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Review Article

Body habitus and coronary heart disease in men

A review with reference to methods of body habitus assessment

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Introduction

Despite recent reports of an encouraging decline in the coronary heart disease mortality rate[1,2], it remains a leading cause of morbidity and mortality in many modern societies.

During the last several decades, a great deal of attention has been focused on the identification of potentially modifiable biological, physiological and biochemical risk factors[3] that place the individual at an increased risk of developing atheromatous lesions in the coronary blood vessels. The degree of overweight or obesity (and the two are not synonymous), are two possible risk factors that have attracted a great deal of research attention in men[4–12,15–44]. Height has also been studied as a potential marker for ischaemic coronary heart disease[4–14].

Despite this abundance of information, contrasting findings suggest that the exact position of overweight or obesity in the aetiology of coronary heart disease remains unclear. One possible explanation for this disparity is that the measurement techniques employed do not satisfactorily estimate body fatness. More recent evidence suggests that these inconsistencies can also be partly explained by the distribution of body fat[16,22,24–27,30–32,43,44]. As the metabolic complications associated with excess body fat may require a prolonged period of time before their effect on cardiovascular disease mortality is observable, the duration of the obese state may also be an important factor in explaining these inconsistencies[45].

The focus of this review is the association between human body habitus and atherosclerotic coronary heart disease in men. The terms cardiovascular disease, coronary heart disease, coronary artery disease and ischaemic heart disease are not used interchangeably, rather, no attempt has been made to alter the terminology adopted by the original research. Commentary is made on the wide variety of both simple and more complex methods that have been used to assess body habitus. The term body habitus has been chosen to incorporate a number of distinct physical bodily characteristics. These include body weight and height, weight-for-height, relative weight, total body fat, fat distribution, subcutaneous fat pattern and somatotype (body shape). Body weight and height are the simplest, most accessible measurements of body size and are generally reliable with small technical errors of measurements[46]. Thus, they have become important and extensively used epidemiological research tools. However, it is clear they cannot provide information on body composition. To overcome this limitation, there has been continued interest in the development of valid and reliable body composition estimators such as relative weight scores or weight-for-height indices. These have been the most extensively used indicators of overweight. Coronary heart disease mortality and morbidity rates have also been examined in a variety of ways, including the analysis of hospital and physicians' records, self-reporting of coronary events, information from the next of kin, postmortem findings, death certificates and recently coronary angiography. These factors coupled with varying lengths of subject follow-up, contrasting statistical analysis and socio-economic, ethnic and risk-factor variation between subjects from different studies

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makes interpretation of the vast amount of available literature difficult.

Body weight and height

Prospective studies

Amongst the earliest investigations of an association between coronary heart disease and body weight and height are the classic studies of Harvard and Pennsylvania University students[6,7]. Paffenberger and co-workers found that for later coronary decedents, body weight at initial examination was greater than controls. This study also found that compared to controls, a greater percentage of coronary decedents were less than 68 inches (172 cm tall) (32% vs 22%, $P<0.001$). An increased incidence of ischaemic heart disease was reported for shorter London transport workers (height range 151 to 167 cm) compared to their taller counterparts ($P<0.1$)[8]. A study of 17 530 London office workers reported an inverse relationship between height and ischaemic heart disease after 7·5 and 10 years follow-up following multivariate adjustment for age and grade of employment[9,10]. Further research of nearly 18 000 civil servants discovered the highest ischaemic heart disease incidence rate was for subjects shorter than 5 feet 5 inches (165·1 cm)[11]. A 16-year prospective study of almost 1·8 million Norwegians (approximately 900 000 men) found cardiovascular disease mortality was clearly reduced for those who were taller[12]. For males shorter than 160 cm, cardiovascular disease mortality was 50% to 100% greater than the total. For those between 185 and 189 cm, however, cardiovascular disease mortality was only 70% to 80% of the total mortality. The British Regional Heart Study of 7735 middle-aged men demonstrated a similar finding[13]. The mean height of subjects who suffered an ischaemic heart disease event (n=443) was significantly lower than the height of the remaining subjects (171·7 cm vs 173·3 cm, $P<0.001$). Adjustment for age, social class, serum total cholesterol, high-density lipoprotein cholesterol, systolic blood pressure and cigarette smoking weakened the association by over 50%. As height and lung function (forced expiratory volume in one second, FEV_1) were closely correlated (r=0·44, $P<0.002$), and lung function is associated with ischaemic heart disease[47], FEV_1 was added to the multivariate model. The addition of lung function alone ($P=0.25$) or in combination with other confounding variables ($P=0.70$) further weakened the relationship. The height and ischaemic heart disease relationship has recently been reported for 2512 South Wales men (Caerphilly cohort) and 2348 men from the West of England (Speedwell cohort)[14]. After just 61 and 38 months follow-up, respectively, significant inverse trends were found between height and the number of ischaemic heart disease events (both fatal and non-fatal) in the Caerphilly ($P<0.001$) and Speedwell ($P<0.05$) cohorts. Men in the shortest 20% of the height distribution suffered more than double the ischaemic heart disease events than men in the tallest 20%. Adjustment for age, social class, smoking habit and FEV_1 in the Caerphilly cohort weakened the relationship ($P<0.05$). After 26 years follow-up of a select cohort of almost 4000 North American male airline pilots, body weight was significantly greater (76·5 ± 0·5 vs 74·2 ± 0·2, $P<0.01$) and height shorter (175·8 ± 0·3 vs 176·9 ± 0·1 cm, $P<0.01$) in subjects who developed coronary heart disease[15]. Postmortem findings of 71 decedents from the Framingham Study revealed that body weight 1 and 9 years before death independently predicted left ventricular thickness[35]. Height and body weight measured 5 years before death had inverse and positive associations with heart weight, respectively.

Case-control studies

Gertler and co-workers found that men hospitalized with myocardial infarction were approximately 5·0 cm shorter and 3 kg heavier than control subjects[4]. Later analysis found height to be second only to total cholesterol as a predictor of coronary heart disease although cigarette smoking was not considered[5].

Angiography studies

The use of coronary angiography to group subjects into those with significant (>50% stenosis in one, two or three coronary vessels) or insignificant arterial disease (a normal angiogram or <50% stenosis), has recently shown a non-significant association between body weight and disease status ($P>0.05$)[16]. Height and coronary artery disease exhibited a significant inverse relationship following univariate ($P<0.01$) and multivariate analysis ($P<0.05$). Hauner *et al.* found that height was significantly shorter ($P<0.01$) and weight greater ($P<0.05$) in subjects with coronary artery disease and a history of myocardial infarction compared to men free of coronary artery disease[30]. In a further angiography study, there was no difference in height and weight between normal men and women with coronary artery disease[32].

Evaluation of body weight and height as predictors of coronary heart disease

A number of possible explanations have been proposed to give the inverse relationship between height and coronary heart disease a biological basis. As suggested, multicolinearity with lung function as a confounding variable may be one explanation. Inadequate pre-natal, infant and childhood nutrition and the occurrence of illness during the growing years may partly account for some cases of shorter attained adult stature. It is plausible that these factors may also directly affect

pulmonary development and, therefore, explain the association between height and lung function[13]. Based on findings from a large number of studies, Barker has suggested that under-nutrition of the fetus, in utero, can lead to permanent changes in structure, physiology and metabolism that predispose to elevated fibrinogen and factor VII, non-insulin dependent diabetes, hypertension, hyperlipidaemia and, therefore, to an increased risk of cardiovascular disease[48]. Inverse relationships between height and total cholesterol, high density lipoprotein-cholesterol, systolic blood pressure and smoking duration have also been reported[13]. Correlation coefficients are weak however ($r = -0.04$ to -0.11, $P<0.002$), and are significant due to the large sample size. A further possible biological mechanism is that taller individuals have larger coronary arteries than shorter individuals and, therefore, have a lessened risk of occlusion[49]. Support for this mechanism can be derived from studies which have found a higher post coronary bypass surgery mortality rate for shorter individuals compared to taller individuals[50,51].

Results from studies examining the coronary heart disease relationship with body weight are inconclusive. In the Manitoba study, the mean body weight of the coronary heart disease subjects (76·5 kg ± 0·5 kg) can hardly be considered large and although significant, differed from the body weight of subjects free of coronary heart disease by only about 2·0 kg[15]. The striking similarity in the body weight of subjects with significant (77·4 kg ± 9·6 kg) and insignificant (77·8 kg ± 11·3 kg) arterial disease[16] may be partly accounted for by the insensitivity of the disease classification criteria used. Of interest would be a comparison of the mean body weight of asymptomatic subjects and those with evidence of extreme arterial disease. Contrary to this theory however, no difference was found in the height and weight of men free from coronary artery disease when compared to men with angina and an angiogram showing greater than 50% luminal narrowing[32]. From the limited amount of research, it appears that body weight, per se, is not as strong a predictor of cardiovascular disease as height, although the underlying biological mechanism remains to be established.

Weight-for-height ratios

Complex laboratory methods for estimating body composition are inappropriate for large-scale surveys. The simplicity of measurement and availability of normative data have, therefore, contributed to the widespread use of weight-for-height ratios (W/H^p). The power function (^{p}) should be calculated so that the index is highly correlated with body weight and fatness but be independent of height. The most widely used weight-for-height ratio is Quetelets index (W/H^2) or body mass index[15–22,24–32,38,41,43,44]. Other ratios to have been applied in epidemiological studies include W/H[23] and Sheldon's[52] ponderal index ($H/W^{0.33}$)[6,7,38].

Prospective studies

A number of large-scale population studies examining the association between body mass index and coronary heart disease have been performed in both North America and Europe[15–22,24–28,30–32,38,41,43,44]. Jooste and co-workers have examined this relationship in 7188 white South Africans[29]. Data gathered in these studies have produced inconsistent findings.

Dyer *et al.* found a U-shaped curve described the relationship between body mass index and coronary heart disease mortality in 1233 white middle-aged North American men followed for 14 years[17]. Rhoads and Kagan reported this phenomenon in 8006 men aged 45–68 years who were subsequently followed for 10 years as part of the Honolulu Heart Program[18]. In this latter study, excess deaths amongst those in the lower body mass index category were due primarily to cancer and in the upper body mass index groups to coronary heart disease. In South Africa, the incidence of coronary heart disease in relation to body mass index was greater in both the lowest (body mass index <20) ($P>0.05$) and highest (body mass index 30–35 and >35) ($P<0.01$) body mass index categories[29]. A number of studies with varying lengths of follow-up (5–26 years) have shown little or no association between body mass index and coronary heart disease. Keys *et al.* reported no association between coronary heart disease and a variety of physical measurements (including body mass index) in their 23 year study of Minnesota Executives[19]. Similar findings were observed after a 5 year investigation of 11 400 men from Northern and Southern Europe and North America (Minnesota Railroad Workers)[20]. Despite an excessive incidence of coronary heart disease in overweight subjects, after the confounding effects of age, blood pressure, serum cholesterol and smoking were removed the contribution of body mass index to this trend was not significant ($P>0.05$). After 15 years follow-up there was still no relationship[21]. In a further multivariate model, with age, total cholesterol, triglyceride, systolic blood pressure, cigarette smoking, presence of diabetes and a fat distribution index entered as covariates, body mass index was not a predictor of coronary heart disease ($P>0.05$)[22]. The Stockholm prospective study of 3168 men identified smoking and elevated levels of plasma cholesterol and triglycerides as independent risk factors for ischaemic heart disease but not the index W/H[23]. Further Scandinavian research found no association ($P>0.05$) between body mass index and the 13 year incidence of ischaemic heart disease, stroke and death[24]. After adjustment for subscapular skinfold thickness, the independent effect of body mass index on either non-fatal myocardial infarction or death from coronary heart disease was not significant ($P>0.05$) after 12 years follow-up in the Honolulu Heart Program[25]. A recent study has reported that of an original random sample of 107 Edinburgh men, 11 developed clinical coronary heart disease over the subsequent 12 year period[26]. Examination of baseline data revealed the body mass index of coronary heart disease

men (26·7 ± 0·8) was greater ($P<0·05$) than the men who remained free of the disease (24·9 ± 0·3) (values are means ± SEM). Other risk factors (total cholesterol, triglycerides, diastolic blood pressure, indices of glucose–insulin homoeostasis) were not significantly different ($P>0·05$). However, following adjustment for high density lipoprotein-cholesterol, which was lower in coronary heart disease patients ($P<0·05$), body mass index was no longer a significant risk factor ($P>0·05$). Recently, researchers from the Paris Prospective Study found increasing body mass index was modestly associated with cardiovascular disease in subjects with a mean blood pressure less than 96 mmHg, but had no effect in men with higher blood pressure (≥96 mmHg)[44].

In a few instances, large-scale prospective studies have reported a significant independent relationship between body mass index and coronary heart disease. After adjustment for age and blood pressure, body mass index was found to be a significant independent predictor of sudden death ($P<0·01$), coronary insufficiency or suspected myocardial infarction ($P<0·05$) and myocardial infarction ($P<0·05$)[15]. The 7 year follow-up of 3786 men in eastern Finland found men with a body mass index of 28·5 or more experienced a significantly greater incidence of acute myocardial infarction ($P<0·05$)[41]. This effect was independent of age and smoking but not other major coronary risk factors (total cholesterol and blood pressure). In the Framingham cohort, standardized logistic regression analysis controlling the effects of age, serum cholesterol, cigarette smoking, systolic blood pressure, blood glucose and ECG evidence of left ventricular hypertrophy demonstrated a significant ($P<0·05$) positive influence of body mass index on the 22-year incidence of coronary heart disease[27].

Angiography studies

Recent results from the Honolulu Heart Program have shown body mass index to be a significant predictor of both arteriographically diagnosed severe coronary stenosis and incident myocardial infarction after 20 years follow-up of 357 men[28]. However, further recent angiography studies that have provided similar results, conflict with these later findings from the Honolulu Program. Body mass index was not related ($P=0·197$) to coronary artery disease in 286 men following stepwise logistic regression analysis[30]. Chi-square analysis also revealed no difference ($P>0·05$) in the body mass index of men with coronary artery disease (>30% stenosis), men with coronary artery disease plus a history of myocardial infarction, and men without coronary artery disease. Flynn and her colleagues found no relationship ($P>0·05$) between coronary artery disease and body mass index[16]. Other weight-for-height indices, including the risk index of body build [W (kg)/H $(m)^{2\cdot2}$], adipose tissue index [0·75 $(W/H^{0·35}) - 21·4$] and body fat index [0·72 $(W/H^{0·40}) - 23·5$][53] also showed no correlation with coronary artery disease ($P>0·05$). Hodgson and coworkers have recently applied different scoring systems to quantify an extent score (proportion of coronary endothelial surface area affected by atheroma)[54] and a myocardial score (degree of stenosis of any number of arterial branches)[55] in 160 men and 66 women undergoing cardiac catheterization[31]. Spearman's rank correlation coefficients between body mass index and both extent score and myocardial score were not significant ($P>0·05$) for men or women. Non-significant differences ($P>0·05$) have recently been reported for the body mass index of middle-aged normal men (24·5 ± 0·3 kg . m^{-2}), men with angina but a normal angiogram (25·1 ± 0·4 kg . m^{-2}) and men with angina and an abnormal angiogram (25·1 ± 0·3 kg . m^{-2})[32]. Thompson and coworkers found no difference between the mean body mass index of patients with confirmed coronary atherosclerosis (27·0 ± 3·5 kg . m^{-2}), hospitalized controls (27·0 ± 3·7 kg . m^{-2}) and neighbourhood controls (26·4 ± 3·5 kg . m^{-2}) ($P>0·05$)[43].

Evaluation of weight-for-height ratios as predictors of coronary heart disease

The variation in the relationship between weight-for-height indices and cardiovascular disease may be due partly to the inaccuracy of these indices in estimating body fat. The numerator, body weight, is composed of lean as well as fat tissue. Body mass indices are, therefore, as much estimations of musculoskeletal mass as fat mass. An individual with a considerable muscle, bone and organ mass relative to height may be classified as obese even though they may not have a large fat mass. Similarly, in individuals with small muscle and bone masses relative to height, body fat will be underestimated[56]. In a population sense this may be unimportant as the main cause of excessive weight-for-height is an increased fat mass[57]. However, using simple weight-for-height ratios to compare different populations is particularly unreliable if they differ in ethnicity and socio-economic status[57]. For instance, high body mass indices found amongst the Canadian Inuit were explained by short stature and well developed musculature rather than excessive body fat[58,59]. The genetically homogeneous Pima Indians, on the other hand, exhibit a high prevalence of obesity[60].

The correlation between body mass index and body fat derived from underwater weighing has been reported to be 0·55 for men[61]. This leaves 70% of the variation in fatness unexplained. Correlations between densitometrically assessed body fat and other weight-for-height indices (W/H, W/H^3, $W^{0·33}/H$, $H/W^{0·33}$ and percentage overweight based on age, sex and height) were of a similar magnitude[61]. Smalley *et al.* found a slightly stronger relationship (r=0·70) between body mass index and relative body fat estimated from densitometry in 150 men[62]. In their study of United States Air Force personnel, Weinsier *et al.* found a correlation of 0·74 between body mass index and relative body fat estimated using tritium dilution[38]. Another study reported a common variance of 41% between body mass

index and relative body fat in 342 males[63]. Micozzi *et al.* reported a correlation of 0·77 between body mass index and subscapular skinfold thickness in men from the National Health and Nutrition Examination Survey (NHANES 1)[46]. Even though these correlations indicate a stronger relationship, with respect to body fat estimation, body mass index still has little predictive power. Garn *et al.* found a significant correlation (r=0·65) between body mass index and lean body mass in their analysis of data from the Tecumseh Community Health Survey[64]. Body mass index was also related to radiogrammatically determined bony chest breadth in men aged 50–60 years (r=0·67)[64]. This supports the notion that body mass index is as much a reflection of lean body mass as it is fat mass.

Weight-for-height indices are also supposed to dissociate height. Data from NHANES 1 show a non-significant association between height and body mass index in men[46]. Garn *et al.*, however, have shown a relationship between relative sitting height (sitting height/stature) and body mass index in men aged 20–35 and 36–50 (r=0·21)[64]. This suggests body mass index is also influenced by body proportions and means that shorter-legged individuals can have higher body mass index values by as much as 5 units[64].

The consequences of these limitations is that to describe individuals as obese on the basis of a W/H index is unfounded and potentially misleading. The term obesity refers to excess body fat and should, therefore, be applied when more precise measurements of body fat are used. As weight-for-height indices simply describe body weight in relation to height, the term overweight is preferable as their validity as an indicator of fatness is questionable.

Relative weight

Relative weight is obtained by expressing the individual's bodyweight as a percentage of some reference weight. This reference data, usually based on a large, random, cross-sectional sample can be obtained from a regression equation or chart[65] or more frequently a set of height–weight tables. Although relative weight implies no value judgment[66], correlations with mortality has led to the application of the concept of 'desirable'[66] or 'ideal' weight. These terms are used to describe individuals at lowest risk of premature mortality and as the standard for weight reduction targets.

Prospective studies

The relationship of Framingham Relative Weight (deviation of body weight from the median weight of the population distribution) to the 12 year incidence of coronary heart disease suggested an excess risk of angina and sudden death in 'obese' men[33]. This excess risk existed in the absence of elevated blood pressure and serum cholesterol. After 18 years follow-up, a positive linear association was observable in the male population[34]. An autopsy study of 127 Framingham decedents found relative weight 9 years prior to death was an independent predictor of heart weight but not left ventricular muscle thickness, percentage luminal involvement or percentage luminal insufficiency[35]. Hubert and her colleagues later gathered data on 2252 Framingham men[36]. Metropolitan Relative Weight (ratio of actual to desirable weight) independently predicted the 26 year incidence of angina, coronary disease other than angina, coronary death and congestive heart failure. Desirable weight was derived from Metropolitan Life Insurance Company height–weight tables[67] by taking the midpoint of the weight range for a medium build at a specified height. Metropolitan height–weight tables[67] were also used to calculate excess weight in a group of 200 'morbidly obese men' (mean excess bodyweight=130%) aged 23 to 70 years[37]. After 7·6 years follow-up, the total number of deaths was 50. Cardiovascular disease was the most common cause in the study subjects (54·0%) and the U.S. male population (40·3%). Compared to the general population, life-table techniques demonstrated a 12-fold excess mortality in subjects aged 25 to 34 years and a 6-fold excess in subjects aged 35 to 44 years. This ratio continued to diminish with advancing age.

The final report of the Pooling Project suggested relative weight was associated with an increased risk of a first coronary event only in younger men aged 40–44 years ($P<0·01$) and 45–49 years ($P<0·05$)[42]. Keys *et al.* found no association between coronary heart disease and relative weight in 279 men after 20 years follow-up[19]. Later multivariate analysis also found no association between relative weight and coronary heart disease in larger male cohorts from the United States, southern Europe and northern Europe after 5[20] and 15[21] years follow-up.

Evaluation of relative weight as a predictor of coronary heart disease

As with weight-for-height ratios, one unequivocal limitation of the relative weight concept is its inability to differentiate fat and lean tissues and, therefore, satisfactorily predict adiposity. The 1959 Metropolitan height–weight tables[67] were first to consider the significance of skeletal mass by introducing the 'frame-size' concept. Later, anthropometric measurements were introduced to give this concept some objectivity. The frequently used biepicondylar elbow breadth, however, which is used to categorize frame-size in the 1983 Metropolitan tables[68] has a poor correlation with other measures of skeletal size, bone density and, thus, bone mass[65]. Furthermore, considerable inter-individual variation in bone mineral density means that even if bone size is controlled, bone mass may still differ markedly. Anthropometric bone diameters are also influenced by subcutaneous adipose tissue and skin thickness. This means that frame size tends to be overestimated in fatter subjects and underestimated in lean subjects.

Further limitations of the relative weight concept are discussed in-depth by Harrison[66] based on Knapp's earlier discourse[69]. First, the quality of data used to construct height–weight tables is in some instances questionable. For example, about 10% of weights and heights used to construct the 1983 Metropolitan tables[68] were self-reported. In addition, the clothing of those who were measured in this study (Build Study, 1979)[70,71] was not standardized. Second, few studies, including the Build Study[70,71] have adequately controlled variables known to have a confounding influence on the weight and mortality relationship, most noticeably cigarette smoking[72]. Third, describing weight as a percentage of a reference value does not represent a constant degree of overweight. For example, 40% overweight could describe both a person weighing 84 kg whose 'desirable' weight is 60 kg, or a person weighing 140 kg whose 'desirable' weight is 100 kg. Finally, even some of the largest data sets[70,71,73] may not be representative of populations as a whole. This means that for some under-represented sections of the population (e.g. lower socio-economic groups, non-caucasians and those older than 60 years) the tables may not be a valid indicator of the weight-for-height relationship with mortality.

The two-component model

Evaluation of absolute and relative fat mass

The lack of validity of weight-for-height ratios and relative weight as body fat surrogates is one possible explanation for the variation in the relationship between obesity and coronary heart disease[74]. The more precise measurement of body composition should, therefore, yield a stronger correlation between body fatness and coronary heart disease. Several studies have used more valid body composition measurement methods and adopted a two-component model that includes fat and fat-free masses. In this respect, skinfold thickness measurement, which has been used in both cross-sectional[16] and prospective studies[19–21,24], has predominated. Others have used more sophisticated methods including underwater weighing[19], tritium dilution[38] and dual energy X-ray absorptiometry[32].

Underwater weighing is widely used in the estimation of whole body density from body volume. The difference between the weight of the body in air and weight of the body submerged in water, corrected for the density of the water at the time of measurement and residual lung volume, which ideally should be measured during submersion, is the body volume[75]. Body density values are then often used to calculate relative body fat from a standard equation such as that of Siri[76].

As approximately 50% to 70% of the body's total fat content is located subcutaneously, skinfolds are associated with total body fatness[77]. The alluring feature of skinfolds for the estimation of body fat in large-scale surveys is their inexpense and relative simplicity. Based on the inverse relationship with skinfold thickness, there are many regression equations for the prediction of body density and subsequently relative body fat available in the literature. Rather than convert skinfold values into body fat estimations in this way, some researchers have used the sum of skinfold values in their own right as the indicator of body fatness[19–21,24].

Tritium dilution allows measurement of total body water from which fat-free mass can be estimated (assuming a fixed hydration of this tissue component, usually 73%) i.e. fat-free mass=total body water/0·73. The method is based on the assumption that the radioisotope tritium (^3H), which is measured with liquid scintillation counting, has the same distribution volume as water. The subject is given an accurately measured oral or intravenous dose of labelled water, followed by an equilibration period of at least 2 h before sampling a body fluid, either saliva, blood or urine[75]. The accuracy of both isotope dilution and underwater weighing is in the range of 1–2%[75].

Dual energy X-ray absorptiometry allows the precise measurement of total and regional body composition with a very low radiation exposure. As the dual energy radiation source scans soft tissue, the relative attenuation of the photons changes in proportion to the fat content[56]. The short-term precision of dual energy X-ray absorptiometry for measuring the relative fat in soft tissue has been reported as 1–2%[78].

Prospective studies

Recognizing that neither relative weight nor body mass index provide satisfactory estimates of body fat, Keys *et al.* examined the coronary heart disease relationship with the sum of triceps and subscapular skinfolds[19,20] and whole body density derived from underwater weighing[19]. Neither exhibited a significant relationship with coronary heart disease incidence ($P>0·05$). The sum of triceps, subscapular and parathoracic skinfolds were found to be unrelated to cardiovascular disease in Gothenberg men[24]. Weinsier *et al.* estimated relative body fat from total body water measured by the tritium dilution technique and found no difference ($P>0·05$) between those with coronary heart disease (23%) and those without (21·1%)[38].

Angiography studies

Flynn *et al.*[16] estimated relative body fat from the sum of biceps, triceps, subscapular and suprailiac skinfolds using the regression equation of Durnin and Womersely[79]. They found a significant difference ($P<0·05$) between men with insignificant disease (27·7% ± 6·0%) and men with significant disease (29·3% ± 5·2%). In a multivariate model, however, relative body fat was not an independent predictor of cardiovascular disease. More recently, no differences ($P>0·05$) in absolute fat mass measured by dual energy

X-ray absorptiometry were found between normal healthy men (17·1 kg ± 0·6 kg), men with angina and a normal angiogram (18·6 kg ± 0·9 kg) and men with angina and an abnormal angiogram (17·0 kg ± 0·6 kg)[32].

Evaluation of fat mass as a predictor of coronary heart disease

If total body fat is important in the pathogenesis of atherosclerotic cardiovascular disease the results from these studies[16,19–21,24,32,38] are perhaps somewhat surprising. Neither long-term prospective nor case-control designs, including angiography, have shown a relationship between cardiovascular disease and body fat.

The estimation of relative body fat from body density relies on several unsound assumptions[80], perhaps the most notable being that the fat-free mass has a chemical composition resulting in a density of $1{\cdot}10\ g\ .\ ml^{-1}$. For this reason, the calculation of relative body fat from body density has been criticized and the use of density in its own right advocated[81]. As Keys *et al.*[19] used body density rather than relative body fat in their analysis, the inaccurate estimation of body fat cannot be a contributory factor in the explanation of these findings.

The validity for the prediction of relative body fat from skinfold thickness is based on the inverse relationship with body density. The estimation of relative body fat from skinfold thickness is, therefore, subject to the same assumptions[80] plus others. These include, constant compressibility of the skinfolds, negligible or constant skin thickness, fixed adipose tissue patterning, constant fat portion of adipose tissue and a fixed proportion of deep to subcutaneous fat. Cadaver dissections suggest that, in the elderly at least, these assumptions should be rejected[81,82]. For this reason, it may also be more desirable to use skinfolds and their sum totals in their own right rather than force them into an equation to predict relative body fat. The evidence to date, however, suggests that a limited number of skinfolds treated in this way or in a relative body fat equation are poor predictors of cardiovascular disease.

Body fat distribution

Evaluation of fat distribution

Since the results of two large prospective studies in Scandinavia[24,83] confirmed the findings originally reported by Vague *et al.*[39,40], the focus of research in the area of obesity and cardiovascular disease has shifted. Evidence is accumulating in support of the hypothesis suggesting the anatomical distribution of body fat is a stronger predictor of susceptibility to coronary heart disease mortality and morbidity, than measures of overweight or obesity, per se.

A variety of anthropometric indices have been used to describe the distribution of fat on the human body in relation to cardiovascular disease. Major prospective studies such as the Paris Prospective Study[22], Honolulu Heart Program[25], and Framingham Study[27] used skinfolds on the trunk and limbs to assess subcutaneous fat pattern. Others, including the Scandinavian studies[24,83], and recent work embracing coronary angiography[16,30,31,43] have relied on circumference measurements of the waist and hips to distinguish upper and lower trunk fatness. Waist and hip circumference measurements are generally used to compute a ratio, i.e. waist-to-hip circumference ratio. The underlying theory of the waist-to-hip circumference ratio, is that it discriminates between fat deposited in the upper (waist and abdomen areas) and lower trunk (hips and buttocks). As a predominance of fat in the upper trunk is primarily a masculine characteristic, and predominance in the lower trunk feminine, the terms android and gynoid obesity[40] are used to characterize these types of fat distribution. Later analysis of the Paris cohort also included the ratio of iliac-to-left thigh circumference, termed the circumference index, as an indicator of abdominal obesity[44].

Coronary heart disease in relation to subcutaneous fat pattern: prospective studies

Extending the period of follow-up to 30 years, Stokes *et al.* published further data from the Framingham Study[27]. They claimed the results not only reconfirmed earlier findings[36], but indicated that upper trunk (subscapular) and arm (triceps) skinfolds were better coronary heart disease predictors than skinfolds measured at the waist (abdominal) or front thigh. In the Paris Prospective Study, 6718 men aged 42 to 53 years were followed for an average of 6·6 years[22]. Coronary heart disease was classified as angina pectoris, non-fatal myocardial infarction or sudden death due to coronary heart disease. Trunk skinfolds (subscapular, axillary and subumbilicus) were the strongest predictors of coronary heart disease ($P<0{\cdot}05$), whereas thigh skinfolds (anterior, posterior, internal and external) were not associated with coronary heart disease ($P>0{\cdot}05$). The trunk to thigh skinfolds ratio was a highly significant predictor of angina pectoris ($P<0{\cdot}0001$) and to a lesser extent sudden death and myocardial infarction ($P<0{\cdot}01$). The association between the skinfold ratio and total incidence of coronary heart disease was also highly significant ($P<0{\cdot}00001$). In multivariate analysis, with cholesterol, cigarette habit, blood pressure, diabetes, age, body mass index and triglycerides as co-variables, the skinfold ratio remained a significant predictor ($P<0{\cdot}025$). A third large-scale prospective study, examined the relationship between definite coronary heart disease (non-fatal myocardial infarction and death from coronary heart disease) and subscapular skinfold thickness in 7692 men from the Honolulu Heart Program[25]. For a given body mass index, subscapular skinfold remained a significant

predictor of coronary heart disease after adjustment for several established risk factors ($P<0.05$ for the highest vs lowest tertile of subscapular skinfold and $P<0.01$ for the middle vs lowest tertiles). The study of Edinburgh men found baseline abdominal skinfold thickness was significantly greater ($P<0.05$) in the 11 men who developed coronary heart disease than the 96 men who remained free of the disease[26]. There was no difference in triceps and subscapular skinfold thicknesses ($P>0.05$). After adjustment for high density lipoprotein-cholesterol, abdominal skinfold thickness remained an independent predictor of coronary heart disease ($P<0.05$).

Evaluation of skinfold distribution as a predictor of coronary heart disease

Whilst these findings[22,25–27] exhibit some commonality, there are distinctive differences. Excluding the Edinburgh men[26], subscapular skinfold consistently appears as a stronger predictor of coronary heart disease than any other skinfold. However, it only accounted for approximately 10% (R^2) of the total variance across the entire age spectrum[27]. Examined in relation to specific age groups, subscapular skinfold presents as the strongest predictor in subjects less than 50 and 50–59 years of age ($R^2=15\%$ and 16% respectively) but the weakest predictors in subjects older than 60 years ($R^2=5\%$)[27]. In these older subjects, thigh skinfold showed the strongest association with the 22 year incidence of coronary heart disease ($R^2=15\%$). This contradicts the results of the Paris cohort for whom thigh skinfolds clearly exhibited the weakest relationship with coronary heart disease incidence[22]. The claim that triceps skinfold is generally a stronger coronary heart disease predictor than abdominal or thigh skinfolds[27] appears to be exaggerated. The results show that for each age stratum, coefficients of multiple logistic regression between triceps skinfold and coronary heart disease incidence are lower than those for both abdominal and thigh skinfolds. This may be suggestive of an alternative phenomenon. That is, it is truncal deposition of subcutaneous fat that is associated with increased coronary heart disease risk. This appears particularly apparent for younger subjects.

Coronary heart disease in relation to gynoid or android obesity: prospective studies

In Sweden, 13 years follow-up in men revealed significant associations between the waist-to-hip circumference ratio and the occurrence of stroke ($P=0.002$) and ischaemic heart disease ($P=0.04$) but not death ($P=0.053$)[24]. After the confounding effects of body mass index and the sum of three skinfolds were removed, waist-to-hip circumference ratio remained a long-term predictor of stroke and myocardial infarction and also correlated with death ($P<0.001$). Following adjustment for other major risk factors (smoking, systolic blood pressure and total cholesterol), the waist-to-hip circumference ratio was not a predictor of any of the end-points. Extension of the follow-up period by 5-years revealed that only 1·7% of men in the lowest 10% of the waist-to-hip circumference ratio distribution suffered cerebral infarction compared with 18·9% in the upper 10%[84]. Whilst the waist-to-hip circumference ratio was no longer an independent predictor of myocardial infarction, either in univariate or multivariate analysis, the risk of myocardial infarction was greater in the upper 10% compared to the lowest 10% of the waist-to-hip circumference ratio distribution. This difference, however, was markedly reduced at 72 years of age (after 18 years follow-up) compared to the maximal risk difference observed after 13 years. In the study of Parisian men, the ratio of iliac-to-left thigh circumference was a weak predictor of cardiovascular disease mortality in men with a lower mean blood pressure (<96 mmHg) but a stronger predictor in men with a higher mean blood pressure ($\geq$96 mmHg)[44].

Angiography studies

Hauner *et al.* examined the degree of coronary stenosis and several established risk factors in 286 men aged 30 to 74 years[30]. Coronary stenosis (>30%) or occlusion of one or more of the coronary arteries was present in 207 men. Those remaining were free of coronary heart disease and served as controls. There were no significant differences ($P>0.05$) between control and coronary heart disease subjects with respect to circumference measurements at the waist (midway between xiphoid process and umbilicus), umbilicus, or hips (level of greater trochanter). The waist-to-hip circumference ratio was also not significantly different ($P>0.05$). Stepwise logistic regression analysis revealed that in addition to low-density lipoprotein cholesterol ($P=0.0001$) and age ($P=0.0005$), an abdominal type fat distribution was a significant predictor ($P=0.0129$) of coronary heart disease. This association was independent of total cholesterol, high density lipoprotein-cholesterol, triglycerides, fasting insulin and systolic and diastolic blood pressures. A similar study found the waist-to-hip circumference ratio was related (Spearman's rank correlation) to both an extent ($r_s=0.18$, $P<0.05$) and myocardial score ($r_s=0.17$, $P<0.05$)[31]. After adjusting for several co-variables (age, body mass index, smoking habit, total cholesterol, low density lipoprotein-cholesterol, high density lipoprotein-cholesterol, apolipoprotein A1, apolipoprotein B and triglycerides) these relationships lost their significance ($r_s=0.17$ and 0·05 respectively, $P>0.05$). Ley and co-workers used dual energy X-ray absorptiometry, following procedures outlined by Mazess *et al.*[78] to evaluate android and gynoid fat in 77 men aged 31 to 60 years who presented with chest pain typical of angina pectoris[32]. Android fat was measured by selecting a region from the superior iliac crest upward to include all abdominal and thoracic soft tissue laterally. Gynoid fat was measured as a region of the same

length as the android fat region, from the lower sacral border downward to include all soft tissue laterally. Angiography revealed 39 men had greater than 50% luminal stenosis in one or more epicardial coronary arteries. The remainder had no detectable abnormality on their angiogram (<50% stenosis of any epicardial coronary artery). A further 40 men of similar age and weight and who were apparently asymptomatic were studied as a control group. Men with angina had a greater proportion of android fat than men without ($P<0.05$). Consequently, there was a trend towards a greater proportion of gynoid fat in asymptomatic men compared with men with angina but a normal angiogram ($P>0.05$), and men with angina and an abnormal angiogram ($P<0.05$). Thompson *et al.* found the waist-to-hip circumference ratio of patients with atherosclerosis was significantly greater than the waist-to-hip circumference ratio of subjects recruited from the same neighbourhood and matched for age, sex and race (0.96 ± 0.05 vs 0.92 ± 0.06, $P<0.025$)[43]. Flynn and her colleagues found the waist-to-thigh circumference ratio ($P<0.005$) and the waist-to-hip circumference ratio ($P<0.05$) were independently associated with coronary artery disease[16]. Whilst the waist-to-thigh circumference ratio was positively associated with coronary artery disease, in contrast to other prospective[24] and angiography studies[30,31,43] the waist-to-hip circumference ratio was inversely related to coronary artery disease.

Evaluation of anthropometric circumference measurements as predictors of coronary heart disease

Individuals characterized by an android fat distribution may represent a sub-group of obese individuals at increased risk of cardiovascular disease. This may partly explain the somewhat weak associations between cardiovascular disease and obesity per se[45]. Consequently, it has been suggested that as android obese individuals appear to be those at increased risk, the gynoid obese, whose risk of cardiovascular disease is elevated only slightly, should be considered to have a cosmetic rather than clinical problem[85]. Evidence from two prospective studies suggest anthropometric indicators of abdominal obesity are stronger predictors of coronary heart disease than body mass index[24,44]. After adjustment for three risk factors, however, the waist-to-hip circumference ratio lost its predictive power[24]. Furthermore, results from case-control studies with angiographically diagnosed coronary artery disease are not convincingly supportive of the ability of the waist-to-hip circumference ratio to predict coronary heart disease[16,30,31]. Indeed, some evidence is entirely conflicting[16]. This highlights the complex nature of the fat distribution relationship with coronary artery disease and supports the conclusion that more than one measure of obesity and fat distribution should be included in future research designs[86].

The waist-to-hip circumference ratio is an imprecise measurement as it includes several adipose tissue depots that anthropometric circumference measurements cannot separate. The waist circumference includes subcutaneous abdominal and intra-abdominal adipose tissue. Adipose stores contained within the hip circumference are the intermuscular, intramuscular and subcutaneous layer. This limiting feature of the waist-to-hip circumference ratio is highlighted by the results of several investigations that have used either computed tomography or magnetic resonance imaging to determine fat deposition in the thorax and abdomen. Fujioka *et al.* demonstrated how two individuals with an almost identical body mass index can differ substantially in intra-abdominal fat volume[87]. Seidell *et al.* reported a significant relationship between intra-abdominal fat area measured by computed tomography and the waist-to-hip circumference ratio in 71 men ($r=0.75$, $P<0.001$)[88]. Kvist *et al.* and Sjostrom and Kvist found a relationship of similar magnitude between visceral adipose tissue volume and the waist-to-hip circumference ratio in a group of 24 men ($r=0.77$, $P<0.001$)[89,90]. Pouliot *et al.* reported a correlation of 0.71 ($P<0.0001$) between the waist-to-hip circumference ratio and visceral adipose tissue area in 81 men[91]. Ross and co-workers have recently reported a significant correlation between the waist-to-hip circumference ratio and visceral adipose tissue volume measured by magnetic resonance imaging in 27 men ($r=0.85$, $P<0.001$)[92]. The relationship between the waist-to-hip circumference ratio and abdominal subcutaneous adipose tissue measured by computed tomography ($r=0.68$, $P<0.001$)[88,91] and magnetic resonance imaging ($r=0.61$, $P<0.05$)[92] is weaker. Thus, despite being highly significant, approximately 30% to 50% of the variation in the relationship between the waist-to-hip circumference ratio and intra-abdominal adipose tissue remains unexplained. Computed tomography and magnetic resonance imaging therefore, remain the only methods available for the measurement of intra-abdominal fat mass, although there are a few less satisfactory alternatives for quantifying either total abdominal fat (dual photon absorptiometry and dual energy X-ray absorptiometry) or intra-abdominal depth (ultrasound)[93]. Anthropometric measurements will remain valuable research tools for epidemiological surveys, especially if valid regression equations for the prediction of intra-abdominal fat can be developed[93]. High costs and exposure to ionizing radiation limits the large-scale use of computed tomography. Magnetic resonance imaging however, whilst still involving a high financial cost, provides detailed anatomical images in vivo, without the risks of computed tomography and offers an exciting development with regard to intra-abdominal fat assessment. As this depot has been closely linked with metabolic and physiological disturbances associated with cardiovascular disease (for reviews see[74,86,94]. considerable significance should be attached to measuring this depot when evaluating cardiovascular disease risk.

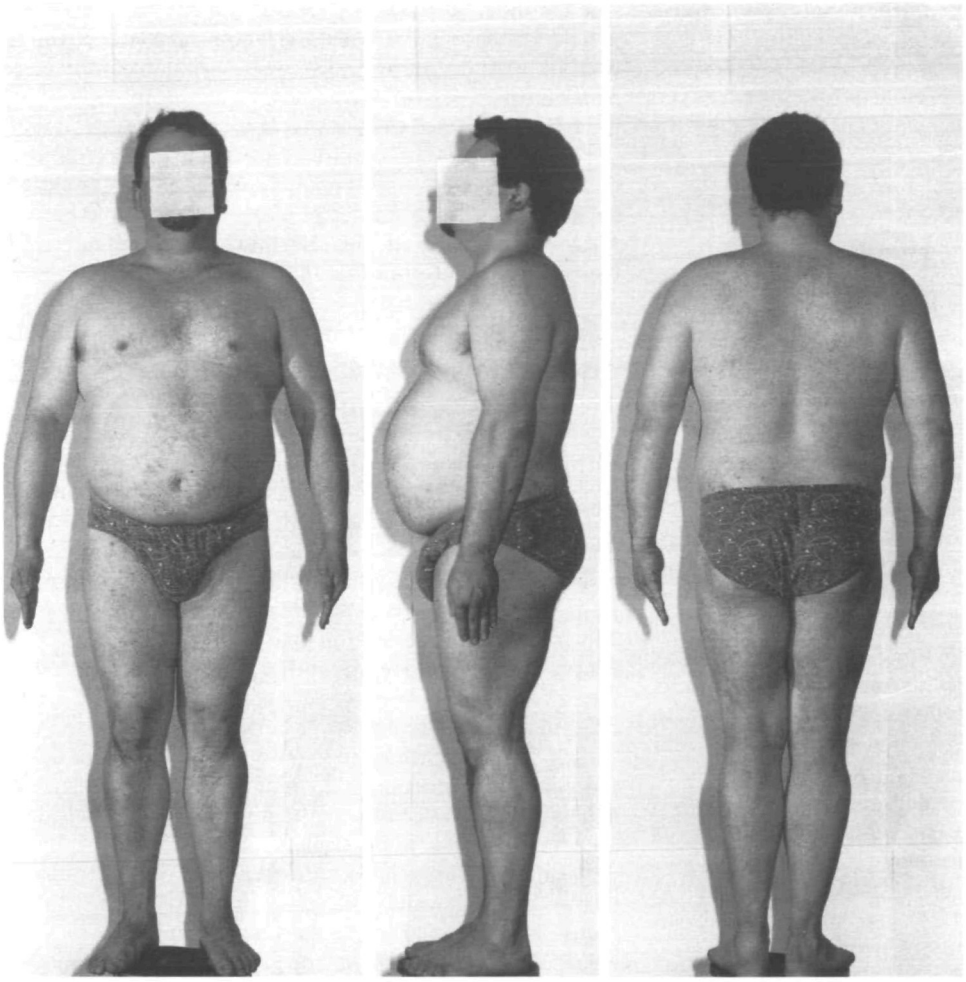

Figure 1 **Standard somatotype photograph of an abdominally obese, 30-year-old male who exhibits extreme levels of both endomorphy and mesomorphy but minimal ectomorphy. This subjects body mass index, waist-to-hip circumference ratio and somatotype were 33·4, 1·06 and 7–7·5–0·5 respectively.**

Somatotype

Classification of somatotype

The somatotype is a classification of human physique based on the concept of body shape independent of size[95]. In the somatotype, body shape is expressed as a series of three numbers each representing a particular component. These are always recorded together and in the same order. The first figure represents a rating of endomorphy, the second mesomorphy, and the third ectomorphy[95]. Dominant in the early development of somatotype methodology was the work of Sheldon *et al.*[52], in which ratings began at zero and had a fixed upper point of seven. More recently, the method developed originally by Heath and Carter[96] has predominated. This method uses much of Sheldon's original vocabulary although some of the fundamental ideas have been revised. A detailed description of this method has been provided recently[95]. Briefly, Heath–Carter somatotype classifications can be obtained either by inspection of a standard somatotype photograph, from a series of anthropometric measurements, or preferably, from a combination of photoscopic inspection and anthropometric measurements[95]. A physique attributed a high endomorphy rating is characterized by a large subcutaneous fat deposit, or noticeable relative fatness. High ratings in mesomorphy signify a large musculature and bone mass relative to stature. High ratings in ectomorphy describe a physique with little mass relative to stature and relatively elongated limb segments[95]. Component ratings still begin theoretically at zero but have no fixed upper-end points. In general, component ratings of 0·5 to 2·5 are regarded as low, 3 to 5 as midrange, 5·5 to 7 as high and greater than 7 extremely high. Thus, the classification 7–1–1 represents an extreme endomorph, 1–7–1 represents an extreme mesomorph and 1–1–7 an extreme ectomorph. A 3–3–3 or 4–4–4 classification represents a central or balanced somatotype, 4–5–1 an endomorphic–mesomorph and 2–3–5 a mesomorphic–ectomorph. Extreme examples for each of these components would be an obese

individual (endomorphy), a body-builder (mesomorphy) and the Nilote people of Sudan who exhibit extreme ectomorphy[95]. Figure 1 shows front, left-side and rear views of a 30-year-old male whose somatotype (7–7·5–0·5) we have classified with Heath and Carter's method[95].

Coronary heart disease in relation to somatotype

The relationship between somatotype and coronary heart disease attracted attention in the United States in the 1950s and 1960s[4,5,97–100] and later in South Africa[101].

Of 97 men and three women who experienced a non-fatal myocardial infarction before 40 years of age, 42% were found to be dominant mesomorphs, 26% dominant endomorphs, 25% were in the mid-range (no dominant component) and only 7% were dominant ectomorphs[4,5]. In 1953, the first of three papers examining the somatotype–coronary heart disease relationship was published[97]. This reported the autopsy findings on 111 consecutive white males under 46 years of age. Of these, 38 had suffered death secondary to coronary artery disease and 73 had died suddenly and unexpectedly by violent means (suicide, homicide, accident) or some other non-cardiac condition. Of the 38 who died from coronary disease, 24 were classified as being dominant mesomorphs, three endomorphs, three ectomorphs and eight were in the mid-range. In the 73 apparently healthy males, the degree of atherosclerosis was found to be distinctly more pronounced in mesomorphic individuals compared to those of ectomorphic dominance. A second post-mortem study also revealed that the extent of coronary atherosclerosis was markedly greater in mesomorphic compared to ectomorphic individuals[98]. Of 64 consecutive autopsy examinations involving sudden death from coronary occlusion, 44 cases were classified as dominant mesomorphs. In a third study, the incidence of coronary heart disease amongst 5000 males aged 36–50 years was three times greater for endomorphic–mesomorphs (9·2%) compared to dominant ectomorphs (3·0%)[99]. This further evidence led to the conclusion that individuals characterized by mesomorphic dominance, were at greater risk of coronary heart disease than their ectomorphic counterparts[99]. This was attributed to the mesomorphs' large relative muscle mass, which was hypothesized to have a more direct association with atherosclerotic coronary heart disease than adipose tissue[99]. The examination of 87 men aged 40 to 55 years, failed to support these earlier findings[100]. It was reported that endomorphic dominance was important since there was an excess of coronary cases in the group characterized by endomorphy ($P<0·01$). Further examination, however, show that whilst the difference between observed to expected coronary cases (myocardial infarction, angina pectoris, death from coronary heart disease) was greatest in the endomorphic sub-sample (19 observed/13 expected), the total number of cases was greatest in the mesomorphic group. In these individuals, for whom mesomorphy was dominant and endomorphy greater than ectomorphy, 37 confirmed cases were found, one more than may have been expected. There was also a significant number of cases in the group for whom mesomorphy and endomorphy were approximately equal (15 observed/11 expected). Of further interest is the lower than expected number of cases in the ectomorphic dominant and mesomorphic-ectomorphic individuals.

Evaluation of somatotype classification as a predictor of coronary heart disease

These early studies[4,5,97–100] can be criticized on several grounds. Most notably, the subjectivity of the photoscopic somatotype technique[52], the lack of statistical analysis or control of covariables and failure to recognise the somatotype as a Gestalt. Despite these limitations, the findings were later confirmed in a study of 146 cardiac rehabilitation patients (mean age=52·7 years)[101]. Using Heath and Carter's technique[95] a mean somatotype of 4–5·5–1 was reported, the majority of patients being endomorphic–mesomorphs.

The overwhelming number of cardiac cases amongst mesomorphic individuals necessitates further explanation. Predominant mesomorphs show considerable variation in body density, hence mesomorphy is only modestly associated with measures of pure muscularity[102]. An equally plausible interpretation is that many large-framed muscular older males also have enlarged fat stores[102]. As body fat distribution appears to be particularly important in the relationship between body habitus and cardiovascular disease, the association between somatotype and fat distribution is of great interest and may help explain the abundance of coronary heart disease amongst mesomorphic individuals. Among 824 men, those classified as android obese (mean somatotype 4·67–4·21–1·89) were reported to be significantly more mesomorphic and less endomorphic than those with gynoid obesity (mean somatotype 5·91–2·16–1·84) ($P<0·01$)[103]. Mesomorphy is also a masculine characteristic, and as reported for non-insulin-dependent diabetes mellitus, there appears to be an assemblage of male differentiation factors amongst individuals at increased risk of coronary heart disease[103].

Summary

Table 1 is a synopsis of the major findings from an extensive literature on the association between human body habitus and coronary heart disease. Whilst some studies have used quite sophisticated laboratory procedures to quantify body fat[19,32,38] most have relied upon anthropometric measurements to determine some component of body habitus. Of these, body weight and height are the simplest measurements and are, therefore,

Table 1 Synopsis of major findings from studies that examined the relationship between a variety of physical characteristics and coronary heart disease in men

Reference and study design	Physical characteristic(s)	Findings
Gertler *et al.*[4] case-control study of 100 post MI patients and 146 controls	weight, height, somatotype	CHD patients were on average 5 cm shorter and 3 kg heavier than controls. The CHD group were predominantly mesomorphs (42%) with only 7% dominant in ectomorphy.
Paffenbarger *et al.*[6] prospective study of 40 000 students (25 year follow-up)	weight, height, PI	Body weight of coronary decedents was greater than controls at time of initial examination. More coronary decedents were shorter than 68 inches tall. 10% of coronary decedents had a PI of <12·5.
Paffenbarger *et al.*[7] prospective study of 42 000 students (30 year follow-up)	weight, height, PI	As above for body weight and height 10% of controls and 17% of coronary decedents had a PI of <12·5.
Morris *et al.*[8] prospective study of 687 busmen (5 year follow-up)	height, subcutaneous fat pattern	Non-significant trends for increased incidence of IHD amongst shortest men and by increasing fatness, particularly suprailiac skinfold.
Marmot *et al.*[9,10] prospective study of 17 530 civil servants (7 5 year follow-up)	height, BMI	Inverse association between height and CHD independent of age and employment grade.
Morris *et al.*[11] prospective study of 17 944 office-workers (8·5 year follow-up)	height, BMI	Inverse trend between height and CHD. Positive trend between BMI and CHD.
Waaler[12] prospective study of 900 000 men (16 year follow-up)	height, BMI	Clear inverse trend between height and CVD.
Walker *et al.*[13] prospective study of 7735 men (7·5 year follow-up)	height	Risk of MI was approximately twice as great in the shortest quitile compared to the tallest. Adjustment for several risk factors caused the relationship between height and MI to disappear.
Yarnell *et al.*[14] prospective study of 4860 men (38 and 61 months follow-up)	height	Men in the shortest fifth of the height distribution experienced twice the IHD events compared to men in the tallest fifth. The trend across the distribution remained significant after adjustment for several co-variables.
Rabkin *et al.*[15] prospective study of 3983 airline pilots (26 year follow-up)	weight, height, BMI	Body weight and BMI were greater and height shorter in those who developed CHD than those who did not. After adjustment for age and BP, BMI was an independent predictor of CHD.
Flynn *et al.*[16] case-control (angiography) study of 186 men, 72 with insignificant CAD and 114 with significant CAD	weight, height, W:H indices (i, ii, iii, iv), various circumferences, relative BF (v), subcutaneous fat pattern, fat distribution	Significant differences between the groups for the variables; height, CHR, ATI, WTR and relative BF. Height and WHR were inverse independent predictors of CAD and WTR a positive independent predictor.
Dyer *et al.*[17] prospective study of 1233 men (14 year follow-up)	BMI, MRW	BMI was found to have a significant quadratic relationship to CHD.
Rhoads and Kagan[18] prospective study of 8006 men (10 year follow-up)	BMI	Age adjusted mortality from CHD was related to BMI at both time of examination and at 25 years of age.
Keys *et al.*[19] prospective study of 279 men (20 year follow-up)	height, BMI, MRW, body density (vi), sum of skinfolds	No association between any of the physical characteristics and CHD.
Keys *et al.*[20] prospective study of 11 400 men (5 year follow-up)	BMI, MRW, sum of skinfolds	No association between any of the physical characteristics and CHD.
Keys *et al.*[21] prospective study of 11 579 men (15 year follow-up)	BMI	No association between BMI and CHD.
Ducimetiere *et al.*[22] prospective study of 6718 men (6·6 year follow-up)	BMI, subcutaneous fat pattern	Truncal fat deposition was a significant predictor of CHD after adjusting for BMI and other risk factors.
Carlson and Bottiger[23] prospective study of 3168 men (9 year follow-up)	W:H	No association between W:H and IHD.
Larsson *et al.*[24] prospective study of 792 men (13 year follow-up)	BMI, sum of skinfolds, waist and hip circumferences, WHR	WHR of stroke and IHD cases was greater than non-cases. WHR was associated with stroke, IHD and death after adjustment for sum of skinfolds but not when other risk factors considered.

Table 1 continued on next page

Table 1 Continued from previous page

Reference and study design	Physical characteristic(s)	Findings
Donahue *et al.*[25] prospective study of 7692 men (12 year follow-up)	BMI, subscapular skinfold	Subscapular skinfold was an independent predictor of CHD after adjustment for BMI and several other co-variables.
Hargreaves *et al.*[26] prospective study of 107 randomly chosen men (12 year follow-up)	BMI, subcutaneous fat pattern, WHR	Baseline BMI and abdominal skinfold measurements were greater in subjects who subsequently developed CHD.
Stokes *et al.*[27] prospective study of 1834 men (22 year follow-up)	BMI, subcutaneous fat pattern, various circumferences.	Subscapular and abdominal skinfolds, waist circumference and BMI were all independent predictors of CHD. Subscapular skinfold was the strongest predictor in men <60 years old and abdominal skinfold the strongest in older men (60+ years).
Reed and Yano[28] prospective study of 7591 men and case-control (angiography) study of 357 men after 20 year follow-up	BMI	BMI was a significant independent predictor of MI and coronary stenosis following cohort analysis but not with stenosis in the angiography series.
Jooste *et al.*[29] prospective study 3357 men (10 year follow-up)	BMI	In the BMI groups 30–35 and >35 kg m^{-2}, CHD was present in approximately 34% of men aged 45–64 years. This was greater than the BMI group 20·0–24·9 kg . m^{-2}.
Hauner *et al.*[30] case-control study of 112 men with CAD+MI. 95 men with CAD and 79 controls (CAD diagnosed by angiography)	weight, height, BMI, various circumferences, WHR	Body weight was lower in the CAD+MI men compared to controls. The CAD+MI and CAD men were shorter than controls. WHR was an independent predictor of CAD.
Hodgson *et al.*[31] case-control (angiography) study of 159 men	BMI, WHR	No association between BMI or WHR with CAD.
Ley *et al.*[32] case-control (angiography) study of 77 non-obese men with anginal chest pain	weight, height BMI, absolute and relative body fat by DXA android and gynoid fat by DXA	Men with AP and abnormal angiogram had lower absolute and relative gynoid fat and greater relative android fat than men with AP and normal angiogram and controls. Men with AP and normal angiogram had a greater proportion of android fat than controls.
Kannel *et al.*[33]prospective study of 5127 men (12 year follow-up)	FRW	Antecedent FRW and weight gain after age 25 were related to AP and sudden death but not MI.
Kannel and Gordon[34] prospective study (18 year follow-up)	FRW	Linear association between FRW and CHD.
Feinleib *et al.*[35] prospective study (1, 5 and 9 years follow-up) and postmortem examination of 71 decedents	weight, height, FRW	Body weight 1 and 9 years before death was an independent predictor of LV thickness. Five years before death, body weight was positively and height inversely related to heart weight. FRW was an independent predictor of heart weight.
Hubert *et al.*[36] prospective study of 2252 men (26 year follow-up)	MRW	MRW was an independent predictor of CHD, AP, CHD other than AP, sudden death from CHD, CHF, total CVD and death from CHD.
Drenick *et al.*[37] prospective study of 200 overweight men (7·5 year follow-up)	MRW	A 12-fold excess risk of CVD amongst men aged 25–34 years and a 6-fold excess risk for men aged 35–44 years.
Weinsier *et al.*[38] case-control study of 1483 male airforce personnel	BMI, PI, W:H, relative body fat (vii)	No relationship between any of the physical characteristics and CHD.
Tumomilehto *et al.*[41] prospective study of 3786 men (7 year follow-up)	BMI	Significant association between BMI and CHD independent of age and smoking but not serum cholesterol and BP.
Pooling Project[42] combined data from several major prospective studies	MRW	MRW associated with an increased risk of a first coronary event in men aged 40–45 and 45–49 years.
Thompson *et al.*[43] case-control (angiography) study of 100 cases and 118 controls	BMI, WHR	No differences in BMI. WHR of coronary cases was greater than controls.

Table 1 continued on next page

Table 1 Continued from previous page

Reference and study design	Physical characteristic(s)	Findings
Filipovsky *et al.*[44] prospective study of 7312 men (15–20 year follow-up)	BMI, CI	BMI and CI were poor predictors of CVD mortality in men with lower mean BP. CI was a stronger predictor in men with higher BP.
Spain *et al.*[97] case-control (postmortem) study of 38 men with CAD and 73 men who died from non-cardiac causes	somatotype	24 of the 38 men who died from CAD were dominant mesomorphs. In the non-cardiac cases, the degree of atherosclerosis was greater in the dominant mesomorphs than the dominant ectomorphs.
Spain *et al.*[98] descriptive postmortem study of 157 individuals who died suddenly by violent means or from coronary occlusion	somatotype	Of the 64 cases of sudden death from coronary occlusion 44 were dominant mesomorphs.
Spain *et al.*[99] prospective study of 5000 men (5 year follow-up)	MRW, somatotype	After adjustment for diabetes and hypertension, MRW was not associated with CAD incidence. A greater incidence of CAD amongst endo-mesomorphs than ectomorphs.
Paul *et al.*[106] prospective study of 1989 men (4 year follow-up)	weight, height, subcutaneous fat pattern, somatotype	Triceps and subscapular skinfolds were greater in subjects who developed CHD. Height and weight were not different. A greater number of CHD cases amongst those of endomorphic and mesomorphic dominance.
Smit *et al.*[101] descriptive study of 146 patients in cardiac rehabilitation	somatotype	Majority of patients were endomorphic — mesomorphs.

(i) BMI, (ii) risk index of body build, (iii) adipose tissue index, (iv) body fat index, (v) by Durnin and Womersley's sum of 4 skinfolds, (vi) by underwater weighing, (vii) by tritium dilution.
W=body weight; H=height; PI=ponderal index; BF=body fat; CHD=coronary heart disease; IHD=ischaemic heart disease; BMI=body mass index; MI=myocardial infarction; FRW=Framingham relative weight; AP=angina pectoris; BP=blood pressure; LV=left ventricular; CAD=coronary artery disease; WHR=waist-to-hip ratio; CHR=chest-to-height ratio; ATI=adipose tissue index; WTR=waist-to-thigh ratio; CVD=cardiovascular disease; MRW=Metropolitan relative weight; CHF=congestive heart failure; DXA=dual energy X-ray absorptiometry: CI=circumference index (ratio of iliac to left thigh circumference).

well-suited to large-scale prospective studies. Height and weight are highly reproducible measurements, although in the short term, weight can have considerable physiological variation associated with gastric emptying and state of hydration[104]. Less reliable measurements than height and weight are skinfolds and body circumferences, both of which have been used extensively in cross-sectional and prospective analyses. For skinfolds, both the inter and intra-observer variability is affected by the measurement technique, location of the skinfold site, the skinfold caliper used and skinfold compressibility[56]. As measurement error has been shown to be a function of skinfold thickness[105], accurate and repeatable skinfold measurements are particularly difficult to make in the obese. In these subjects, it is not always possible to locate a specific anatomical bony landmark or to pull a parallel skinfold away from the underlying tissue. Furthermore, in the extremely obese it is sometimes possible for a skinfold to be thicker than the jaws of the currently available commercial calipers[106]. Alternately, body circumferences are obtainable in all subjects and have greater reproducibility than skinfolds[106]. They are, therefore, the preferred method in obese subjects[106]. However, there is considerable work to be done to establish their association with body fatness.

The evidence examined in this review suggests that body weight is a poor predictor of coronary heart disease. Some studies have reported no difference in the body weight of coronary heart disease patients compared to subjects free of the disease[16,32,100], others found the body weight of subjects with coronary heart disease to be slightly greater[4,6,7,15,35], and one found the body weight of cardiac patients to be less than controls[30]. Height, however, is associated with coronary heart disease in prospective studies with long-term[6–13,15] and shorter-term[14] follow-up periods and case-control designs[4,5,16,30]. Fetal, infant and childhood under-nutrition may link shorter adult height and susceptibility to cardiovascular disease[48].

Many researchers have studied the relationship between overweight and coronary heart disease by using a surrogate measurement of body fatness such as relative weight or a weight-for-height index. In general, results produced by these studies suggest weight-for-height indices, particularly the often used body mass index, are not strong predictors of coronary heart disease. Indeed case-control designs have consistently failed to show a relationship between body mass index and coronary heart disease. Inconsistent results from prospective studies, however, are difficult to interpret. To further confuse the situation, the body mass index has been

examined in relation to different coronary heart disease end-points and adjusted for different confounding variables. Explaining the inconsistent results on the basis of length of follow-up is also not straightforward. When follow-up periods exceed 20 years[15,27], and sample size is large, the body mass index exhibits a stronger relationship with coronary heart disease. When sample size is small, however, this closer association has not been found, even with a long follow-up period[19]. Whilst some studies have found no association after 15, 13 and 12 years[21,24,25] others have reported a relationship after 8·5, 10, 12, 10 and 7 years[11,18,26,29,41]. The 22 year follow-up evidence from the Framingham Study shows the strongest 'independent' association between body mass index and coronary heart disease[27]. Despite adjusting for several established risk factors (age, total cholesterol, systolic blood pressure, cigarette smoking, blood glucose and ECG evidence of left ventricular hypertropy), the 'true' relationship between body mass index and coronary heart disease awaits more extensive adjustment for factors associated with the overweight state.

As body mass index has been shown to have only a moderate correlation with body fatness[38,46,61–63], future research should establish whether body mass index is a valid predictor of body fat in that particular population before the term obesity is adopted. Ideally, the power function of height should be calculated so that the index exhibits the strongest possible relationship to body fatness and is independent of height. Unless this prior validation is performed, the term overweight is preferable.

Relative weight, a further simple index of overweight based on height and weight alone, has been used less extensively than body mass index. Data from the Framingham Study suggests relative weight can predict coronary heart disease in the short[33,34] and long term[36]. However, there is contradictory evidence from studies, with follow-up periods ranging from 5 to 20 years[19–21]. The principal limiting feature underpinning measures of relative weight is the same as for body mass index, i.e. an inability to reflect adiposity.

It has been suggested that the failure of weight-for-height indices and relative weight to reflect adiposity may partly account for the inconsistency in the relationship between 'obesity' and cardiovascular disease[74]. If this is so, then the more 'direct' measurement of body fat should theoretically produce a closer association between obesity and cardiovascular disease. However, studies reviewed here suggest that this is not the case. Neither prospective[19–21,24] or case-control[16,32,38] studies that assessed body fat by more direct methods have shown a relationship between the level of fatness and cardiovascular disease. This is not to say that obesity is unimportant in the pathogenesis of cardiovascular disease. Studies of obese and overweight men have shown a relationship between fat loss and weight reduction and improvements in blood pressure and blood lipids[107–112]. Thus, despite the lack of an independent statistical association between obesity and cardiovascular disease, the avoidance of obesity or the loss of excess fat with subsequent maintenance of the lower level should be an important aspect of cardiovascular disease risk reduction[113].

As the combination of various skinfolds seem unrelated to coronary heart disease when summed[19–21,24] or used to estimate relative body fat[16], it is perhaps surprising that several studies have found that individual skinfolds treated as discrete variables independently predict coronary heart disease. It appears that central or truncal skinfolds are stronger predictors than limb or peripheral skinfolds. However, which truncal skinfold is the strongest predictor remains unclear. For instance, one study found subscapular skinfold to be a better predictor than abdominal skinfold[27]. The study of Edinburgh men, however, found that baseline abdominal skinfold was significantly greater in men who developed coronary heart disease compared to those who did not[26]. There was no difference in subscapular skinfold thickness.

Results from two prospective studies[24,44] suggest that abdominal obesity, as measured by the waist-to-hip circumference ratio and ratio of iliac-to-left thigh circumference respectively, is important in the evaluation of cardiovascular disease risk. However, more recent findings from case-control studies[16,30,31] indicate that the waist-to-hip circumference ratio is not closely associated with coronary artery disease. This may well be due to the fact that the waist-to-hip circumference ratio exhibits only a moderate relationship with intra-abdominal fat. It is this fat compartment, particularly the metabolically unique omental and mesenteric adipose tissues that drain into the portal circulation[114], that have been linked with the metabolic complications associated with cardiovascular disease[74,86,94]. As magnetic resonance imaging allows repeated scanning without exposure to radiation, the long-term study of cardiovascular disease in relation to intra-abdominal fat volume may now be feasible. Financial constraints, however, are likely to limit this possibility.

The studies that examined coronary heart disease in relation to somatotype revealed very similar findings. Men with an endomorphic–mesomorphic physique appear to experience a far greater incidence of coronary events than other somatotypes. Ectomorphic dominance appears to be the somatotype least associated with coronary heart disease. Although somatotyping does not allow the quantitative assessment of body composition compartments, it could provide an alternative method for the early identification of the individual at risk of coronary heart disease. The somatotype could be particularly useful when used in conjunction with other well established risk factors. The latest methodological advances allows somatotype classification to be made objectively and relatively easily[95]. Reliability of the classification depends entirely on the reliability with which several anthropometric measurements can be made[95].

It is clear from the literature examined in this review that a wide variety of bodily physical characteristics have been studied in relation to the incidence of coronary heart disease. These characteristics have ranged

from the most basic and easily quantified to the profoundly more complex. Results suggest that it is not necessarily the more complex that are most closely associated with coronary heart disease. The variability in findings suggest that future research in this area should include a wide variety of measurements in order to identify the strongest predictor for that particular population.

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