity, is included. The closed part of the questionnaire consists of stem questions to elicit the presence or absence of 40 symptoms. (The 40 symptoms were developed from the diagnostic criteria related to the mortality classification.) More detailed questions on duration, severity and other qualities of these symptoms form sub-questions which are only asked if the answer to the stem question is positive.

Translation of the VA took place during the pilot visit. Two medically trained individuals were asked to translate the English version into Iban independently of each other. Their results were then compared and discussed in the presence of the author. A rough draft was produced. Non-medically trained Iban individuals were then asked to explain what they understood the questions to mean. Eventually, a final draft (written in both English and Iban) was produced. This is shown in Appendix A6.1.

In all cases the VA was applied to the most appropriate respondent the author could find. The most appropriate respondent would be the person who cared for the deceased during their final illness. The respondent had to be able to remember, recollect and give an accurate account of the circumstances leading to the death and the signs and symptoms of the illness. In most cases, more than one respondent participated in the interview as the burden of caring for the deceased was shared between family members.

Two local physicians were asked to examine the completed VAs and apply their knowledge of symptomatology and the disease patterns of the area in order to reach a diagnosis of the cause of death. The VA has a mortality classification which includes six groups of causes of death. These are subdivided into 25 subgroups or categories and some of these are further divided into specific causes (see Appendix A6.2). Where no consensus as to the cause of death was found the VA was passed on to the original author of the VA (Chandramohan) who acted as an "arbitrator". Appendices A6.3 and A6.4 show the rates and principle causes of death by age and sex group.

2.6.2 Validation of the Verbal Autopsy

Ideally, a VA is validated by applying the questionnaire to the relatives of a deceased individual for whom cause of death was independently recorded by a physician. This method of validation was not used in this study because no certified death records were available. Instead, the validation simply involved asking two local physicians and Chandramohan to diagnose the cause of death from twenty-five VAs completed during the pilot. A comparison of their diagnoses revealed that the three physicians agreed on a diagnosis in twenty-four cases at the "group" level of mortality classification (a consensus rate of 96%). They also agreed on twenty-one diagnoses at the "subgroup" level of mortality classification (a consensus rate of 84%).

2.7 Morbidity method

As stated in the introduction, the relationship between adult anthropometrical status and morbidity experience is complex and may be circular. In order to examine this relationship it is necessary to study the morbidity experience of initially healthy adults longitudinally. Thus the method of morbidity data collection in this study had to include a way in which to (i) differentiate between chronically ill and healthy individuals at the baseline and (ii) record morbidity data longitudinally.

Morbidity measures can be classified as either observed or self-perceived. Observed morbidity consists of reports from clinicians or other investigators about illnesses they have observed in the people they examine or test. Self-perceived morbidity consists of reports from people about their own illnesses (Murray et al. 1992). The deviation from a normal state of health is the usual definition of morbidity. This concept is closer to illness in a self-perceived survey, but is closer to disease in an observed survey.

Observed measures of morbidity generally involve measurement of physical and vital signs, physiological and patho-physiological indicators, functional tests and clinical diagnoses. This method would probably have been the ideal way to assess the presence of chronic disease at the baseline. However, such a survey could not be undertaken because of the cost and logistical problems involved. Instead, measures of self-perceived morbidity were used. The major problem of this method is that self-perceived measures of morbidity depend not only on the subjects' actual morbidity experience but also on their perception of illness and their illness behaviour.

Morbidity is notoriously difficult to measure and the restraints imposed upon this part of the project by budget and time are apparent. It must be noted that the method described below is less than ideal and represents a compromise between scientific ideals and practical restraints.

2.7.1 Methods employed in measuring morbidity

1. In the first round the author applied a morbidity recall questionnaire to each subject. In addition to this a further questionnaire was applied to assess the presence of chronic illness at the baseline. At this stage the author administered a sputum test for Tuberculosis (TB) to each subject.
2. During the second round of visits the author re-applied the morbidity questionnaire to each subject. Anaemia status was assessed by measuring haemoglobin levels.
3. Between the author's visits local interviewers collected basic information on health from each subject every two weeks.

The questionnaires which were employed can be found in Appendix A7.1. A two week morbidity recall period was used as this was long enough to include a reasonable number of illness episodes within the sample size and short enough to minimise the problems of recall errors (Ross & Vaughn, 1986, Kroeger, 1983, Murray et al, 1992, Martorell et al, 1976).

The morbidity recall questionnaire started with the stem question "have you been ill at all in the last two weeks ?". If the answer to this question was negative the interviewer would immediately skip to the chronic illness questionnaire. If the answer to the stem question was positive the interviewer would use the symptom tracer list to assess the nature of the individuals illness. The stem question was used before the tracer list in order to reduce the possibility of over-reporting due to prompting (Kroeger, 1983, Belcher et al, 1976).

The symptom list was developed after a survey completed in the pilot study. Longhouse residents were asked by a local doctor and the author to describe the illnesses / symptoms they had suffered from over the past six months. Medical records were also referred to. The seven most commonly reported symptoms (accounting for approximately 80% of complaints) were originally utilised.

Having run through the symptom list, more detailed questions were posed about the duration, severity and treatment of each symptom. The activity restriction questionnaire was then applied to all individuals who reported illness in the two weeks prior to interview. This questionnaire attempted to determine whether ill health had prevented the respondent from carrying out his normal activities in the previous 14 days. The respondent was simply asked if they had had to curb their normal activities during the past two weeks because of illness or injury. Further questions assessed how many (whole or partial) days of work were missed because of the illness.

The type of work (farm / household / other) affected and the "causal" symptom was also recorded.

Questions concerning functional ability or "restricted activity" are highly dependent on the nature of the individual's usual activity; an action criterion may mean very different things for a housewife with small children and an elderly retired man (Ross & Vaughn, 1986; Kroeger, 1983). Care was taken to ensure that the subjects understood that an interruption of their normal activities due to illness should be reported regardless of what their normal activity might be. Thus it was hoped that elderly grandmothers who were too ill to do the cooking were as likely to report a break in their activities as young men who were too ill to work on the farm.

On the basis of the questions concerning activity restriction, three categories of morbidity were employed during the analysis (i) illness during the two weeks prior to interview, (ii) illness on the day of interview, (iii) illness which resulted in the individual having to alter their normal activity patterns (including visits to the doctor). Category (i) was a general overview of the subject's health experience two weeks prior to the time of interview. Category (ii) was a tighter measure of illness experience than (i) and is less subject to the vagaries of recall, but yields a lower overall morbidity rate. By employing a salience principle, category (iii) was the most stringent measure of morbidity.

The questionnaire method was also used to assess the presence of chronic illness. Hence "chronic illness" was a self-perceived condition. The author made an arbitrary decision to classify a chronic illness as an illness or complaint which an individual had been suffering for more than one month. All the subjects were asked whether or not they suffered from a long term illness. Questions concerning the nature, severity, length and treatment of the chronic complaint(s) were posed. A question asking whether or not this complaint had prevented the individual from undertaking their usual activities attempted to classify the chronic illness as debilitating or not.

The author took a sputum sample from each subject during the first round of visits in order to diagnose TB. This was included in the protocol because it was felt to be particularly important to identify individuals with a chronic disease at the baseline which effects nutritional status. TB, which is endemic in Sarawak (Medical and Health Services Department Sarawak, 1990), is known to affect nutritional status. The Sarawaki Health Department undertook to analyse the sputum for TB as part of their communicable diseases programme.

Haemoglobin (Hb) status was measured using the HemoCue Haemoglobinometer for all adults who consented at visit B. This instrument photometrically determines the Hb concentration of a whole-blood sample without dilution, using disposable cuvettes containing three reagents. Blood samples were taken from a fingertip. The calibration was checked before use by means of a cuvette fitted with a red filter. The WHO cut-off points for anaemia (Herceberg et al. 1991) were used i.e.: 13.0g/dl for men and 12.0g/dl for women.

In order to collect morbidity data from the subjects between the author's visits (i.e.: repeated measures of morbidity over a six month interval), volunteers from each longhouse were trained by the author to collect morbidity data using a simple questionnaire. Unfortunately this part of the method was unsuccessful. The problems encountered are described below.

2.7.2. Problems with the morbidity method

As stated above, this method resulted from a series of compromises and was not perfect. Several major problems arose during the main phase of the study which had not been foreseen in the pilot these included - (i) hypertension, (ii) the TB tests and (iii) the data collection by local interviewees.

Hypertension (HT) was frequently cited as a chronic disease but did not appear on the original tracer list of symptoms. It was not considered a potential symptom in the pilot study because its diagnosis cannot be made by non-medical personnel. Thus an individual who reported being hypertensive only knew this because a doctor or nurse had told them, and those hypertensive individuals who had never seen a doctor (or at least not since they had developed HT) would be unaware of the problem. Hence a bias in reporting of HT could occur between individuals who visited the doctor or nurse and those who did not.

At the start of the main phase the author decided to include HT in the questionnaire and analysis for four reasons (i) access to medical care in the study area is relatively easy and most of the respondents were aware that HT could cause headaches (and other symptoms) and hence would go to the hospital if they had recurring headaches, thus it seemed likely that most cases of HT would be diagnosed, (ii) HT was widespread, (iii) HT is associated with other symptoms which may also be symptoms of other (infectious) diseases - it was important in the analysis to attempt to separate out the different disease types, and (iv) HT is known to be associated with nutritional status.

All the results of the TB tests were negative. Both the author and the Sarawaki medical authorities found this result difficult to believe given the levels of TB in Sarawak. It is possible that there was some error either in the method collection of the sputum or the analysis. The technicians in the hospital may also have been overwhelmed by the number of tests they were required to carry out in a relatively short period of time and may not have examined the slides carefully enough. In an attempt to rectify this situation, two new questions concerning TB were added to the morbidity questionnaire in the second round. The subjects were asked if they had ever suffered form TB and, if so, if this had occurred since 1990.

An attempt was made to train local interviewers to apply morbidity questionnaires to the inhabitants of a longhouse for the interval between the author's visits to the longhouses. Unfortunately this part of the project failed. This was probably due to a combination of reasons:- (i) poor training of the interviewers by the author - the training period was short, (ii) the interviewers' apathy - possibly because they were not paid enough, and (iii) the lack of enthusiasm amongst the interviewees for the project - possibly because the purpose of the project was not well enough explained. Following this failure it was decided that the morbidity analysis would focus on the data collected by the author at each of her two visits to the longhouse.

2.7.3 Morbidity data organisation

There is no biological basis for assuming that the BMI or MUAC and all the symptoms in the questionnaire are associated in the same manner. The symptoms are allied with many different diseases the physiological mechanisms of which are varied and hence the relationships observed between the symptoms and the BMI or MUAC will not all be identical or, indeed, in the same direction.

Studies assessing the relationship between the BMI and mortality risk have frequently reported a U- or J-shaped relationship (see chapter one). Mortality risk may be raised in subjects with both low and high BMI. Certain diseases are positively associated with the BMI and others are negatively associated with it. Waaler (1984) refers to the high-weight and low-weight groups of disease. In the developed world, typical high-weight or high-BMI diseases include the cardiovascular and cerebrovascular diseases, breast cancer and late onset NIDDM and its sequelae. Typical low-weight or low-BMI diseases include tuberculosis, obstructive lung diseases, certain types of cancer e.g. lung cancer (Kabat et al, 1992). In the developing world, infectious diseases may make up much of the morbidity burden of a population. Many important

infectious diseases such as acute diarrhoeal disease (Scrimshaw et al. 1968; Briend, 1990; Tomkins & Watson, 1989) are associated with low-weight in the developing world.

Table 2.12 shows the various symptoms and the direction in which they may be expected to relate to the BMI or MUAC, taking biological disease processes into account. One of the difficulties in using a symptom list is that it is not possible to determine the underlying disease with which a symptom may be associated. Certain symptoms such as cough, chest pain and respiratory disease may, for example, be associated with cardiovascular problems or result from a bacterial infection. An attempt to disaggregate these symptoms has been made by treating any headache or cough, chest and respiratory problem reported in conjunction with hypertension separately from a headache, cough, chest or respiratory problem reported without hypertension. Also, epigastric pain may, for example, be due to a cancer or an intestinal infection. Gastric cancer may be associated with high BMI in young adults (Hansson et al, 1994), on the other hand the a priori expectation of intestinal infections is that they would associate negatively with the BMI. Given that the study population lives in the developing world and has a relatively high exposure to infectious disease, epigastric pain has been placed in the negative association group as it is expected that infectious diseases would be the most common cause of epigastric pain in this population.

Table 2.12: The direction of the association between the BMI or MUAC and various symptom types expected

Incidental association	Positive association	Negative association
Injury	HT	Fever
Muscle ache	Headache (assoc. with HT)	Diarrhoea
Headache (not assoc. with HT)	Cough (assoc. with HT)	Gastric pain
Other	Chest (assoc. with HT)	Cough (not assoc. with HT)
	Respiratory (assoc. with HT)	Chest pain (not assoc. with HT)
		Respiratory (not assoc. with HT)

It can be seen that the diseases which are expected to associate positively with the BMI or MUAC are similar to Waaler's high-weight group. The negative association group represent symptoms which are commonly found with infectious diseases (e.g.: fever, diarrhoea) and various lung diseases. The incidental group represent diseases which are not expected to associate with the BMI or MUAC in a particular direction. For example, it is possible that an injury or muscle ache could result in leanness due to an inability to work or, as one study reported, that obesity is

associated with increased severity of ankle fracture following low velocity injuries (Spaine & Bollen, 1996).

It should be noted that the associations shown above are not necessarily assumed to be causal. For example, fever may be associated with low BMI or MUAC because (a) a thinner person may be more likely to catch a febrile disease, or (b) an individual with a fever may have a depressed appetite and hence become leaner, or (c) an individual with a fever may have an increased basal metabolic rate and hence greater energy expenditure, or, finally, (d) a person with a febrile disease may be unable to work and hence have less to eat. Note that these explanations are not exclusive and febrile disease and low BMI or MUAC may be linked for more than one reason.

In order to overcome the problem of small numbers of individuals reporting certain diseases some of the symptom categories have been combined. A group known as 'incidental' is made up of injury, muscle ache and headaches not associated with hypertension. A group known as 'respiratory with hypertension' includes cough, chest pain and respiratory problems in conjunction with hypertension. A group known as 'epigastric' will include gastric pain and diarrhoea. A group known as 'respiratory no hypertension' includes cough, chest pain and respiratory problems not in conjunction with hypertension. Note that an individual may report more than one symptom group.

Thus in this study, "illness" is defined as a complaint of either epigastric illness, fever or respiratory problems not found in conjunction with HT.

2.7.4 Validation of the morbidity data

Validation of morbidity data is almost as complex as the actual collection of the data itself (Schulpen & Swinkwels, 1980; Cochrane et al, 1951; Ross & Vaughn, 1986; Kroeger, 1983). The repeatability of the morbidity questionnaire was tested in the pilot study by a doctor applying the questionnaire to the same 25 individuals the next day. No substantial under- or over-reporting was found. None of the subjects was physically examined by the doctor (which would have been a more stringent method of validation). However, medical cards were examined and these were found to be in agreement with the subjects' answers in 96% of the cases.

2.8 Sample Size Calculations

The section below describes the sample size calculations undertaken by the author when she first proposed to undertake the study. Some of these calculations (particularly those for the morbidity section) may not be appropriate as the methods changed during the course of the study. However, the original calculations are shown.

2.8.1 Follow-up study

The sample size of the follow-up study was fixed. In 1990 Strickland & Ulijaszek (1990) measured and interviewed 1047 adults who formed the basis of the follow-up study. Thus true sample size calculations were not estimated. Instead the calculations show the size of the relative risk (RR) this study could expect to find, given a certain power.

In order to perform a sample size or power calculation for this part of the study an estimate of the mortality of the population during the period 1990-1996 had to be made. Ideally, the number of expected deaths in the 1990 population should have been calculated by applying age specific death rates (ASDRs) for a rural Iban population to the 1990 age and sex distribution data.

However, accurate data on Iban mortality was not readily available. ASDRs for the Iban were calculated by Lam (1981) using the intercensual survival technique (Shyrock & Siegel, 1976) on the 1947, 1960 and 1970 Sarawak censuses. These ASDRs were too dated to be of use to the project as Sarawak's mortality levels have decreased considerably since Lam's work (Lam, personal communication). An attempt was made to use the intercensual survival technique to deduce ASDRs indirectly from the 1980 and 1990 Malaysian censuses. This effort failed as the two censuses were not directly comparable.

The only alternative left to the author was to select a model life table on which to base mortality estimates. After discussion with Timaeus and Lam and reference to Leete (1994) it was decided that Coale & Demeny's (1983) West models (levels 18 and 22) would probably be the most appropriate models to predict the minimum and maximum number of deaths expected over the period. The mortality rates of these model populations were applied to the 1990 data. The results can be seen in Appendix A8. Using West level 22, 88 deaths can be expected over 7 years, using level 18, 119 deaths could be expected by the end of 1996.

The following assumptions were made in order to calculate the RR of mortality for low BMI adults which the study could detect:

1. The mortality estimates calculated suggested that the mortality of the adults from the 1990 study would probably be in the region of 10% by the end of 1996.
2. In 1990 there were 190 low BMI (BMI<18.5 kg/m^2) adults and 822 normal BMI adults (BMI>18.5kg/m^2).
3. Following normal conventions, the power of this study would be 80% and a 5% significance level would be sought.

The equation below was used to estimate the power which the study could expect to find (Kirkwood, 1992):

$$(p_1-p_2)^2 = p_1\frac{(1-p_1)}{n_1} + p_2\frac{(1-p_2)}{n^2} * (Z_{\alpha/2} + Z_\beta)^2$$

Where, p_1=rate of mortality in normal BMI subjects

p_2=rate of mortality in low BMI subjects

n_1=number of normal BMI subjects in 1990

n_2=number of low BMI subjects in 1990

$Z_{\alpha/2}$=percentage point of the normal distribution corresponding to a significance level of 5%

Z_β=percentage point of the normal distribution corresponding to 100%-80% (power)

The author concluded that the study would have 80% power to detect a relative risk of mortality of 1.85 between low BMI adults and normal BMI adults, assuming a significance level of 5%. Given that the only other study which has looked at this RR in the developing world (Satyanarayana et al. 1991) found increased risks of mortality of between 1.1 and 2.7 among CED subjects compared to non-CED subjects, the author suggested that the power to detect an RR=1.7 was a reasonable basis for this study.

2.8.2 Longitudinal Study

The following assumptions were made when calculating the size effect which could be shown with this study -

1. The maximum number of subjects involved in the study would be 792. This figure was reached by allowing for a mortality rate of 10% since 1990 and assuming that 13% of the remaining population had migrated out of the area (Padoch, 1982).
2. A similar BMI distribution to that seen in 1990 was assumed. In 1990 19% of the population were CED. Thus it was assumed that 150 adults would be CED in 1996.
3. The calculation was dependent on the probability of missing work due to illness for a given number of days over six months. Following a survey in the pilot study, it was assumed that the probability of missing work for zero day in six months would be 50% and that the probability of missing one day of work would be 10%. These probabilities were crucial to the calculation, so the calculations were repeated assuming the probability of missing work for zero days would be either 25% or 75%.

The calculation involved in predicting the sample size was complex as the outcome of interest (numbers of days unable to work over six months) was an ordered scale, not simply a binary result. Following the method of Campbell et al (1995) the following formula was used -

$$m=\frac{6(Z_{\alpha/2} + Z_{\beta})^2/(\log OR)^2}{[1- \sum^{k}_{i=1} p_i^3]}$$

Where m=sample size (assuming equal sized groups, see correction below)

OR=odds ratio of an individual being in morbidity category i or less for CED compared to non-CED individuals

k=number of categories

p_i=is the mean proportion expected in morbidity category i

$Z_{\alpha/2}$=as above

Z_{β}=as above

Unequal sample size correction formula:

$$n=\frac{r+1}{2r} * m$$

Where r is the allocation ratio (642/150=4.28)

The author concluded that the study would have 80% power to detect an odds ratio between morbidity categories of 1.7 between CED and non-CED adults if the probability of missing zero days work in six months equals 50%, assuming a significance level of 5% and a power of 80%.

If the probability of missing zero days work in six months was equal to 25% or 75% then the study would be able to show RRs of 1.6 and 1.8 respectively.

Given that Pryer's (1990, 1994) cross-sectional study of days of work missed due to illness and BMI in Bangladeshi men found that the RR of being incapacitated from wage work for one or more days was between 1.8 and 5.9 times higher for Bangladeshi men with low BMI compared to those with normal BMI the author concluded that the morbidity section of the study would be worthwhile

2.9 Analysis

Both Epi-info 6.04b and STATA 5.0 were used for data analysis. A brief outline of the analytical techniques employed is found below.

The initial stage of analysis involved calculating descriptive statistics for the data set (ranges, means, medians and SDs). The normality of distributions was examined by assessing normal plots and undertaking formal tests for skewness and kurtosis. Cross-tabulations, graphs and frequency tables allowed the distribution of the variables to be observed. Further analyses used orthodox methods of Chi square, t-test, analysis of variance (ANOVA), simple and multivariate linear regression, logistic regression and Cox regression. The chi square test p values given are those after the continuity correction has been applied. The p values for t-tests, F-test and Chi square all apply to two-tailed tests unless stated otherwise.

2.9.1 Anthropometry

Most of the anthropometric variables had a skewed distribution. Thus the variables were normalised using box-cox transformations when their determinants were examined (chapter 3). The differences between the 1990 and 1996 anthropometric measurements were normally distributed for all the measurements except the body composition variables and only those which were skewed were transformed.

Changes in the anthropometrical status of adults over the six year and six month periods were analysed using t-tests. The effects of age, sex and other SES and "lifestyle" variables were assessed by examining the amount of variance in the anthropometric indices explained when these variables were added to backwards stepwise regressions (see below).

2.9.2 Mortality

Survival analysis techniques were employed to relate anthropometry to mortality. Cox regression was used as the preferred technique as it takes survival time into account, although logistic regression could have been employed. A hazard ratio (HR) of survival was obtained either using the anthropometric measurements as continuous variables, or by comparing the survival of those subjects with values of a given anthropometric measurement in the lowest quintile compared to the rest of the population. An HR of more than 1.00 implies that an increase in a measurement will result in an increased risk of mortality, conversely an HR of less than 1.00 implies that as the anthropometric variable decreases the risk of mortality increases. An HR equal to 1.00 implies that the risk of mortality does not change as the anthropometrical variable increases or decreases.

2.9.3 Morbidity

Initial analysis included tables showing the point prevalence of different types of illness by sex. Further analysis employed logistic regression to obtain odds ratios (ORs) relating the anthropometric variables to morbidity. The anthropometric measurements were entered either as continuous variables, or as CED non-CED. Similar to the HR, an OR of less than 1.00 indicates that as the anthropometric variable decreases the odds of reporting illness increase and the converse is true for an OR of more than 1.00. At an OR equal to 1.00 the odds of reporting illness do nor change as the anthropometric variable increases or decreases.

2.9.4 Potential confounding factors

Potential confounding factors such as age, socio-economic status, geographical location and smoking habits were taken into account when examining the relationships between anthropometrical status and mortality and morbidity.

Given the co-linearity of many of the SES variables, during the analysis the SES variables were entered into the regressions using backwards elimination stepwise methods. Those candidates with the highest p-values (using the log likelihood ratio test) were removed from the regression until only those with a p-value<0.05 remained. Thus the method "weeded-out" the co-linear SES variables and kept only the most significant confounders.

CHAPTER THREE

ANTHROPOMETRY 1990-1996

The purpose of this chapter is to describe the distribution of the anthropometric variables in the study population in 1996. Changes in the anthropometrical variables over the follow-up period will also be assessed. Furthermore, the determinants of both the 1996 anthropometrical variables themselves and the changes observed since 1990 will be examined. This chapter will assess whether or not each of the anthropometric variables are (i) independent of sex and age, and (ii) associated with other (socio-economic or lifestyle) variables.

3.1 Anthropometry in 1996

3.1.1 Distribution of the anthropometrical variables in 1996

The data presented in this section are cross-sectional. This may result in difficulties in explaining any differences seen between age groups. If a difference exists between generations in cross-sectional data it is not possible to state whether the difference exists because of a secular trend or because the older age group has undergone real physiological changes (Borkan, 1986). The longitudinal data presented later in the chapter should help to clarify the causes for these differences.

Figures 3.1-3.10 show the study population's distribution of height, weight, BMI, MUAC and energy stores with respect to age and sex in comparison to the NHANES reference data (Frisancho, 1990). Figures 3.1-3.10 show the medians and the 95% confidence intervals of the ICGN data compared to the 5th and 50th centiles of the NHANES data. The NHANES data was representative of non-institutionalised U.S. civilians aged twenty-five to seventy-four years and was collected in a cross-sectional survey by the NCHS (National Center for Health Statistics). Some authors have suggested that these references may not be appropriate for comparative purposes as the levels of obesity found in N. America are too high and fat patterning may differ between the races (Wang, 1994; Davies et al, 1986; Jones et al, 1976). The data have been used in this study for two reasons - (i) they are the most complete references available and (ii) although the populations are compared in terms of anthropometric status, no value judgements as to which is "best" in terms of health are made.

Figures 3.1 and 3.2 show median height by age for the Iban men and women measured in 1996 compared to American reference data. Both the male and female Iban groups are shorter than even the 5th centile of the NHANES reference data. Male Iban are taller than the females at all ages. From aged 40+ the older groups of both men and women have lower median heights than the younger groups. These differences are greater above aged 60.

Figures 3.3-3.8 show the median weight, BMI and MUAC for Iban men and non-pregnant women compared to their American counterparts. The distribution of these variables are remarkably similar. In Iban men the median values of weight, BMI and MUAC decrease as age increases after age 30. The median male Iban values remain well below the American's median values at all ages and, for weight and MUAC at least, closely resemble the American 5th centile group.

Iban women's weight, BMI and MUAC distributions with age are similar. However, the women's pattern of change in these variables with age is distinct from both that of Iban men and American women. The median weight, BMI and MUAC of the youngest Iban women are much higher than those of the older groups - a steep decline in median values of these variables is seen with increasing age. Indeed, the median BMI and MUAC of the youngest Iban women are greater than those of the Americans, but the oldest groups' median values are below the 5th centile. Thus the Iban women show a much greater spread of weight, BMI and MUAC than the other groups described. This greater spread is also evident when examining the error bars within each age group - the women have greater SEs than the men.

Figures 3.9 and 3.10 show the median fat percent for Iban men and non-pregnant women in 1996 compared to the American reference data (Frisancho, 1990). It should be noted that the fat percent is probably under-estimated throughout this study because individuals who had at least one skinfold reading of more than 40mm were discarded from this analysis (see methods section).

At any given age, the women have a higher percent fat than the men (this is true for both populations). The median fat percent of the male Iban population is between the 50th and 5th centile of the American population at all ages. The lowest median fat percent is found in the youngest and oldest groups of male Iban, the middle-aged men have higher values of fat percent. The younger Iban women have a higher percent fat than the 50th centile of the young American women, however after age 45 the Iban women show a decrease in percent fat and drop below the 50th centile of the NHANES data. In contrast, the American women increase their fat percent steadily with increasing age. As most of the fattest women were found in the younger age groups

but were excluded from the analysis it is possible that in reality a decrease in percent fat (and FM) actually exists with increasing age across the whole female Iban population.

Figure 3.1: Median male height by age in 1996 (n=361)

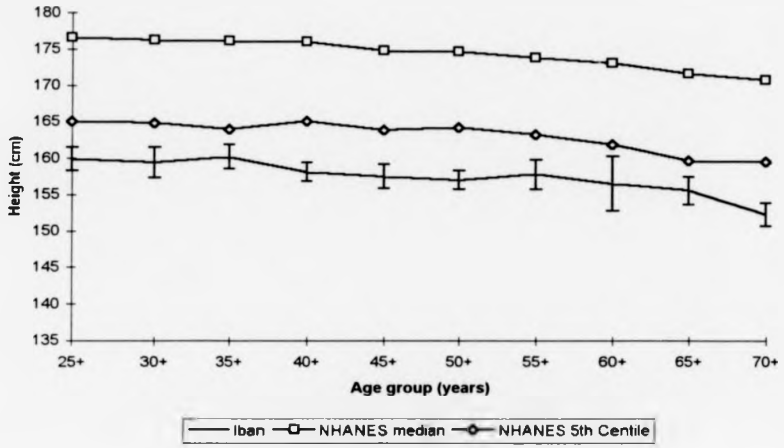


Figure 3.2: Median female height by age in 1996 (n=511)

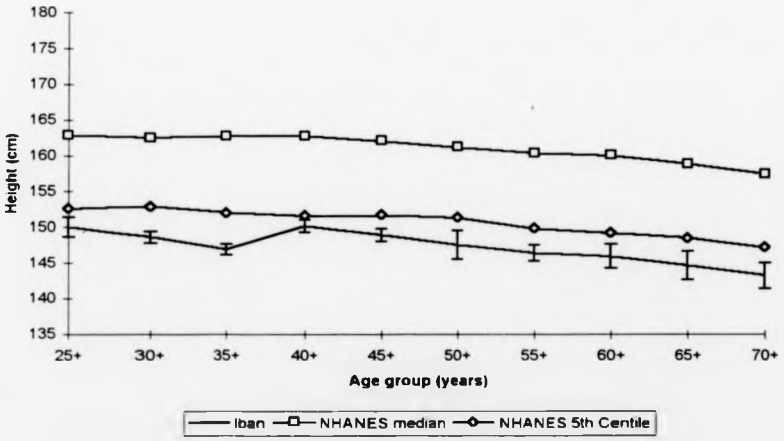


Figure 3.3: Median male weight by age in 1996 (n=364)

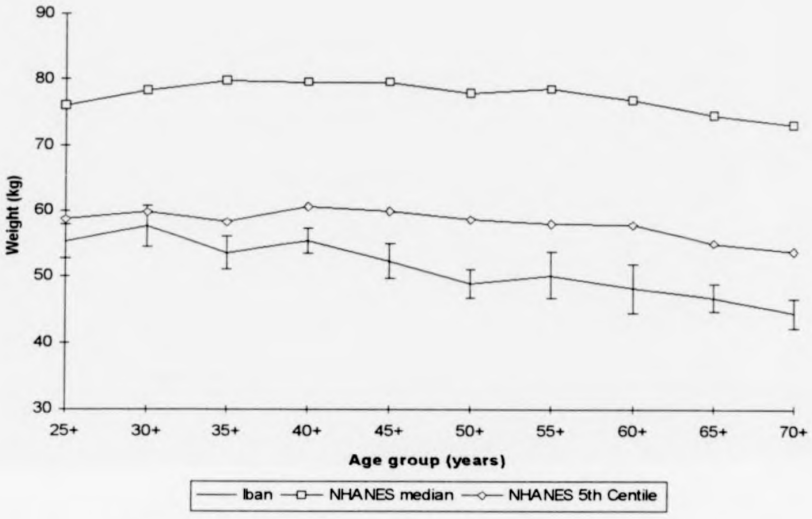


Figure 3.4: Median female weight by age in 1996 (n=495)

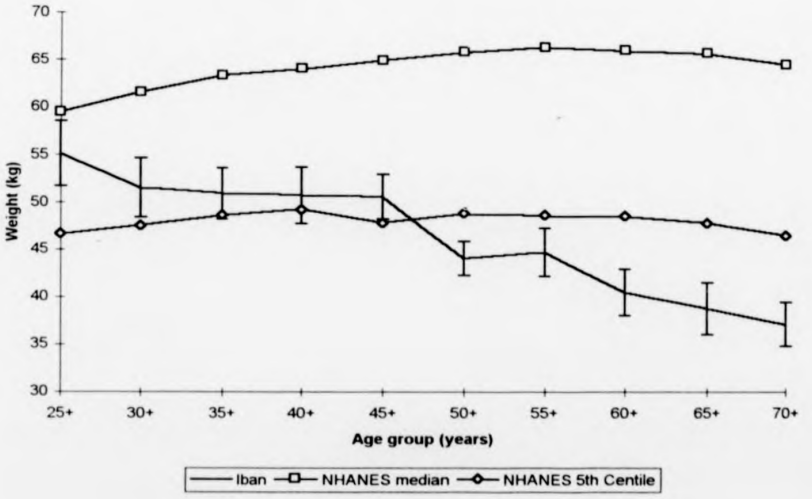


Figure 3.5 Median male BMI by age in 1996 (n=360)

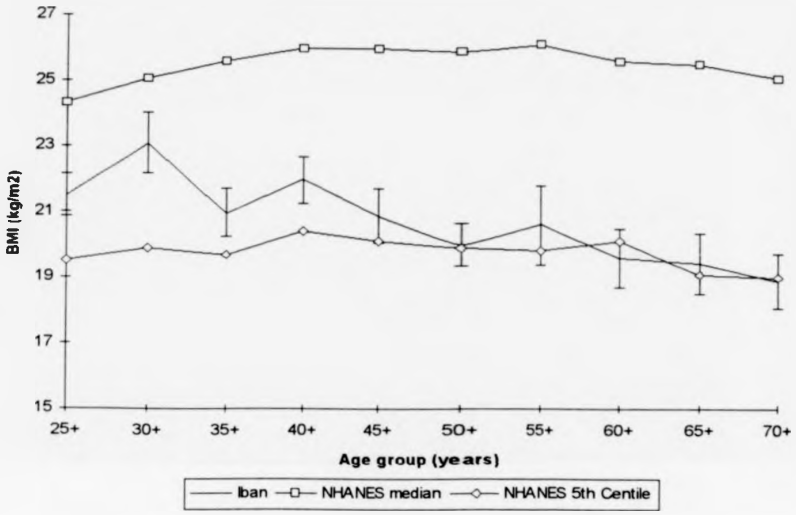


Figure 3.6 Median female BMI by age in 1996 (n=490)

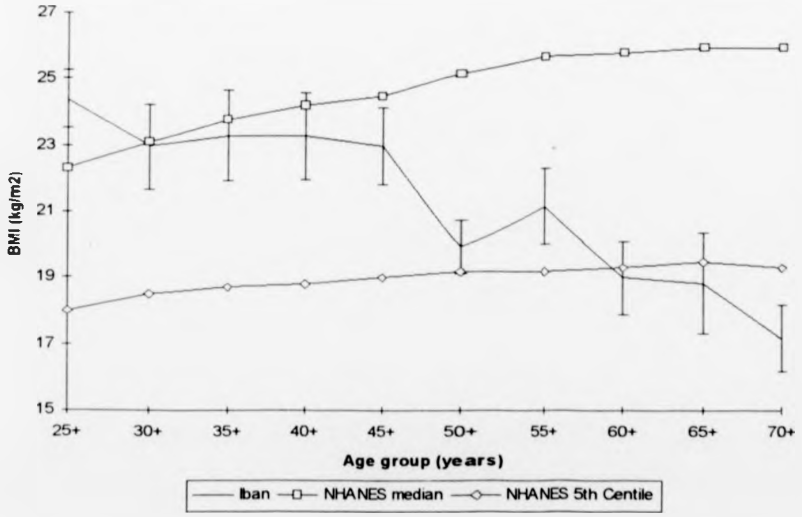


Figure 3.7: Median male MUAC by age in 1996 (n=364)

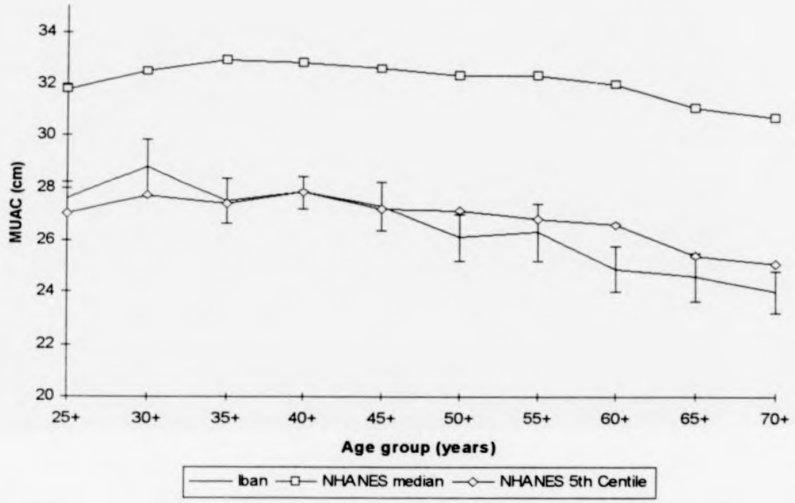


Figure 3.8: Median female MUAC by age in 1996 (n=494)

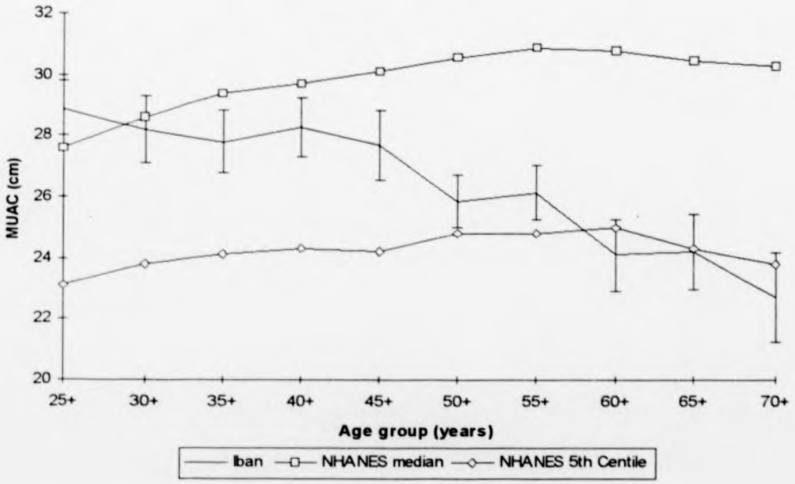


Figure 3.9: Median male percent fat mass by age in 1996 (n=331)

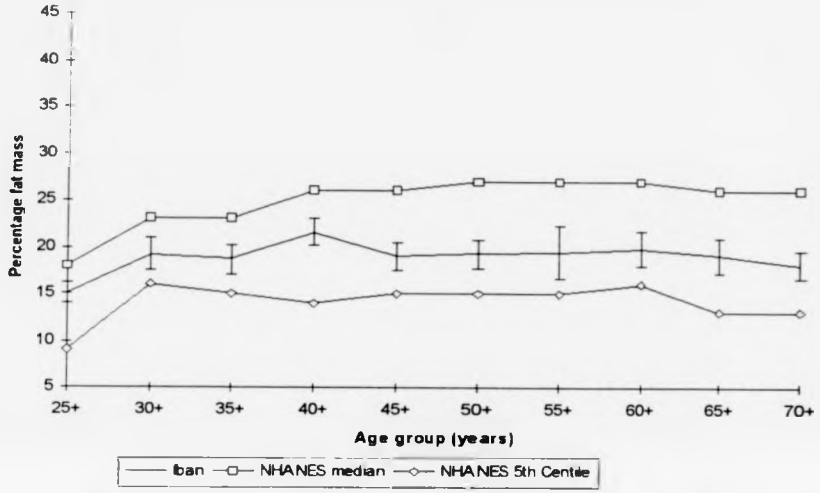


Figure 3.10: Median female percent fat mass by age in 1996 (n=360)

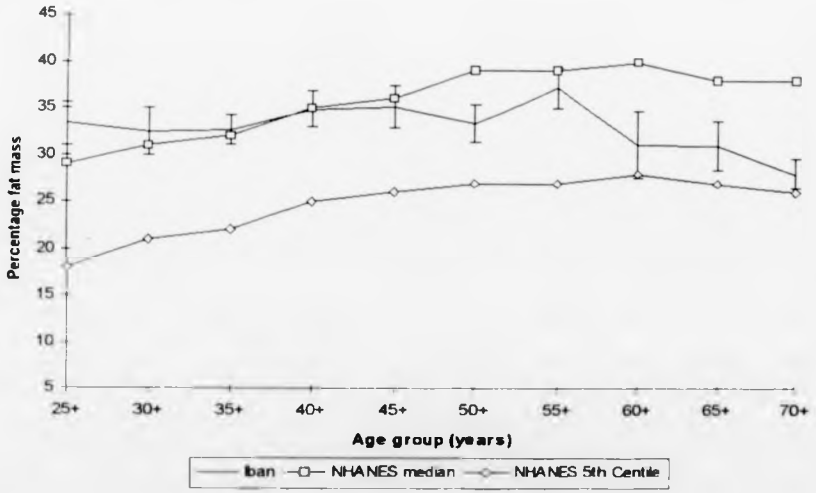


Table 3.1 shows the distribution of BMIs from a selection of countries in the developing world. This table is only intended as a basic overview of BMI data from the developing world and it should be interpreted carefully as some of the studies were conducted more than 15 years ago and some may not be representative of the whole population. As some of the studies shown assess all age groups and some concentrate only on younger age groups both types of data have been given for the Iban in 1996. Note that where possible mean age or age range has been given. The mean Iban BMIs are greater than those found in the Indian, Ethiopian or Vietnamese study and are similar to those found in China or PNG.

Table 3.1 *Mean and Median BMIs by sex from various studies conducted in the developing world*

Location	N	Sex	Age	Mean	Median	SD	C.V. (%)
PNG[1]	527	Male	40	21.9	21.8	2.0	
	546	Female	38	20.9	20.8	2.3	
Ethiopia[2]	159	Male	40	19.0	18.9	1.7	
	197	Female	35	19.2	18.9	2.1	
Rwanda[3]	964	Male		20.6	20.4		9.9
	1,104	Female		21.2	21.0		12.2
India[4]	9,447	Male		18.9	18.6		13.7
	11,914	Female		19.0	18.6		15.5
Brazil[5]	60,296	Male		22.4	21.9		14.4
	62,343	Female		22.9	22.1		18.8
China[6]	963	Male		21.1	20.7		11.4
	1,463	Female		21.6	21.2		13.9
Vietnam[7]	3,213	Male	30-39	19.6		1.9	
	3,921	Female	30-39	19.4		2.0	
Nepal[8]	231	Male	48		20.7	2.1	
	186	Female	43		20.4	2.3	
Iban	118	Male	25-40	22.3	21.8	2.9	
	169	Female	25-40	24.1	23.5	4.2	
	363	Male		21.1	20.7	2.9	
	500	Female		22.0	22.3	4.3	

Sources: 1. Papua New Guinea (Norgan & Ferro-Luzzi, 1982) 2. Ethiopia (Ferro-Luzzi, 1990) 3. Rwanda (Francois, 1990, adapted from Shetty & James, 1994) 4. India (NIN, adapted from Shetty & James, 1994) 5. Brazil (IBGE, adapted from Shetty & James, 1994) 6. China (NINFH, adapted from Shetty, 1994) 7. Vietnam (Giay & Khoy, 1994) 8. Nepal (Strickland & Tuffrey, 1997)

3.1.2 Distribution of CED and overweight grades in 1996

Table 3.2 gives the 1996 distribution of CED and overweight grades as defined by the BMI alone. The majority of the population have a "normal" BMI and few are classified as either obese or grade III CED. Women show a larger spread of values than men.

Table 3.2: The % distribution of CED and overweight grades defined by the BMI alone

	Men (n=361)	Women (n=498)	Total (n=859)
Grade III CED	1.4	5.2	3.6
Grade II CED	2.8	3.8	3.4
Grade I CED	11.9	10.3	10.9
Normal	73.9	57.6	64.5
Overweight	7.5	16.5	12.7
Obese	2.5	6.6	4.9

Table 3.3 below shows the distribution of CED individuals using the three different methods described in chapter one. It can be seen that using the MUAC alone reduces the number of individuals classified as CED by approximately half in both sexes compared to using the BMI alone. Eight individuals who were not diagnosed as CED using the MUAC alone were diagnosed as CED when the BMI and MUAC were used in conjunction. These eight individuals had a BMI <17.0 but a normal MUAC.

Table 3.3 The % distribution of CED individuals diagnosed using either the BMI, the MUAC or both in combination

	BMI	MUAC	BMI & MUAC
Men (n=361)	16.1	8.9	8.0
Women (n=498)	19.3	9.8	11.2
All (n=859)	17.9	9.4	9.9

3.2 The associations between the anthropometrical variables, sex and age in the 1996 data

The basis of the analyses in this section is the data set for the 601 non-pregnant individuals for whom all anthropometric and socio-economic data are available. All the anthropometric measurements and indices were normalised using box-cox transformations (Box & Jenkins, 1964) to reduce their skew.

Examination of the cross-sectional data above shows that all of the anthropometrical variables considered in this study are associated with age and / or sex in some way. The analyses below assess the nature of these relationships and calculate the amount of the variance in the distribution of the variables which can be explained by age and sex. As the anthropometric measurements and indices are not all associated with age and sex in the same manner four models were examined for their fit to the data. The models were as follows -

I $var=a + b(sex) + c(age)$

II $var=a + b(sex) + c(age^2)$

III $var=a + b(sex) + c(age) + d(sex*age)$ where * = interaction

IV $var=a + b(sex) + c(age^2) + d(sex*age^2)$

The regression which explained the most variance is taken to give the best explanation for the relationship. The associations of the BMI, weight and FM with age and sex are best explained by equation IV. These variables decrease with increasing age in a non-linear manner which differs between the sexes, i.e. men and women's BMI, weight and FM decrease with age at different rates (women's decrease more steeply). Stature, sitting-height, MUAC and FFM differ from the other variables in that no interaction terms are seen between age and sex i.e. the gradients are the same for men and women but the intercepts are different. The relationships of height and MUAC with sex and age are best explained by equation II, i.e. they decrease in a non-linear manner with age. FFM decreases in a linear fashion with age and is best explained by equation I. Fat percent decreases linearly with age in women but increases slightly for men (equation III). The Cormic Index (CI) also decreases with increasing age because sitting height decreases more than leg length with ageing (equation IV). Demi-span decreases in men, but not women indicating that some secular trend is seen in male growth in this population (equation I). Figures 3.11-3.19 clearly show the relationships described above.

Figure 3.11: The male and female distributions of weight by age (equation IV)

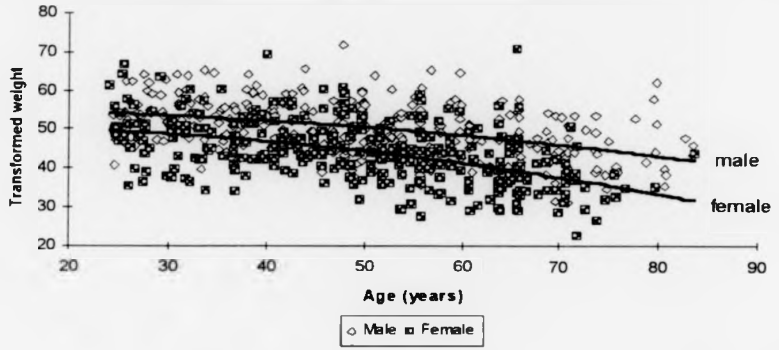


Figure 3.12: The male and female distributions of height by age (equation II)

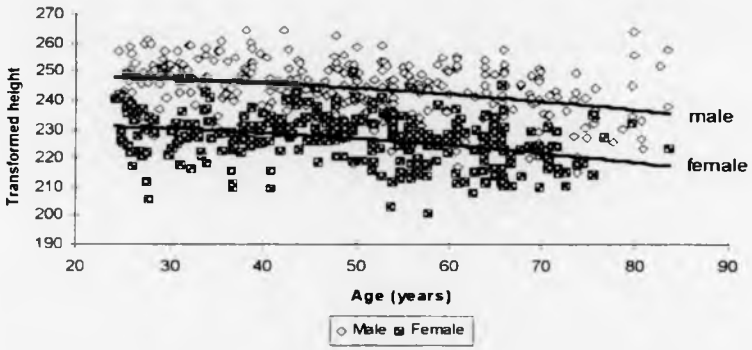


Figure 3.13: The male and female distributions of BMI by age (equation IV)

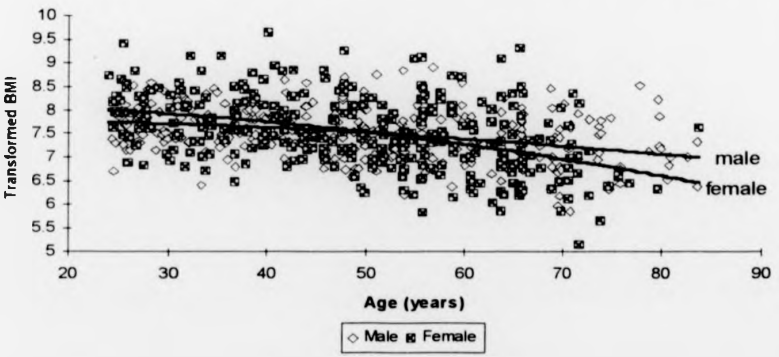


Figure 3.14: The male and female distributions of MUAC by age (equation IV)

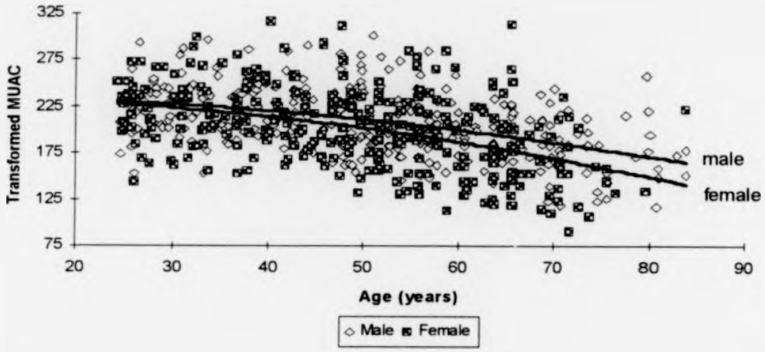


Figure 3.15: The male and female distributions of fat mass by age (equation IV)

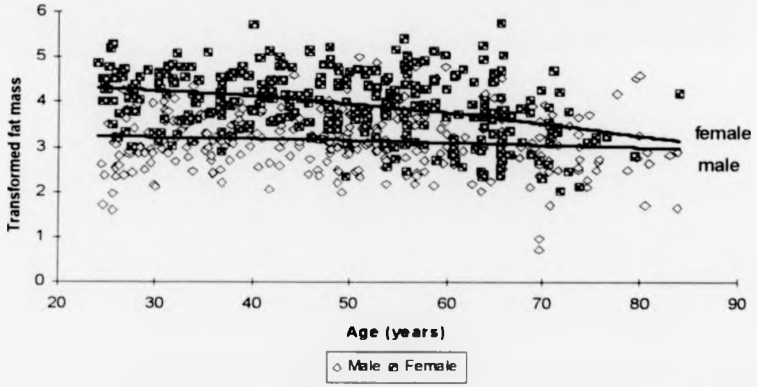


Figure 3.16 The male and female distributions of fat-free mass by age (equation I)

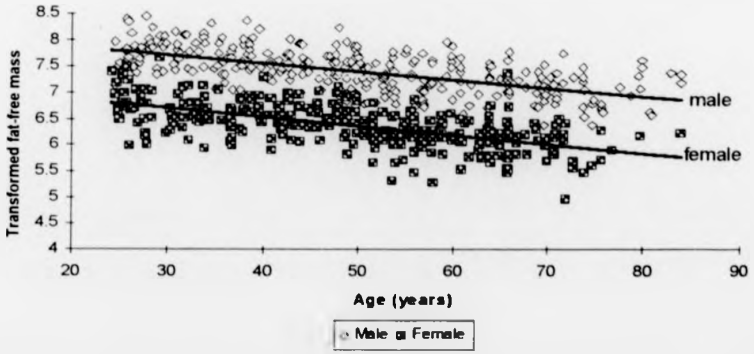


Figure 3.17: The male and female distributions of fat percent by age (equation III)

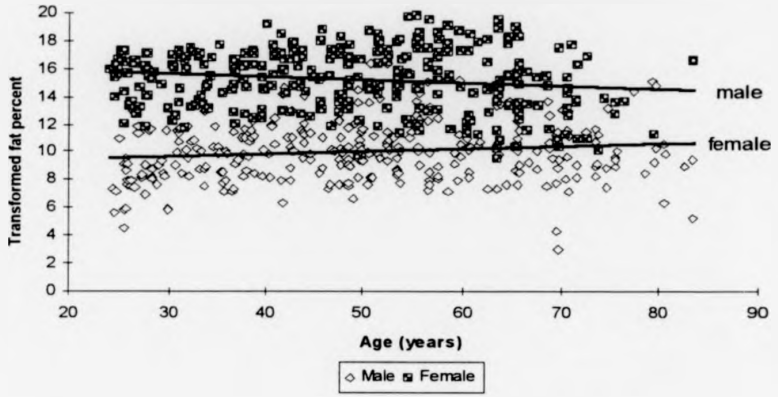


Figure 3.18: The male and female distributions of Cormic Index by age (equation IV)

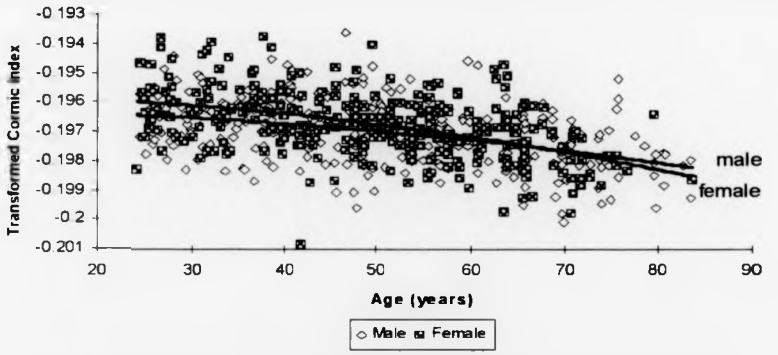
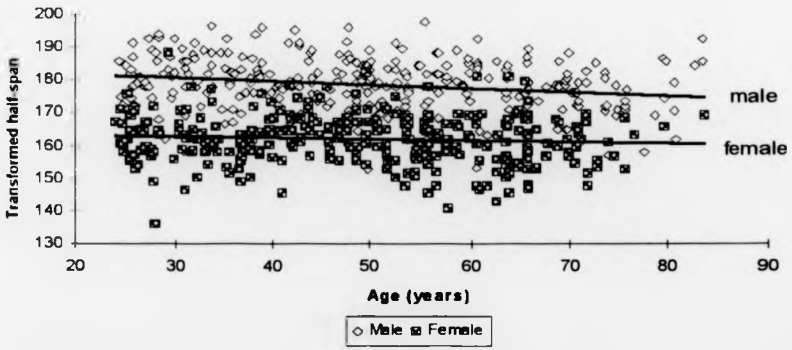


Figure 3.19: The male and female distributions of half-span by age (equation I)



The amount of variation explained by age and sex using the equations described above varies between the anthropometric measurement and indices. This can be seen in table 3.4 below. Over 50% of the variance in height, half-span, FFM and fat percent is associated with the age and sex distribution of the population. Less of the variance in weight, BMI, MUAC, CI and fat mass can be explained by age and sex.

Table 3.4. Variance in the distribution of the anthropometric variables which can be explained by sex and age (N=601)

Anthropometric variable	Variance explained by sex and age (adjusted r^2 * 100)
Weight	32.7
Height	53.6
BMI	16.6
MUAC	20.6
Fat mass	30.0
Fat-free mass	74.7
Fat percent	58.9
Cormic index	21.0
Demi-span	53.3

In this population the probability of being CED (defined by the BMI) or overweight (BMI>24.9 kg/m^2) is also affected by sex and age. Table 3.5 shows the results of the logistic regressions of CED and overweight with sex and age. As age increases so does the risk of being CED, conversely as age decreases the risk of being overweight increases. Women, who were shown earlier to have a greater spread of BMI than men, are more likely to be either CED or be overweight than men in this population.

Table 3.5. The associations between CED and overweight and age and sex (N=601)

	N	Odds Ratio	Confidence intervals	p-value
CED				
Age	127	1.070	1.052-1.087	0.001
Sex	127	1.946	1.261-3.004	0.003
Overweight				
Age	38	0.968	0.944-0.991	0.008
Sex	38	2.228	1.098-4.524	0.027

p-value based on log likelihood ratio test

3.3 Other determinants of the anthropometric variables in 1996

Age and sex explain more of the variance seen in the anthropometric variables in this population than any other factors. However "other" factors are also associated with the anthropometric variables. Factors which were assessed included - geographical location, educational level, quality of building materials used for the *bilek*, water supply, wealth, quality of land, cash cropping, smoking habits, alcohol consumption, physical activity level and HH composition data. In order to test for any associations between these disparate factors and the anthropometrical variables, regressions were run with each anthropometric measurement or index and these "other" factors controlling for age and sex in the most appropriate manner (described in section 3.2).

Of all these factors, only four broad categories are associated with anthropometry in this study - geographical location, wealth, smoking habit and physical activity level. No other factors were significantly associated with the anthropometric variables after controlling for age and sex in this cross-sectional analysis. Table 3.6 shows the direction of the associations after controlling for age and sex. Note that age and sex, although not shown in this table, remain significant in all the analyses.

Table 3.6: The association of SES and other factors with the anthropometric variables (controlling for age and sex) using separate regressions (n=601)

	GEOGRAPHICAL LOCATION		WEALTH				SMOKING	PHYSICALLY ACTIVE
	Migrant status	Distance to closest town	Savings	Material possessions scale	A member of HH receives wages	Electricity		
Weight	+	-	+	+	+			
Height	+							
BMI	+	-	+	+			-	+
MUAC	+	-	+	+			-	+
Fat mass	+			+	+	+	-	+
Fat-free mass	+	-	+	+				
Fat percent	+			+	+	+	-	+

+ = positive association. - = negative association

The geographical location category is split into two parts - migrant status and distance to the closest town. Those individuals who were living in a town (as opposed to a rural longhouse) were classified as migrants and were found to have higher mean values of all the anthropometric variables. Similarly, those individuals who lived furthest away from a town (more than two hours travelling) were found to have lower mean values of weight, BMI, MUAC and FFM.

The second category is concerned with wealth, measured either as access to cash (savings, wage receiver in the HH) or by the material possessions scale. The factors in this category are all related positively to the anthropometric variables, although not all are significantly associated with each measurement. The material possessions scale variable is probably the most important of these measures as it is significantly associated with all the anthropometric measurements except height in these cross-sectional analyses.

The third category is the individuals' smoking habit. Smokers were found to have lower BMIs, MUACs, FMs and fat percents. (Alcohol consumption was not significantly associated with any anthropometric variable). Finally, those individuals who were physically active during the previous year (either in the farm or at another job) had higher mean BMIs, MUACs, FMs and fat percents.

Following this initial analysis, stepwise regressions were run to see which combination of factors were significantly associated with the anthropometric variables. The results of these regressions can be seen in table 3.7.

Table 3.7 The association of SES and other factors with the anthropometric variables (controlling for age and sex) using stepwise regression (n=601)

	GEOGRAPHICAL LOCATION		WEALTH			SMOKING	ACTIVITY LEVEL	Adjusted r^2*100
	Migrant status	Distance to closest town	Savings	Material possessions scale	A member of HH receives wages	Electricity			
Weight	+	-		+			-	+	36.2
Height	+								54.0
BMI	+	-		+			-	+	21.1
MUAC	+	-					-	+	25.6
Fat mass	+	-		+			-	+	33.9
Fat-free mass	+								75.0
Fat percent	+	-			+	+	-	+	61.2

+ = positive association, - = negative association

On looking at table 3.7 it is apparent that, when all the factors which are associated with the anthropometric variables are entered into one grand regression, the "wealth" category becomes least important. The material possessions scale remains significantly associated only with weight, the BMI and FM. Having savings is no longer associated with any anthropometric variable.

Migrant status remains associated with all the anthropometric measurements, and distance to the nearest town is also important (although no longer so for FFM). Smoking is negatively associated with all the variables except height and FFM, and physical activity shows a positive association with the same variables. The extra variance explained by these SES and other "lifestyle" variables compared to that explained by age and sex alone is low. This is especially true for height and FFM. Approximately 5% of the variance in the BMI and MUAC is associated with these "other" factors.

CED and overweight are also associated with factors other than age and sex. The results of a stepwise logistic regression with CED (BMI<18.5kg/m^2) and the variables described above are shown in table 3.8. Those living further from the town are more likely to have CED as are those who smoke. Subjects who were physically active were less likely to have CED. Migrant status is not associated with the risk of CED. Most of these factors are not associated with the risk of being overweight (table 3.9). In a multiple logistic regression only smoking and age were found to be significantly related to risk of overweight. These associations were both negative, i.e. as age increased the OR of overweight decreased, smokers were less likely to be overweigh than non-smokers.

Table 3.8. The associations of SES and other factors with CED

	Odds Ratio	Confidence intervals	p-value
Age	1.064	1.045-1.082	0.001
Sex	2.235	1.378-3.625	0.001
Distance from town	2.210	1.385-3.526	0.001
Smoking habit	1.802	1.094-2.969	0.021
Physical Activity Level	0.372	0.209-0.664	0.001

p-value based on log likelihood ratio test

Table 3.9 The associations of SES and other factors with overweight

	Odds Ratio	Confidence intervals	p-value
Age	0.966	0.943-0.989	0.005
Smoking	0.305	0.131-0.709	0.006

p-value based on log likelihood ratio test

3.4 Changes in anthropometry between 1990 and 1996

3.4.1 Description of changes in anthropometry between 1990-1996

The data and analyses presented above focus on the cross-sectional associations between the anthropometrical variables and sex, age and other SES factors in 1996. Although an association between age and a certain anthropometrical variable may exist, it is not possible to assess whether the association seen is due to secular trend or physiological changes with ageing. The data and analyses below examine the changes in anthropometrical status observed between 1990 and 1996. These analyses may be useful in assessing whether the associations between age and anthropometrical status described above are due to physiological or secular change.

As explained in the methods section, the 1990 data has been transformed to adjust for inter-observer error. All the figures and tables presented below are based on age in 1990. Paired t-tests are used in all the analyses and thus the p-values recorded are probabilities for paired t-tests testing a mean difference of zero. When assessing the changes seen, the TEMs reported earlier should be borne in mind as differences less than these may be irrelevant.

Figures 3.20 and 3.21 and table 3.10 show that only small differences in height between 1990-1996 are observed in men up to age 40y. A slight increase in height (0.2cm) is seen up to aged 30y. This late growth may be the result of delayed puberty and the slower growth associated with poor environmental conditions (as reported by Strickland and Tuffrey, 1997 and Cameron, 1991). Alternatively the increase in height seen may be due to measurement error. After age 40y a decrease in height is observed which is particularly marked in those over 60y. During the six year interval a decrease of approximately 0.24 cm per year is seen in men over 60y.

Figure 3.20: Median male heights in 1990 and 1996 (n=355)

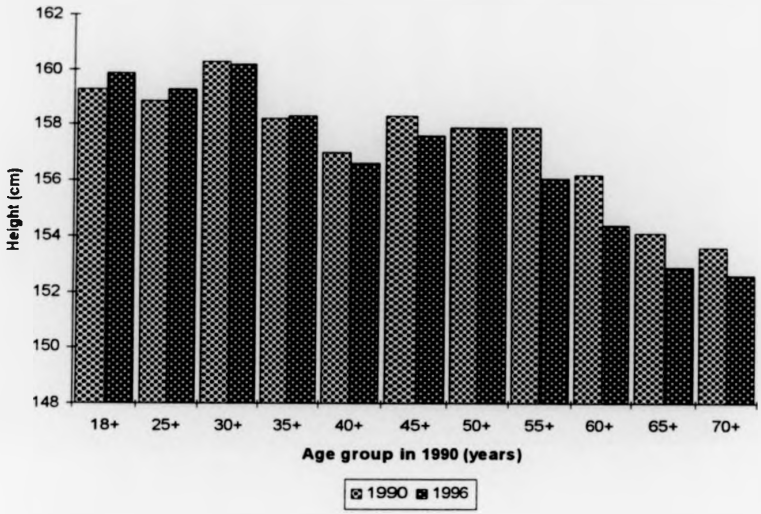
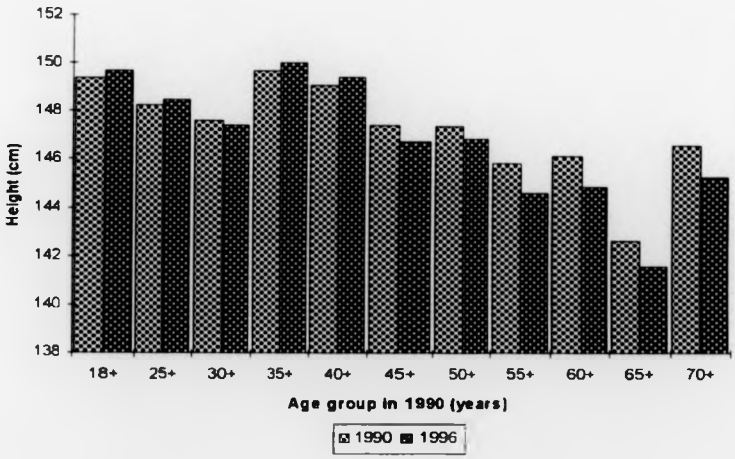


Figure 3.21: Median female heights in 1990 and 1996 (n=508)



In women a slightly larger late growth spurt (0.4cm) is observed up to age 30y. It seems unlikely that women would still be growing up to age 40y and this result (an increase of 0.1cm) may be due to measurement error. Above age 45y the women begin to lose height and this process accelerates above age 55y. During the six year interval women above age 60y decrease in height by approximately 0.25cm/year.

Table 3.10: Mean heights in 1990 and 1996

Age in 1990	N	Mean height in 1990	Mean height in 1996	Mean difference	p-value
MEN					
18.0-29.9	86	159.3 (5.5)	159.6 (5.5)	0.3 (0.7)	0.004
30.0-39.9	68	159.2 (4.3)	159.2 (4.2)	0.0 (0.7)	0.787
40.0-49.9	90	157.1 (5.0)	156.6 (5.2)	-0.5 (0.8)	0.001
50.0-59.9	53	157.2 (5.1)	156.5 (5.3)	-0.7 (0.9)	0.001
60.0-69.9	43	154.7 (4.6)	153.3 (5.0)	-1.4 (1.2)	0.001
70.0+	15	155.6 (6.9)	154.1 (7.6)	-1.5 (1.3)	0.001
All ages	355	157.7 (5.3)	157.3(5.6)	-0.4 (1.0)	0.001
WOMEN					
18.0-29.9	138	148.7 (4.5)	149.1 (4.5	(0.7)	0.001
30.0-39.9	120	148.5 (4.0)	148.6 (4.0	(0.7)	0.049
40.0-49.9	110	148.4 (4.8)	148.1 (4.9)	3(0.7)	0.001
50.0-59.9	92	146.0 (4.9)	144.9 (5.1)	-1.1 (1.2)	0.001
60.0-69.9	42	145.1 (4.2)	143.8 (4.2)	-1.3 (1.3)	0.001
70.0+	6	145.2 (4.2)	143.9 (5.4)	-1.3 (1.3)	0.219
All ages	508	147.8(4.7)	147.5(4.9)	-0.3 (3.9)	0.001

Figure 3.22 and table 3.11 show that the young Iban men (up to age 35y in 1990) put on a significant amount of weight during the 1990-1996 period. Those men who were aged above 50y in 1990 lost weight in the six years before re-measurement. A decrease in weight of approximately 0.25kg/year is seen in men older than 60y during the 1990-1996 period. Figure 3.23 shows that, on average, the youngest group of Iban women have put on 4kg in the six year interval. The older women have lost weight at approximately 0.34kg/year.

Figure 3.22: Median male weights in 1990 and 1996 (n=366)

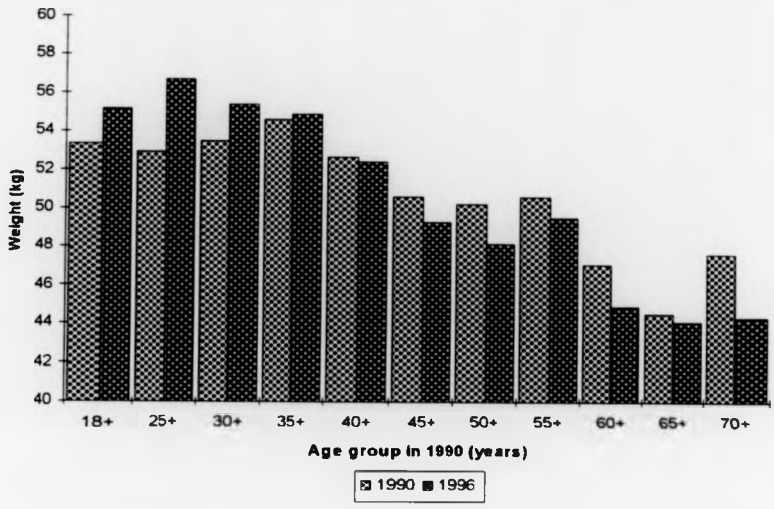


Figure 3.23: Median female weights in 1990 and 1996 (n=477)

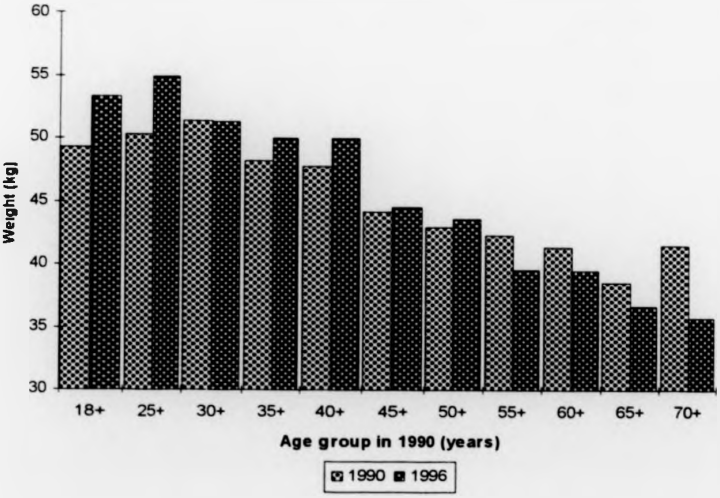


Table 3.11: Mean weights in 1990 and 1996

Age in 1990	N	Mean weight in 1990	Mean weight in 1996	Mean difference	p-value
MEN					
24.0-29.9	91	54.4 (6.4)	57.2 (9.2)	2.8 (4.5)	0.001
30.0-39.9	68	54.0 (6.1)	55.5 (7.1)	1.5 (3.6)	0.001
40.0-49.9	93	52.3 (7.6)	52.3 (8.9)	0.0 (3.2)	0.899
50.0-59.9	56	50.7 (6.5)	49.4 (6.7)	1.3 (3.2)	0.004
60.0-69.9	43	46.3 (5.2)	45.0 (5.7)	-1.3 (2.8)	0.004
70.0+	15	48.3 (8.4)	46.2 (7.6)	-2.0 (2.4)	0.006
All ages	366	52.0 (7.1)	52.6 (8.9)	0.5 (3.9)	0.008
WOMEN					
24.0-29.9	106	50.6 (7.9)	54.6 (9.9)	4.0 (5.3)	0.001
30.0-39.9	112	40.4 (8.5)	52.4 (10.0)	1.9 (3.8)	0.001
40.0-49.9	113	47.6 (8.8)	47.9 (9.7)	0.2 (3.8)	0.530
50.0-59.9	95	43.4 (8.3)	42.4 (8.7)	-1.0 (3.9)	0.018
60.0-69.9	42	41.0 (7.1)	39.5 (8.2)	-1.6 (3.0)	0.002
70.0+	9	40.9 (7.2)	36.4 (7.8)	-4.5 (3.5)	0.004
All ages	477	47.4 (8.9)	48.4 (10.9)	1.0 (4.6)	0.001

Figures 3.24 and 3.25 and table 3.12 show the changes in BMI (derived from standing height) between 1990 and 1996 according to age group in 1990. There has been a significant increase in BMI for both sexes in those aged 40y or below in 1990 in the six years before re-measurement. These increases have been especially large for women. Above age 50y both sexes have shown small decreases in BMI during the 1990-1996 period. The mean rates of BMI decrease in the over sixties were approximately 0.05kg/m^2 per year for men and 0.09kg/m^2 for women.

Figures 3.26 and 3.27 and table 3.13 show the changes in BMI derived from half-span between 1990 and 1996 according to age group in 1990. Half-span was not measured by Strickland and Ulijaszek in 1990 and hence it has been assumed that half-span remained constant during this period. When half-span is used to calculate the BMI the effect is to make the BMI lower (compared to a BMI derived from standing height) as the population ages. Thus it can be seen that the differences between the BMI measured in 1990 and 1996 become more accentuated and more closely resemble the pattern seen in the weight changes. Using this half-span derived BMI it can be seen that the median BMIs of both the Iban men and women decrease significantly with increasing age above 50 years.

Figure 3.24: Median male BMIs (derived from stature) in 1990 and 1996 (n=353)

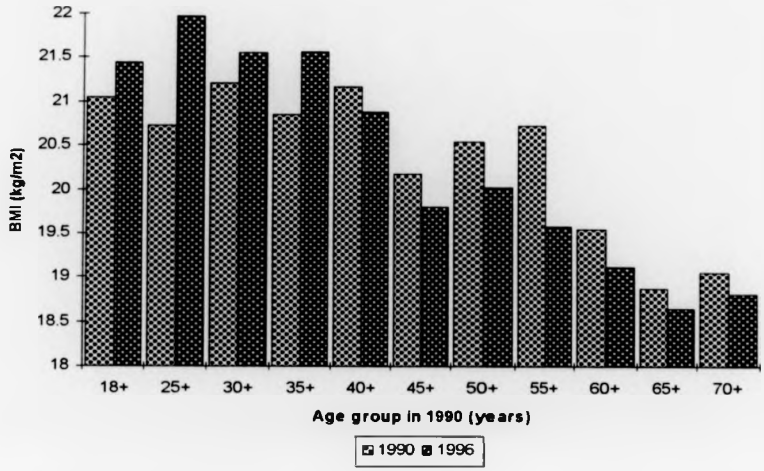


Figure 3.25: Median female BMIs (derived from stature) in 1990 and 1996 (n=466)

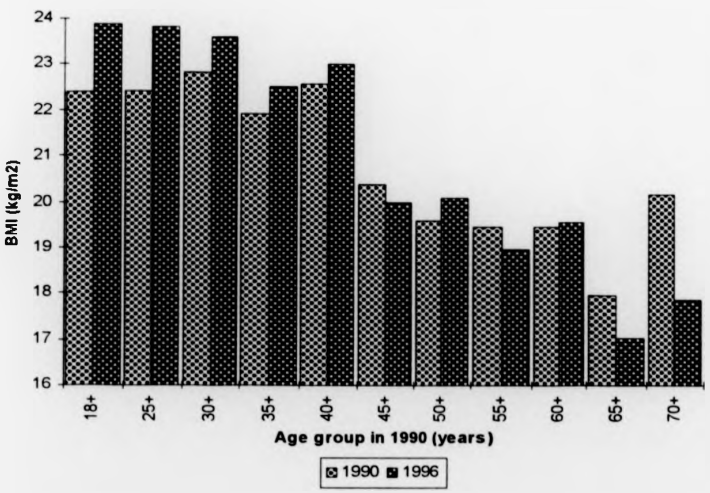


Figure 3.26: Median male BMIs (derived from half-span) in 1990 and 1996 (n=353)

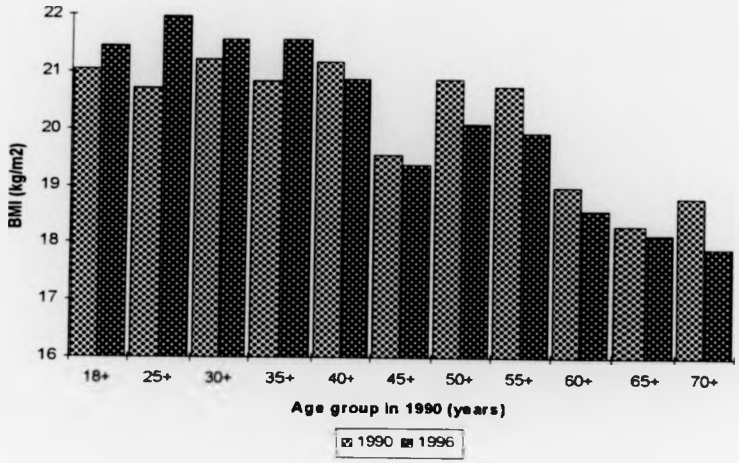


Figure 3.27: Median female BMIs (derived from half-span) in 1990 and 1996 (n=466)

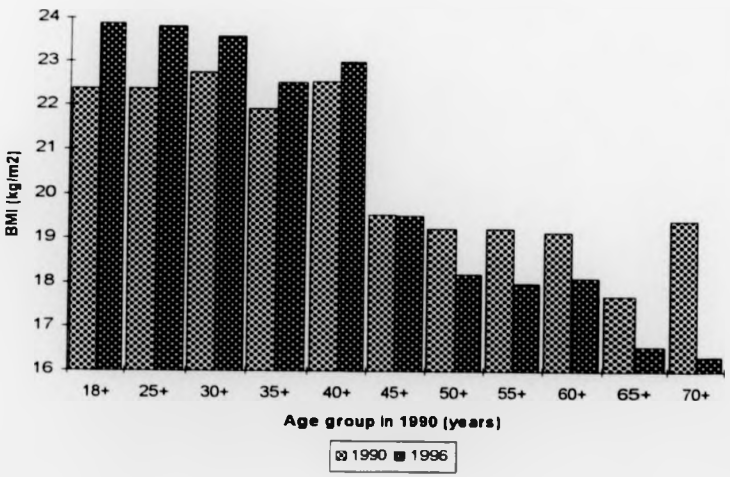


Table 3.12: Mean BMIs in 1990 and 1996 (derived from stature)

Age in 1990	N	Mean BMI in 1990	Mean BMI in 1996	Mean difference	p-value
MEN					
18.0-29.9	86	21.3 (2.0)	22.3 (2.9)	1.0 (1.7)	0.001
30.0-39.9	67	21.3 (2.2)	21.9 (2.6)	0.6 (1.4)	0.001
40.0-49.9	90	21.0 (2.4)	21.0 (2.9)	0.1 (1.3)	0.536
50.0-59.9	54	20.5 (2.3)	20.1 (2.3)	-0.4 (1.3)	0.035
60.0-69.9	43	19.4 (1.9)	19.1 (2.3)	-0.2 (1.3)	0.270
70.0+	15	19.9 (2.5)	19.4 (2.4)	-0.4 (1.0)	0.089
All ages	353	20.8 (2.3)	21.1 (2.9)	0.3 (1.5)	0.001
WOMEN					
18.0-29.9	104	22.8 (3.3)	24.4 (4.1)	1.6 (2.3)	0.001
30.0-39.9	111	22.9 (3.5)	23.7 (4.1)	0.9 (1.7)	0.001
40.0-49.9	109	21.6 (3.6)	21.8 (3.9)	0.2 (1.7)	0.372
50.0-59.9	94	20.4 (3.4)	20.1 (3.6)	-0.2 (1.8)	0.202
60.0-69.9	42	19.5 (3.3)	19.0 (3.9)	-0.4 (1.4)	0.050
70.0+	6	19.7 (2.4)	18.5 (3.2)	-1.1 (1.4)	0.087
All ages	466	21.7 (3.6)	22.2 (4.4)	0.5 (2.0)	0.001

Table 3.13: Mean BMIs in 1990 and 1996 (derived from half-span)

Age in 1990	N	Mean BMI in 1990	Mean BMI in 1996	Mean difference	p-value
MEN					
18.0-29.9	86	21.3 (2.0)	22.3 (2.9)	1.0 (1.8)	0.001
30.0-39.9	67	21.3 (2.2)	21.9 (2.6)	0.6 (1.4)	0.001
40.0-49.9	90	21.0 (2.4)	20.9 (3.0)	0.1 (1.4)	0.755
50.0-59.9	54	20.3 (2.5)	19.8 (2.4)	-0.5 (1.3)	0.003
60.0-69.9	43	19.0 (1.9)	18.4 (2.1)	-0.5 (1.2)	0.004
70.0+	15	19.6 (3.1)	18.8 (2.9)	-0.8 (0.9)	0.005
All ages	353	20.7 (2.4)	20.9 (3.0)	0.1 (0.8)	0.081
WOMEN					
18.0-29.9	103	22.8 (3.3)	24.4 (4.1)	1.6 (2.3)	0.001
30.0-39.9	111	22.9 (3.5)	23.7 (4.1)	0.9 (1.7)	0.001
40.0-49.9	109	21.6 (3.6)	21.6 (3.9)	0 (1.9)	0.933
50.0-59.9	94	19.9 (3.4)	19.4 (3.7)	-0.5 (1.7)	0.014
60.0-69.9	42	18.9 (3.2)	18.2 (3.7)	-0.7 (1.4)	0.002
70.0+	6	18.6 (2.2)	17.2 (2.6)	-1.4 (1.5)	0.025
All ages	459	21.5 (3.7)	21.9 (4.5)	0.4 (2.1)	0.001

Figures 3.28 and 3.29 and table 3.14 show that the young men's MUACS have significantly increased from 1990-1996. The MUAC of the men over 50 years old has significantly decreased. The pattern is similar for women, although their MUACs have increased up to the age of 50 years and did not decrease significantly until aged 60 or above. The increases in MUAC amongst the younger age groups are particularly interesting as these cannot be deduced from the cross-sectional data in 1996.

Table 3.14: Mean MUACs in 1990 and 1996

Age in 1990	N	Mean MUAC in 1990	Mean MUAC in 1996	Mean difference	p-value
MEN					
18.0-29.9	90	27.2 (2.2)	28.1 (2.7)	0.9 (1.5)	0.001
30.0-39.9	67	27.4 (2.0)	27.8 (2.4)	0.4 (1.1)	0.003
40.0-49.9	91	27.0 (2.5)	27.1 (3.0)	0.1 (1.3)	0.612
50.0-59.9	57	25.7 (2.3)	25.3 (2.2)	-0.4 (1.3)	0.031
60.0-69.9	44	24.7 (2.0)	24.0 (2.7)	-0.8 (1.7)	0.004
70.0+	15	25.0 (2.8)	24.0 (2.6)	-1.0 (1.6)	0.025
All ages	364	26.6	26.7	0.1 (1.5)	0.096
WOMEN					
18.0-29.9	104	26.9 (2.8)	28.8 (3.3)	1.9 (2.0)	0.001
30.0-39.9	112	27.4 (3.2)	28.4 (3.6)	1.0 (1.6)	0.001
40.0-49.9	113	26.3 (3.4)	26.8 (3.5)	0.5 (1.6)	0.001
50.0-59.9	91	25.1(3.3)	25.0 (3.5)	-0.1 (1.8)	0.665
60.0-69.9	43	24.1 (3.1)	23.5 (3.4)	-0.6 (1.6)	0.011
70.0+	9	24.7 (3.0)	22.3 (3.4)	-2.4 (1.5)	0.001
All ages	472	26.3	26.9	0.6 (1.9)	0.001

Figure 3.28: Median male MUACs in 1990 and 1996 (n=364)

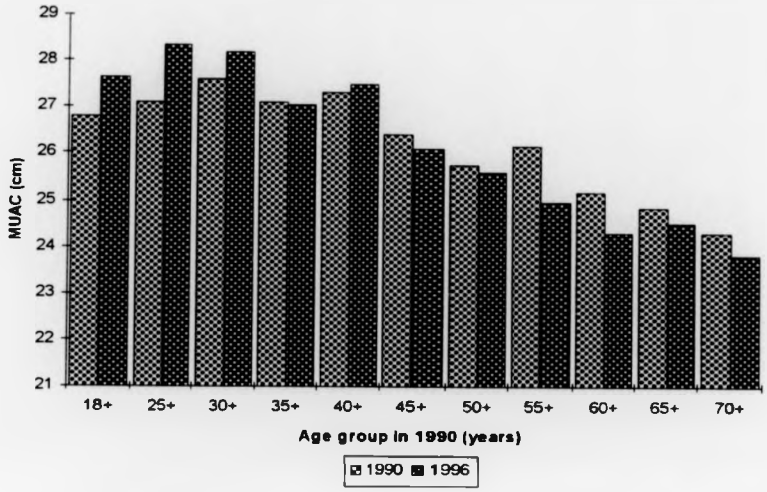
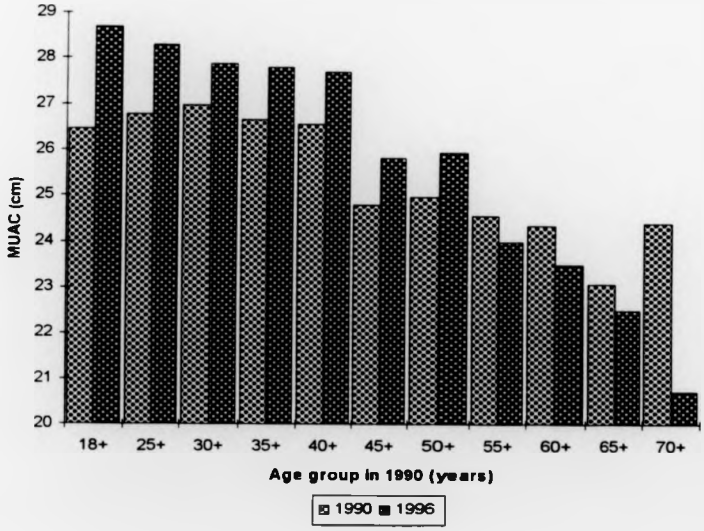


Figure 3.29: Median female MUACs in 1990 and 1996 (n=472)



Figures 3.30-3.36 and tables 3.15-3.17 show the changes in body energy and body composition seen since 1990. The median male FM values of all age groups has increased or remained constant since 1990. The increases seen are more pronounced in the younger age groups than in the elderly. In women, median FM has also increased up to age 50y. After age 60y the women's median FM has decreased. The median FFM values of men and women of all ages has decreased since 1990. This loss of FFM is greater amongst the elderly. The net result of these changes in energy stores are changes in body composition over the six year period. The younger adults of both sexes have experienced a significant (approximately 5%) increase in percentage body fat. The older men have experienced a smaller increase in percentage body fat and the older women have undergone a slight decrease in percentage body fat.

Table 3.15: Mean Fat mass in 1990 and 1996

Age in 1990	N	Mean fat mass in 1990	Mean fat mass in 1996	Mean difference	p-value
MEN					
18.0-29.9	79	6.4 (1.8)	9.5 (3.2)	3.1 (2.5)	0.001
30.0-39.9	60	8.7 (2.0)	11.0 (3.0)	2.3 (2.1)	0.001
40.0-49.9	81	9.1 (3.1)	10.8 (4.3)	1.7 (2.4)	0.001
50.0-59.9	53	9.1 (3.3)	10.2 (4.0)	1.1 (2.5)	0.001
60.0-69.9	42	8.0 (2.1)	8.6 (3.0)	0.6 (1.8)	0.001
70.0+	14	9.1 (4.4)	9.7 (4.9)	0.5 (1.6)	0.001
All ages	329	8.3 (2.8)	10.1 (3.7)	1.9 (2.5)	0.001
WOMEN					
18.0-29.9	61	13.1 (3.8)	16.2 (4.4)	3.1 (3.3)	0.001
30.0-39.9	69	13.6 (3.4)	16.4 (4.6)	2.8 (3.0)	0.001
40.0-49.9	89	13.8 (4.1)	15.7 (4.7)	1.9 (2.5)	0.001
50.0-59.9	75	13.2 (4.7)	13.8 (5.4)	0.5 (3.2)	0.001
60.0-69.9	34	11.6 (3.2)	11.3 (3.9)	-0.4 (2.3)	0.001
70.0+	4	11.9 (3.4)	10.3 (4.1)	-1.6 (2.5)	0.170
All ages	332	13.3 (4.0)	15.0 (5.0)	1.7 (3.1)	0.001

Table 3.16 Mean Fat-free mass in 1990 and 1996

Age in 1990	N	Mean fat-free mass in 1990	Mean fat-free mass in 1996	Mean difference	p-value
MEN					
18.0-29.9	79	46.7 (4.1)	45.3 (4.3)	-1.4 (2.3)	0.001
30.0-39.9	60	44.4 (4.0)	43.0 (3.7)	-1.3 (2.0)	0.001
40.0-49.9	81	42.3 (4.4)	40.1 (4.5)	-2.2 (1.6)	0.001
50.0-59.9	53	41.6 (4.1)	39.2 (3.7)	-2.4 (2.0)	0.001
60.0-69.9	42	38.4 (3.8)	36.3 (3.7)	-2.0 (1.7)	0.001
70.0+	14	39.7 (4.7)	37.1 (3.9)	-2.7 (1.7)	0.001
All ages	329	43.0 (4.9)	41.1 (5.1)	-1.9 (2.0)	0.001
WOMEN					
18.0-29.9	61	34.6 (3.7)	33.4 (3.9)	-0.9 (2.0)	0.001
30.0-39.9	69	33.4 (3.1)	31.7 (3.0)	-1.7 (1.7)	0.001
40.0-49.9	89	31.9 (3.7)	29.5 (3.3)	-2.4 (1.5)	0.001
50.0-59.9	75	29.2 (3.5)	27.6 (3.1)	-1.6 (1.7)	0.001
60.0-69.9	34	28.1 (2.8)	26.7 (2.9)	-1.4 (1.3)	0.001
70.0+	4	28.3 (4.0)	26.2 (2.8)	-2.1 (1.5)	0.022
All ages	332	31.6 (4.1)	29.9 (4.0)	-1.7 (1.7)	0.001

Table 3.17 Mean Fat percent in 1990 and 1996

Age in 1990	N	Mean fat % in 1990	Mean fat % in 1996	Mean difference	p-value
MEN					
18.0-29.9	79	12.0 (2.8)	17.1 (4.3)	5.1 (3.7)	0.001
30.0-39.9	60	16.3 (2.9)	20.1 (4.2)	3.8 (2.9)	0.001
40.0-49.9	81	17.4 (4.0)	20.7 (3.6)	3.3 (3.6)	0.001
50.0-59.9	53	17.6 (4.3)	20.1 (5.6)	2.5 (3.8)	0.001
60.0-69.9	42	17.1 (3.2)	18.7 (4.9)	1.6 (3.3)	0.001
70.0+	14	18.0 (5.5)	19.9 (7.0)	1.8 (2.7)	0.001
All ages	329	15.9 (4.2)	19.4 (5.2)	3.4 (3.6)	0.001
WOMEN					
18.0-29.9	61	27.2 (5.2)	32.1 (4.9)	4.9 (4.7)	0.001
30.0-39.9	69	28.6 (4.4)	33.5 (5.3)	5.0 (3.9)	0.001
40.0-49.9	89	29.8 (4.9)	34.1 (6.0)	4.3 (3.5)	0.001
50.0-59.9	75	30.3 (5.8)	32.2 (7.3)	1.8 (4.6)	0.001
60.0-69.9	34	28.8 (4.8)	28.9 (5.8)	0.1 (3.7)	0.001
70.0+	4	29.3 (4.2)	27.5 (6.2)	-1.7 (4.5)	0.001
All ages	332	29.1 (5.1)	32.6 (6.2)	3.5 (4.5)	0.001

Figure 3.30: Median male fat mass in 1990 and 1996 (n=329)

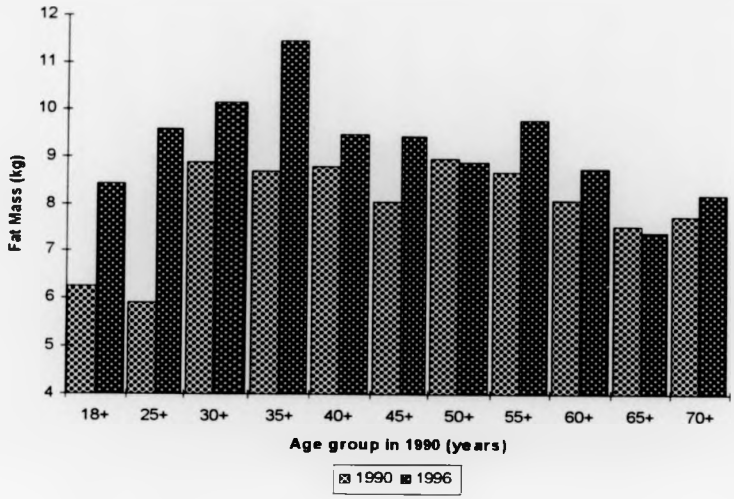


Figure 3.31 Median female fat mass in 1990 and 1996 (n=332)

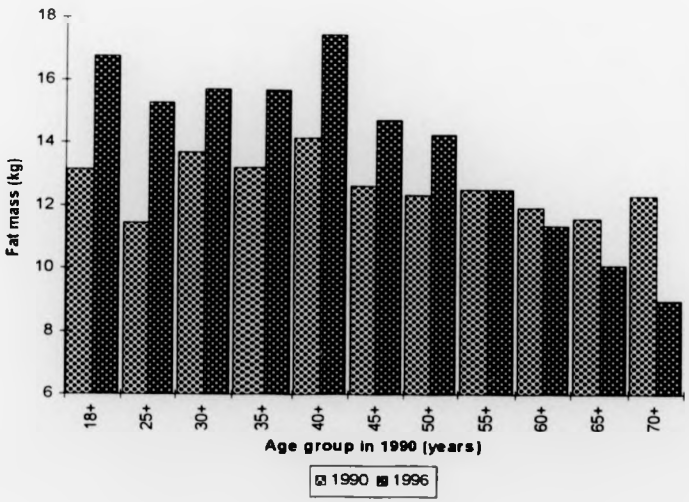


Figure 3.32: Median male fat-free mass in 1990 and 1996 (n=329)

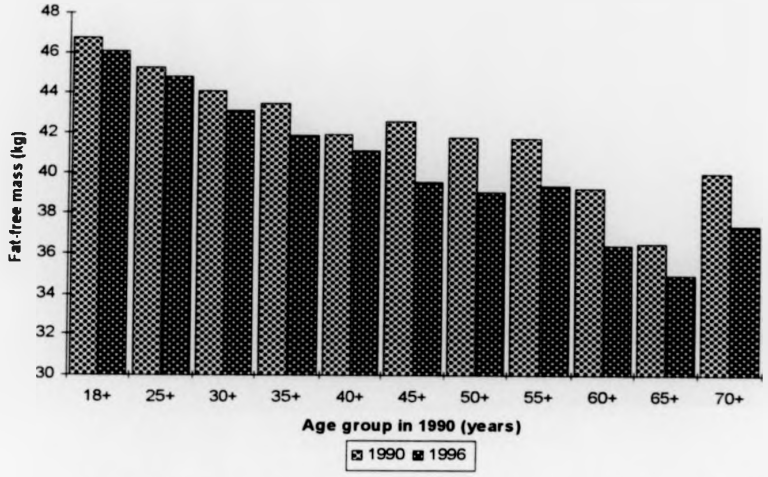


Figure 3.33: Median female fat-free mass in 1990 and 1996 (n=332)

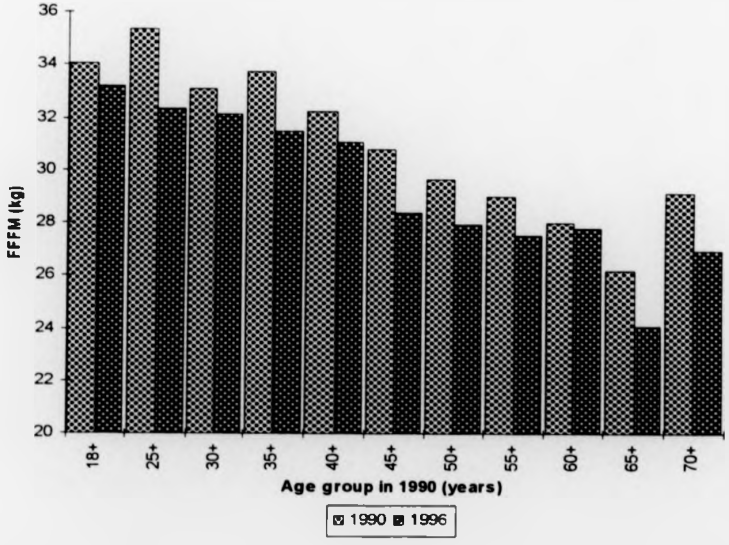


Figure 3.34 Median male fat percent in 1990 and 1996 (n=329)

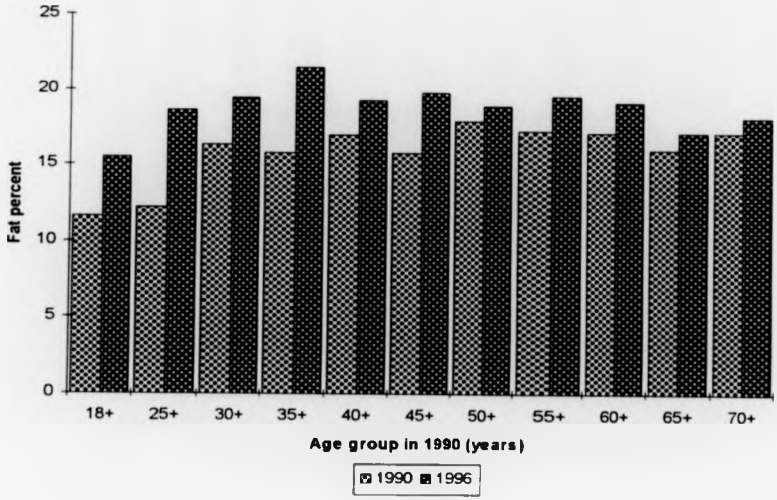
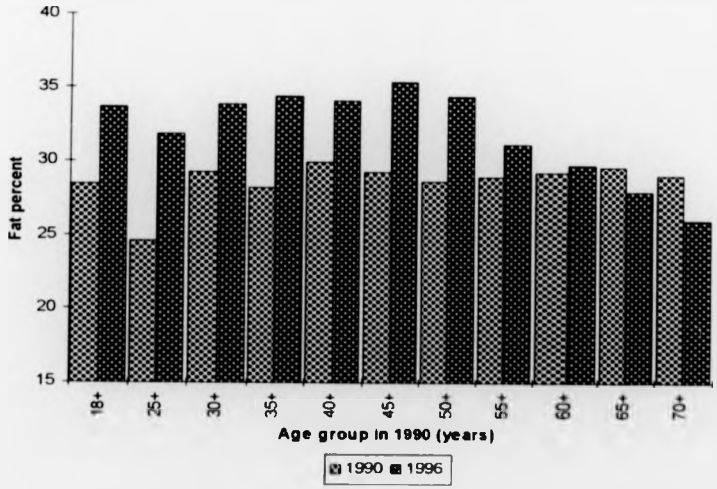


Figure 3.35 Median female fat percent in 1990 and 1996 (n=332)



3.4.2 Changes in the amount of CED or overweight between 1990-1996

Table 3.18 shows the change in the amount of CED diagnosed by the BMI alone between 1990 and 1996. It should be noted that although the data in this table compares the same individuals in 1990 and 1996 it is not necessarily an accurate reflection of the amount of CED or obesity in 1990 as the non-survivors and migrants are not included in the analysis. The table shows an increase in the amount of grade III CED and obesity in both sexes. As a result of this increase in the spread of the data fewer of the individuals are defined as having a normal BMI in 1996.

Table 3.18: CED (diagnosed by the BMI alone) in 1990 and 1996 (N=802)

	% diagnosed as CED		
	1990	1996	Change
Men (n=348)			
Grade III CED	0.0	1.4	+1.4
Grade II CED	3.7	2.9	-0.8
Grade I CED	10.3	11.8	+1.5
Normal	82.2	74.4	-7.8
Overweight	3.2	7.2	+4.0
Obese	0.6	2.3	+1.7
Women (n=454)			
Grade III CED	2.2	5.5	+3.3
Grade II CED	4.2	4.2	0.0
Grade I CED	11.9	10.1	-1.8
Normal	62.6	57.1	-5.5
Overweight	16.3	16.5	+0.2
Obese	2.9	6.6	+3.7
All (n=802)			
Grade III CED	1.2	3.8	+3.6
Grade II CED	4.0	3.6	-0.4
Grade I CED	11.2	10.8	-0.4
Normal	71.1	64.6	-6.5
Overweight	10.6	12.5	+1.9
Obese	1.9	4.7	+2.8

3.5 Age and sex as determinants of change in the anthropometric variables between 1990-1996

The basis of the analyses in this section is the data set for the 553 non-pregnant individuals for whom all anthropometric and socio-economic data are available in both 1990 and 1996.

The results in the section above show that the changes in the anthropometric variables between 1990-1996 are dependent on age. The implication of these results is that physiological change in anthropometrical status does occur with ageing and hence that some of the differences observed in the cross-sectional data are due to ageing and not only to secular trends.

If the physiological change is seen as a continuous variable ranging from loss to gain, a general pattern emerges whereby the younger groups have the greatest change in the six year period (i.e. the mean values of their measurements tend either to increase or to decrease only slightly) and the oldest groups have the smallest change (i.e. the mean values of their measurements increase less or decrease more than the younger groups). Naturally not all the variables change in the same manner, e.g. in men mean FM increases with age but mean FFM decreases with increasing age, and the changes may not be identical in both sexes, e.g. mean fat percent in men increased in all age groups, but decreased in older women. In order to assess the nature of change in relationship with sex and age, regressions similar to those in section 3.2 were run for each variable. As before, the regression which explained the most variance is taken to give the best explanation for the relationship.

Changes in the BMI, weight, height and FFM were best explained simply by age. No significant differences between the sexes were found, i.e. both men and women lost height in a linear fashion at the same rate (equation I). The change in CI was also best explained by equation I - there was a similar gradient of decrease in both sexes but the intercepts were different. The changes in MUAC were best explained by equation III, i.e. the change in MUAC was linear with age and both the gradients and intercepts were different between men and women. The changes in fat percent and FM were best described by equation IV, i.e. the change with age was non-linear and the gradients and intercepts were different for men and women. No data are available for change in half-span between 1990-1996 as it was not measured in 1990.

The amount of variation in the changes seen, explained by age and sex using the equations described above, varies between the anthropometric measurement and indices. This can be seen in table 3.19. Compared to the figures in section 3.2 the amount of variance explained by age

and sex is low particularly for FFM, although 30% of the variance in the change in height can be explained by age

Table 3.19: Variance in the distribution of the change in the anthropometric variables (1990-1996) which can be explained by sex and age (N=553)

Anthropometric variable	Variance explained by sex and age (adjusted r^2*100)
Weight	14.8
Height	29.7
BMI	8.1
MUAC	16.4
Fat mass	14.1
Fat-free mass	3.2
Fat percent	11.8
Cormic index	5.2

37 individuals who were not classified as CED in 1990 were diagnosed as CED in 1996. Stepwise logistic regression revealed that the risk of changing from non-CED in 1990 to CED in 1996 was not associated with sex, but was significantly associated with age (O.R. = 1.031, 95% C.I. 1.008-1.055, $p<0.009$). However, the variance explained by this regression was low - only 2.6%.

3.6 Other determinants of the change in anthropometric variables between 1990-1996

Factors other than age and sex are also associated with the change in anthropometry seen since 1990. The analysis below is similar to that in section 3.3 in that it will assess which "other" factors (geographical location, educational level, quality of building materials used for the *bilek*, water supply, wealth, quality of land, cash cropping, smoking habits, alcohol consumption, physical activity level and household composition data) are associated with the changes in anthropometric status since 1990. Note that SES in 1996 is used as a proxy of SES for the whole six year period because no detailed baseline data on SES was available from 1990. Stepwise multiple regressions were run with each anthropometric measurement or index and the "other" factors, controlling for age and sex as described above. The results of these regressions can be found in table 3.20.

Table 3.20: The association of SES and other factors with the change in anthropometric variables (controlling for age and sex) using stepwise regressions (N=553)

	GEOGRAPHICAL LOCATION	WEALTH			SOLD CASH CROPS	SMOKING	ALCOHOL	PHYSICALLY ACTIVE	ADJUSTED r^2*100
	Distance to closest town	Material possessions scale	A member of HH receives wages	Remittances					
Weight	-				-	-	+		18.1
Height									29.7
BMI	-				-	-	+		11.2
MUAC				+				+	17.8
Fat mass	-	+				-		+	18.1
Fat-free mass	-								4.5
Fat percent	-		+		-	-		+	16.7

+ = positive association; - = negative association

When examining table 3.20 it must be remembered that change since 1990 has been entered as a continuous variable. Thus a positive association means that those who, for example, scored highly on the material possessions scale, were likely to gain more FM (younger groups) or lose less (older groups) than those who were poor. A negative association means that those who were, for example, living further from a town were more likely to gain less weight (younger groups) or lose more (older groups) than those who lived closer to a town.

The first point to note about table 3.20 is that none of the SES or "other" factors makes any significant difference to the rate of change in height - this is determined by age alone. However, the rates of change in the other anthropometric measurements or indices are associated with at least one SES or "other" factor. Distance to the nearest town and smoking are both negatively associated with change in many of the anthropometric variables, i.e. those who smoke were more likely to lose more weight or gain less than non-smokers. Physical activity and some of the wealth variables were both positively associated with change in some of the anthropometric measurements or indices. Those who were physically active were more likely to have larger increases or smaller decreases in MUAC, FM and fat percent between 1990 and 1996. These results were all expected given the associations seen in the cross-sectional data, and smoking, distance to the nearest town, PAL and some of the wealth variables are probably true determinants of anthropometric status in this population.

Some differences are observed in the associations of these factors with the cross-sectional data and these longitudinal data. Firstly, migrant status is no longer an important factor. Secondly, although some of the wealth variables are associated with change these associations are not as common or as strong as those in the cross-sectional data. This may be because the HH wealth variables in 1996 are used as a proxy for economic wealth between 1990-1996 as no baseline data on HH wealth are available. Thus change in wealth over the period cannot be assessed. HH wealth may be sensitive to short term changes in environmental and economic conditions but this study cannot analyse such changes or their effects on the anthropometric variables. Also, alcohol consumption is positively associated with change in weight and BMI. Those who drank at least once a week were more likely to gain more weight or lose less but this association was not seen in the cross-sectional data. Finally, the selling of cash crops was negatively associated with change in weight, BMI and fat percent.

As for the cross-sectional data, the increase in variance explained by the SES and "other" factors compared to that explained by age and sex is not great. The increase ranges from 0% (height) to 5% (FM).

Table 3.21 shows the results of regressions between change in the anthropometrical variables and initial (1990) values of the anthropometrical variables and the SES and other "lifestyle" factors. This analysis aims to assess whether or not initial anthropometrical status (in 1990) affects the rate of change between 1990-1996 whilst controlling for age, sex and SES and other "lifestyle" factors. The table shows that initial anthropometrical status is negatively associated with change for all the anthropometrical variables except for height and weight, e.g. those with higher values of BMI are more likely to put on less weight (younger groups) or lose more (older groups) than those with lower values of BMI in 1990. The affects of the SES and "other" factors remain almost the same. The extra variance observed in the changes explained by initial anthropometry is not great except for FFM where the extra variance explained is approximately 8%.

If a regression between BMI in 1996 and BMI in 1990 controlling for SES and other factors is run, the coefficient of BMI in 1990 is 0.903, i.e. an association of less than 1.00 is found between the two measurements - as described above. Part of this association can be ascribed to the phenomenon of regression to the mean (Fuller, 1987). After accounting for regression to the mean (by comparing the partial correlation coefficients between the two BMI measures in 1996 and the regression between BMI in 1990 and 1996), the negative association seen between the BMI in 1990 and 1996 is reduced and is only partially significant. This is also true for weight, fat percent and FM; only the MUAC and FFM show significant negative associations between 1990 and 1996 after regression to the mean is taken into account.

The only factor other than age which was found to be significantly associated with the risk of changing from non-CED in 1990 to CED in 1996 was initial BMI. Subjects with low BMI in 1990 were more likely to become CED in 1996.

Table 3.21. The association of SES, "other" factors and 1990 anthropometrical status with the change in the anthropometric variables (controlling for age and sex) using stepwise regressions (N=553)

	1990 VALUE OF VARIABLE	GEOGRAPHICAL LOCATION	WEALTH		SOLD CASH CROPS	SMOKING	ALCOHOL	PHYSICALLY ACTIVE	ADJUSTED r^2*100
		Distance to closest town	Material possessions scale	A member of HH receives wages					
Weight		-			-	-	+		18.1
Height									29.7
BMI	-	-	+ (savings)		-	-	+		12.9
MUAC	-	-				-		+	21.4
Fat mass	-	-	+			-		+	18.1
Fat-free mass	-	-					+		12.2
Fat percent	-	-	+	+	-	-		+	17.3

+ = positive association, - = negative association

3.7 Changes in anthropometrical status between visit A and visit B in 1996

Complete anthropometric and SES data are available on 462 non-pregnant and non-migrant individuals at both visits A and B. these data are compared in this section.

No significant differences in the anthropometrical status of the women was found between visits A and B. Men aged 40y+ had a significantly lower weight and BMI at visit B. However, these differences were small - the mean difference in weight was 0.4kg and in BMI was 0.2kg/m^2. No significant changes in the MUAC, FM, FFM or fat percent were observed.

When assessing the changes seen in the whole population using multiple regression techniques, the only significant determinants of weight loss were sex, the presence of piped water in the *bilek* and initial BMI at visit A. The group who had piped water in their *bilek* were less likely to lose weight. Initial BMI was weakly negatively associated with change i.e. those with high initial BMI were more likely to lose weight than those with a lower initial BMI. Age was not a significant determinant of change and nor was PAL, i.e. those who were physically active in the farm at some point during the year were equally likely to lose weight as those who were not (physically active). The variance in change in weight or BMI explained by all these variables was low - only 3.5%.

It is tempting to attribute any changes observed between visits A and B to seasonality as the measurements were made at different times of the agricultural calendar. However, it is not possible to assess whether or not the differences are due to seasonal effects (e.g. increased PAL or decreased energy intake) or are part of the ageing process or simply that the men's environmental conditions deteriorated over the period for some other reason. A third group of measurements in 1998 taken in the same months as those made for visit A would be required to assess the causes for these differences.

3.8 Discussion

The results above present the anthropometric measurements and indices calculated for the Iban compared to those of other populations. The anthropometric variables are also assessed in terms of their properties as indices of nutritional status. In particular the analyses focus on whether or not the variables are (i) independent of sex and age, and (ii) sensitive to SES and other lifestyle factors.

Compared to the Americans in the NHANES data set, the Iban have low median values of height and weight at all ages, but their values are similar to those reported for other populations in S Asia (Strickland & Tuffrey, 1997). Adult height is largely determined by an individual's genotype and childhood nutritional experience, and large variations in the average heights of adults have been reported elsewhere (Eveleth & Tanner, 1976). In general, the young Iban women's median BMIs, MUACs, FM and FFM resemble their American counterparts but the older females and all the male Iban have median values which are considerably lower than the NHANES reference set. The older Iban women and the men have anthropometric values which are more comparable to those of other groups of the developing world.

More individuals were classified as CED using the BMI diagnosis than if the MUAC was used in conjunction with the BMI. Fewer individuals were classified as CED if the condition was diagnosed using the MUAC alone. The authors who originally proposed that CED could be diagnosed in terms of the BMI in conjunction with MUAC were aware that the method would result in a decrease in the number of individuals defined as CED. Indeed, the purpose of adding the low MUAC criterion (which implies peripheral tissue wasting) was to prevent healthy but thin individuals being wrongly classified as CED. Alemu and Lindtjorn (1997) reported considerable discrepancies in assessing the prevalence of malnutrition using the BMI or MUAC in two Ethiopian communities, use of the MUAC actually increased the amount of CED diagnosed in one population.

The mean values of all the anthropometrical variables differ according to sex. As reported in most other populations men have greater heights and weights than women. Men also have greater values of MUAC (Wang, 1994, James et al, 1994b). Conversely, women have higher fat percent than men (Norgan, 1994b, Shetty & James, 1994, Gallagher et al, 1996, Wormersley & Durnin, 1977). In this study the young women have a higher BMI than the young men but the converse is true for the older groups. Most of the evidence for the developed world (Rolland-Cachera et al, 1991, Norgan, 1990, Frisancho, 1990) suggests that the BMI of men is generally greater than that of women. Apparently this sex difference reflects the higher lean body mass of men (Forbes, 1976). However, data from the developing world suggest that the BMIs of the women are either equal to or slightly higher than those of the men (see table 3.1). Alemu and Lindtjorn's study (1997) in Ethiopia found that female BMI was higher in one region but that male BMI was higher in another. It is difficult to compare many of these studies directly as their subjects may be of different ages.

Female BMIs appear to be more widely spread (i.e. larger SDs and a greater percentage of CED or obesity than men) in both the developed and undeveloped world (James & Francois, 1994a). This may reflect the fact that women can tolerate lower BMIs than men (Shetty & James, 1994), although it does not prove that women benefit from having a different range of BMIs (James & Francois, 1994a).

In the cross-sectional analyses it appears that the women's median weight, BMI, MUAC and body composition variables decrease faster than the men's. However, the longitudinal analysis shows that the rate of decrease is similar for height, weight, BMI and FFM for both sexes. The latter result does not support the hypothesis that the higher incidence of osteoporosis in women leads to statural loss with age being greater in women (Cline et al, 1989). However changes in FM, fat percent and MUAC were different between the sexes. A further difference between the sexes is that the decreases appear to be delayed in the women (around 60y) compared to the men (around 50y).

The values of all the anthropometric indices vary with age. This can be seen from both the cross-sectional and longitudinal data above. It is important to note that any studies which assess changes in anthropometrical status with age may be subject to survivor bias. The individuals described in this chapter have all survived from 1990-1996 and, in this sense, they may represent a "biological elite" (Exton-Smith, 1982). This is also true for the subjects who are described in other similar studies. However, the bias towards mortality or survival may be different in distinct populations, e.g. it may be possible that a large change in BMI would be life-threatening in Sarawak but not in Europe. If the bias towards survival is different in distinct populations, then one would expect to find larger changes of BMI in the European survivors than in the Iban survivors. This point should be borne in mind when considering any comparison in anthropometric change between distinct populations.

It is generally accepted that there is a progressive decline in absolute height with ageing due to compression of the spinal vertebrae and kyphosis. This phenomenon has been well reported in Caucasians above 50-60y, often as a result of cross-sectional studies and sometimes as a result of longitudinal studies (Borkan & Norris, 1977; Noppa et al, 1980; Chumlea et al, 1988). A recent longitudinal study by Chumlea et al (1988) of white, healthy, middle class elderly men and women (aged 60-80y) found the rate of decrease in stature was estimated to be 0.5 cm/y irrespective of sex and age cohort. The SENECA investigation study (de Groot et al, 1996) observed a gradual 1-2 cm decrease in height for both men and women above 70y over 4 years. A cross-sectional study of elderly females in peninsular Malaysia (Yassin & Terry, 1991) found

that the mean height of 55-59 year olds was 4.8 cm more than the mean height of those in the 70y+ age group. The decrease found in this study of 0.25cm/year above 60y is comparable to that found by Cline et al (1989).

It is probable that a secular trend in growth does exist in this population, at least for men. A decrease in half-span length is seen as the age of each group increases. Also, in the cross-sectional data, lower median heights are progressively seen in each age group even under aged 50y, i.e. before adults usually lose height because of kyphosis etc. It is probable that the younger groups were better nourished as children than the older groups or suffered fewer childhood diseases and hence grew closer to their genetic potential. The exception to this rule is the women aged 30-40y who have lower median heights than the women aged 40-50y. It is possible that these women were exposed to adverse environmental conditions during their childhood and hence became stunted. Alternatively, selective out-migration of the stronger individuals from the longhouses may have occurred within this age group (see below).

This study has found that body weight increases in middle aged and decreases above age 50y in both sexes. This pattern is similar to that described by other authors, although the age at which weight starts to decrease may be higher in the developed world (Noppa et al, 1980; Borkan & Norris, 1977; Burr & Philips, 1984; Norgan & Ferro-Luzzi, 1982; Shephard, 1991). Chumlea et al (1988) found a loss of approx. 1.0 kg per decade for those aged 60-80y in his study. The Malaysian study found that a difference of 11.7kg between the 70y+ group and the 55-59y group.

The pattern of change in the BMI and MUAC, particularly the BMI derived from the half-span, is similar to that described for weight i.e. the measurements increase in early middle age and decrease after age 50y. This has been observed in studies in the developed world (Rolland-Cachera et al, 1991; Burr & Philips, 1984; Smith et al, 1995). Smith et al (1995) reported an annual decrease in BMI in Canadian men and women aged 55 to 86 years of 0.083 kg/m^2(men) and 0.065 kg/m^2 (women). A similar phenomenon has been noted in the developing world. De Vasconcellos (1994) found that the probability of having a low BMI was greater in young people and decreased with age up to 50y (after controlling for SES); above 50-60y he found that the BMI started to decrease. Delpeuch et al (1994), Giay and Khoy (1994), Ge et al (1994), Gopalan (1988) and Yassin and Terry (1991) found similar results, although the ages at which the BMI started to decrease were slightly different in each study.

In this study FM increases with age in men, although the increase is smaller at older age groups. In women FM decreases in the oldest groups. FFM decreases as age increases in both sexes.

These results are similar to those reported by other authors (Novak, 1972; Forbes, 1976; Noppa et al, 1980; Borkan & Norris, 1977; Shephard, 1991; Munro & Danforth, 1989; Yassin & Terry, 1991). The existing evidence suggests that the greatest age related gains occur in subcutaneous tissues of the lower part of the trunk. The thickness of subcutaneous and internal adipose tissues are reported to increase in the trunk area with increasing age (Borkan & Norris, 1977; Shephard, 1991). Adipose tissue thicknesses decrease or remain constant on the arms and legs with age (Young et al, 1963; Shephard, 1991). This results in a reduced ratio of subcutaneous fat to total body fat. The result of these changes is that the body undergoes reciprocal changes in lean body mass and body fat (Bowman & Rosenberg, 1982) and hence that older adults will have a different body composition compared to younger adults. This is reflected in the figures of change in fat percent with age (figures 3.34-3.35) and is discussed further in the following chapter.

If the BMI or MUAC or any other anthropometric measurement which is associated with age is to be used to assess current nutritional status, the cause of the association between the measurement and age should be elucidated. Are the changes in age due to environmental conditions or physiological processes?

In the analyses above age remains associated with the BMI and MUAC even after controlling for SES factors or environmental conditions. De Vasconellos (1994) reported a similar finding in a cross-sectional survey in Brazil. The BMI increased in the youngest age groups and then decreased within all SES groups, however the mean age at which BMI began to decrease was markedly later in rich adults compared to the poorer groups.

Unfortunately the SES factors in this study have been crudely measured, and no attempt was made to ascertain SES individually - only HH SES data are available. In this population it is difficult to assess individual SES because earnings from the farm or an external job may be pooled within a *bilek*. However, some individuals working outside the longhouse may keep their wage separately from the HH finances in some cases. The problem with HH SES data is that it is not possible to ascertain how the resources are distributed. Thus it is not possible to know if, for example, as much money is spent on the doctor's fees for a sickly elderly individual as it would be for a young man who was actively bringing in the family income. Nor is it possible to know whether or not older people are given (or take) the same amount of food as those who are physically active in the farm. In the analyses in this study it is assumed that the resources are equably distributed between a bilek's members and thus that all the individuals in a certain HH have the same SES.

Various physiological processes could account for the changes in anthropometry with ageing - (i) the prevalence of chronic disease is higher among the elderly (Meydani & Blumberg, 1989). This may result in secondary malnutrition due to malabsorption or to tissue losses through the disease process. Furthermore, because of the increase in the prevalence of diseases, the elderly use prescription drugs more than other age groups. Drug and nutrient interaction can result in anorexia or weight loss (Fischer, 1990). (ii) Elderly individuals are more likely to have dental problems and this may result in their not being able to obtain the quantities or quality of nutrients they require. (iii) Degenerative physiological changes during the ageing process, for example, changes in the gastrointestinal tract, may give rise to altered nutrient requirements and status. There is some evidence that nutrient (e.g. zinc and ascorbic acid) uptake by cells can decrease with age (Munro & Danforth, 1989). Exton-Smith (1982) also suggests that there is some evidence that the elderly can become nutrient deficient more easily (than the general population) in response to stressful illness because their metabolism is already compromised by the ageing process.

If the change in anthropometrical status seen with ageing is due mainly to physiological processes is a low BMI in an older person equivalent (in terms of health and functional outcomes) to a higher BMI in a younger person? A similar question could be posed for sex. Probably the only way to answer these questions is to assess the impact of low BMI or MUAC on health within different age groups.

All of the anthropometric variables discussed in this chapter are associated with factors other than age or sex. The most prominent of these factors is migration. The group of individuals who were working in a town and not usually resident in their rural longhouses at visit A were taller, heavier and fatter than their counterparts who were resident in the longhouses. The height of these individuals had not changed in the intervening period and hence a taller group of individuals must have migrated to the towns. When assessing the cross-sectional data it is not immediately obvious whether these individuals were heavier and fatter because they had migrated to the towns or that a group which was heavier and fatter had migrated to the towns since 1990. One way of establishing this is to (a) assess the BMI or MUAC of the migrant group in 1990 and (b) examine the change in their BMI compared to the rest of the population.

By using a multiple regression controlling for age and sex, it was found that the mean 1990 BMIs and MUACs of those who were migrant in 1996 were higher ($p<0.05$) than those who remained resident in the longhouses. However, the migrants' changes in BMI and MUAC were not different from non-migrants'. These results imply that individuals with higher BMIs and

MUACs were more likely to migrate to the towns than individuals with lower values of these variables in 1990. This phenomenon, known as selective migration, has been described elsewhere (Mascie-Taylor, 1984; Macbeth, 1984; Bogin, 1988). It is probable that SES confounds any unique biological difference seen between the migrants and sedentes, i.e. that migrants tend to be heavier (and healthier) but also tend to have a higher SES than non-migrants (Bogin, 1988). Given that SES is crudely measured in this study and that the number of migrants is relatively small, it is difficult to assess any association between SES and migration more thoroughly.

Distance to the nearest town is also an important confounding factor. The group of individuals who live more than two hours away from the town have lower mean BMIs and MUACs as well as FM and weight than the group which lives closer to a town. Thus the more rural group has lower values of these anthropometric variables. The group who live closer to a town were also more likely to put on more weight and FM and increase their BMI and MUAC between 1990-1996 than the group who live further away. Other authors have reported similar results, finding either a lower mean BMI or more CED in rural areas (Giay & Khoy, 1994; Delpeuch et al, 1994; de Vasconcellos, 1994) or more overweight in the urban areas (Ge et al, 1994; Berdasco, 1994).

For the Iban, distance to the nearest town measures the ease of access to the shops and markets, and thus food not produced on the farm, and also to medical care. Those inhabitants who live further away are more self-reliant in terms of depending on the food they grow or catch rather than being able to go to the shops. This may result in those living further from town not only having less processed food, but also having to work harder on the farm in order to make sure it can provide everything they need. The fact that crop selling tended to be negatively associated with change between 1990-1996 may also be explained in terms of this phenomenon. One might expect that selling crops would lead to an higher income and hence expect a positive association. However, crop selling and distance to the closest town are associated in this population. Those who live further from town are more likely to sell crops (χ^2=5.6, pr=0.02).

In summary, the rural group, who have lower mean values of BMI, MUAC and weight, have a more "traditional" lifestyle than those who live closer to a town. Hence this study supports the hypothesis that mean adult weight and the prevalence of obesity rise with modernisation (McGarvey et al, 1989).

Other SES variables associated with anthropometrical status in this study were concerned with the HH's wealth. This type of variable is difficult to measure and assess as not only is the subject a sensitive one to ask questions about, but also each HH has many ways in which to use its income, for example they could buy more land, or a television, or put the money in the bank,

or even pay for a relative's operation. Despite these difficulties and the resulting crude measures, cross-sectionally, the MUAC and the BMI were found to be positively associated with the HH score on the material possessions scale. The group of individuals who scored more than average on this scale were found to have higher mean BMIs and MUACs than those who scored less. The mean MUAC was also higher in the group which had savings. These results are similar to those described by other authors (Naidu & Rao, 1994; Kennedy & Garcia, 1994) although most do not report a linear association with the variables but an increase in the amount of low BMI and decrease in the probability of high BMI in the lower income groups (de Vasconcellos, 1994; Delpeuch et al, 1994; Branca et al, 1993). Other authors have not found an obvious association between the BMI and SES variables (Gray & Khoy, 1994). In terms of longitudinal change, the BMI, FM and fat percent were also found to be associated with the material possessions scale. The implication of these results is that the BMI and MUAC are sensitive to the variables used to measure HH wealth.

Both cross-sectionally and longitudinally smoking was found to be negatively associated with the BMI, MUAC, weight, FM and fat percent. The trend for current smokers to be lighter than non-smokers or ex-smokers is well established (Molarius et al, 1997; Bermingham et al, 1996; Rasky et al, 1996) although the physiological processes which lead to this are not completely understood.

In the 1996 data, the group of individuals who were physically active at some point in the last farming year had a higher mean BMI, MUAC, FM and fat percent than the rest of the population. Similarly, physical activity was positively associated with change in MUAC, FM and fat percent between 1990-1996. This finding could mean that those who were physically active became heavier or that those who were thin were less likely to be physically active. Other research has shown that as food intake is reduced activity patterns change and that less energy is expended on non-obligatory activities (Ferro-Luzzi et al, 1992) or, when access to food is severely limited, that inactivity replaces normal behaviour (Keys et al, 1950). However a lack of physical activity could lead to wasting or a reduced income and hence lower BMIs. Some adults may retire from physical work because of old age (62.4% of the non-active individuals are 60y+) or because they develop a chronic illness. In this population 21% of those who were ill were inactive compared to 11% of those who were healthy (χ^2=10.5, p=0.001). The association between low BMIs or MUACs and inactivity may be explained by illness. It should be noted that other authors (Bermingham et al, 1996) found no association between exercise patterns and the BMI.

In this study, after controlling for age and sex, no significant associations were found between the anthropometric variables and educational level, quality of building materials used for the *bilek*, water supply or quality of land. Some studies have found that educational level is associated with the BMI or MUAC (Baqui et al, 1994; Berdasco, 1994) or with type of housing (Baqui et al, 1994). Bermingham et al (1996) found no association with alcohol intake. No association between the anthropometric variables and HH composition data (e.g.: total number of occupants or ratio of adults: children) was found. Some studies have reported that household size may affect child growth (Russel, 1976; Stinson, 1980) but others have not found this effect (Malina et al, 1985).

3.9 Conclusions

- The anthropometrical status of the study population was similar to that described for other populations in the developing world except the youngest female group who more closely resembled Americans.

- The distributions of all the anthropometrical variables were related to sex and age, although the BMI and the MUAC were least affected by these factors.

- Most of the anthropometric measurements were sensitive to SES and other lifestyle factors.

CHAPTER FOUR

ASSOCIATIONS BETWEEN THE ANTHROPOMETRIC MEASUREMENTS AND INIDCES

The purpose of this chapter is to assess the relationships between the anthropometric measurements and the indices themselves. By examining the associations seen it should be possible to conclude whether (i) the indices are independent of height and shape, and (ii) the indices reflect current body energy stores.

4.1 Associations between the anthropometric measurements

The basis of this analysis is the data set for the 601 non-pregnant individuals for whom all anthropometric and socio-economic data are available. All the anthropometric measurements and indices were normalised using box-cox transformations to reduce their skew.

The correlation coefficients of stature with the other bony measurements and the CI are shown in table 4.1. Stature is highly correlated with both sitting height and half-span, although the correlations are slightly higher between stature and sitting height - particularly in the older groups. This may be because sitting height and stature both decrease with age but long bone length does not and thus stature and sitting height are more evenly correlated in all age groups. The CI is not significantly associated with stature except weakly in the middle aged male and youngest female groups.

Table 4.1 Correlations of stature with sitting height, half-span and Cormic index

	Age group	N	Sitting Height	Half-span	Cormic Index
Men	24.0-39.9	91	0.841*	0.795*	-0.070
	40.0-59.9	125	0.805*	0.802*	-0.181+
	60+	80	0.858*	0.786*	-0.078
	All	296	0.848*	0.803*	0.013
Women	24.0-39.9	89	0.832*	0.796*	-0.248+
	40.0-59.9	136	0.846*	0.837*	-0.052
	60+	80	0.823*	0.804*	-0.105
	All	305	0.841*	0.796*	0.017

Table 4.2 shows the correlations of weight with body composition variables. Weight is significantly correlated with all the measures of body energy stores and these correlations are

higher in women than in men. The weight-triceps correlations are the lowest, presumably because the triceps measurement only considers the peripheral subcutaneous fat in the arm and does not take into account the central mass of fat found in the trunk. FM is more highly correlated with weight than is fat percent. FFM is more highly correlated with weight than is FM in men, but the converse is true for women.

Table 4.2: Correlations of weight, with the triceps skinfold, fat mass, fat-free mass and fat percent.

	Age group	N	Triceps	Fat mass	Fat-free mass	% fat
Men	24.0-39.9	91	0.486*	0.731*	0.877*	0.499*
	40.0-59.9	125	0.566*	0.820*	0.837*	0.616*
	60+	80	0.623*	0.860*	0.870*	0.719*
	All	296	0.566*	0.754*	0.873*	0.496*
Women	24.0-39.9	89	0.719*	0.936*	0.913*	0.737*
	40.0-59.9	136	0.734*	0.921*	0.845*	0.713*
	60+	80	0.829*	0.947*	0.875*	0.827*
	All	305	0.805*	0.930*	0.891*	0.725*

Tables 4.3 a & b present the correlations of several weight-for-height indices with the MUAC and height, weight, body composition variables and the CI. The tables are presented in order to compare the properties of the various weight-for-height indices and the MUAC in this population. W/H^p represents Benn's index (see methods chapter).

W/H, W/H^3 and MUAC are significantly correlated with height in at least one age group in one of the sexes, although in women W/H^3 is not related to height within the age bands. The correlations between W/H^p and height are not significant for either sex, even when all the age groups are combined. The BMI is significantly correlated with height in both sexes if age is not taken into account. However, the correlations within the age bands are not significant and range from -0.03 to 0.18.

As expected, the weight-for-height indices and the MUAC are all significantly positively correlated with weight, more so for women than men. In both sexes W/H and BMI are more highly correlated with weight at all ages than either W/H^3 or W/H^p. The MUAC is almost as highly correlated with weight as the BMI is.

Table 4 3a *Correlation coefficients of the various height-for-weight indices with the MUAC and other anthropometric and body composition variables in men*

INDEX	Age group	N	Height	Weight	FM	FFM	Fat %	CI
W/H	24 0-39 9	91	0.402*	0 965*	0 757*	0 810*	0 547*	0.175
	40 0-59 9	125	0 353*	0 974*	0 858*	0 758*	0 682*	0 150
	60+	80	0 295!	0 960*	0 894*	0 772*	0 784*	0.035
	All	296	0 428*	0 972*	0 797*	0 801*	0 567*	0.253*
W/H^2	24 0-39 9	91	0 077	0 823*	0 710*	0 645*	0 548*	0 211+
	40 0-59 9	125	0 088	0 875*	0 842*	0 616*	0 712*	0 228+
	60+	80	-0 032	0 814*	0 842*	0 581*	0 780*	0 065
	All	296	0 145+	0 860*	0 787*	0 650*	0 611*	0 277*
W/H^3	24 0-39 9	91	-0 249+	0 593*	0 583*	0 415*	0 485*	0 223+
	40 0-59 9	125	-0 189+	0 707*	0 758*	0 429*	0 682*	0 292*
	60+	80	-0 328!	0 601*	0 715*	0 352*	0 703*	0 086
	All	296	-0 159!	0 664*	0 702*	0 438*	0 596*	0 277*
W/H^F	24 0-39 9	91	-0 067	0 731*	0 664*	0 551*	0 528*	0 219+
	40 0-59 9	125	-0 030	0 811*	0 815*	0 541*	0 706*	0 259!
	60+	80	-0 166	0 727*	0 795*	0 484*	0 754*	0 075
	All	296	0 015	0 785*	0 760*	0 566*	0 612*	0 280*
MUAC	24 0-39 9	91	0 230!	0 801*	0 674*	0 630*	0 512*	0 243+
	40 0-59 9	125	0 198+	0 845*	0 795*	0 607*	0 663*	0 104
	60+	80	0 241+	0 843*	0 802*	0 671*	0 711*	-0 011
	All	296	0 336*	0 861*	0 723*	0 700*	0 525*	0 256*

$W/H^F = W/H^{2.42}$

Table 4.3b *Correlation coefficients of the various height-for-weight indices with the MUAC and other anthropometric and body composition variables in women*

INDEX	Age group	N	Height	Weight	FM	FFM	Fat %	CI
W/H	24.0-39.9	89	0.431*	0.978*	0.957*	0.844*	0.801*	0.159
	40.0-59.9	136	0.278!	0.980*	0.954*	0.758*	0.790*	0.152
	60+	80	0.333!	0.986*	0.961*	0.820*	0.866*	0.220
	All	305	0.421*	0.985*	0.950*	0.835*	0.780*	0.334*
W/H^2	24.0-39.9	89	0.185	0.892*	0.925*	0.708*	0.833*	0.243+
	40.0-59.9	136	0.060	0.913*	0.947*	0.623*	0.841*	0.171+
	60+	80	0.147	0.935*	0.944*	0.730*	0.881*	0.257+
	All	305	0.228*	0.929*	0.938*	0.736*	0.815*	0.357*
W/H^3	24.0-39.9	89	-0.096	0.730*	0.820*	0.506*	0.805*	0.316!
	40.0-59.9	136	-0.168	0.796*	0.891*	0.452*	0.851*	0.182+
	60+	80	-0.054	0.845*	0.886*	0.608*	0.861*	0.285+
	All	305	0.008	0.824*	0.879*	0.594*	0.814*	0.365*
W/H^P	24.0-39.9	89	-0.091	0.734*	0.823*	0.510*	0.806*	0.315!
	40.0-59.9	136	-0.164	0.799*	0.893*	0.456*	0.852*	0.182+
	60+	80	-0.049	0.847*	0.888*	0.611*	0.862*	0.284+
	All	305	0.012	0.826*	0.881*	0.597*	0.814*	0.365*
MUAC	24.0-39.9	89	0.287!	0.860*	0.894*	0.682*	0.810*	0.204
	40.0-59.9	136	0.144	0.884*	0.933*	0.584*	0.839*	0.139
	60+	80	0.238+	0.912*	0.943*	0.680*	0.894*	0.197
	All	305	0.313*	0.909*	0.929*	0.708*	0.816*	0.325*

$W/H^P = W/H^{2.58}$

The correlations between the weight-for-height indices or MUAC and body composition variables are all higher in women than men, particularly for fat percent. W/H and the BMI have the highest correlations with FM in both sexes. Relatively similar correlations are found between percentage fat and all the weight-for-height indices. The BMI and the other weight-for-height indices are more highly correlated with FM than fat percent. FFM is most highly correlated with the W/H and BMI in both sexes; the BMI is more highly correlated with FM than FFM in this population. The MUAC's correlations with body composition variables are lower than those of the BMI or W/H but higher than those of W/H^p or W/H^3. In general, the correlations between the weight-for-height indices and MUAC and FM or fat percent increase with age whereas those with FFM decrease with age.

Figures 4.1 and 4.2 show the relationships between the BMI and body energy stores. Note that many of the individuals with the highest levels of FM and fat percent are not shown on this graph as they were excluded from analysis during data cleaning (see methods chapter). At a given BMI, FFM is greater than FM in both sexes. However, men generally have a higher FFM than women and women have a higher FM at any BMI value. This is reflected in figure 4.3 which shows that women have a higher percentage fat than men at any given BMI. An increase in the BMI is concurrent with a linear increase in both FM and FFM although the two components do not increase in an identical manner in each sex. FM increases more steeply than FFM in women which results in the gap between the amounts of FFM and FM being reduced at the higher values of BMI. The relationship between the MUAC and body composition variables are similar to those of the BMI (figures 4.4-4.6). In a regression model fat percent was found to account for 22.1% of the variance in BMI and 12.7% of the variance in MUAC.

The CI is significantly associated with all the weight-for-height indices in at least one age band except with W/H. The strongest correlations are found with W/H^1 and W/H^p. The CI is also correlated with the BMI in all age groups except the oldest men. Within the separate age bands the MUAC is not significantly correlated with the CI. However, if all the age groups are pooled then even the MUAC is found to be significantly associated with the CI.

Figure 4.1: BMI and fat mass and fat-free mass in men at visit A (n=296)

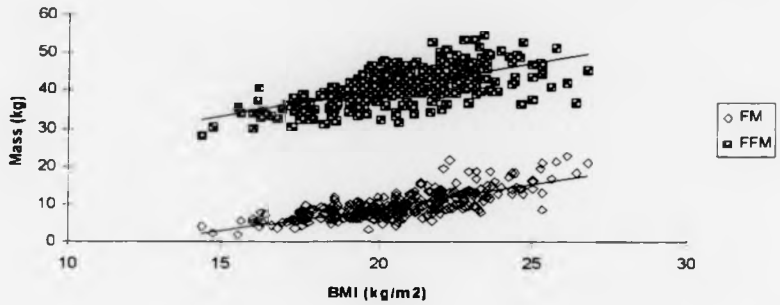


Figure 4.2: BMI and fat mass and fat-free mass in women at visit A (n=305)

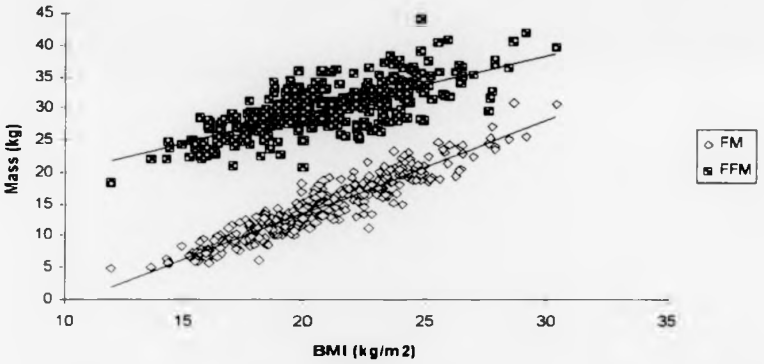


Figure 4.3: BMI and fat percent in men and women at visit A (n=601)

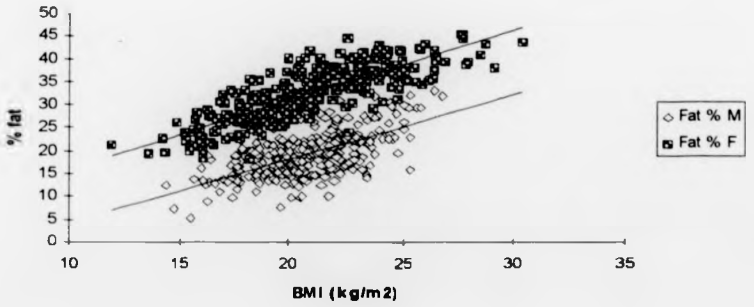


Figure 4.4: MUAC and fat mass and fat-free mass in men at visit A (n=296)

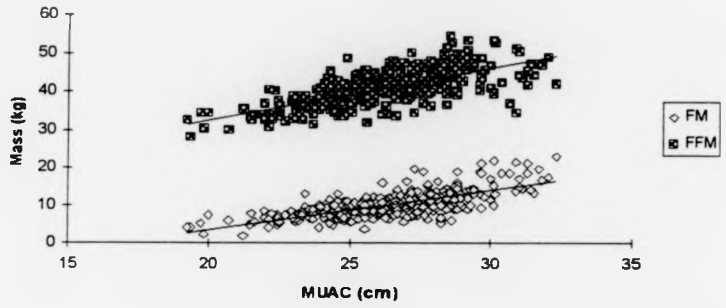


Figure 4.5: MUAC and fat mass and fat-free mass in women at visit A (n=305)

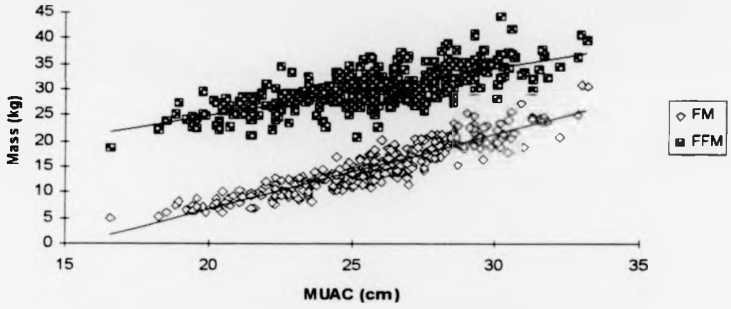
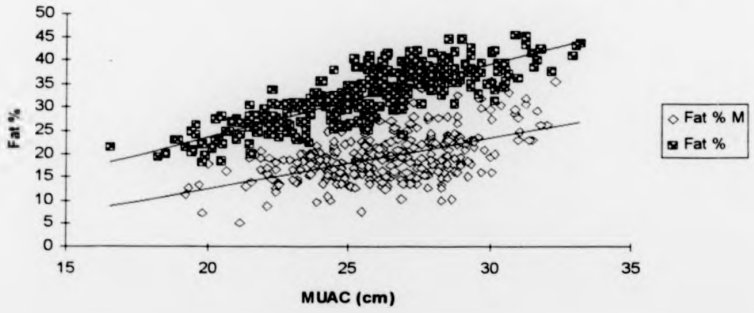


Figure 4.6: MUAC and fat percent in men and women at visit A (n=601)



All the weight-for-height indices and the MUAC are highly correlated with each other. The correlations range from 0.77 to 1.00 (see tables 4.4a and b). In particular, the BMI explains 82.1% of the variance seen in the MUAC. These correlations are slightly higher for women than men.

Table 4.4a. The correlations between the weight-for-height indices and MUAC in men (n=296).

	W/H	W/H^2	W/H^3	W/H^p	MUAC
W/H	1.000*				
W/H^2	0.955*	1.000*			
W/H^3	0.819*	0.951*	1.000*		
W/H^p	0.907*	0.991*	0.984*	1.000*	
MUAC	0.899*	0.875*	0.767*	0.838*	1.000*

p=2.42

Table 4.4b. The correlations between the weight-for-height indices and MUAC in women (n=305).

	W/H	W/H^2	W/H^3	W/H^p	MUAC
W/H	1.000*				
W/H^2	0.979*	1.000*			
W/H^3	0.909*	0.975*	1.000*		
W/H^p	0.911*	0.976*	1.000*	1.000*	
MUAC	0.939*	0.938*	0.892*	0.893*	1.000*

p=2.98

4.2 The associations between the anthropometric variables controlling for age and sex

The correlations between the anthropometric variables vary in strength according to age and sex. Hence the addition of age and sex to these correlations (using the partial correlation method) may alter the strength of some of the correlations. Table 4.5 below shows, for the whole population, the associations between the BMI and MUAC and other anthropometric variables using both zero-order correlations and the partial correlations (bold type) controlling for age and sex.

Table 4.5: Zero order and partial correlations (controlling for age and sex) between the BMI and MUAC and other anthropometric variables.

	Height	Weight	Fat mass	Fat-free mass	Fat percent	Cormic index
BMI	0.129!	0.832*	0.757*	0.433*	0.471*	0.321*
	0.057	**0.878***	**0.869***	**0.622***	**0.771***	**0.170***
MUAC	0.302*	0.859*	0.668*	0.522*	0.359*	0.278*
	0.196*	**0.858***	**0.846***	**0.604***	**0.755***	**0.117!**

The partial correlations are generally similar to the zero-order correlations, particularly for weight. However, the addition of age and sex does decrease the strength of the association between height or the CI and the BMI or MUAC. In the case of the BMI and height, once sex and age are controlled, no significant association is seen between the two variables. The correlations for the body composition variables are all made stronger by controlling for age and sex.

The regressions between the BMI or MUAC and body composition variables can be improved by adding sex and age into the equations. Table 4.6 shows the variance explained if age and sex are added to the regressions between the BMI and MUAC and FM, FFM and fat percent. No interaction terms were significant. It can be seen that addition of sex to these equations greatly increases the amount of variance explained. This is especially true for the FFM and fat percent regressions. The addition of age also strengthens the regressions.

Table 4.6 Variance explained by the regressions between BMI or MUAC and FM, FFM and fat percent controlling for sex and or age.

	Fat mass (r^2*100)	Fat-free mass (r^2*100)	Fat percent (r^2*100)
BMI	57.2	18.6	22.1
BMI + sex	81.6	79.4	80.1
BMI + age	58.6	23.2	25.5
BMI + sex + age	83.2	84.6	84.0
MUAC	44.6	27.1	12.7
MUAC + sex	77.5	79.7	78.0
MUAC + age	45.8	29.5	15.1
MUAC + sex + age	79.9	84.1	82.6

4.3 Discussion

In reviewing the results above, this discussion will assess the relative utility of the various weight-for-height indices and the MUAC in terms of their properties as ideal indices of adult nutritional status. In particular the following properties will be examined: (i) independence from height and shape, and (ii) correlations with body energy stores.

Billewicz et al (1962) noted that an (obesity) index that is correlated with height, even moderately, cannot be safely used in comparison studies, since it could potentially produce misleading results. Thus, in terms of height, W/H^p is the best index of nutritional status as it has no significant correlations with stature (Lee et al, 1981). All the other indices show a significant association with height if age is not controlled for. The BMI is the least highly correlated and does not show any significant association if age is taken into account. Other authors have reported similar results, i.e., that in men W/H^2 correlates less highly with height than W/H or W/H^3 (Evans & Prior, 1969; Goldbourt & Medalie, 1974; Wormersley & Durnin, 1977; Seod et al, 1984; Garn et al, 1986; Keys et al, 1972; Micozzi et al, 1986; Khosla & Lowe, 1967; Revicki & Israel, 1986; Smalley et al, 1990) although some report (Lee et al, 1981; Micozzi et al, 1986) that either W/H or $W/H^{1.5}$ have lower correlations with height than the BMI in women. In this population W/H^p for women is close to W/H^3 and hence an index of W/H or $W/H^{1.5}$ would be inappropriate.

Few data are available on the relationship between MUAC and stature, but in this population MUAC correlates more strongly with height than the BMI and some of the correlations are still significant after controlling for age. Thus, if Billewicz et al's condition must be met, then the MUAC may not be an ideal index of nutritional status.

It should be noted, however, that although there is no a priori reason to believe that in the general population less tall individuals tend to have greater or smaller skinfold thickness than taller individuals, some studies have observed a correlation between height and body fat measured by skinfolds (Wormersley & Durnin, 1977; Norgan & Ferro-Luzzi, 1982). Other authors such as Micozzi et al (1986) found that stature is unrelated to subscapular skinfold thickness for men and only slightly correlated for women or that stature is unrelated to total body fat in adults (Roche et al, 1984; Rush et al, 1997). If height is associated with skinfold thickness then an appropriate anthropometric index will be correlated with height in a similar manner (Norgan & Ferro-Luzzi, 1982). In this study the correlations between height and fat percent are all non-significant and range from 0.05-0.12 in men and -0.08-0.14 in women (see table 4.7). These correlations are of

the same magnitude as those between the BMI and height and hence indicate that the BMI is an appropriate index. As the MUAC and height are more highly correlated MUAC may not be so appropriate in this respect.

Table 4.7: The correlations between height and fat percent

	Age group	N	% fat
Men	24.0-39.9	91	0.124
	40.0-59.9	125	0.054
	60+	80	0.115
	All	296	0.035
Women	24.0-39.9	89	0.145
	40.0-59.9	136	-0.079
	60+	80	0.137
	All	305	0.108

The CI in this population ranges from 0.49-0.57 (mean=0.54; SD=0.01) which is similar to that described by Norgan (1994b) for other Asian populations. The correlations between the BMI and the CI range from 0.07 to 0.26. These are comparable to those reported by Garn et al (1986) in NHANES data and those reported by Norgan (1994a) for individuals from Papua New Guinea. Regressions between the BMI and CI show that approximately 10.7% of the variance in the BMI is associated with the CI, although this figure is reduced to approximately 3% if age is controlled. This implies that the BMI is, to some extent, affected by shape. If this is true then the BMI may be an invalid index of nutritional status - at least unless shape is taken into account.

The MUAC is also correlated with the CI if age is not taken into account. Approximately 7.6% of the variance in MUAC may be associated with the CI in this population. However, once age is controlled for only 1% of the variance in MUAC is associated with the CI. As far as the author knows, no data on the relationship between MUAC and CI has been published. If the results of this study can be generalised then in this respect the MUAC may be a better measure of nutritional status than the BMI.

In this population the BMI-weight correlations range from 0.814-0.935. These results are consistent with those found by other authors (Khosla & Lowe, 1967; Evans & Prior, 1969; Goldbourt & Medalie, 1974; Keys et al, 1972; Wormersley & Durnin, 1977; Lee et al, 1981; Miccozzi, 1986; Smalley et al, 1990; Norgan, 1990; Revicki & Israel, 1986; Garrow, 1988; Gallagher et al, 1996). Similarly, the MUAC is also strongly correlated with weight (James et al, 1994b).

The weight-for-height indices and MUAC are all strongly associated with FM as reported by other authors (including Smalley et al. 1990; Roche et al. 1981; Revicki & Israel. 1986. Norgan. 1990. 1982. 1994; Durnin & Wormersley. 1977; Goldbourt & Medalie. 1974; Evans & Prior. 1969; Micozzi et al. 1986; Keys et al. 1972; Scod et al. 1984; Rush et al. 1997. Strickland & Ulijaszek. 1993 ; Strickland & Tuffrey. 1997; Garrow. 1988; Deurenberg et al. 1989. 1991; Khosla & Lowe. 1967; Gallagher et al. 1996; Wang. 1994; Luke et al. 1997). The BMI is also a measure of FFM. The correlations reported by other authors (Norgan. 1994; Strickland & Tuffrey. 1997) between the BMI and FFM range from 0.57 to 0.76 in several ethnic groups and are of the same magnitude as those reported in this study. although Immink et al (1992) found a much lower correlation (0.37) in Guatemala.

Most researchers find the relationship between BMI and body composition variables to be linear over 'normal' levels (Norgan. 1994b; Garrow. 1988; Gallagher et al. 1996; Wang. 1994) and this is true for the current data although Luke et al (1997) described a quadratic relationship for black women.

The correlations between all the weight-for-height indices and the MUAC are lower with fat percent than FM. Other authors have also reported this phenomenon (Norgan. 1994b; Strickland & Tuffrey. 1997; Gallagher et al. 1996). The correlations reported are usually in the range from 0.49 to 0.91 for F% and from 0.64 to 0.95 for FM. However. Immink et al (1992) found much lower correlations (F% 0.12-0.23 and FM 0.32-0.4) in a rural Guatemalan population.

The correlations between the weight-for-height indices themselves are all high and are consistent with results reported by other authors (Evans & Prior. 1969. Goldbourt & Medalie. 1974. Scod et al. 1984. Garn et al.1986. Khosla & Lowe. 1967. Keys et al. 1972; Wormersley & Durnin. 1977. Lee et al. 1981. Miccozzi. 1986. Smalley et al. 1990) The correlations between the BMI and MUAC are similar to those described by other authors (Collins. 1996. James et al. 1994b. Alemu & Lindtjorn. 1997).

The results of chapter 3 show that none of the anthropometric measurements are independent of age or sex. indeed much of the variation in these measures is associated with these two factors. In itself this is an interesting result and may lead to different cut-offs being employed for the diagnosis of CED in different age or sex groups. However. what is equally interesting is that the relationships between the BMI or MUAC and body composition variables vary according to age and sex.

The relationship between the BMI or MUAC and "fatness" is unquestionably different between the two sexes. Although no interaction between sex and the BMI or MUAC is seen, the regressions for men and women are different. Women have a higher fat percent than men at all BMIs. This result is reported elsewhere (Norgan, 1990, 1994b; Gallagher et al, 1996; Wang, 1994; Luke et al, 1997; Strickland & Ulijaszek, 1993; Strickland & Tuffrey, 1997). Differences in the absolute amounts of FM and FFM at a given BMI level are found between the sexes and are also reported by other authors. It is probable that a real biological difference (i.e. genetic difference) exists between the sexes as the disparity is found in other species as well. Presumably this reflects the female's greater need for energy stores to maintain a buffer against a nutrition-induced failure in reproductive function and allows women to cope with the substantial energy demands of lactation (Shetty & James, 1994).

In general women also seem to have a greater spread of BMI or MUAC values than men (James & Francois, 1994a). This may explain why the correlations between the weight-for-height indices or MUAC and body composition variables are higher in women than men. Males show a smaller range which automatically reduces the strength of such correlations. It is probable that this sex difference may represent a real characteristic of the study population as it was observed in this study and two previous studies of the same population (Strickland and Ulijaszek, 1990; Wells & Strickland, 1996). Norgan (1994b) and Wang (1994) reported similar results.

Age is also a confounding factor in the associations between the BMI or MUAC and the body composition variables, although it does not affect the relationships as profoundly as sex. The correlations between FM and fat percent and the BMI or MUAC increase in strength as age increases and, conversely, those with FFM decrease with age. Norgan (1994b) and Wormersley and Durnin (1977) also reported lower correlations between FM and the BMI in younger groups. Presumably these changes are seen as the proportion of weight made up of FM increases with increasing age and hence the BMI is a better reflection of FM than FFM in older age groups and vice versa. In this study the addition of age to the regressions did increase the variance explained in the relationship between the BMI or MUAC and body composition variables. Similar results were found by Norgan (1990), Gallagher et al (1996) and Wang (1994), although other authors (Micozzi et al, 1986; Immink et al, 1992; Norgan, 1994b; Luke et al, 1997) did not consistently find age to be an important confounder.

4.4 Conclusions

- The BMI and MUAC are both highly correlated with body energy stores but are not entirely independent of height or shape. Moreover the relationships between the BMI or MUAC and body energy stores are both sex and age dependent. Neither the BMI nor the MUAC should be used to predict accurate levels of body composition in this population without using age and sex specific equations.

- Much of the work criticising the use of the BMI as an index of nutritional status has focused on its use as an index of obesity i.e. a measure of fatness. In the context of this study the focus is on its use as an index of under-nutrition i.e. as a measure of body energy stores, which are made up of both FM and FFM. There can be no doubt that a low BMI approximates to low weight, FM and FFM in this population, even if the proportions of FM varies by age and sex group.

CHAPTER FIVE

MORTALITY

The purpose of this chapter is to examine the associations between anthropometric variables and mortality between 1990 and 1996. The central question to be addressed is "were those individuals who were malnourished in 1990 more likely to die between 1990 and 1996 than those who were well-nourished?" The majority of the analyses and discussion in this chapter focus on the relationship between the BMI or MUAC and mortality as these are the anthropometric variables of most interest to this project. However, the results of the analyses run for the other anthropometric variables are also be presented as these data are unique.

5.1 Description of the sample used for the mortality analyses

Follow-up on the mortality of the 1990 population of 1047 adults was considered 100% complete by the author. A total of 84 deaths were recorded by verbal autopsies. This figure is very close to that predicted by Coale and Demeney's (1983) West level 18 model (see methods chapter). 897 individuals were measured and interviewed by the author in 1996. A further 8 individuals were seen by the author but refused to be interviewed or measured in 1996. 58 subjects were not seen by the author. Of these 58, one was reported to have died but his family had migrated. The remaining 57 individuals were migrants (both temporary and permanent) and detailed information as to their whereabouts was obtained from relatives or friends in the same longhouse. Given the structure of Iban society and their traditions concerning death, the author is confident that all these migrants were alive at the time of interview with their relatives and friends.

Full verbal autopsies were applied by interview to the relatives of 84 of the deceased. In only one case was the author unable to interview the relatives of a non-survivor. This death was known to be non-accidental but the longhouse inhabitants did not know when the subject had died or what illness caused his death. The death was thus assumed to have occurred mid-way through the study from some unknown, non-accidental cause. The second round of interviews started on August 1st 1996 and this date was used as the census point for all the subjects; any deaths after this date were discounted. The three accidental deaths recorded were treated by excluding the individuals at the time of their deaths.

Subjects who were pregnant (n=35) in 1990 are excluded from all the analyses. All those who were obese in 1990 (n=21) are also excluded from the analyses. The obese are excluded as

almost all mortality studies (see introduction) report an excess of mortality above a BMI of 30kg/m^2. This study is interested in the relationship between mortality and low values of anthropometry and hence subjects with high values of anthropometry are excluded to prevent a "U" shaped relationship between the anthropometrical variables and mortality complicating the analyses. The bulk of the analyses have also excluded those individuals who were overweight (BMI>24.9kg/m^2) in 1990 (n=111). This is because many of the studies assessing the relationship between the BMI and mortality find that the risks of mortality start to rise above a BMI of 26-28kg/m^2. By excluding the overweight group this study can focus on the different risks of mortality between low and normal BMI individuals.

After the exclusions and censoring described above, a total of 991 individuals were included in the initial analysis (excluding only obese individuals). Of these, 80 had died from non-accidental causes by August 1st 1996 and 911 survived. When the overweight individuals are excluded the sample size falls to 880 (805 survivors and 75 fatalities).

5.2 Age and sex as confounding factors

Age is clearly closely associated with mortality rates. The result of a Cox regression between age and mortality with the non-obese sample can be seen in table 5.1. A similar regression was found using the non-overweight sample. Age appears to be linearly related to mortality, i.e.: age-squared is less significantly associated with mortality than age. Hence age is entered as a confounding factor in all the analyses described in this chapter.

Table 5.1 The association between age and mortality

	H R	95% Confidence Intervals	P-value
Age	1.111	1.091-1.132	0.001

p-value for the maximum likelihood ratio test

Sex is not significantly associated with mortality in this population. However, given that sex is associated with the anthropometric variables it was entered as a confounder into the regressions between the anthropometrical variables and mortality. Sex is not found to be a significant confounding factor in these relationships except when FFM was considered.

5.3 Initial analyses

Two types of analyses were run between mortality and each anthropometric variable. Firstly, the anthropometric measurement or index was entered as a continuous variable. Secondly, the group with values in the lowest quintile of a given anthropometric measurement in 1990 was compared to the rest of the population. Thus in the second type of analysis the anthropometric measurements were treated as categorical variables. Later analyses will assess the relative risk of mortality for CED and non-CED groups.

Tables 5.2 and 5.3 report hazard ratios and the 95% confidence intervals for mortality and the anthropometric measurements controlling for age and (where applicable) sex in the non-obese population. Table 5.2 shows that a unit increase in the MUAC or FFM significantly reduced the HR of mortality in the six year interval at the population level. The analysis of the data entered as categorical variables (table 5.3) shows that the groups whose values of weight and MUAC were in the lowest quintiles in 1990 had an increased risk of mortality compared to the rest of the population. The BMI has a borderline significant association with mortality in this section of the population.

Table 5.2. *HRs of mortality for the anthropometric measurements (entered as continuous variables) amongst all subjects who were not pregnant or obese in 1990 controlling for age and sex (N=991)*

	H.R.	95% C.I.	p-value
Height	0.995	0.966-1.025	0.741
Weight	0.978	0.950-1.005	0.114
BMI	0.928	0.857-1.006	0.069
MUAC	0.919	0.852-0.992	0.030
Fat mass	0.961	0.910-1.014	0.145
Fat-free mass	0.920	0.867-0.977	0.006

p-value for the maximum likelihood ratio test

Table 5.3: *RRs of mortality for the section of the population with the lowest quintile of anthropometric measurements compared to the rest of population amongst all subjects who were not pregnant or obese in 1990, controlling for age and sex (N=991).*

	R R	95% C I	p-value
Height	1.351	0.851-2.145	0.202
Weight	1.619	1.022-2.567	0.040
BMI	1.567	0.987-2.490	0.057
MUAC	1.631	1.031-2.580	0.036
Fat mass	1.591	0.990-2.557	0.055
Fat-free mass	1.712	0.783-3.746	0.178

p-value for the maximum likelihood ratio test

Tables 5.4 and 5.5 are equivalent to tables 5.2 and 5.3 except that the analyses are conducted only on those individuals whose BMI in 1990 was normal or low, i.e. the overweight individuals were excluded from these analyses. The HRs for each anthropometrical variable and mortality remain in the same direction as those seen above but they have become stronger (except for height). In these analyses a unit increase in the BMI as well as the MUAC and FFM result in a significantly lower HR of mortality at the population level. Similarly in the categorical analyses, the groups with the lowest quintile values of BMI or MUAC were more likely to die than the rest of the population. If this non-overweight section of the population is considered, those in the lowest quintile of weight no longer had an increased risk of mortality.

Table 5.4 *HRs of mortality for the anthropometric measurements (entered as continuous variables) amongst all subjects who were not -pregnant or overweight in 1990, controlling for age and sex (N=880)*

	H R	95% C I	p-value
Height	0.996	0.964-1.024	0.666
Weight	0.972	0.942-1.003	0.078
BMI	0.893	0.810-0.985	0.024
MUAC	0.888	0.814-0.969	0.007
Fat mass	0.932	0.869-1.001	0.052
Fat-free mass	0.908	0.851-0.969	0.003

p-value for the maximum likelihood ratio test

Table 5.5: RRs of mortality for the section of the population with the lowest quintile of anthropometric measurements compared to the rest of population amongst all subjects who were not pregnant or overweight in 1990, controlling for age and sex (N=880)

	R.R.	95% C.I.	p-value
Height	1.427	0.886-2.297	0.144
Weight	1.506	0.934-2.428	0.093
BMI	1.689	1.049-2.720	0.031
MUAC	1.824	1.136-2.927	0.013
Fat mass	1.617	0.987-2.648	0.056
Fat-free mass	2.042	0.862-4.838	0.105

p-value for the maximum likelihood ratio test

The exclusion of the overweight group strengthens the associations seen between the BMI or MUAC and mortality. It is therefore probable that excess of mortality is seen in the overweight group in this population and by excluding these individuals a more accurate analysis of the risks of mortality associated with low values of anthropometric variables can be obtained. Hence the rest of the analyses presented will focus on the section of the population which was not overweight or obese in 1990.

5.4 Analyses controlling for SES and other lifestyle factors.

The results above are interesting, but some authors (Kushner, 1993; Gronback et al, 1994) have shown that confounding factors such as smoking or alcohol intake may account for the associations seen. Educational level and access to medical care may also be important. Thus it is necessary to control for these and other SES and lifestyle factors as described in the methods chapter.

Unfortunately, no information on the subjects' education, smoking habits or alcohol consumption was collected in the 1990 survey. Nor were any detailed data collected on the household's SES or other lifestyle factors. However, data concerning these topics were collected during the interviews with all survivors in 1996, and relatives of the deceased were asked similar questions about those who had died. No information of this nature was collected for the 58 individuals who were not seen by the author or for those who refused. In hindsight it would have been useful, and probably fairly easy, to pose questions to the relatives and friends of the migrants - at least about their smoking habits.

Thus gaps in the socio-economic data exist. In total, after excluding pregnant and overweight individuals, there are 58 subjects for whom no data on confounding factors are available. This group of individuals, who will be referred to as the "non-interviewees", are a mixture of those who refused to be interviewed and those who were migrants. Thus they are a self-selected group and may not be typical with respect to either anthropometry or SES compared to the rest of the population.

The analyses shown below which control for smoking, drinking, SES and "other" lifestyle factors have been run excluding the 58 "non-interviewees". These analyses could be biased if being one of the 58 subjects is associated with anthropometric status in 1990, e.g. if high BMI in 1990 predicted being a migrant in 1996 then the survival of those with high BMI is underestimated. An association between anthropometric status and migration has already been reported in this study (see chapter 3). If the 58 "non-interviewees" are similar to the migrants actually measured in 1996 then a bias may exist. If migration or refusing interview in 1996 is not associated with anthropometrical status in 1990 but is associated with some or all of the confounding factors then no bias should exist. Equal proportions of survivors with high and low BMI would become "non-interviewees" and hence the relative risk would be unbiased overall.

Table 5.6 shows the 1990 mean anthropometric values of both those for whom information on SES and other "lifestyle" factors is available and the 58 "non-interviewees" for whom it is not. Analysis of variance was used to see if any of the differences between the two groups were significant. No significant differences were found between the female groups but some significant differences were found between the male groups. It can be seen that those men who were not interviewed were significantly younger than those who were (i.e. the migrants were younger). After controlling for age, the heights, weights, FM and FFM of the non-interviewees were significantly larger than those of the interviewees, however no significant differences were found in BMI or MUAC. This implies that not being interviewed was associated with anthropometry in 1990 and hence a bias may exist with respect to height, weight, FM and FFM.

Table 5.6. Summary characteristics of the study population in 1990. Means and SDs (excluding pregnant and overweight subjects).

	N	Age (yrs)	Height (cm)	Weight (kg)	BMI (kg/m^2)	MUAC (cm)	Fat mass (kg)	Fat-free mass (kg)
Interviewed in 1996								
Men	385	45.3 (16.2)	157.4 (5.4)	50.7 (6.2)	20.4 (2.0)	25.9 (2.4)	7.8 (2.6)	42. 9 (5.1)
Women	437	44.8 (15.9)	147.0 (5.1)	43.8 (6.8)	20.2 (2.5)	24.8 (2.9)	12.3 (3.5)	31.5 (4.2)
Both	822	45.0 (16.0)	151.9 (7.4)	47.0 (7.4)	20.3 (2.3)	25.3 (2.8)	10.2 (3.9)	36.8 (7.3)
Not interviewed in 1996								
Men	37	33.3 (13.6)*	161.0 (6.8)*	54.4 (7.0)*	21.0 (2.0)	26.6 (2.4)	8.3 (3.2)*	46.1 (5.4)*
Women	21	38.7 (18.1)	148.9 (5.5)	46.1 (6.9)	20.8 (2.7)	24.8 (2.5)	13.0 (3.5)	33.1 (4.5)
Both	58	35.2 (15.5)*	156.6 (8.6)*	51.4 (8.0)*	20.9 (2.2)	26.0 (2.6)	10.0 (4.0)	41.4 (8.1)*

*p<0.05 ANOVA

In order to test if a bias does exist, analyses identical to those run in table 5.4 and 5.5 were run on the group of subjects about whom information on confounding factors were available. The resulting HRs were almost identical to those in tables 5.4 and 5.5 and can be found in Appendix A9. The major differences seen between these analyses and those described above are, as table 5.6 suggests, in the analyses where height and FM are entered as categorical variables. The RRs of mortality for the groups whose height or FM were in the lowest quintile of the population in 1990 are greater than those reported earlier, and for FM and FFM become significant in this analysis. The RRs for the BMI and MUAC remain almost the same. Thus a bias in this subgroup of analyses does exist - the risks of mortality for the groups with low values of height, FM or FFM are increased. This should be borne in mind throughout the rest of the analyses.

Tables 5.7 and 5.8 show the results of stepwise Cox regressions between the anthropometric variables and mortality controlling for SES and other lifestyle factors. The confounding factors originally entered into the regressions included data on each individuals' smoking habits, alcohol consumption, education and cormic index and for each HH information on the distance to the nearest town, material possessions scale, savings, remittances, electricity, piped water, paid employment and crop selling.

Table 5.7: HRs of mortality for the anthropometric measurements (entered as continuous variables) amongst all subjects who were not pregnant or overweight in 1990 and for whom SES data were available, controlling for sex, age and SES factors. The associations of the significant confounding factors are shown.(N=822)

	HR	95% CI	p-value	CONFOUNDERS						
				Distance to closest town	Piped water	Smoking	School	Alcohol	Sex	Age
Height	0.996	0.935-0.997	0.041	+	-	+	+			+
Weight	0.959	0.929-0.990	0.011	+	-	+	+			+
BMI	0.896	0.810-0.992	0.035	+	-	+				+
MUAC	0.865	0.789-0.947	0.002		-			+		+
Fat mass	0.936	0.870-1.007	0.079		-			+		+
Fat-free mass	0.901	0.844-0.962	0.002	+	-				-	+

p-value for the maximum likelihood ratio test

Table 5.8: RRs of mortality for the section of the population with the lowest quintile of anthropometric measurements compared to the rest of the population amongst all subjects who were not pregnant or overweight in 1990 and for whom SES data were available, controlling for age, sex and SES factors. The associations of the significant confounding factors are shown.(N=822)

	RR	95% CI	p-value	CONFOUNDERS						
				Distance to closest town	Piped water	Smoking	School	Alcohol	Sex	Age
Height	2.107	1.241-3.578	0.006	+	-	+	+			+
Weight	1.801	1.098-2.954	0.020	+	-	+				+
BMI	1.694	1.045-2.745	0.032	+	-	+				+
MUAC	2.349	1.431-3.856	0.001		-			+		+
Fat mass	1.714	1.042-2.820	0.034		-			+		+
Fat-free mass	1.632	0.975-2.731	0.063	+	-	+	+			+

p-value for the maximum likelihood ratio test

A unit increase in weight, height, BMI, MUAC and FFM are all associated with a decrease in the risk of mortality at the population level. Those groups of subjects with values of height, weight, BMI, MUAC and FM in the lowest quintile had a significantly higher risk of mortality than the rest of the population. The addition of the confounding factors to the regressions does not alter the HRs for the BMI very much, i.e. the HRs are similar to those described in tables 5.4 and 5.5. However, the associations seen between mortality and MUAC, weight and height are stronger when the confounding factors are controlled; and those for height and weight become significant. The effects of controlling the confounding factors in the FM and FFM analyses are erratic. If the body composition variables are entered as continuous factors then FFM is significantly associated with mortality at the population level and FM is not. However, if the variables are entered categorically then the group with the lowest values of FM has increased RRs of mortality, but not the lowest FFM group.

Other than age, the most important confounding factor in these analyses is the presence of piped water in the *bilek*, the group which had piped water had decreased risks of mortality. Those living furthest from a town and those who smoked or drank more than once a week had increased risks of mortality. Surprisingly, those who had attended school had increased risks of mortality in the height regressions. None of the "wealth" variables were significantly associated with the risk of mortality in any of these analyses.

5.5 Analyses allowing for a "wash-out period"

One further refinement of the mortality analysis should be considered - the allowance of a "wash-out period". Some studies in the West have found that excess mortality in the underweight may occur early in the follow-up period, while that of overweight may be delayed (Manson et al, 1987). It has been suggested that this may be due to confounding between low measures of anthropometrical status and chronic illness at the time of measurement and that a wash-out period, ignoring the first few years of mortality, should be employed during analysis to allow for this.

However, the empirical evidence for the necessity of a wash-out period is varied and some recent studies suggest that excluding the first 2, 5 or 6 years of follow-up of mortality may make no difference to the resulting relationship between BMI or body weight and height (Tuomilehato et al, 1987; Lee et al, 1993; Folsom et al, 1993; Gronback et al, 1994; Waaler, 1984).

Ideally, information on morbidity in 1990 would be used to exclude any subject who was chronically ill at the time of measurement from the analysis. Unfortunately, the morbidity data from 1990 was too limited to be used for such a purpose. Data on the type and length of chronic diseases was acquired from those interviewed in 1996. However no information of this nature was asked about the deceased as it was considered that it would be difficult for the respondents to remember it accurately. Hence no data on the chronic illness status of the whole 1990 population is available. This study will treat the issue of a wash out period empirically and examines the effects of allowing for early mortality in analysis by excluding the first two years' of mortality data.

Only deaths after May 1st 1992 are assessed. This should at least exclude the subjects who were seriously (chronically) ill in 1990 as they would probably have died within the next twenty-four months. Using a wash out period of two years decreases the number of deaths to 48. If more than two years of wash out were employed the number of deaths would be too small for analysis. Tables 5.9 and 5.10 show the results of stepwise Cox regressions run between the anthropometric variables and mortality between 1990-1996, controlling for all the confounders described above.

Table 5.9 *HRs of mortality pre-May 1st 1992 for the anthropometric measurements (entered as continuous variables) and amongst all subjects who were not pregnant or overweight and for whom SES data were available, controlling for age, sex and SES factors (n=796)*

	H R	95% C I	p-value
Height	0.977	0.939-1.018	0.267
Weight	0.960	0.923-0.999	0.045
BMI	0.873	0.772-0.986	0.029
MUAC	0.878	0.786-0.982	0.022
Fat Mass	0.971	0.893-1.055	0.483
Fat-free Mass	0.957	0.915-1.000	0.051

p-value for the maximum likelihood ratio test

Table 5.10: RRs of mortality pre-May 1st 1992 for the section of the population with the lowest quintile of anthropometric measurements compared to the rest of the population amongst all subjects who were not pregnant or overweight and for whom SES data were available, controlling for age, sex and SES factors (n=796)

	R R	95% C.I.	p-value
Height	1.823	0.987-3.367	0.055
Weight	2.024	1.108-3.698	0.022
BMI	1.945	1.072-3.528	0.029
MUAC	2.101	1.155-3.824	0.015
Fat mass	1.037	0.528-2.036	0.916
Fat-free mass	1.952	1.072-3.555	0.029

p-value for the maximum likelihood ratio test

Table 5.9 shows that, at the population level, a single unit increase in weight, BMI or MUAC is associated with a decreased risk in mortality even when the first two years of mortality are excluded. Similarly, table 5.10 shows that those groups of subjects who had values of weight, BMI, MUAC or FFM in the lowest quintile in 1990 had an increased risk of death compared to the rest of the population. The HRs and RRs seen are roughly similar to those reported when the two earliest years of mortality are included.

Age is a significant confounder in all the analyses in tables 5.9 and 5.10. Alcohol is also a confounder for all the analyses except for those involving the BMI. No other significant confounders are seen in these analyses.

5.6 Analyses with CED

The analyses in this section will be similar to those described in section 5.5 as these are probably the most rigorous way of looking at the associations between the anthropometric variables and mortality. Table 5.11 shows the RRs of mortality between 1990-1996 for those individuals who were diagnosed as CED in 1990 compared to those who were not. CED is diagnosed according to the three definitions described in the introduction.

Table 5.11: RRs of mortality pre-May 1st 1992 for CED compared to "normal" and non-pregnant subjects for whom SES data were available, controlling for age, sex and SES factors (n=796)

	No CED	R R	95% C I	p-value	Alcohol	Age
BMI<18.5kg/m^2	165	1.869	1.031-3.386	0.039		+
Low MUAC	90	1.516	0.805-2.854	0.197	+	+
BMI<18.5kg/m^2 & low MUAC	94	1.534	0.824-2.855	0.177	+	+

p-value for the maximum likelihood ratio test

It can be seen that in this population only the group of individuals diagnosed as CED by the use of the BMI alone have a significantly increased risk of mortality compared to the rest of the population. The group defined as CED diagnosed using the MUAC or the MUAC in conjunction with the BMI did not show a significant increase in the risk of mortality compared to the "normal" population.

This result may seem surprising given that the MUAC has been more strongly associated with mortality than the BMI in the other analyses in this chapter. It may be that, because a much smaller number of subjects were actually diagnosed as CED by the MUAC in 1990 compared to the number diagnosed by the BMI, that this result is due to a small numerator and is not in fact evidence for the null hypothesis that mortality and CED are not associated. If an analysis similar to that reported in table 5.8 is employed (i.e. including deaths in the first two years) then the sample size is increased and all three definitions of CED are significantly associated with increased risk of mortality (see table 5.12). However, it may simply be that the CED diagnosed by the BMI is more strongly associated with mortality than the CED defined by the MUAC.

Table 5.12: Risk ratios of mortality for CED compared to "normal" and non-pregnant subjects for whom SES data were available, controlling forage, sex and SES factors (n=822)

	No. CED	R.R.	95% C.I.	p-value	CONFOUNDERS				
					Distance to closest town	Piped Water	Smoking	Alcohol	Age
BMI<18.5kg/m^2	178	1.618	1.000-2.626	0.050	+	-	+		+
Low MUAC	104	2.136	1.292-3.530	0.003		-		+	+
BMI<18.5kg/m^2 & low MUAC	105	1.961	1.173-3.276	0.010		-		+	+

p-value for the maximum likelihood ratio test

5.7 Analyses within different age bands

Age is the grand confounder in all the mortality analyses discussed above. This implies that the BMI or MUAC may be associated with mortality differently in distinct age bands. Ideally, the mortality data could be analysed separately according to age band. However, given the small number of deaths - especially if the first two years of mortality data is excluded - this could be problematic. Table 5.13 below shows the number of deaths recorded within certain age bands if the first two years of mortality are included.

Table 5.13: Number of deaths according to age band (n=882)

	Deaths	Survivors	Total
18.0-39.9	1	330	331
40.0-59.9	20	332	352
60.0+	51	159	159

It is clear that no mortality analyses can be conducted on the youngest age group. Table 5.14 shows the results of the analyses conducted on the middle-aged group and table 5.15 the results of the analyses conducted on the oldest group. Note that these analyses are similar to those discussed in tables 5.7 and 5.8 (i.e. with the first two years of mortality included). Table 5.14 shows that, at the population level, a unit increase in the BMI reduces the HR of mortality in the middle-age group. Those defined as CED by the BMI were also found to have an increased RR of mortality in this age group. No significant associations were seen between the MUAC and mortality risk in this group.

Conversely, table 5.15 shows that, at the population level, a unit increase in MUAC in older adults results in a decreased HR of mortality and that the group defined as CED by using the MUAC alone have significantly higher RRs of mortality than the non-CED group. No associations are seen between the BMI and mortality risk in the oldest group.

Table 5.14: HRs and RRs of mortality for the anthropometric measurements amongst all subjects aged 40-59.9 who were not pregnant or overweight in 1990 and for whom SES data were available, controlling for sex, age and SES factors. The associations of the significant confounding factors are shown.(N=332)

	No. CED	HR or RR	95% CI	p-value	CONFOUNDERS						
					Distance to closest town	Piped water	Smoking	School	Alcohol	Sex	Age
BMI		0.651	0.525-0.807	0.001						+	
MUAC		0.907	0.767-1.072	0.251			+				+
BMI<18.5kg/m2	89	4.085	1.610-10.363	0.003			+	+			+
Low MUAC	43	1.867	0.682-5.105	0.224			+				+

p-value for the maximum likelihood ratio test

Table 5.15: HRs and RRs of mortality for the anthropometric measurements amongst all subjects aged 60+ who were not pregnant or overweight in 1990 and for whom SES data were available, controlling for sex, age and SES factors. The associations of the significant confounding factors are shown.(N=159)

	No CED	HR or RR	95% CI	p-value	CONFOUNDERS						
					Distance to closest town	Piped water	Smoking	School	Alcohol	Sex	Age
BMI		0.937	0.829-1.058	0.295	+	-			+		+
MUAC		0.865	0.779-0.960	0.007	+	-			+		+
BMI<18.5kg/m2	68	1.180	0.669-2.083	0.568	+	-					+
Low MUAC	53	2.270	1.248-4.128	0.007	+	-			+		+

p-value for the maximum likelihood ratio test

5.8 Discussion

The results obtained above imply that an association between low anthropometrical status and high mortality does exist in this population. In particular, low values of weight, BMI, MUAC and FFM in 1990 all seem to be associated with increased mortality during the follow-up period. Other authors have also reported such an association (Lew et al. 1979; Waaler, 1984; Jarrett et al. 1982; Rissanen et al. 1989 & 1991; Tuomilehto et al. 1987; Wienpahl & Ragland, 1990; Laara & Rantakallio, 1996; Durazo-Arvizu et al. 1997; Chyou et al. 1997).

The authors above, who included obese subjects in their studies, found either a "U-shaped" association (Waaler, 1984; Rissanen et al. 1989 & 1991; Laara & Rantakallio, 1996; Durazo-Arvizu et al. 1997) or a "J-shaped" association (Lew et al. 1979; Jarrett et al. 1982; Tuomilehto et al. 1987; Wienpahl & Ragland, 1990; Chyou et al. 1997). In this study, where obese individuals were excluded from the analyses, a (direct) negative association was seen.

This point is important. If the whole 1990 population were considered, i.e., even those who were obese, no direct association between mortality and anthropometrical status is observed (not shown). The results above only hold true when comparing low and normal BMI or MUAC groups and thus cannot be generalised to the whole population.

It is also important to note that the risks of mortality do change if either (a) the "non-interviewee" group is excluded or (b) deaths in the first two years of the follow-up are disregarded. The anthropometrical differences found between those for whom SES data was available and those for whom it was not, mean that the RRs and HRs reported for height, weight, FM and FFM after section 5.3 should not be over-interpreted and should be regarded with a certain amount of scepticism. It is difficult to assess whether or not these anthropometric variables are actually associated with mortality risks in this population if SES and "other" lifestyle factors are controlled. Theoretically, an ideal study of the mortality risks of anthropometrical status would be conducted on a group of subjects who have identical SES and other "lifestyle" conditions.

Similarly, the RRs and HRs of mortality for low BMIs and MUACs are slightly stronger when the first two years of mortality are excluded. The Framingham heart study compared mortality risks in adults over 65 years in relation to their BMI. They found that the mortality risks associated with low BMI were higher in the first four years than in later years. The authors interpreted this to indicate a greater effect of baseline morbidity, but felt that to exclude this four year period would eliminate a period of substantial risk for other subjects and therefore no

"wash-out" period was used in later analysis (Harris et al. 1988). This author would probably take a different view and would prefer to quote risks which have excluded the first two years of mortality. In the morbidity data presented in chapter six, the cross-sectional associations between low BMI or MUAC and chronic illness are relatively strong; it therefore seems safer to exclude the first two years of mortality.

At the population level, a unit increase in both the BMI or MUAC is associated with decreased HRs of mortality in the analyses which exclude all "non-interviewees" and the first two years of mortality. These results imply that both of the variables may be useful as predictors of mortality. FFM but not FM is also significantly associated with mortality at the population level, but given the bias discussed above the importance of this result should not be overstated. The group diagnosed as CED by the BMI in 1990 have a RR of 1.87 for mortality compared to the non-CED group. However, the group diagnosed as CED by the MUAC do not have a significantly increased RR of mortality compared to the non-CED group. Thus if the *a priori* expectation of a diagnosis of CED is that it is associated with mortality risk then the CED diagnosed by the BMI is a more meaningful measure of nutritional status in this analysis of the whole population.

Age is an important confounding factor in all the mortality analyses except for that between the RRs of CED (defined by the BMI) and non-CED in the middle-aged group. The implication of this is that the BMI or MUAC may be associated with mortality differently in distinct age groups. The problem with the data set in this study is that there have not yet been enough deaths to analyse the relationships separately in each age band; and thus the first two years of mortality have had to be included in these analyses.

The limited analyses in section 5.7 suggest that the BMI may be a sensitive predictor of mortality in the middle-aged group. In this age group a unit increase in the BMI results in a decreased risk of mortality at the population level and the group who were diagnosed as CED by the BMI alone have an RR of 4.08 for mortality compared to the rest of the population. However, no significant associations between the MUAC and mortality are found at the population level in this age group. Nor do the group which are diagnosed as CED by the BMI alone have a significantly higher RR of mortality than the non-CED group.

Conversely, the MUAC may be a better predictor of mortality in the elderly than the BMI. In the oldest group a unit increase in MUAC is associated with a decreased risk of mortality at the population level, and the group who were diagnosed as CED by the MUAC have a RR of 2.27 for mortality compared to the non-CED group. However, no significant associations between the

BMI (entered as a continuous or a categorical variable) and mortality are found at the population level in this age group.

These result are contrary to the author's expectations. As described in the introduction, the bulk of the available evidence suggests that moderate degrees of fatness are perhaps of no medical or physical disadvantage to the elderly of either sex and may constitute a benefit. Thus the expectation is that a low BMI is particularly harmful for the elderly and thus that those who were diagnosed as CED by the BMI would have an increased risk of mortality compared to the non-CED group.

It is probable that the BMI derived from half-span measurements is more sensitive to mortality than the BMI derived from the stature in the elderly. This seems to be the case for morbidity. Unfortunately, no half-span measurements were made in 1990 and hence no analyses looking at the relationship between the BMI derived from half-span and mortality have been undertaken.

It is probable that, although in some analyses a cut-off point of BMI<18.5kg/m^2 does seem to incur an increased risk of mortality, the relationships between either the BMI or MUAC and mortality are continuous. This finding is similar to that described by Pelletier (1991) in his review of childhood anthropometry and mortality; he described the relationship as a continuous exponential function. Conversely, Chen et al's (1980) study found a distinct "threshold" or breakpoint exists in the relationship between growth and mortality, with lower levels of growth deficit showing little if any association with between size and the risk of death but higher deficits showing a strong positive correlation.

The major weakness of this part of the study is that no allowance has been made for cause of death. At the moment it would be not possible to analyse the data in terms of cause of death as only a small number of deaths have occurred. I suggest that this part of the study should be followed up by returning to Sarawak after a few more years to record any further deaths. This would also allow a longer period for "wash-out" techniques to be employed.

5.9 Conclusions

- Low values of the BMI and MUAC are associated with increased risks of mortality.
- The BMI may be more strongly associated with mortality than the MUAC in the middle-aged group, however the converse may be true for the older group.
- In the analysis excluding early mortality, those with CED (BMI<18.5kg/m^2) had an RR of 1.87 of mortality compared to the non-CED group.

CHAPTER SIX

MORBIDITY

The purpose of this chapter is to examine the relationship between anthropometry and self-reported morbidity. Most of the analyses and discussion will focus on the associations seen between the BMI or MUAC and morbidity as these are the two anthropometric variables that are of most interest to this project.

Initial analyses examine the 709 non-pregnant and non-obese individuals for whom data on the BMI, MUAC, morbidity, SES and other "lifestyle" factors are available. Obese individuals have been excluded from all the analyses in this chapter because obesity is known to be associated with ill-health (see chapter one) and this project is concerned with the relationship between low values of anthropometry and morbidity. Pregnant women are also excluded from the analyses.

6.1 Rates of reported illness

Table 6.1 shows the point prevalence of the different morbidity categories at visit A according to sex. A decrease in prevalence is seen as the stringency of the definition of illness increases, i.e.: more subjects report morbidity category (i) - illness in the past two weeks - than category (iii) - loss of activity due to illness. This is probably a reflection of the smaller number of disabling illnesses as opposed to relatively less serious illness. Women report significantly more illness than men in all categories except "loss of activity". Figure 6.1 shows the amount of illness reported by age group. As expected, more illness is reported by the older age groups.

Figure 6.1 The cross-sectional association between age and illness at visit A (sexes combined)

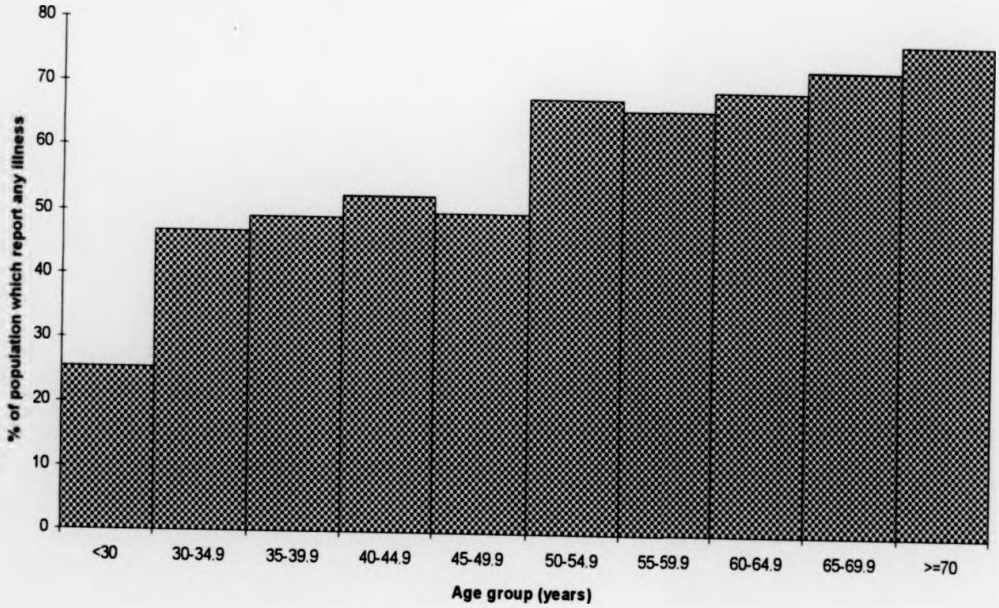


Table 6.1 The point prevalence of the different morbidity categories at visit A

	Men (n=319)	Women (n=390)	Total (n=709)	p-value
Any illness in previous two weeks	47.6	64.1	56.7	0.001
Any illness today	38.9	53.3	46.8	0.001
Loss of activity due to illness in previous two weeks	19.7	23.1	21.6	0.313
Any chronic illness	38.6	49.7	44.7	0.003

p-value for Fisher's exact test

Table 6.2 shows the point prevalence of the different morbidity categories at visit B according to sex. Less illness is reported at visit B than at visit A. If the proportion of illness reported is compared between the individuals who were interviewed about their illness at visits A and B (using a two-sample test of proportion with the null hypothesis that the difference between the proportions is zero), then significantly less illness is reported at visit B ($p<0.001$). There are no significant differences in the amount of illness reported between the sexes at visit B.

Table 6.2 The point prevalence of the different morbidity categories at visit B

	Men (n=249)	Women (n=350)	Total (n=599)	p-value
Any illness in previous two weeks	44.2	48.7	46.8	0.281
Any illness today	40.2	45.0	43.0	0.242
Loss of activity due to illness in previous two weeks	18.1	15.5	16.6	0.435

p-value for Fisher's exact test

Table 6.3 shows the types of symptom reported amongst those who report illness in the two weeks prior to visit A. Headache, muscle ache and gastric pain are the most frequently reported symptoms, cough and chest pain are also highly reported. Some sex differences exist in the type of symptom reported - women report significantly more headaches and men report significantly more coughing, chest pains and other respiratory problems.

Table 6.3: The % frequency of reported symptoms amongst those who reported illness in the two weeks prior to visit A

	Men (n=152)	Women (n=250)	Total (n=402)	p-value
Headache	27.0	45.2	38.3	0.001
Fever	9.2	7.6	8.2	0.578
Diarrhoea	5.3	6.4	6.0	0.829
Gastric	18.4	26.0	23.1	0.088
Cough	27.6	15.2	19.9	0.003
Chest	21.0	21.4	15.7	0.024
Muscle	30.9	37.6	35.1	0.196
Injury	3.9	2.8	3.2	0.568
Respiration	11.2	4.0	6.7	0.007
HT	11.8	14.8	13.7	0.456
Other	9.9	8.0	8.7	0.585

p-value for Fisher's exact test

Table 6.4 shows the number of symptoms reported by individuals who complained of any illness. It can be seen that 46.3% of those reporting any illness reported more than one symptom. Many of the symptoms on the list may commonly be found in conjunction with each other e.g. cough and chest pain. No significant difference exists between the number of symptoms reported by each sex.

Table 6.4 The % frequency of the number of symptoms reported amongst those who reported illness at visit A

No. of Symptoms	Men (n=152)	Women (n=250)	Total (n=402)
One	56.6	52.0	53.7
Two	25.0	34.8	31.1
Three	15.8	9.6	11.9
Four	2.6	3.2	3.0
Five	0.0	0.4	0.3

Amongst those missing any activity in the two weeks prior to visit A, table 6.5 shows the type of activity missed. Some individuals may have missed more than one type of work. When they were ill, men were more likely to miss farm work than house work. Women, however, were almost equally likely to miss either household work or farm work. These differences may be explained by the division of labour between the sexes in the Iban - although women do work in the farm they are almost entirely responsible for the housework. The two men who missed "other

work" were labourers who had developed a fever.

Table 6.5. The % frequency of each activity the respondents had to miss because of illness, amongst those who missed any activity at all at visit A

	Men (n=63)	Women (n=90)	Total (n=153)	p-value
Farm work	73.0	61.1	66.0	0.165
House work	28.6	50.0	41.2	0.012
Other work	3.2	0.0	1.3	0.281

p-value for Fisher's exact test

6.2 Cross-sectional associations between illness and anthropometry

In order to assess the relationship between anthropometry and illness, stepwise logistic regression was used to obtain ORs and confidence intervals for the relationships between the BMI or MUAC and the various symptom types described in the methods chapter (section 2.6.4). Age, sex and the SES and other lifestyle factors discussed in the methods section were entered into the regression models as confounding factors. Note that an individual may report suffering from more than one symptom group. Table 6.6 shows, for each symptom group reported for the two weeks prior to interview, the number of individuals reporting the symptom group and the ORs and confidence intervals for the relationships between the BMI or MUAC and the symptom group.

Table 6.6: The associations between the various symptom groups and the BMI or MUAC at visit A (n=709)

Symptom type	No. ill at visit A	Odds Ratio	95% Confidence Intervals	p-value
Incidental				
BMI	218	1.027	0.969-1.088	0.366
MUAC	218	1.008	0.950-1.070	0.786
Hypertension				
BMI	55	1.228	1.115-1.353	0.001
MUAC	55	1.266	1.138-1.409	0.001
Fever, epigastric or respiratory illness no HT				
BMI	208	0.869	0.820-0.922	0.001
MUAC	208	0.860	0.811-0.911	0.001

p-value for the log-likelihood ratio test

The incidental symptom group show no significant directional association with the BMI or MUAC. HT is significantly positively associated with BMI and MUAC: at the population level, a unit increase in the BMI or MUAC results in an increased odds of reporting HT. Complaints of fever or epigastric illness or respiratory problems not found in conjunction with HT show a significant negative association with the BMI and MUAC. A single unit increase in the BMI or MUAC results in decreased odds of reporting these symptom types at the population level.

No single SES or "lifestyle" variable or other confounding factor (e.g. age or sex) is found to be significantly associated with all the regressions in table 6.6. This is true for all the analyses run in this chapter i.e. similar confounding factors are not significant in all the regressions. The importance of confounding factors will be discussed section 6.8, but note that the results of all the regressions shown below have been adjusted for confounding factors. (Tables 6.20 and 6.21 show which confounding factors are significant in which regression and can be referred to if necessary).

Table 6.6 shows that there are several types of associations between the symptom groups and the BMI or MUAC. The group of symptoms which associate negatively with the BMI and MUAC i.e. fever, epigastric and respiratory problems not in conjunction with HT are the most interesting for this project as it is a study of the consequences of low anthropometrical status. Therefore when assessing the relationship between anthropometry and self-reported morbidity it is these symptoms or sicknesses which are being discussed for the remainder of the chapter. Table 6.7

shows the association between the various morbidity categories shown in table 6.1 and the BMI or MUAC with this new definition of "illness".

Table 6.7: The associations between the BMI or MUAC and illness at visit A (n=709)

	No. ill at visit A	Odds Ratio	95% Confidence Intervals	p-value
Illness in previous two weeks				
BMI	208	0.869	0.820-0.922	0.001
MUAC	208	0.860	0.811-0.911	0.001
Illness today				
BMI	147	0.872	0.816-0.931	0.001
MUAC	147	0.842	0.789-0.898	0.001
Loss of activity due to illness in previous two weeks				
BMI	74	0.881	0.812-0.956	0.002
MUAC	74	0.898	0.829-0.971	0.007
Chronic illness				
BMI	52	0.848	0.750-0.958	0.008
MUAC	52	0.794	0.701-0.899	0.001

p-value for the log-likelihood ratio test

Table 6.7 shows that, at the population level, a unit increase in the BMI or MUAC is associated with decreased odds of reporting illness in this cross-sectional data. The ORs of the BMI and MUAC are similar. The associations between the BMI or MUAC and illness at visit B are shown below in table 6.8. It should be noted that the total number of non-pregnant and non-obese individuals who were eligible for this analysis at visit B was 599.

Table 6.8: The associations between the BMI or MUAC and illness at visit B (n=599)

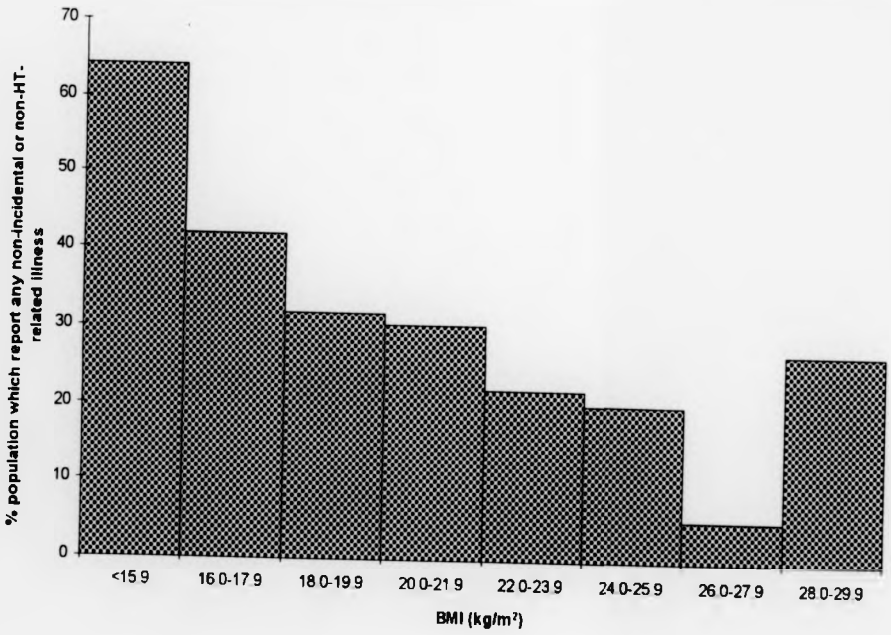
	No. ill at visit B	Odds Ratio	95% Confidence Intervals	p-value
Illness in previous two weeks				
BMI	124	0.891	0.825-0.961	0.003
MUAC	124	0.862	0.804-0.925	0.001
Illness today				
BMI	102	0.905	0.835-0.981	0.016
MUAC	102	0.887	0.818-0.961	0.004
Loss of activity due to illness in previous two weeks				
BMI	49	0.888	0.802-0.982	0.021
MUAC	49	0.866	0.788-0.952	0.003

p-value for the log-likelihood ratio test

It can be seen that the ORs reported above are similar to those reported at visit A. Negative associations are seen between the BMI or MUAC and all the illness categories at visit B in these cross-sectional analyses. The implication of these results is that, at the population level, as the BMI or MUAC decreases the risk of reporting illness increases.

Figure 6.2 shows the cross-sectional relationship between the BMI and illness in the two weeks prior to interview at visit A. It is interesting to note that a BMI of 26.0-27.99 kg/m^2 is associated with the lowest amount of morbidity in this population.

Figure 6.2: The cross-sectional association between the BMI and illness reported in the two weeks prior to interview A (sexes combined)



6.3 Analysis excluding individuals who were ill at the baseline

The analyses above show that associations between the BMI or MUAC and self-reported illness exist in this population. These associations may exist because low BMI or MUAC lead to increased illness, or because illness leads to a decrease in BMI or MUAC. If a causal relationship between the BMI or MUAC and morbidity experience is to be established the element of circularity in the relationship between thinness and illness must be eliminated. Thus the relationship between the BMI and morbidity experience in the subjects who reported being healthy i.e. reported no illness at the baseline (no chronic illness) was examined. Table 6.9 below examines the association between the BMI or MUAC and self-reported illness amongst individuals who did *not* report any non-incidental or non-HT related chronic illness. The visit A data was used as this provides a larger sample size for analysis, a total of 657 individuals were eligible for the calculations.

Table 6.9 Associations between the BMI or MUAC and illness amongst those who did not report any chronic disease at visit A (n=657).

	No ill at visit A	Odds Ratio	95% Confidence Intervals	p-value
Illness in previous two weeks				
BMI	156	0.885	0.830-0.944	0.001
MUAC	156	0.891	0.836-0.949	0.001
Illness today				
BMI	99	0.875	0.809-0.946	0.001
MUAC	99	0.868	0.804-0.937	0.001
Loss of activity due to illness in previous two weeks				
BMI	51	0.927	0.840-1.022	0.129
MUAC	51	0.971	0.882-1.069	0.544

p-value for the log-likelihood ratio test

Amongst those who reported no chronic disease at visit A, a unit increase in the BMI or MUAC is significantly associated with lower odds of reporting illness in the previous two weeks or on the day of interview at the population level. A significant OR is not found when the relationship between loss of activity due to illness and the BMI or MUAC is examined. Note that the ORs for these analyses are weaker than those in table 6.7. This is because there is a significant cross-sectional association between the BMI or MUAC and chronic illness and removing the

chronically ill group therefore reduces the strength of the association.

Table 6.10 is similar to table 6.9 in that it also examines the associations between the BMI or MUAC and reported illness amongst those who reported being healthy at the baseline. In this analysis the baseline is visit A, so all those individuals who reported illness at visit A were excluded from the analysis. The total number eligible individuals was 410. It can be seen that the number of subjects reporting illness at visit B but not at visit A was low.

Table 6.10 The associations between illness and the BMI or MUAC at visit B amongst those who did not report illness at visit A (n=410).

	No. ill at visit B	Odds Ratio	95% Confidence Intervals	p-value
Illness in previous two weeks				
BMI	39	0.858	0.760-0.970	0.014
MUAC	39	0.870	0.777-0.974	0.016
Illness today				
BMI	29	0.928	0.817-1.054	0.250
MUAC	29	0.918	0.811-1.041	0.183
Loss of activity due to illness in previous two weeks				
BMI	17	0.840	0.696-1.013	0.068
MUAC	17	0.804	0.675-0.959	0.015

p-value for the log-likelihood ratio test

Amongst those who reported no illness at visit A, a unit increase in the BMI or MUAC is significantly associated with a decreased odds of reporting illness in the two weeks prior to interview B at the population level. No significant associations were found between the BMI or MUAC and illness reported on the day of interview. A significant association between the MUAC and loss of activity due to illness was found, but this association was not significant for the BMI.

6.4 Longitudinal analyses

When epidemiologists attempt to separate causal from non-causal explanations they usually assess a relationship in terms of at least three criteria - (i) biological plausibility, (ii) consistency of the findings, and (iii) lack of temporal ambiguity (Briend, 1990; Susser, 1991; Rothman & Greenland, 1998). None of these criteria are in themselves sufficient to prove causality and only (iii) is necessary - in that a putative cause C must precede a putative effect D (Rothman &

Greenland, 1998). However, all the criteria do lend support to the hypothesis that a causal relationship exists. The results described above meet criteria (i) and (ii), but they do not meet criterion (iii) as the analyses have all been cross-sectional.

Two methods may be used to show that a lack of temporal ambiguity exists between the BMI or MUAC and illness experience in this type of study. The first of these methods would be to look at the relationship between BMI or MUAC at visit A and subsequent illness experience at visit B amongst those individuals who reported being healthy at the baseline. A comparison could then be made of the illness experience at visit B between those with low and those with high BMIs or MUACs at visit A. The second method, similar to that described by Sommer (1996) in his Vitamin A studies, would be to assess the relationship between CED and illness in four different groups of individuals. These groups would be defined by the absence and presence of CED at visits A and B as seen below in Table 6.11. The expectation would be that the rates of illness in group N-N would be less than in the other groups and that they would be highest in group C-C. Also, that group C-N would behave more like group N-N at visit B and more like group C-C at visit A.

Table 6.11 The groups of different individuals found using a method similar to Sommer

CED status at Visit A	CED status at Visit B	Group Name
Normal	Normal	N-N
CED	CED	C-C
CED	Normal	C-N
Normal	CED	N-C

Using the first method, table 6.12 examines the relationships between the MUAC or BMI at visit A and illness at visit B, excluding the individuals who reported any chronic illness at visit A. Table 6.13 also examines this relationship but excludes the individuals who reported any illness (chronic or otherwise) at visit A. Only the category "illness reported in the previous two weeks" is shown because such small numbers are found in the other categories.

Table 6.12: The associations between the BMI or MUAC at visit A and illness in the two weeks prior to interview B for individuals who reported no chronic illness at visit A (n=551)

	No. ill at visit B	Odds Ratio	95% Confidence Intervals	p-value
BMI	156	0.914	0.842-0.992	0.031
MUAC	156	0.906	0.834-0.983	0.018

p-value for the log-likelihood ratio test

Table 6.13: The associations between the BMI or MUAC at visit A and illness in the two weeks prior to interview B amongst those who reported no illness at visit A (n=410)

	No. ill at visit B	Odds Ratio	95% Confidence Intervals	p-value
BMI	39	0.907	0.807-1.021	0.107
MUAC	39	0.873	0.777-0.981	0.023

p-value for the log-likelihood ratio test

Amongst the population who reported no illness (chronic or otherwise) a unit increase in the MUAC at visit A is associated with significantly decreased odds of reporting illness at visit B. A similar result is seen for the BMI when those with chronic illnesses are excluded. However when those with any illness at visit A are excluded, the BMI at visit A is not significantly associated with the odds of reporting illness at visit B, although the association is in the direction expected. Note that the ORs recorded in table 6.10 are stronger than those found in table 6.12, thus it seems that associations between the BMI and illness are stronger cross-sectionally than prospectively.

6.5 Analyses using CED

This section of analysis aims to assess the powers of the various definitions of CED in predicting morbidity experience. Do CED groups have a higher risk of morbidity than non-CED groups? The analyses in this section will be similar to those described in table 6.13 as these are probably the "strictest" way of looking at the associations between the anthropometric variables and morbidity. Table 6.14 shows the RRs of reporting illness in the two weeks prior to visit B among individuals who reported no illness at visit A comparing those individuals who were

diagnosed as CED at visit A to those who were not. CED is diagnosed according to the three definitions described in the introduction.

Table 6.14: RRs of illness in two weeks prior to visit B for CED compared to "normal" subjects amongst those who reported no illness at visit A (n=410)

	No. CED at visit A	R.R.	95% Confidence Intervals	p-value
BMI<18.5kg/m^2	66	3.002	1.345-6.700	0.007
Low MUAC	28	2.462	0.886-6.842	0.084
BMI<18.5kg/m^2 & MUAC	30	2.335	0.844-6.433	0.103

p-value for the log-likelihood ratio test

In the study population only the group of individuals diagnosed as CED by the use of the BMI alone have a significantly increased risk of morbidity compared to the rest of the population. This result may seem surprising given that the MUAC has been more strongly associated with morbidity than the BMI in the most of the other analyses in this chapter. Only a small number of subjects were diagnosed as CED by the MUAC. Thus, this result may be due to a small numerator yielding wide confidence intervals, and may be an artefact rather than evidence for the null hypothesis. In the cross-sectional analyses such as those seen in table 6.10, the CED groups defined by the MUAC or the MUAC in conjunction with the BMI have significantly increased risks of self-reported illness compared to the rest of the population (not shown).

Table 6.15 shows the results of the analysis similar to that described by Sommer where the illness category is any symptom reported during the two weeks prior to visit B amongst those who reported no illness at visit A. CED is diagnosed by the BMI alone and the N-N group is taken as the baseline. The group which have CED at both visits A and B report a significantly higher amount of illness at visit B than the group which is normal at both visits. The C-N and N-C groups have small numbers and thus the confidence intervals are large.

Table 6.15: The associations between the Sommer-type groups and reported illness at Visit B (n=410)

Group type	No. in each group	Odds Ratio	95% Confidence Intervals	p-value
N-N	332	1.000		
C-C	58	3.245	1.401-7.514	0.006
C-N	12	1.749	0.204-14.967	0.610
N-C	8	1.956	0.211-18.086	0.554

p-value for the log-likelihood ratio test

6.6 Analyses using the BMI derived from half-span measurements

In chapter 3 it was shown that if the BMI is derived using half-span measurements the values of the BMI are lower in the older age groups compared to the values of the BMI derived from stature. The analysis below examines the relationship between the BMI derived from half-span at visit A and illness at visit B, excluding those individuals who reported any illness at visit A.

Table 6.16 The associations between the BMI derived from half-span at visit A and illness in the two weeks prior to interview B amongst those who reported no illness at visit A (n=410)

	No. ill at visit B	Odds Ratio	95% Confidence Intervals	p-value
BMI (half-span)	39	0.912	0.815-1.021	0.110

p-value for the log-likelihood ratio test

Table 6.16 shows that when the BMI is derived from half-span for the whole population (i.e. all ages) no significant association is seen between the BMI and morbidity at the population level. In table 6.17 the BMIs for the younger (<50y) section of the population are derived from height and those for the older groups (>50y) are derived from half-span. When this "mixed" BMI is entered into the analysis it can be seen that, amongst the population who reported no illness (chronic or otherwise), a unit increase in the BMI at visit A is associated with a significant decrease in the odds of illness at visit B.

Table 6.17: The associations between the BMI derived from half-span or stature at visit A and illness in the two weeks prior to interview B amongst those who reported no illness at visit A (n=410)

	No. ill at visit B	Odds Ratio	95% Confidence Intervals	p-value
BMI (mixed)	39	0.887	0.792-0.994	0.039

p-value for the log-likelihood ratio test

The results in tables 6.16 and 6.17 should be compared to that in table 6.13 where an identical analysis was run using the BMI derived from stature for all ages. At the population level, a single unit increase in the BMI derived from stature is not significantly associated with illness in this longitudinal analysis.

6.7 Haemoglobin levels

Haemoglobin values were recorded for 486 non-pregnant individuals at visit B. Mean values of haemoglobin were lower for women ($p<0.001$) and decreased with increasing age. Using linear regression techniques similar to those described in section 3.2 it was found that age and sex accounted for 16.9% of the variance in the distribution of haemoglobin values in this population.

Table 6.18 shows the distribution of anaemia by age group for each sex. A relatively high proportion of the population are classified as anaemic, especially amongst those aged 50 years or more. No significant differences in the frequency of anaemia is found between the sexes.

Table 6.18: The % frequency of anaemia by sex and age group at visit B

Age group	Men (n=269)	Women (n=217)	Total (n=486)
<30	28.6	25.0	26.2
30.0-39.9	18.5	41.2	33.3
40.0-49.9	42.1	29.2	33.3
50.0-59.9	50.0	50.7	50.4
60.0-69.9	67.7	51.9	57.8
70.0+	76.0	70.0	73.3
All	48.4	42.3	44.6

The BMI and MUAC are associated with haemoglobin values or risk of anaemia in this

population. Table 6.19 below shows the cross-sectional associations seen between the BMI or MUAC and anaemia at visit B among non-pregnant individuals. The SES and other "lifestyle" factors described above have been entered into these analyses. At the population level, a single unit increase in either the BMI or MUAC is associated with a decreased OR of anaemia.

Table 6.19: The associations between the BMI or MUAC and anaemia at visit B (n=471)

	No. anaemic	Odds Ratio	95% Confidence Intervals	p-value
BMI	203	0.890	0.837-0.945	0.001
MUAC	203	0.883	0.827-0.944	0.001

p-value for the log-likelihood ratio test

Haemoglobin value is not significantly associated with reported illness at visit B in this population. In order to assess if haemoglobin value is a confounding factor in the relationship between the BMI or MUAC and morbidity, regressions similar to those in table 6.8 were run and haemoglobin value was entered as an extra confounding factor. Haemoglobin value was not a significant confounder in the relationship between BMI or MUAC and morbidity.

6.8 Confounding factors

As stated above, confounding factors differ for many of the analyses in this chapter. There may be at least three explanations for this - (i) the sample sizes are small, or (ii) the confounding factors are crudely measured, or (iii) no particular confounder measured by this study is important in the relationships between the BMI or MUAC and self-reported morbidity in this population. Tables 6.20a, 6.20b and 6.21 show which confounding factors are significantly associated with which analyses and the direction of these associations.

Table 6.20a: Confounding factors significantly associated with the BMI regressions in tables 6.6-6.10

	Age	Sex	Smoke	Alcohol	Active	School	Mat. Pos.	Savings	Sold	Pd. wk	Remit.	Electric	Water
X-sectional at A (table 6.6)													
Incidental	+	+		-			-						
HT	+		-										
X-sectional at A (table 6.7)													
Two weeks			+	-		-							
Today					-	-							
Activity				-									
Chronic	+	-			-				-				
X-sectional at B (table 6.8)													
Two weeks	+											-	-
Today	+												-
Activity									-				
X-sectional at A, no chronic (table 6.9)													
Two weeks		+	+	-							-		+
Today						-							
Activity				-									+
X-sectional at B, no ill at A (table 6.10)													
Two weeks							+			+		-	-
Today						-							-
Activity													

Table 6.20b: Confounding factors significantly associated with the MUAC regressions in tables 6.6-6.10

	Age	Sex	Smoke	Alcohol	Active	School	Mat. Pos	Saving	Sold	Pd. Wk.	Remit	Electric	Water
X-sectional at A (table 6.6)													
Incidental	+	+		-			-						
HT	+		-										
X-sectional at A (table 6.7)													
Two weeks			+	-		-							
Today						-							
Activity		-		-									
Chronic	+	-			-				-				
X-sectional at B (table 6.8)													
Two weeks					-	-							-
Today	+												-
Activity									-				
X-sectional at A, no chronic (table 6.9)													
Two weeks		+	+	-							-		+
Today						-							
Activity				-									+
X-sectional at B, no ill at A (table 6.10)													
Two weeks							+			+		-	-
Today						-							-
Activity									-				

Table 6.21: Confounding factors significantly associated with the regressions for the BMI and MUAC in tables 6.12-6.19

	Age	Sex	Smoke	Alcohol	Active	School	Mat. Pos.	Saving	Sold	Pd. Wk.	Remit	Electric	Water
Longitudinal, not chronically ill at A (table 6.12)													
BMI						-						-	
MUAC						-						-	
Longitudinal, no ill at A (table 6.13)													
BMI							+			+		-	-
MUAC							+			+		-	-
CED longitudinal, no ill at A (table 6.14)													
BMI<18.5							+			+		-	-
Low MUAC							+			+		-	-
BMI & MUAC							+			+		-	-
Sommer analysis (table 6.15)							+			+		-	-
Longitudinal, not chronically ill at A (table 6.16)													
Half-span BMI							+			+		-	-
Anaemia (table 6.19)													
BMI	+	-											
MUAC	+	-											

Tables 6.20a and 6.20b examine the confounding factors in the cross-sectional analyses between the BMI or MUAC and self-reported illness at visits A and B. It can be seen that some of the confounding factors are generally negatively associated with the risk of morbidity. These include the presence of electricity in the *bilek*, some schooling, selling crops, being physically active in the farm and the consumption of alcohol at least once a week. Other factors result in an increased risk of morbidity reporting. Surprisingly, these include scoring highly on the material possessions scale and someone in the *bilek* receiving wages. Age is also positively associated with morbidity in some of the cross-sectional analyses. Other factors are significantly associated with morbidity in different directions in distinct analyses. These include the presence of piped water in the *bilek*, sex and smoking. In the regressions assessing the risks of HT, smoking is actually found to be positively associated with reported good health. It is possible that this result is observed because the health workers have encouraged those with HT to give up. In the other analyses smoking is associated with poor health.

Table 6.21 examines the confounding factors in the longitudinal analyses between the BMI or MUAC at visit A and self-reported illness at visit B. The confounding factors are more constant than those found in table 6.20, presumably because most of these regressions involve the same group of subjects. It can be seen that the presence of piped water and electricity in the *bilek* are associated with lower ORs of self-reported morbidity. This result is not surprising, the availability of piped water and electricity both probably help HH members to keep the *bilek* cleaner and hence reduce the incidence of infectious diseases. However a more surprising result was that, at the population level, those HHs which scored more highly on the material possessions scale or had a member of their *bilek* receiving paid employment had higher ORs of morbidity. This finding is contrary to the author's expectation that poorer HH would suffer more ill-health than those which are richer. It is possible that the richer HHs were more at leisure to be ill, i.e. could afford to be ill, but this seems unlikely given that those who reported illness at visit A were excluded from the longitudinal analyses. It may simply be that the material possessions scale was not an accurate reflection of the HH's wealth. It is interesting to note that there are no significant associations seen between age or sex and morbidity in the longitudinal analyses. However, age (and smoking) are both positively associated with the ORs of anaemia as reported elsewhere (Gibson, 1990).

The cormic index was not a significant confounder in the relationship between the BMI or MUAC and morbidity in any of these analyses, i.e. shape does not seem to alter these associations. Distance to the nearest town and migrant status were also non-significant

confounders in these analyses, as were the HH composition variables.

One major failure of this study is that no allowance has been made for the clustering of illness within a longhouse. A higher prevalence of illness may exist in certain regions or longhouses than in others. Due to the 1990 sampling methods, this data set is not composed of a selection of individuals within a population, but is made up of individuals within selected longhouses within a population. Thus the association between poor nutritional status and self-reported morbidity may be biased if clustering of illness does exist. Multi-level modelling techniques are usually employed to remove the effects of clustering in studies similar to this project. Unfortunately this technique could not be used here as the sample sizes within some of the longhouses are too small, hence in this study the data are treated as though they were sampled at a population level.

6.9 Discussion

All the analyses above show a negative association between the BMI or MUAC and self-reported illness in the study population. Subjects with low BMI or MUAC are more likely to report non-incidental or non-HT-related illness than subjects with a high BMI or MUAC. Some of the associations seen are not significant (several are borderline) but a trend exists. The three criteria commonly used to assess causality are all supported, to some extent, by the results.

The main problem with the later analyses in this chapter is that only a small number of subjects reported being ill at visit B and not at visit A. This is because (i) many of those who reported illness at visit B also reported being ill at visit A and (ii) a higher proportion of the population reported being ill at visit A than at visit B. Point (i) is not surprising, many of those individuals who reported illness at visit A were chronically ill and hence were still ill at visit B, however point (ii) is more interesting - why should proportionally more individuals have reported illness at visit A than at visit B? There may be three explanations for this phenomenon - (a) some over-reporting of illness took place at visit A, (b) seasonal differences in the activity patterns of the population resulted in a difference in reporting rates and (c) the true underlying prevalence rates of morbidity differed between the visits. Each of these possibilities will be examined in turn.

It is possible that during the first round of visits some of the subjects may have over-reported illness. Although every effort was made to fully explain to the subjects that no medicines could be given to anyone whether or not they were ill, it is possible that - in the first round of visits - some of the individuals thought that by complaining of illness they would be able to obtain medicine from the author or the accompanying nurse. Thus they might have complained of an illness which did not exist, or had existed in the past, or exaggerated the seriousness of a minor complaint in order to obtain medicine which they could store for future needs. By the second round of visits the subjects may have realised that no medicines were given to anybody and hence that there was no point in reporting illness in order to obtain medicine. It is also possible that a "boredom factor" set in by visit B - the subjects were asked exactly the same morbidity questions at visits A and B and hence may have become bored of them. The quickest reply to the morbidity questionnaire was simply "no, I have not been ill" and thus illness at visit B could have been under-reported.

Seasonal differences in physical activity patterns were found between the two visits: a higher proportion of the population was engaged in hard farm work at visit B as opposed to relatively

easier farm work at visit A. This hard farm work at visit B basically entailed clearing the land in order to plant more hill padi and is essential. Thus it is possible that illness was under-reported at visit B because the subjects had "no time to be ill", or no time to think about being ill.

Finally, it is possible that seasonal differences in morbidity prevalence do exist. Unfortunately no data concerning seasonality of illness in Sarawak is available to the author. Such seasonality has been reported in other tropical climates (Adams, 1995) and may exist in Sarawak. In order to assess whether or not morbidity prevalence fluctuates seasonally a further assessment of the population's health status should have been carried out from Feb-June in 1997.

Given points (a) and (b) above, in the author's view, the analyses in tables 6.12, 6.13, 6.15 and 6.16 are the most rigorous test of causality presented. They exclude the group of subjects who might over-report illness or are chronically ill at the baseline. Furthermore, those who reported illness at visit B did so at the busiest time of the year (in terms of farm work) and hence probably truly were "ill". Thus the discussion below will focus mainly on these analyses.

Unfortunately the results of these analyses are ambiguous and it is difficult to state categorically that they show that anthropometrical status is causally linked to morbidity experience. Although all the associations are in the direction expected, not all are significant. This is especially true for the BMI. However, a single unit increase of MUAC is associated in a decrease in the OR of subsequent illness at the population level. Thus it appears that the MUAC may be a useful predictor of morbidity in this population.

The results of the analyses examining the longitudinal relationship between the BMI and morbidity are more complex. If the BMI derived from stature or half-span are entered as continuous variables, a significant increase in the OR of morbidity is not seen. Conversely, if the younger age group's BMI is derived from stature and the older group's BMI is derived from half-span then a significant negative association between this "mixed" BMI and morbidity is observed at the population level.

The BMI derived from arm length measurements has been suggested as a proxy for the BMI derived from stature (Bassey, 1986). However, the distributions of the two BMIs are distinct because the BMI derived from half-span decreases proportionally more than the BMI derived from stature as age increases above 50y. Thus the relationships of the two BMIs with morbidity are not identical.

This study can not examine associations between anthropometrical status and morbidity in distinct age or sex bands. The sample size is too small to undertake this type of analysis. It is possible that no single BMI measure is suitable as a predictor of morbidity in this population. If the *a priori* expectation of an anthropometrical index of nutrition is that it is associated with measures of health, then the BMI derived from half-span may be a better proxy for nutritional status in the elderly than the BMI derived from stature, the converse may be true for younger groups. The analysis in table 6.16 seem to support this suggestion.

In terms of the CED cut-offs, table 6.14 shows that, amongst those individuals who were healthy at the baseline, the CED group (defined by the BMI alone) had an RR of 3.00 of reporting subsequent illness compared to the non-CED group. This RR is similar to those described by other authors in their cross-sectional analyses (Pryer, 1990 & 1994; de Vasconcellos, 1994). Unfortunately it was not possible to examine the associations between morbidity and BMI<17.0 (CED grade II) as only 20 subjects who were healthy at the baseline were CED grade II.

No cut-offs for diagnosing CED have been suggested for the BMI derived from half-span, and hence no analysis using the CED defined by the BMI derived from half-span has been attempted. The CED group defined by the MUAC was not significantly more likely to report subsequent illness than the non-CED group. This result may be due to the small numerator - only 28 individuals who were healthy at the baseline were diagnosed as CED using the low MUAC definition.

It is probable that, although a cut-off point of BMI<18.5kg/m^2 does seem to incur an increased risk of morbidity, the relationships between either the BMI or MUAC and morbidity are continuous. No distinct threshold of BMI below which morbidity increases steeply can be seen in figure 6.2, rather the prevalence of morbidity increases incrementally as the BMI decreases.

In summary, taken in isolation tables 6.12-6.16 do not provide convincing evidence that the BMI or MUAC can predict subsequent morbidity experience. However, when all the analyses are considered together, the results are suggestive of a causal relationship between anthropometric status and self-reported illness in this population.

It is important to note that low BMI or MUAC may be associated with under-nutrition of other types. For example, Garn et al (1983) found that lean men had lower levels of vitamin A and carotene than obese men. The association between low BMI and morbidity may not be caused by the biological effect of the thinness itself, but may be due to factors associated with thinness. In

this study haemoglobin level was found to be significantly positively associated with the BMI or MUAC, and at the population level those with low BMI or MUAC had increased ORs of being anaemic. Haemoglobin level was not, however, a significant confounding factor in the relationships between BMI or MIUAC and morbidity. Alternatively, deficiencies of vitamins and micro-nutrients could play a role in the nutrition-infection relationship in conjunction with protein or energy under-nutrition (Payne & Lipton, 1994). Until further prospective studies controlling for other nutrients are undertaken, it will not be possible to assess the true cause of the association seen.

The aim of this study was to assess the BMI and MUAC in terms of indices of current adult energy status. If low BMI and MUAC are associated with poor nutritional status of other nutrients then this may make them more useful indices of nutrition, particularly if the other nutrients are also associated with illness.

6.10 Conclusions

- Low BMI and MUAC are both associated with increased risk of non-incidental or non-HT-related illness cross-sectionally, although age may alter this relationship.

- MUAC may be weakly predictive of subsequent morbidity status in the section of the population which is healthy at the baseline.

- In the longitudinal analyses of the over fifties, the BMI derived from stature may not be as strongly associated with morbidity as the BMI derived from half-span. The converse may be true for a younger group.

- Amongst the group of subjects who were healthy at the baseline, those with CED ($BMI<18.5 kg/m^2$) had an RR of 3.00 of subsequently reporting morbidity compared to the non-CED group.

CHAPTER SEVEN

CONCLUSION

This study is concerned with the use of anthropometric measurements and indices as tools to assess current adult nutritional status. This involved examining the practical characteristics, distribution and determinants of the anthropometric variables and also their relationships to morbidity and mortality. The anthropometric variables considered were weight, height, BMI (derived from stature or half-span), MUAC, FM and FFM.

This chapter will begin by discussing the methods employed. An examination of the findings will follow. Finally implications for future research will be suggested.

7.1 Discussion of the Method

This study is unique in that it is the first prospective longitudinal study of the relationship between anthropometry, mortality and morbidity in adults in the developing world in a non-famine situation. Certain aspects of the methodology worked well. The follow-up section of the study used the most straightforward methods and was probably also the most successful part of the project. Of the 962 survivors at visit A in 1996 only 65 refused re-measurement or could not be interviewed by the author. Of the 85 non-survivors only one VA could not be obtained. Given the six year interval this follow-up of 94% was remarkable. Furthermore, given the nature of Iban society, the author is confident that the 6% who could not be interviewed were alive at the time of interview with their relatives and friends. The success of this part of the study must be attributed to the Iban themselves; they were extremely helpful and willing to co-operate with the author at all times.

The methods used in the morbidity section of the project were more complex and problematic than those described above. Accurate measures of morbidity status and history are notoriously difficult to obtain (Ross & Vaughn, 1986). In this study, self-reported measures of morbidity were obtained. This type of definition of morbidity is closer to the concept of illness than that of disease. The major problem with perceived morbidity is that it depends not only on the subjects' actual patho-physiological experience, but also on their perception of illness and their illness behaviour. By excluding those individuals who reported being ill at the baseline it was hoped not only that the chronically ill would be removed from any association seen (in order to exclude circularity) but also that individuals who tended to exaggerate their illnesses were removed from

the analyses.

In the methods section it was explained how the various symptoms were grouped together into symptom types. The groupings are crude and some overlap may exist between the categories. Also, the bias in reporting HT may be problematic. If many of the subjects' illnesses were wrongly classified as being associated with HT rather than not associated with HT or vice versa then the analyses of morbidity will be biased.

A weakness of the morbidity method is that the study was unable to answer questions concerning the frequency of illness. If it had been possible to collect data on morbidity between the author's visits some of these questions may have been answered. Data on the incidence or duration of illness have been used to assess causal links between diarrhoea and malnutrition in young children (Briend, 1990). However, it should be noted that any serial collection of morbidity data may be difficult as (a) the subjects may become bored with the repetitive questioning, and (b) it may not be ethical to ask questions about illness over a prolonged period of time and not give medicines or other treatments to alleviate the problem; if the problems are alleviated the study may become invalid (Sommer, 1996).

In general, the major weakness of this study was the sample size. The sample size of the analyses considering the relationships between anthropometrical status and mortality or morbidity in distinct age groups was relatively small. It is evident that the sample size of the follow-up study was fixed. The 1990 project was not undertaken with a follow-up study in mind and thus the authors of the original study did not calculate sample sizes for this project. If the follow-up period had been extended more deaths would have been recorded. However, extending the follow-up would have been problematic itself in terms of accuracy of the VA (Garenne, 1990) and tracing of migrants. Theoretically, the sample size of the morbidity section of the study could have been enlarged - the newcomers to the original longhouses since 1990 or the residents of other longhouses could also have been assessed. Funding and time budgets prevented this.

Many of the analyses assessing the significance of the MUAC cut-off points for CED had small numerators and were unsatisfactory. This occurred because only a small percentage of the study population is diagnosed as CED using the MUAC definition. It should be noted that when the author started work on this study the authors of the scheme which uses MUAC cut-offs for diagnosing CED had not yet published their proposals (James et al, 1994b). Thus no allowances for this type of analysis were made during the sample size calculations.

A further consequence of the study's relatively small sample size is that no allowance could be made for the possibility of deaths or reports of morbidity being clustered together. It is possible that in some longhouses levels of certain disease types were higher than others e.g.: malaria levels may have been raised in longhouses near stagnant water. These types of analyses are best undertaken using multi-level modelling techniques and require larger sample sizes.

SES and other "lifestyle" factors were mainly measured at the HH level in this study. Thus an assumption was made that all the individuals in a certain HH would have the same SES. This assumption may not have been valid for all the subjects, particularly for the temporary migrants. When considering those who lived permanently in the longhouses the assumption was perfectly acceptable for some factors, e.g.: the presence of piped water in the *bilek*. However the assumption may have been invalid for the wealth factors, e.g.: the savings variable, as one or more of the individuals in the HH may have had easier access to the "communal" savings than the others. In defence of the method used it should be noted that, given the Iban's social structure, it would have been awkward to ask questions concerning personal as opposed to HH wealth. However, it might have been useful to record each individual's professional category for example, farmer or labourer or teacher.

The SES factors were relatively crudely measured (e.g.: the material possessions scale) and the variance explained by these factors was generally low. Many epidemiological studies of this nature rely on proxy indicators of SES (Nutrition CRSP, 1993) and the concept which these indicators denote is imprecise or loose - hence they are not expected to be strongly associated with anthropometric status.

More detailed information on the PAL of the subjects might have been useful in elucidating the causes of the age related changes in anthropometrical status observed. These kinds of data could also have been used to examine the anthropometry and morbidity or mortality links seen.

7.2 Summary of findings

Given the drawbacks described above, this section will discuss the general findings of the study. Weight, height, BMI (derived from stature or half-span), MUAC, and skinfold thicknesses were assessed in terms of their characteristics as ideal indices of current adult nutritional status, so that they should be -

(i) highly correlated with body energy stores

(ii) independent of body height

(iii) independent of sex and age

(iv) highly correlated with health and/or functional outcomes

(v) fulfil all the practical characteristics (i.e. be simple to measure and interpret, accurate, valid and precise)

It is difficult to obtain accurate and precise skinfold measures (Garrow, 1988). Moreover, their interpretation as FM and FFM using Durnin and Wormersley's (1974) equations is not straightforward. Thus skinfold measurements are not ideal anthropometric indices of current adult nutritional status. Measures of height, half-span, weight and MUAC are generally easier to obtain than skinfolds, although even these measurements may be harder to make in the elderly. Stature, in particular, is difficult to obtain in the elderly. It is evident that neither weight nor height fulfil characteristic (ii). Given that only the BMI and the MUAC fulfil characteristics (ii) and (v) the discussion below will focus on these two indices.

Both the BMI and MUAC are highly correlated with body energy stores. The BMI is more highly correlated with either FM or FFM than is the MUAC in this population. The relationships between the BMI or MUAC and FM or FFM are altered by age and sex. At a given BMI or MUAC women have a higher mean FM and lower mean FFM than men. At a given BMI or MUAC younger groups have a higher mean FFM and lower mean FM than older groups. Thus identical BMIs or MUACs do not necessarily equate to identical body energy stores in distinct age or sex groups in this population.

Neither the BMI or MUAC are completely independent of stature or shape. Those with a low CI (i.e. individuals with relatively long legs) are more likely to have a low BMI (Norgan, 1994a & 1994b) or MUAC. The BMI is less highly correlated with stature than the MUAC; the MUAC is less highly correlated with shape.

The distributions of both the BMI and MUAC can be partially explained by age and sex in this population. In the cross-sectional analyses, 17% of the variance in the BMI and 21% of the variance in MUAC are explained by age and sex. Mean MUAC is significantly different for men and women; this is accounted for in the scheme using MUAC cut-offs to diagnose CED (James et al 1994b). Mean BMIs do not differ between men and women if the whole population is compared, but the women's BMI is significantly higher than the men's in the 20-40 year age

range. In fact, most of the variance in the BMI and the MUAC is explained by age. As age increases the risk of CED increases and that of being overweight decreases. A further 5% of the variance in the BMI and MUAC can be explained by SES and other "lifestyle" variables.

None of the findings described above are unique to this study and most were discussed by the authors of the cut-off schemes when assessing how to diagnose adult CED (Ferro-Luzzi et al, 1992; James et al, 1994b; Shetty & James, 1994; Norgan 1994b). The importance of the findings lies in whether or not these differences in body composition or mean values of BMI and MUAC result in different functional outcomes. For example, if FFM is more important in determining immunocompetence than FM, and women have a lower FFM than men at a given BMI, should women have an elevated BMI cut-off in order to elicit an identical response to illness as men? Similarly, are the functional consequences of having low BMI or MUAC the same in older and younger groups, and if not, should the same methods be used to diagnose CED for all ages?

Low values of the BMI and MUAC are associated with increased risks of mortality after controlling for SES and other "lifestyle" factors and excluding early mortality. A hierarchy of evidence suggests that low values of the BMI and MUAC are associated with increased risk of self-reported non-incidental or non-HT related illnesses. Cross-sectional and longitudinal analyses excluding those with chronic illness show that, in the study population, an increase in either the BMI or MUAC results in a decrease in the odds of reporting illness. In the longitudinal analyses excluding all individuals who reported any illness at the baseline, a unit increase in the BMI derived from stature did not result in significantly decreased odds of reporting illness at the population level. However a unit increase in the MUAC did result in significantly decreased odds of reporting illness in an identical analysis.

Age, but not sex, is a significant confounding factor in the relationship between the BMI or MUAC and mortality in all the analyses run. Small sample sizes prevented the most rigorous testing of the associations between the BMI or MUAC and mortality in distinct age groups and the analyses run at different ages included early deaths. These analyses suggest that the BMI may be a better predictor of mortality in the middle aged group (40-60 years) but that the MUAC may be a better predictor of mortality in the older group (>=60 years). Age and sex are not significant confounders in the longitudinal analyses of the BMI or MUAC and morbidity. However, the BMI derived from half-span may be a better predictor of morbidity than the BMI derived from stature in the group over 50 years and the converse may be true for the younger group.

In this study it is not truly possible to assess the relative merits of the different methods of diagnosing CED. This is because only a small number of subjects in either 1990 or 1996 are actually diagnosed as CED when low MUAC is a criterion of diagnosis. A BMI<18.5kg/m^2 is associated with increased risks of mortality and morbidity if the whole population is examined. In the elderly, this cut-off point is not significantly associated with an increase in mortality, but the cut-offs for low MUAC are significant. The converse is true for the middle-aged groups. Given that MUAC (entered as a continuous variable) is associated with both mortality and morbidity in many of the analyses presented it is probable that the cut-off scheme utilising the MUAC will also be useful. This study cannot ascertain the added value of using the MUAC in conjunction with or separately from the BMI to diagnose CED.

It is important to stress that the relationship between the BMI or MUAC and mortality or morbidity may not best be described in terms of a threshold. Given the results of the analyses when the indices are entered as continuous variables it is possible that a dose-response relationship may exist. There is some debate in the literature as to whether or not the relationship between growth and mortality in children is best described as a threshold or continuum (Pelletier, 1991; Chen et al, 1980). Further studies in adults are required in order to describe the shapes of these relationships in more detail.

On the basis of the results from this study it is difficult to assess whether the BMI or MUAC is the most useful index of current adult nutritional status. MUAC is generally easier to measure and less complicated to calculate; it also less highly correlated with shape. However, the BMI is more highly correlated with body energy stores and more independent of height. In the analyses described both the BMI and MUAC are associated with mortality when the whole population is considered. The BMI may be a better predictor of mortality in those aged less than 60 years and the MUAC may be a better predictor of mortality in those aged more than 60 years. If the whole population is considered, then the MUAC may be a better predictor of morbidity than the BMI derived from stature, however the BMI derived from half-span may also be a useful predictor of morbidity for older groups.

The results of this study suggest that the BMI derived from stature may not be the best index of current energy nutritional status in older adults. This may be because stature is generally difficult to measure in this group or because kyphosis of the spine results in the BMI derived from stature actually being higher than the subjects' true BMI (i.e. their BMI if their spines were straight). The BMI derived from half-span or MUAC may be better indices of current nutritional

status for the older groups. Alternatively, the BMI cut-off points for CED could be raised in older adults in order to compensate for their decrease in stature.

The use of BMI alone to diagnose CED will classify more adults as undernourished than if the BMI and the MUAC are used in conjunction. The authors of the BMI and MUAC cut-off scheme put this idea forward in order to prevent thin but healthy adults being classified as CED (James et al. 1994b). Some of the analyses presented in this project show that CED diagnosed by the BMI alone is associated with increased risks of mortality and morbidity. Thus excluding individuals who have low BMI but not low MUAC in a diagnosis of CED may result in the exclusion of some subjects who have a sufficiently poor nutritional status to compromise their health.

7.3 Implications for future research

The findings of this study suggest that the BMI and MUAC may be useful indices of current adult anthropometric indices. However, further studies are required to confirm these results. The discussion below suggests some types of study which may be useful to this end.

Both the mortality and morbidity section of this project are hampered by relatively low statistical power. Power could be improved either by increasing the sample sizes or by studying a population with a higher proportion of CED subjects. In particular, it is important for future studies to have enough power to assess the affects of age in the relationships between anthropometric status and mortality or morbidity.

For the mortality section a further follow-up study of the current population would be useful as this would allow assessment of the long-term effects of low anthropometrical status. Furthermore, when more deaths have been recorded it may be possible to assess the associations between anthropometrical status and specific causes of death.

This study has shown that an association exists between self-reported morbidity and anthropometric status. It would be useful to assess whether or not an association exists between anthropometric status and observed morbidity. Observed measures of morbidity generally involve measurement of physical and vital signs, physiological and patho-physiological indicators, functional tests and clinical diagnoses. This method would probably be an ideal way to assess the presence of chronic disease at the baseline, even if it could not be used on a longitudinal basis. A further refinement of the morbidity study would be to examine morbidity

over a whole year thus excluding any effects of seasonality.

This study has not assessed whether the associations between low BMI or MUAC and morbidity are due to the biological effect of the thinness itself or due to other factors associated with thinness. Deficiencies of vitamins and micro-nutrients could play a role in the nutrition-infection relationship in conjunction with protein or energy under-nutrition. Until further prospective studies controlling for other nutrients are undertaken, it will not be possible to assess the causes of the associations seen.

Given that this study is the first of its type in the developing world it is important to see if these results can be generalised to other populations. Thus it may be useful to undertake other studies assessing the relationship between anthropometric status and morbidity or mortality in distinct populations.

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APPENDICES

Appendix A1: Household questionnaire

Longhouse _____ Head of HH _____

Bilik Number _____ HH number _____

ID	Name	Sex	1990 Age	1996 Age	Rel to HH	Anth 1990	Status I	New address	Status II	New address

ADDITIONAL MEMBERS OF THE HH (SINCE 1990)

ID	Name	Visit	Sex	Age	Rel to HH	Status I	New address	Status II	New address

Appendix A2: SES questionnaire

Table A2.1: SES Questionnaire

FLOOR MATERIAL		MATERIAL POSSESSIONS	
Linoleum		Chainsaw	
Concrete/brick		Outboard motor	
Wood		Cassette player	
Bamboo		Electricity	
Other		Television	
ROOF MATERIAL		OCCUPATION	
Zinc		Paid employment	
Asbestos		Government service	
Palm/atap		School teacher	
Other		Labourer	
WALL MATERIAL		Timber camp worker	
Concrete/brick		Other	
Plank		REMITTANCES	
Plywood		Do you receive any?	
Other		LAND	
WINDOWS		No swidden farm	
Glass		Hill padi	
Wire netting		Swamp/ wet padi	
Nothing		Kerukoh & kakah (< 1 year of growth)	
Other		Dijap (2 / 3 years growth)	
WASHING WATER		Pengerang (2ndry forest)	
Piped into residence		CASH CROPPING	
Piped into yard or plot		Do you grow cocoa	
Public tap		Do you grow coffee	
River		Do you grow rubber	
Rainwater		Do you grow fruit	
Other		Do you grow elipe nuts	
DRINKING WATER		Have you sold any cocoa	
Same source as washing water?		Have you sold any coffee	
Piped into residence		Have you sold any rubber	
Piped into yard or plot		Have you sold any fruit	
Public tap		Have you sold any elippe nuts	
River		Other cash crops	
Rainwater		SAVINGS	
Other		Do you have any?	
TOILET FACILITIES		How much?	
Private with flush			
Private no flush			
Shared/Public			
No toilet			
Other			

Table A2.2: Education questionnaire

Have you ever attended school?	
What is the highest level of school you attended: primary, secondary or university?	
What is the highest (grade, form, year) you completed at that level?	
Can you read or understand a letter or newspaper easily, with difficulty, or not at all?	
Do you usually read a newspaper or magazine at least once a week?	

Appendix A3 : SES Variables

Material possessions:	0=score less than the median value of the scale
	1=more than or equal to the median value of the scale
Savings:	0=no savings
	1=savings
Paid work:	0=no-one in the *bilek* has paid work
	1=someone in the *bilek* has paid work
Remittances:	0=those remaining in the *bilek* do not receive remittances
	1=remittances are received by those remaining in the *bilek*
Sold crops:	0=no cash crops sold in the last year
	1=some cash crops sold in the last year
Electricity:	0=the *bilek* has no electricity
	1=the *bilek* does have electricity
Water source:	0=the *bilek* does not have piped water
	1=the *bilek* does have piped water
Toilet:	0=the *bilek* does not have a toilet
	1=the *bilek* does have a toilet
Window materials:	0=the *bilek* does not have glass windows
	1=the *bilek* does have glass windows
Floor material:	0=the *bilek* does not have lineoleum floors
	1=the *bilek* does have lineoleum floors
Distance to nearest town:	0=less than 2 hours to the closest town
	1=more than 2 hours to the closest town

Appendix A4: Physical activity questionnaire

Table A4.1: Physical activity questionnaire

Place of Work	Yes/No	Activity	Yes/No
LONGHOUSE		Washing	
		Cooking	
		Child care	
		Handiwork	
		None of the above	
FARM		Clear	
		Weed	
		Plant	
		Harvest	
		Threshing	
OUTSIDE THE FARM		Labourer	
		Desk job	

Appendix A5: Equations used to transform the 1990 data.

AD-SSS equations for correction for bias

Height = (height*1.003973)-0.6339314
Sitting height = (sitting height*1.00212)-0.4690306
MUAC = (muac*1.012571)-0.167275
Biceps = (biceps*1.356792)-1.240613
Triceps = (triceps*1.323751)-1.479241
Subscapular = (subscapular*0.9174224)-1.27194
Suprailliac = (suprailliac*1.035866)+1.977697

AD-SJU equations for correction for bias

Height = (height*0.989636)+1.712982
Sitting height = (sitting height*1.011643)-0.7210039
MUAC = (muac*0.9706294)+1.302426
Biceps = (biceps*0.794581)+1.281839
Triceps = (triceps*0.9299889)+1.122887
Subscapular = (subscapular*1.067416)+1.488283
Suprailliac = (suprailliacs*0.962834)+1.417679

Appendix A6.1: Verbal autopsy

Respondent Identification Form

Name of the deceased _____ ..... ID No. |_|_|_|_|

I. List of potential respondents

Names of potential respondents	Age	Sex	Relationship to deceased	Appropriateness	Availability	Participation

Relationship to deceased: RECORD THE RELATIONSHIP OF THE RESPONDENT TO THE DECEASED (e.g. If the deceased is a man and the respondent is his daughter, then the relationship is daughter not father)

Appropriateness: REFER TO THE INSTRUCTION MANUAL FOR DEFINITIONS (very appropriate; appropriate; probably appropriate; may be appropriate)

Availability: REFER TO THE INSTRUCTION MANUAL FOR DEFINITIONS (present; absent; unavailable)

Participation: TICK AT THE END OF THE INTERVIEW IN THE BOXES OF THOSE WHO PARTICIPATED

II. Identification & Demographic Data of Principal Respondent

Q1. Name of respondent _____

Q2. Age of respondent ..... |_|_|

Q3. Sex of respondent (*male=1; female=2*) ..... |_|

Q4. Relationship of respondent ..... |_|
(spouse=1; daughter=2; son=3; mother=4; father=5;
others=6 (specify) _____)

Q5. Years of formal education of respondent ..... |_|_| .....

Q6..... First language of the respondent _____

III..... Information about the visits

7.Date of the Visit ..... |_|_|/|_|_|/|_|_|

8Date of the second Visit..... |_|_|/|_|_|/|_|_|

9Date of the third Visit..... |_|_|/|_|_|/|_|_|

10.Reason(s) for abandoning the interview (IF YOU COMPLETE THIS SECTION, DISCUSS WITH YOUR SU

VERBAL AUTOPSY QUESTIONNAIRE FOR ADULT DEATHS

I. Identification & Demographic Data of Deceased

Q1. Name _____---_ Q2. ID NO |_|_|_|_| .....IDN

Q3. Address _____

Q4. Age of deceased ..... |_|_| AOD

Q5. Sex of deceased (*male=1; female=2*) ..... |_| SXD

Q6. Marital status of deceased ..... |_| MSD
(single=1; married=2; divorced/separated=3; widowed=4)

Q7. Years of formal education of deceased ..... |_|_| YED

Q8. Occupation of deceased _____ |_| OCC

II. Circumstance of Death

Q9. For how many days was s/he ill before s/he died? (DK=999) |_|_|_| .....DID

Q10. Date of death (*dd/mm/yy*) ..... |_|_|/|_|_|/|_|_| DOD

Q11. Place of death (*home=1; hospital/clinic=2; others=3*) ..... |_| POD
(IF THE ANSWER IS HOME OR OTHER PROCEED TO Q12)
a. Name of the hospital where s/he died _____
b. Did anyone from the hospital tell you why s/he died? ..... |_| RIF

Q12. Do you know what disease (s) was the cause (s) of his/her death? ..... |_| RKC
(*no=0, yes=1, NS=9*)
a. IF THE ANSWER IS YES PROBE TO SPECIFY THE CAUSE(S)
cause (1) _____ ..... |_|_| RD1
cause (2) _____ ..... |_|_| RD2

Q13. (ASK WHETHER S/HE HAD ANY OF THE FOLLOWING ILLNESS)
Hypertension ..... (no=0; yes=1; NS=9) ..... |_| HYP
Diabetes ..... (no=0; yes=1; NS=9) ..... |_| DIA
Epilepsy ..... (no=0; yes=1; NS=9) ..... |_| EPI
TB ..... (no=0; yes=1; NS=9) ..... |_| TB
HIV ..... (no=0; yes=1; NS=9) ..... |_| HIV

ID NO. |_|_|_|_| ..... IDN

III. Respondents Account of Final Illness

Summary of symptoms & signs reported by Respondent

Symptoms	Duration	Severity
1.		
2.		
3.		
4.		
5.		
6.		
7.		
8.		

ID NO. |_|_|_|_| ..... IDN

IV. Specific questions to elicit symptoms & signs of the final illness

S1. Did s/he have fever (*no=0; yes=1; don't know (DK)=9*) ..... |_| FEV
(IF THE ANSWER IS NO OR DK PROCEED TO S2)
a. For how many days did s/he have fever? (*DK=999*) ..... |_|_|_| DPE
b. Was the fever severe? (*severe=1; mild=2; DK=9*) ..... |_| SFE
c. Was the fever present continuous or on and off? ..... |_| TFE
(*continuous=1; on and off=0; DK=9*)

S2. Did s/he have a rash? (*no=0; yes=1; DK=9*) ..... |_| RAS
(IF THE ANSWER IS NO OR DK PROCEED TO S3)
a. For how many days did s/he have the rash? (*DK=999*) ..... |_|_|_| DRA
b. What did the rash look like? (*measles rash=1; rash with clear fluid=2;*
rash with pus=3; others=4; DK=9) ..... |_| TRA
c. Did s/he have sore eyes? (*no=0; yes=1; DK=9*) ..... |_| SEY
d. Did s/he have itching of the skin? (*no=0; yes=1; DK=9*) ..... |_| ITC

S3. Has s/he lost weight recently before death? (*no=0; yes=1; DK=9*) ..... |_| LOW
(IF THE ANSWER IS NO OR DK PROCEED TO S4)
a. Was the loss of weight severe? (*severe=1; moderate=2; DK=9*) ..... |_| SLW

S4. Did s/he have swelling around ankles? (*no=0; yes=1; DK=9*) ..... |_| SAA
(IF THE ANSWER IS NO OR DK PROCEED TO S5)
a. How many days did s/he have the swelling? (*DK=999*) ..... |_|_|_| DSA

S5. Did s/he have puffiness of the face? (*no=0; yes=1; DK=9*) ..... |_| PUF

S6. Did s/he look pale (anaemic)? (*no=0; yes=1; DK=9*) ..... |_| PAL

S7. Did s/he have yellow discoloration of the eyes? ..... |_| JAU
(*no=0; yes=1; DK=9*)

S9. Did s/he have swelling in the NECK? (*no=0; yes=1; DK=9*) ..... |_| SWN

S9. Did s/he have swelling in the axilla? (*no=0; yes=1; DK=9*) ..... |_| SWA

S10. Did she have swelling in the groin? (*no=0; yes=1; DK=9*) ..... |_| SWG

S11. Did s/he have any other swelling or ulcers? (IF THE ANSWER IS YES PROBE FOR THE SITE AND DURATION) _____

_____

S12. Did s/he have cough? (*no=0; yes=1; DK=9*) ..... |_| COU
(IF THE ANSWER IS NO OR DK PROCEED TO S13)
a. How many days s/he had cough? (DK=999) ..... |_|_|_| DCO
b. Was the cough productive (sputum)? (*no=0; yes=1; DK=9*) ..... |_| PCO
c. Did s/he cough blood? (*no=0; yes=1; DK=9*) ..... |_| BCO

S13. Did s/he have shortness of breathing? (*no=0; yes=1; DK=9*) ..... |_| DIB
(IF THE ANSWER IS NO OR DK PROCEED TO S14)
a. Was the shortness of breathing continuous or on and off? (*no=0; yes=1; DK=9*) ..... |_| TDB
b. How many days s/he had breathlessness? (*DK=999*) ..... |_|_|_| DDB
c. Did s/he have wheezing? (*no=0; yes=1; DK=9*) ..... |_| WHE

S14. Did s/he have chest pain? (*no=0, yes=1; DK=9*) ..... |_| CHP
(IF THE ANSWER IS NO OR DK PROCEED TO S15)
a. Where was the pain? ..... |_| SCP
(*over the sternum=1; over the heart=2; others=3; DK=9*)
b. Was the pain continuous (*=1*) or on and off (*=2*)? (*DK=9*) ..... |_| TCP
c. When s/he had an attack of severe pain, how long did it last? ..... |_| DCP
(*< 30 min=1, < 30 min but < 24 hrs=2; > 24 hrs=3; DK=9*)

S15. Did s/he have diarrhoea? (*no=0; yes=1; D=9*) ..... |_| DI
(IF THE ANSWER IS NO OR NS PROCEED TO S16)
a. How many days s/he have diarrhoea? (DK=999) ..... |_|_|_| DDI
b. Was the diarrhoea continuous (*=1*) or on and off (*=2*)? (*DK=9*) ..... |_| TDI
c. When the diarrhoea was severe, how many times did s/he pass stool in a day? ..... |_|_| FDI
(*DK=99*)
d. What did the stool look like? ..... |_| TST
(*watery=1; loose but not watery=2; bloody=3; DK=9*)

S16. Did s/he pass blood in the stool? (*no=0; yes=1; DK=9*) ..... |_| BST

S17. Did s/he have vomiting? (*no=0; yes=1; DK=9*) ..... |_| VOM
(IF THE ANSWER IS NO OR NS PROCEED TO S18)
a. How many days s/he have vomiting? (DK=999) ..... |_|_|_| DVO
b. Was the vomiting continuous (*=1*) or on and off (*=2*)? (*DK=9*) ..... |_| TVO
c. When the diarrhoea was severe, how many times did s/he pass stool in a day? ..... |_|_| FVO
(*DK=99*)
d. What did the vomitus look like? ..... |_| CVO
(*watery fluid=1; yellowish fluid=2; coffee coulored fluid=3; blood=4; faecal matter=5,*
other=6 _____ *; DK=9*)

S18 Did s/he have abdominal pain? (*no=0; yes=1; DK=9*) ..... |_|ABP
(IF THE ANSWER IS NO OR DK PROCEED TO S19)
a. What was the type of pain? ..... |_|CAP
(cramps=1; dull ache=2; burning pain=3; others=4; DK=9)
b. How many days s/he had the pain? (DK=999) ..... |_|_|_|DAP
c. Where exactly was the pain? ..... |_|SAP
(lower abdomen=1; upper abdomen=2; all over the abdomen=3; others=4; DK=9)
d. What was the severity of the pain? ..... |_|TAP
(severe=1; moderate=2; mild=3; DK=9)
e. Was s/he unable to pass stool for some days before death? ..... |_|CON
(able to pass=0; unable to pass=1; DK=9)

S19 Did s/he have distension of the abdomen? (*no=0; yes=1; DK=9*) ..... |_|ABD
(IF THE ANSWER IS NO OR DK PROCEED TO S20)
a. How many days s/he had abdominal distension? *(DK=999)* ..... |_|_|_|DAD
b. Did the distension develop rapidly with in days or slowly over weeks? ..... |_|TAD
(rapid=1; slowly=2; DK=9).

S20 Did s/he have distension of the abdomen? (*no=0; yes=1; DK=9*) ..... |_|DSW
(IF THE ANSWER IS NO OR DK PROCEED TO S21)
a. How many days s/he had difficulty in swallowing? *(DK=999)* ..... |_|_|_|DDS

S21 Did s/he have any mass in the abdomen? (*no=0; yes=1; DK=9*) ..... |_|ABM
(IF THE ANSWER IS NO OR DK PROCEED TO S22)
a. Where exactly was the mass? ..... |_|SAM
(Rt upper abdomen=1; Lt upper abdomen=2; Lower abdomen=3;
others (specify _____ =4; DK=9)
b. How many days s/he had the mass? (*DK=999*) ..... |_|_|_|DAM

S22 Did s/he have headache? (*no=0; yes=1; DK=9*) ..... |_|HEA

S23 Did s/he have a stiff neck? (*no=0; yes=1; DK=9*) ..... |_|STN
(IF THE ANSWER IS NO OR DK PROCEED TO S24)
a. If yes, for how many days? *(DK=999)* ..... |_|_|_|DSN

S24 Did s/he have any change in the level of consciousness? (*no=0; yes=1; DK=9*) ..... |_|LUC
(IF THE ANSWER IS NO OR DK PROCEED TO S25)
a. What was the level of consciousness? ..... |_|_|_|TUC
(confused=1; unconscious=2; others _____ =4; DK=9)

b. If confused or unconscious, for how many days? (DK=999) ..... |_|_|_|DUC
c. How did it start? ..... |_|OUC
(suddenly=1; rapidly within a day=2; slowly over few days=3; DK=9)

S25. Did s/he have fits? (*no=0; yes=1; DK=9*) ..... |_|FIT
(IF THE ANSWER IS NO OR DK PROCEED TO S26)
a. How many days s/he had fits? *(DK=999)* ..... |_|_|_|DFI
b. (ASK THE RESPONDENT TO DESCRIBE THE FITS) ..... |_|TFI
(repetitive jerking of whole body=1; others _____
_____ *=2; DK=9)*
c. When fits were most frequent, how many per day? (*DK=99*) ..... |_|_|FFI
d. Between fits was s/he awake (*=1*) or unconscious (*=2*)? (*DK=9*) ..... |_|BFA

S26. Did s/he have difficulty in opening the mouth? ..... |_|LOC
(able to open=0; unable to open=1; DK=9)

S27. Did s/he have stiffness of the whole body? (*no=0; yes=1; DK=9*) ..... |_|OPI
(IF THE ANSWER IS NO OR DK PROCEED TO S28)
a. How many days s/he had the stiffness? *(DK=999)* ..... |_|_|_|DOP

S28. Did s/he have paralysis of one side of the body? (*no=0; yes=1; DK=9*) ..... |_|HEM
(IF THE ANSWER IS NO OR DK PROCEED TO S29)
a. How many days s/he had the paralysis? *(DK=999)* ..... |_|_|_|DHE

S29. Did s/he have paralysis of one side of lower limbs? (*no=0, yes=1, DK=9*) ..... |_|PAR
(IF THE ANSWER IS NO OR DK PROCEED TO S30)
a. How many days s/he had the paralysis? *(DK=999)* ..... |_|_|_|DPA

S30. Was there any change in the colour of urine? ..... |_|CCU
(IF THE ANSWER IS NO OR DK PROCEED TO S31)
a. What was the colour of the urine? ..... |_|TCC
(dark yellow=1; coffee like=2; blood stained=3; DK=9)
b. How many days s/he had the change in colour? *(DK=999)* ..... |_|_|_|DCC

S31. Was there any change in the amount of urine s/he passed daily? ..... |_|CQU
(*no=0; yes=1; DK=9*)
(IF THE ANSWER IS NO OR DK PROCEED TO S32)
a. How much urine did s/he pass in a day? ..... |_|AQU
(too much=1, too little=2, no urine at all=3; DK=9)

b. How many days s/he had the change in amount of urine? (DK=999) ..... |_|_|_| DQU

S32. Did s/he have difficulty in passing urine? (*no=0; yes=1; DK=9*)..... |_| DPU
(IF THE ANSWER IS NO OR DK PROCEED TO S33)
a. What type of difficulty did s/he have? ..... |_| TDP
(unable to pass urine=1; continuous dribbling of urine=2;
burning sensation while passing urine=3; others=4; DK=9)

S33. Did s/he have any operation before death? (*no=0; yes=1; DK=9*)..... |_| HOP
(IF THE ANSWER IS NO OR DK PROCEED TO S34)
a. How many days before death s/he had the operation? *(DK=999)* ..... |_|_|_| OPD
b. (ASK FOR THE SITE OF THE OPERATION) ..... |_| OPS
(abdomen=1; others=2 _____; DK=9)

IF THE DECEASED IS A FEMALE AND >50 YRS OLD PROCEED TO S37

IF THE DECEASED IS A MALE PROCEED TO S39

S34. Was she pregnant at the time of death? (*no=0; yes=1; DK=9*)..... |_| PRE
(IF THE ANSWER IS NO OR DK PROCEED TO S35)
a. How many months was she pregnant? *(DK=99)* ..... |_|_| MPR

S35. Did she deliver within 45 days before death? (*no=0; yes=1; DK=9*)..... |_| DEL
(IF THE ANSWER IS NO OR DK PROCEED TO S36)
a. How many days before her death did she deliver? (*DK=99*) ..... |_|_| EDD
b. Where did she deliver? (*home=1; clinic=2; hospital=3; DK=9*) ..... |_| PDE
c. How long was she in labour? (*< 24hrs; > 24hrs; DK=9*) ..... |_| DDE
d. Did she have too much bleeding during delivery? (*no=0; yes=1; DK=9*)..... |_| BDE
e. (IF YES PROBE TO FIND OUT WHETHER THE BLEEDING STARTED BEFORE OR AFTER THE DELIVERY OF FOETUS) ..... |_| HDE
f. What was the mode of delivery? ..... |_| MDE
(vaginal delivery; vacuuum or forceps delivery=2; abdominal operative delivery=3; DK=9)
g. Is the baby alive? (IF NO PROBE FOR THE TIME OF DEATH) ..... |_| PNC
(alive=1; still born=2; died within 7 days=3; died after 7 days=4)
h. Did she have any previous complicated delivery? (*no=0; yes=1; DK=9*)..... |_| PCD

S36. Did she have an abortion within 45 days of death? (*no=0; yes=1; NS=9*)..... |_| ABO

S37. Did she have any irregular bleeding per vagina? (*no=0; yes=1; NS=9*)..... |_| ABV

S38. Did she have any swelling or ulcer in the breast? (*no=0; yes=1; NS=9*)..... |_| BT

S39. Did s/he sustain any injury which lead to his/her death? (*no=0; yes=1; NS=9*)..... |_| BT
(IF THE ANSWER IS NO OR DK PROCEED TO S40)
a. (IF THE ANSWER IS YES, PROBE FOR THE TYPE OF INJURY) ..... |_| INJ
(assault=1; road traffic accident=2; war injury=3; animal bite=4; fire accident=5
accidental poisoning=6; others=7 (specify) _____ *)*
b. How many days before death s/he had the injury? (NS=999)..... |_|_|_| DIN

S39. Do you think that s/he committed suicide? (*no=0; yes=1; NS=9*)..... |_| SUI
(IF THE ANSWER IS NO OR DK PROCEED TO S40)
a. How did s/he commit suicide? ..... |_| TSU
(hanging=1; poisoning=2; burns=3; others=4 _____ *)*

V. Interviewer's comments and observations

..... Interviewer's assessment of cause of death
Cause of death 1 _____
Cause of death 2 _____

Interviewer's ID NO ..... |_|_| IID
Date of Interview .....(dd\mm\yy)..... |_|_|/|_|_|/|_|_| DOI

Appendix A6.2: Verbal autopsy - mortality classification

1 Communicable diseases

1.0 Unspecified communicable diseases

1.1 Acute Febrile illness
1.1.1 Unspecified acute febrile illness
1.1.2 Malaria
1.1.3 Meningitis
1.1.4 Typhoid
1.1.5 Hepatitis
1.1.6 Acute respiratory infections
1.1.7 All other specified acute febrile illnesses

1.2 Tuberculosis / AIDS
1.2.1 Unspecified TB/AIDS
1.2.2 Pulmonary TB
1.2.3 AIDS

1.3 Gastro-enteritis / Dysentary

1.4 Tetanus

1.9 All other specified communicable diseases

2.0 Maternal causes

2.1 Early maternal causes
2.1.0 Unspecified early maternal causes
2.1.1 Abortion
2.1.2 All other specified early maternal causes

2.2 Late maternal causes
2.2.1 Unspecified late maternal causes
2.2.2 Eclampsia
2.2.3 Obstructed labour / ruptured uterus
2.2.4 Antepartum Haemorrhage
2.2.5 Postpartum Haemorrhage
2.2.6 Puerperal Haemorrhage
2.2.7 Puerperal Sepsis
2.2.8 Indirect maternal causes
2.2.9 All other specified late maternal causes

3 Non-Communicable diseases

3.0 Unspecified non-communicable diseases

3.1 Cardiovascular disorders
3.1.0 Unspecified cardiovascular disorders
3.1.1 Congestive cardiac failure
3.1.2 Ischaemmic heart disease
3.1.3 Cerebrovascular disease
3.1.4 All other specified cardiovascular disorders

3.2 Chronic obstructive airways disease

3.3 Liver cirrhosis

3.4 Acute abdominal conditions

3.5 Diabetes

3.6 Neoplasms
3.6.0 Unspecified neoplasms
3.6.1 Hepetoma
3.6.2 Carcinoma of breast
3.6.3 Carcinoma of cervix / uterus
3.6.4 Carcinoma of oesophagus
3.6.5 All other specified carcinomas

3.7 Renal disorders

3.8 Central nervous system disorders

3.9 All other specified non-communicable diseases

4. Symptoms, signs and syndromes not classified elsewhere

4.1 Anaemia

4.9 All other symptoms, signs and syndromes not classified elsewhere

5 External Causes

5.0 Unspecified external causes

5.1 Unintentional injuries
5.1.0 Unspecified unintentional injuries
5.1.1 Transport
5.1.2 Falls
5.1.3 Fires
5.1.4 Poisoning
5.1.5 Drowning
5.1.6 All other specified unintentional injuries

5.2 Intentional injuries
5.2.0 Unspecified intentional injuries
5.2.1 Suicide
5.2.2 Homicide
5.2.3 War
5.2.4 All other specified intentional injuries

6 Undetermined

APPENDIX A6.3: MORTALITY RATE

Table A6.1 Number of deaths per person months lived during six years of follow-up by sex and age group per thousand people

Age group and Sex	2/5/90-1/5/91	2/5/91-1/5/92	2/5/92-1/5/93	2/5/93-1/5/94	2/5/94-1/5/95	2/5/95-1/5/96
Male						
18.0-29.9	0.000	0.000	0.000	0.000	0.765	0.000
30.0-39.9	0.000	0.000	0.000	0.000	0.000	1.080
40.0-49.9	0.835	0.000	0.000	0.000	0.000	0.850
50.0-59.9	3.608	2.539	2.599	1.337	1.352	2.754
60.0-69.9	4.036	8.709	6.261	1.653	5.094	3.604
70.0+	0.000	12.701	4.513	4.888	4.959	5.304
Total	1.326	2.138	1.383	0.601	1.209	1.438
Female						
18.0-29.9	0.000	0.000	0.000	0.000	0.000	0.000
30.0-39.9	0.000	0.000	0.000	0.000	0.000	0.000
40.0-49.9	0.000	0.000	1.361	0.000	0.694	0.699
50.0-59.9	0.000	0.825	0.841	0.846	0.000	1.732
60.0-69.9	1.436	2.876	9.212	1.698	0.000	5.234
70.0+	6.651	7.245	25.063	17.472	6.917	25.203
Total	0.417	0.697	2.123	0.724	0.291	1.325
All	0.801	1.299	1.814	0.672	0.676	1.372

APPENDIX A6.4: PRINCIPLE CAUSES OF DEATH

Table A6.2 Percentage of deaths by cause by sex and age group

	Pulmonary Tuberculosis	Other Communicable Diseases	Cardiovascular disorders	Chronic Obstructive Lung Diseases	Neoplasms	Other non-communicable diseases	Accidents	Undetermined
Male								
18.0-39.9						1.18	3.53	
40.0-59.9	3.53		1.18	3.53	4.71	1.18		3.53
60.0+	2.35	1.18	15.29		4.71	7.06		
Total	5.88	1.18	16.47	3.53	9.42	9.42	3.53	3.53
Female								
18.0-39.9								
40.0-59.9			7.06		1.18	1.18		1.18
60.0+	3.53	5.88	9.41	1.18	8.23	2.35		5.88
Total	3.53	5.88	16.47	1.18	9.41	3.53		7.06
All	9.41	7.06	32.94	4.71	18.82	12.94	3.53	10.59

Appendix A7: Morbidity questionnaire

Table A7.1: Two-week morbidity recall questionnaire

Symptom	Yes / No	No. of days	Today	Suffer	Doctor	Medicine
	1= yes 0= no		1= yes 0= no	1=v.severe 2= serious 3 = mild 9= D.K.	1= doctor 2= M.A. 3= nurse 0= no	1= prescription 2= over the counter 0= none
Headache						
Fever						
Diarrhoea						
Abdominal or Gastric Pain						
Cough						
Chest pain						
Muscle or bone pain						
Injury						
HT						
Other						

Table A7.2: Activity during previous 14 days questionnaire

	Affected yes /no	No. of whole days of work missed	No. of days of work partially missed	Illness
	1= yes 0= no			1= headache, 2= fever, 3= diarrhoea, 4= gastric pain, 5= cough, 6= chest pain, 7= muscle / bone pain, 8= injury, 9= other
Farm work				
Household work				
Other work				

Table A7.3: Chronic Illness questionnaire

Symptom	How long?	Doctor	Medicine	Usual activities
1= headache, 2= fever, 3= diarrhoea, 4= gastric pain, 5= cough, 6= chest pain, 7= muscle / bone pain, 8= injury, 9= other		1= doctor 2= M.A. 3= nurse 0= no	1=prescription 2= over the counter 0= none	1= restricted 0 = not restricted

Appendix A8: Sample size calculations

Table A8.1: Mortality predictions using Coale & Demeny Model West level 18

Age in 1990	Men			Women			Total		
	1990	1997	Deaths	1990	1997	Deaths	1990	1997	Deaths
18.0-19.9	23	22.55	0.45	25	24.59	0.41	48	47.14	0.86
20.0-24.9	48	46.89	1.11	61	59.78	1.22	109	106.67	2.33
25.0-29.9	40	38.97	1.03	43	42.00	1.00	83	80.97	2.03
30.0-34.9	41	39.72	1.28	55	53.50	1.50	96	93.22	2.78
35.0-39.9	34	32.63	1.37	52	50.28	1.72	86	82.92	3.08
40.0-44.9	57	53.89	3.11	57	54.60	2.40	114	108.50	5.50
45.0-49.9	38	35.09	2.91	51	48.09	2.91	89	83.18	5.82
50.0-54.9	36	32.07	3.93	55	50.57	4.43	91	82.64	8.36
55.0-59.9	24	20.24	3.76	36	31.75	4.25	60	51.99	8.01
60.0-64.9	35	27.18	7.82	49	40.36	8.64	84	67.54	16.46
65.0-69.9	36	24.64	11.36	35	25.75	9.25	71	50.39	20.61
70.0-74.9	15	8.41	6.59	26	15.93	10.07	41	24.34	16.66
75.0-79.9	11	4.62	6.38	8	3.76	4.24	19	8.38	10.62
80.0-84.9	9	2.51	6.49	3	0.95	2.05	12	3.46	8.54
85.0-89.9	0	0	0	4	0.70	3.30	4	0.70	3.30
90+	0	0	0	4		4.00	4	0	4.00
Total	447	389.42	57.58	564	502.61	61.39	1011	892.03	118.97

Table A8.2 Mortality predictions using Coale & Demeney Model West level 22

Age in 1990	Men			Women			Total		
	1990	1997	Deaths	1990	1997	Deaths	1990	1997	Deaths
18.0-19.9	23	22.81	0.19	25	24.88	0.12	48	47.68	0.32
20.0-24.9	48	47.53	0.47	61	60.61	0.39	109	108.15	0.85
25.0-29.9	40	39.57	0.43	43	42.66	0.34	83	82.23	0.77
30.0-34.9	41	40.45	0.55	55	54.42	0.58	96	94.87	1.13
35.0-39.9	34	33.34	0.66	52	51.22	0.78	86	84.56	1.44
40.0-44.9	57	55.28	1.72	57	55.72	1.28	114	111.00	3.00
45.0-49.9	38	36.17	1.83	51	49.27	1.73	89	85.44	3.56
50.0-54.9	36	33.26	2.74	55	52.15	2.85	91	85.40	5.60
55.0-59.9	24	21.17	2.83	36	33.07	2.93	60	54.24	5.76
60.0-64.9	35	28.75	6.25	49	42.56	6.44	84	71.31	12.69
65.0-69.9	36	26.40	9.60	35	27.56	7.44	71	53.96	17.04
70.0-74.9	15	9.19	5.81	26	17.40	8.60	41	26.59	14.41
75.0-79.9	11	5.20	5.80	8	4.22	3.78	19	9.42	9.58
80.0-84.9	9	2.90	6.10	3	1.10	1.90	12	4.00	8.00
85.0-89.9	0	0.00	0.00	4	0.84	3.16	4	0.84	3.16
90+	0	0.00	0.00	4	0.00	4.00	4	0.00	4.00
Total	447.00	402.01	44.99	564	517.67	46.33	1011	919.69	91.31

Appendix A9.1: Extra mortality analysis

Table A9.1: HRs of mortality for the anthropometric measurements (entered as continuous variables) amongst all subjects who were not pregnant or overweight in 1990 and for whom SES data was available, controlling for age and sex (N=822).

	H R	95% C I	p-value
Height	0.987	0.956-1.018	0.400
Weight	0.969	0.938-1.000	0.052
BMI	0.894	0.809-0.987	0.027
MUAC	0.887	0.812-0.969	0.008
Fat mass	0.932	0.868-1.001	0.055
Fat-free mass	0.904	0.846-0.965	0.003

p-value for the maximum likelihood ratio test

Table A9.2: RRs of mortality for the section of the population with the lowest quintile of anthropometric measurements compared to the rest of the population amongst all subjects who were not pregnant or overweight in 1990 and for whom SES data was available, controlling for age and sex (N=822).

	H R	95% C I	p-value
Height	1.614	0.997-2.614	0.052
Weight	1.567	0.962-2.551	0.071
BMI	1.710	1.052-2.780	0.030
MUAC	1.822	1.124-2.955	0.015
Fat mass	1.648	1.001-2.712	0.049
Fat-free mass	2.568	1.028-6.418	0.044

p-value for the maximum likelihood ratio test