CORONARY HEART DISEASE RISK FACTORS IN SCHOOLCHILDREN AGED 12 TO 13 YEARS OF DIFFERING SOCIO-ECONOMIC STATUS

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A submission presented in partial fulfilment of the requirements of the University of Glamorgan/Prifysgol Morgannwg for the degree of Doctor of Philosophy

This research programme was carried out in collaboration with the Biochemistry Departments of The Royal Glamorgan Hospital, Llantrisant, South Wales, and Llandough Hospital, Penarth, South Wales.

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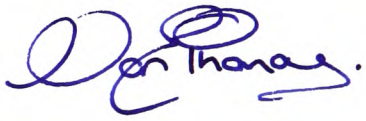
CERTIFICATE OF RESEARCH

This is to certify that the work described in this thesis is the result of my own work. This research programme was carried out in collaboration with the Biochemistry Departments of The Royal Glamorgan Hospital, Llantrisant, South Wales, and Llandough Hospital, Penarth, South Wales.

Neither this thesis, nor any part of it, has been presented, or is currently submitted, in candidature for any degree at any other university.

Professor Bruce Davies

(Director of Studies)



Non Eleri Thomas

(Candidate)

Dedicated to my family whose love and generosity will never cease to warm my heart.

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TABLE OF CONTENTS

LIST OF TABLES

WORK SUBMITTED FOR PUBLICATION FROM THIS THESIS

RECENT SUBMISSIONS

Thomas, N.E., Baker, J.S. and Davies, B. (2003) Established and recently identified coronary heart disease risk factors in young people. The influence of physical activity and physical fitness. Review Article. *Sports Medicine*. Vol. 33, No. 9.

ABBREVIATIONS

CHD	Coronary heart disease
SES	Socio-economic status
BP	Blood pressure
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
BMI	Body mass index
WHR	Waist to hip ratio
$\sum$ SKF	Summation of the thickness of skinfolds measured at four sites (subscapular, suprailiac, biceps and triceps)
FIT	Aerobic fitness
20 MST	20 metre multistage fitness test
PA	Physical activity
Tot fat	Total fat
Sat fat	Saturated fat
TC	Total cholesterol
HDL-C	High density lipoprotein-cholesterol
LDL-C	Low density lipoprotein-cholesterol
TC:HDL-C	Ratio of total cholesterol to high density lipoprotein-cholesterol
TG	Triglycerides
Glu	Glucose
Lp(a)	Lipoprotein(a)
Hcy	Homocyst(e)ine
Fg	Fibrinogen

B_{12}	Vitamin B_{12}
SST	Serum separating tubes
EDTA	Chelating agent - ethylene diamine tetra-acetic (ethanoic) acid
ANOVA	One way analysis of variance
ß	Beta weight
CI	Confidence intervals
df	Degrees of freedom
ES	Effect size
LoA	Limits of agreement
MRA	Multiple regression analysis
$P \leq 0.05$	Level of statistical significance $\alpha = 5\%$
PCA	Principal component factor analysis
r	Correlation coefficient
***t* - test**	Student *t*-test for two independent means

ABSTRACT

From November 2001 through to March 2002, 100 boys and 108 girls, aged 12.0 ± 0.8 years were assessed for coronary heart disease (CHD) risk factors. Rural (school 1) and valley (school 2) comprehensive schools (age range 11 to 18 years) were selected to represent differing socio-economic status (SES). Informed written consent was sought from head teachers, parents, and schoolchildren. All measurements were made in the subjects' respective schools, with the author making a total of 60 visits to these schools. Tests were carried out at the same relative time intervals and by an identical team of researchers. Physical and physiological measurements included stature (m), body mass (kg), body mass index (BMI) (kg m^{-2}), skinfold measurements ($\sum$ SKF) (mm), waist to hip ratio (WHR), aerobic fitness (20 MST) (shuttles), systolic (SBP) (mm Hg) and diastolic blood pressure (DBP) (mm Hg). Total cholesterol (TC) ($mmol \cdot L^{-1}$), high density lipoprotein-cholesterol (HDL-C) ($mmol \cdot L^{-1}$), low density lipoprotein-cholesterol (LDL-C) ($mmol \cdot L^{-1}$), TC: HDL-C, triglyceride (TG) ($mmol \cdot L^{-1}$), lipoprotein(a) (Lp(a)) ($mg \cdot dL^{-1}$), glucose (Glu) ($mmol \cdot L^{-1}$), fibrinogen (Fg) ($mg \cdot dL^{-1}$), folate ($ng \cdot mL^{-1}$), vitamin B_{12} ($pmol \cdot L^{-1}$), and homocyst(e)ine (Hcy) ($\mu mol \cdot L^{-1}$) were measured for haematological concentrations. Lifestyle assessments included dietary and physical activity analysis. Familial risk of coronary heart disease was also determined. Blood analyses were conducted at the Biochemistry Laboratories of the Royal Glamorgan Hospital, Llantrisant, and Llandough Hospital, Penarth.

For all CHD risk factors, one way analysis of variance (ANOVA) revealed relatively few significant differences ($P \leq 0.05$) according to SES (school 1 vs. school 2). Statistically significant differences ($P \leq 0.05$) were however detected according to SES, in SBP (boys only), TC (boys only), LDL-C (boys only), Fg (boys only), and folate (boys and girls). With regards to sex, significant differences ($P \leq 0.05$) were identified in SBP, DBP, aerobic fitness, WHR, $\sum$ SKF, average daily calorie intake, TC, LDL-C, Fg and folate concentration. A correlation coefficient matrix identified a number of significant relationships ($P \leq 0.05$) amongst CHD risk factors.

Clustering of CHD risk factors was evident in this cohort of schoolchildren. More than one risk factor was exhibited in 83.9% of schoolchildren in school 1, and 89.5% of schoolchildren in school 2. Three or more risk factors were exhibited in 66.6% of schoolchildren. Nine CHD risk factors were identified in two individuals and only two pupils were exempt from any risk factor. Principal components factor analysis (PCA) indicated significant clustering of lipids and lipoproteins; homocyst(e)ine, folate and vitamin B_{12}; and, total and saturated fat.

Multiple regression analysis (MRA) revealed that BMI was the strongest predictor of blood pressure. The primary mediator of SBP was central fat, while peripheral fat was the main contributor to DBP. The ratio of total cholesterol to HDL-C, had the greatest effect on all blood lipids and lipoproteins. Body mass index explained the largest proportion of variation in Fg; and as expected, the main predictors of homocyst(e)ine were vitamin B_{12} and folate. The primary mediator of performance in the 20 MST was the summation of four skinfold sites.

This study of Welsh schoolchildren of differing socio-economic status, reveals a high incidence of CHD risk factors. Moreover, it also provides evidence of the development of multiple risk factors from a young age. If the findings of the present study are to be heeded, the primary prevention of coronary heart disease needs to be directed towards young people, irrespective of socio-economic status and sex. These findings should act as a caveat against the dangers of ignoring evidence that the origins of CHD have often begun in youth.

CHAPTER 1

1.0 INTRODUCTION

Current research indicates an epidemic of lifestyle-related chronic diseases such as coronary heart disease (CHD). Coronary heart disease refers to heart disorders arising from disease of the coronary arteries and is one of the most serious eventualities of atherosclerosis (Cunnane, 1993). The precipitating defect in most CHD sufferers is the combined effect of atherosclerosis, hypertension, type 2 diabetes, hyperinsulinemia, obesity and dyslipidemia (Booth, Gordon, Carlson *et al.* 2000). Atherosclerosis itself is a lifelong process, which involves the gradual deterioration of the arteries (Herrick, 1912; Cunnane, 1993; McGill, McMahan, Malcom *et al.* 1997). The pathological process of atherosclerosis has a sustained pre-clinical period that often begins during childhood and becomes manifest in adulthood as CHD (McGill, Geer and Strong, 1963; McGill *et al.* 1997; McGill, McMahan, Herderick *et al.* 2000[a]; McGill, McMahan, Zieske *et al.* 2000[b]). Coronary heart disease has been a major health concern for many years but according to recent evidence there has been an encouraging decline amongst all social denominations over the past two decades (McMenemy, 1999; Shaper, 2001). However, despite most Northern and Western European countries experiencing a fall in CHD related deaths, the disease continues to be a major cause of demise in Western society. According to The Global Burden of Disease Study, coronary heart disease is the most important cause of years of life lost in established market economies (Murray and Lopez, 1996).

A recent report by the British Heart Foundation (British Heart Foundation, 2000) revealed that British men and women were at higher risk of CHD than people living in

many other European countries. The same report identified the disease as being the most common cause of death in Europe and the European Union; accounting for about 2 million deaths per annum in the former, and 600,000 in the latter. In the UK one in four males and one in five females die from CHD each year, and in 1998 at least 170,000 individuals died from the disease (British Heart Foundation, 1998). In Wales, CHD remains a major cause of ill health, and in 1998 the disease caused 7,500 deaths in the principality, the highest incidence of CHD mortality occurring in the South Wales valleys (Hutt, 2001).

Research efforts in CHD tend to concentrate on its secondary and tertiary prevention. Despite the undeniable importance of continued advances in the treatment of CHD, preventing the disease is more humane and less financially draining on a nation's health care system. In the UK alone, CHD costs the economy an estimated £10,000 million per annum (British Heart Foundation, 1998). Of the monies spent on CHD by the health care system, only 1% of this is used in the prevention of CHD (British Heart Foundation, 1998).

Coronary heart disease risk factors are often established during childhood and adolescence. Despite the genetic predisposition of some individuals towards certain risk factors, the majority of young people develop CHD risk factors as a consequence of environmental influences such as physical inactivity and a high-fat diet. There is a need to educate the younger population in the management of healthy living. Evidence would suggest that primary prevention is effective in decreasing coronary heart disease and that immediate action should be taken with our children (Boreham, Twisk, Neville *et al.* 2002).

The aims of this present study are to examine the prevalence of coronary heart disease risk factors in young people of differing socio-economic status and to establish to what extent the clustering of risk factors occur in 12 to 13 year olds.

This thesis contains six chapters the contents of which are briefly outlined below:

CHAPTER 2

The review of literature examines and interprets previous research conducted into CHD risk factors in children and adolescents. This chapter concludes with an experimental rationale and the formulation of a series of experimental aims and null hypotheses.

CHAPTER 3

Chapter 3 outlines in detail the numerous methodological protocols undertaken in this study to examine physical, physiological, haematological and lifestyle risk factors. The statistical procedures applied during the course of this investigation are similarly described, as is a pilot study conducted on twenty schoolchildren.

CHAPTER 4

A results' section presents the data collated and analysed for selected CHD risk factors in 208 schoolchildren. For ease of interpretation, the data are assembled in a series of tables.

CHAPTER 5

Chapter 5 examines the assimilated data and interprets these in the light of findings from previous studies. This chapter concludes with an overview of young peoples' health and fitness.

CHAPTER 6

The realisation of aims is considered prior to an examination of the null hypotheses previously formulated.

REFERENCES

A list of recent and relevant journals and books used during the course of this thesis is described in the reference section.

APPENDICES

To supplement the main text, a number of comprehensive appendices are included in a final section.

† For the purpose of this thesis the phrase young people or young persons embraces both children and adolescents.

CHAPTER 2

2.0 REVIEW OF LITERATURE

2.1 CORONARY HEART DISEASE RISK FACTORS

A risk factor is an identifiable characteristic and when present is considered to put an individual at greater risk of chronic disease (Voller and Strong, 1981). Epidemiological studies have identified several classical risk factors of coronary heart disease (CHD) including, smoking, obesity, physical inactivity, hypertension and hypercholesterolemia (Hubert, Feinleib, McNamara *et al.* 1983; Armstrong, Balding, Gentle *et al.* 1990[a]; Armstrong, Balding, Gentle *et al.* 1990[b]; Baranowski, Bouchard, Bar-Or *et al.* 1992; Boreham, Savage, Primrose *et al.* 1993; Boreham, Twisk, Savage *et al.* 1997; Boreham, Twisk, Murray *et al.* 2001). More recently, clinical indicators such as fibrinogen (Fg), plasminogen activator inhibitor–1 (PAI–1), homocyst(e)ine (Hcy) and C-reactive protein (CRP) have gained prominence (Eliasson, Evrin and Lundblad, 1994; Braunwald, 1997; Danesh, Collins, Appleby *et al.* 1998; Harjai, 1999). According to Cunnane (1993) these risk factors can be strongly indicative of CHD and if more than one is present there is a multiple risk of disease.

Although there is strong evidence relating these risk factors to CHD in adults, there remains a dearth of information on young people. This is unfortunate as evidence suggests that many CHD indicators and risk related behaviour patterns manifest themselves during childhood and adolescence (McGill *et al.* 1963; McGill, 1968; Strong and McGill, 1969; Lauer, Connor, Leverton *et al.* 1975; Gilliam, Katch, Thorland *et al.* 1977; Berenson, McMahan, Voors *et al.* 1980; Armstrong *et al.* 1990[b]; McGill *et al.* 1997; McGill, *et al.* 2000[b]; Boreham *et al.* 2001; Balagopal, Sweeten and Mauras, 2002;

Boreham, *et al.* 2002). Since a number of CHD risk factors are potentially modifiable, it is reasonable to presume that if individuals who are at risk can be identified sufficiently early, preventative strategies could and should be undertaken (Cunnane, 1993; Twisk, van Mechelen, Kemper *et al.* 1997). Preventative schemes might involve activity and nutritional guidelines, and improved knowledge and understanding of healthy living (Boreham *et al.* 1993; Harrell, McMurray, Bangdiwala *et al.* 1996[a]; Harrell, Gansky, McMurray *et al.* 1996[b]). As children and adolescents spend considerable time in school, this instititution lends itself as a key venue for educational and intervention programmes (Fox, 1997). It is essential that every effort be made to reduce CHD risk factor levels in youth. As highlighted by Douthitt and Harvey (1995), adults reach an age when they begin to accept their mortality, adolescents on the other hand, believe that they will live forever.

2.2 ESTABLISHED CORONARY HEART DISEASE RISK FACTORS

2.2.1 Hypertension

The pumping action of the heart facilitates blood flow to the tissues of the body. Blood pressure (BP) is the force exerted by this blood against the walls of the arteries and is the product of cardiac output and total peripheral resistance. During systole, both ventricles contract and blood is ejected from the heart into the aorta and arteries, this results in an increase in arterial blood pressure; as the left ventricle relaxes during diastole, there is a gradual decrease in blood pressure (Frohlich, Grim, Labarthe *et al.* 1988). The minimum pressure of this cardiac cycle is referred to as diastolic blood pressure (DBP), while the peak pressure is called systolic blood pressure (SBP) (Frohlich *et al.* 1988). Traditionally these pressures are recorded in millimetres of mercury (mm Hg) and presented as SBP / DBP.

Blood pressure can be measured by both direct and indirect means. The former method involves inserting a needle or catheter into the arterial tree, this in turn is connected to a pressure transducer. Such a technique is impractical for the mass testing of non-hospitalised subjects; therefore an indirect method of measuring blood pressure is the preferred protocol (Frohlich *et al.* 1988). The indirect method uses a sphygmom-anometer and is based on the occluding-cuff auscultatory technique (Frohlich *et al.* 1988).

The Korotkoff sounds are heard on auscultation over the brachial artery when a pressure cuff is deflated. Korotkoff's first phase (K1), which is the pressure at which the first sound is audible, corresponds to SBP. Korotkoff's fourth (K4; muffling of the pulse sounds) and fifth phases (K5; disappearance of the pulse sounds) are related to DBP (Uhari, Nuutinen, Turtinen *et al.* 1991). Controversy surrounds the measurement of diastolic blood pressure in children and adolescents. Many investigators have suggested that since K5 is often absent in young individuals (National Institutes of Health, 1987), recording K4 should be the preferred option (Lauer *et al.* 1975; Berenson *et al.* 1980). However, Frohlich and co-workers (1988) stated that K4 best represented DBP in children, whereas K5 was most suitable for adolescents. Uhari and colleagues (1991) on the other hand considered K5 to be the most reliable protocol for all age groups of children. The range in protocols for measuring BP in children and adolescents indicates that great care should be taken when opting for suitable methodologies and before making comparisons across studies. Many of the discrepancies in findings may solely be as a consequence of different protocols, especially since many researchers will opt for automated devices.

It is accepted that blood pressure levels rise alongside growth and it is this understanding that has led a number of researchers to ascertain that the processes controlling growth also affects blood pressure (Lever and Harrap, 1992). Since SBP increases more than DBP during childhood and adolescence, the difference between the two pressure levels widens with increasing years (Malina and Bouchard, 1991; van den Bree, Schieken, Moskowitz *et al.* 1996). Difficulties arise when trying to present definitive values for children and adolescents as levels depend on such factors as age, body height and lean body mass. However, for young adults a SBP/DBP of approximately 120/80 mm Hg is considered normal (Joint National Committee, 1988) (Table 2.1).

High blood pressure or hypertension, is a primary risk factor for cardiovascular morbidity or mortality (Caspersen, Nixon and DuRant, 1998; Escobar, 2002). It is considered one of the most potent antecedents of CHD. Elevated BP has also been implicated in nephropathia, retinopathia, and neuropathia in individuals with insulin resistance and type 2 diabetes (Fioretto, Steffes and Mauer, 1991; Friedman, 1996; Cooper, 1998; Comi and Corbo, 1998). Labile hypertension on the other hand, refers to the natural increase in blood pressure that occurs during exercise, stress and very cold weather. Investigations have confirmed that both systolic and diastolic hypertension are associated with an increased risk of developing CHD. However, attention is often focussed upon the latter because of its association with coronary blood flow. For example, MacMahon, Peto, Cutler and colleagues (1990) have reported that a mere 5 mm Hg elevation in diastolic blood pressure results in a 21% increase in CHD incidence. Hypertension is a heterogeneous condition and often manifests alongside many other metabolic disorders including insulin resistance, glucose intolerance, dyslipidemia and obesity (eg syndrome X) (Polare, Lithell, and Berne, 1990; Resnick,

1993; Hardin, Herbert, Bayden *et al.* 1997; American Diabetes Association, 1998). Although the exact aetiology of hypertension is unknown, both genetic and environmental factors are considered to play important roles (Horan and Lenfant, 1990). According to McGill (1968) hypertension leads to atherosclerosis in two ways; firstly, it is thought to have a damaging effect on the vascular endothelial cells; secondly, it is believed that the elevated pressure increases the filtration of lipids into the intimal cells. Increased sympathetic activity is understood to represent an early indication of the development of hypertension, and this scenario is often witnessed in children who have a strong association to parental hypertension (Nho, Tanaka, Kim *et al.* 1998).

Researchers have yet to establish a definitive value for blood pressure above which mortality rate is increased, and below which it remains unaffected, however, the following table (Table 2.1) gives an indication of both 'safe' and 'dangerous' BP levels.

Table 2.1 Classification of blood pressure in adults 18 years or older.

Blood pressure (mm Hg)	Category
Diastolic	
<85	Normal blood pressure
85-89	High normal blood pressure
90-104	Mild hypertension
105-114	Moderate hypertension
>115	Severe hypertension
Systolic	
<140	Normal blood pressure
140-159	Borderline isolated systolic hypertension
>160	Isolated systolic hypertension

Source: Joint National Committee (1988) The 1988 Report of the Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure. *Archives of Internal Medicine*, Vol.148, No. 5, pp 1023-1038.

Hypertension is present in approximately 63 million Americans (American Heart Association, 1993). The Framingham study reported that CHD incidence in men aged 45 to 62 years with blood pressure > 160/95 mm Hg, was five times greater than that of normotensive males (Kannel, Schwartz and McNamara, 1969). The recent MONICA project reported that between 2% and 21% of European men, and 2% and 17% of European women aged 35 to 64 years exhibit a SBP value of 160 mm Hg or more (British Heart Foundation, 2000).

Evidence suggests that hypertension is a process originating from early years. Although the AHA Council on Cardiovascular Disease in the Young recommends early detection of blood pressure elevation (Williams, Hayman, Daniels *et al.* 2002), the need to avoid the premature labelling of adolescents as hypertensive has been stressed by others (Lauer *et al.* 1975; Morgenstern, 2002). Young people will often become normotensive without any intervention, the higher than normal values having solely been a function of growth (Lauer *et al.* 1975; Arensman, Christiansen and Strong, 1989; Twisk, Kemper and Snel, 1995).

Despite childhood blood pressure levels not being a consistent predictor of BP levels in later life, many significant studies have suggested that elevated values tend to track into adulthood (Clarke, Schrott, Leaverton *et al.* 1978; Szklo, 1979; Berenson *et al.* 1980; Voller and Strong, 1981; Whincup, Cook, Shaper *et al.* 1988; Alpert and Wilmore, 1994; Berenson, Wattegney, Bao *et al.* 1995; van de Bree *et al.* 1996). Adopting a diastolic cut-off point of > 90 mm Hg, Heyden, Bartel, McDonough and colleagues (1969) identified hypertension in 11% of 435 adolescents. When 30 of these individuals were re-tested seven years later it was found that five were hypertensive; six were hypertensive with additional vascular complications; and two had died from cerebral haemorrhage. In a similar investigation, Clarke *et al.* (1978) recorded the blood pressure levels of schoolchildren over a five-year period and also found an association between the blood pressure levels obtained in early childhood and values obtained five years later. The authors did concede that since there was much variability in blood pressure values it was difficult to identify those who would develop persistent hypertension. Nevertheless, whilst the tendency for elevated blood pressure levels to track prevails, interventions to prevent the onset of hypertension are desirable (Alpert and Wilmore, 1994; Boreham *et al.* 1997).

The National Institutes of Health (NIH, 1987) Second Task Force on Blood Pressure Control in Children defined blood pressure in children and adolescents according to the following criteria: -

- Normal blood pressure - SBP and DBP less than the 90^{th} percentile for age and sex.

- High normal blood pressure - SBP and/or DBP greater than or equal to the 90^{th} percentile but less than the 95^{th} percentile for age and sex.

- High blood pressure (hypertension) - SBP and/or DBP greater than or equal to the 95^{th} percentile for age and sex.

- Severe hypertension - Blood pressure measurements consistently greater than the 99^{th} percentile for age and sex.

Table 2.2 Classification of hypertension by age group

Age group	Significant hypertension (mm Hg)	Severe hypertension (mm Hg)
Newborn[a]		
7 days	SBP > 96	SBP > 106
8-30 days	SBP > 104	SBP > 110
Infant (< 2 years)	SBP > 112 DBP > 74	SBP > 118 DBP > 82
Children (3-5 years)	SBP > 116 DBP > 76	SBP > 124 DBP > 84
Children (6-9 years)	SBP > 122 DBP > 78	SBP > 130 DBP > 86
Children (10-12 years)	SBP > 126 DBP > 82	SBP > 134 DBP > 90
Adolescents (13-15 years)	SBP > 136 DBP > 86	SBP > 144 DBP > 92
Adolescents (16-18 years)	SBP > 142 DBP > 92	SBP > 150 DBP > 98

Source: "Report of the Second Task Force on Blood Pressure Control in Children – 1987 Task Force on Blood Pressure in Children" by the National Institutes of Health, 1987, *Pediatrics*. Vol. 79, p7.

[a] For newborns, only six SBP were recorded.

Although adult hypertension and its link with CHD and associated risk factors has been comprehensively investigated (Multiple Risk Factor Intervention Trial Research Group, 1982; Wannamethee, Shaper, MacFarlane *et al.* 1995; Dengel, Hagberg, Pratley *et al.* 1998; Orbach and Lowenthal, 1998), definitive data for young people have not been established. Graham, Hines and Gage's, (1945) study was one of the first to address a large cohort of youngsters, and despite the project being undertaken over fifty years ago, the authors of this early investigation had questioned the use of adult cut-off points (140/90 mm Hg) to define hypertension in the younger population. In latter years, Wilmore, Constable, Stanforth *et al* (1982), Fripp, Hodgson, Kwiterovich *et al.* (1985) and Boreham *et al.* (1997) have used the blood pressure cut-off points of 126/82 mm Hg for 12 year olds, and 136/86 mm Hg for 15 year olds to establish their thresholds of CHD risk in schoolchildren. In 1981, Wynder, Williams, Laasko and co-workers conducted a comparative study of children from fifteen countries (Table 2.3). The authors identified an 'optimal level of blood pressure' of 110/60 mm Hg for 12-13 year olds, although they warned that others considered an optimal level of 100/60 mm Hg to be more realistic. Wynder *et al.* (1981) also proposed an arbitrary cut-off point of 130/85 mm Hg for hypertension in 13 year old children (Table 2.3), a threshold adopted in subsequent studies (Armstrong, Balding, Gentle *et al.* 1991[a]). Although the author of the present study adopted the same BP cut-off points, the relatively small cohorts investigated by Wynder and colleagues makes direct comparisons with individual countries questionnable. For the valid identification of thresholds, or the true prevalence of CHD risk factors in young people, a minimum sample number of 2,000 is necessary (personal communication, Professor Rhys Williams, World Health Organisation, 2003).

Table 2.3 Prevalence of selected risk factors among 13 year old children.

	Number		Cholesterol >180mg·dl^{-1}		SBP >130mm Hg	
Country	Males	Females	Males %	Females %	Males %	Females %
West Germany	108	123	47	45	3	6
Finland	999	460	69	70	13	12
France	104	138	-	-	21	13
Greece	157	111	10	9	10	9
Italy	102	98	10	12	1	12
Japan	229	295	13	18	7	6
Kenya	177	113	29	24	1	2
Kuwait	177	233	50	28	12	4
Netherlands	95	102	42	36	8	7
Nigeria[a]	40	20	9	3	0	0
Norway	136	122	52	52	1	3
Taiwan	103	111	-	-	4	0
Thailand	102	102	35	46	3	1
USA	575	538	16	19	3	2
Yugoslavia	77	79	37	48	1	1

[a] Although the numbers in Nigeria were small, the values are consistent with values for 12 and 14 year olds in this country.

Source: Wynder *et al.* (1981) Screening for risk factors for chronic disease in children from fifteen countries. *Preventive Medicine* Vol. 10, pp 121-132.

The importance of repeated measurements in blood pressure readings is well documented and particularly pertinent to the young population (Rames, Clarke, Connor *et al.* 1978; Fixler, Kautz and Dana, 1980; Voller and Strong, 1981). During initial

readings in a group of 6,600 children, Rames *et al.* (1978) identified 13.4% as having SBP/DBP levels greater than 140/90 mm Hg or the 95th percentile, yet when this cohort was re-tested, less than 1% had maintained the elevated pressures. It would appear that without sequential screening it is impossible to differentiate between adolescents with labile hypertension and those with sustained hypertension (Kilcoyne, Richter and Alsup, 1974). Nevertheless, despite recognising the inconsistency in readings, children with occasional high blood pressure levels need to be monitored since one causal elevation of blood pressure might be indicative of the future development of CHD (Armstrong and Davies, 1980). If a child's systolic blood pressure is identified as high more than once, he or she is twice as likely to develop high SBP levels as an adult (Lauer and Clarke, 1989).

Investigations of young people tend to be cross - sectional and little has been done to track blood pressure changes over a period of time. One of the few studies to monitor blood pressure changes at regular intervals was The Amsterdam Growth Study (Kemper, 1995). As a longitudinal analysis of health, fitness and lifestyle, this investigation tracked cardiovascular risk indicators of individuals from ages 13 through to 30 years. Many useful findings were drawn from the study, and with reference to BP, the project concluded that levels during adolescence were not indicative of cardiovascular disease risk indicators in later years. Such a comprehensive study is yet to be undertaken in the United Kingdom.

Over the years, a number of substantial cross-sectional studies have observed blood pressure levels in young people. The Muscatine Study (Lauer *et al.* 1975) investigated 4,829 children and adolescents, and identified elevated BP in 16.7% of 14 to 18 year olds. The Bogalusa Heart Study (Berenson *et al.* 1980) measured body fat distribution and blood pressure in 3,784 children aged 4 to 14 years and similarly concluded that

hypertension begins in childhood (Shear, Freedman, Burke *et al.* 1987). Nevertheless, since the measurements were taken once only, no firm conclusions regarding the prevalence of fixed high levels in children should be made.

Elevated blood pressure levels have been identified in the offspring of hypertensive parents (de Visser, van Hooft, van Doornen *et al.* 1994), and it is presumed that parental history of hypertension can be used as an indicator of risk for the development of the disorder. De Visser *et al.* (1994) compared the fitness status, physical activity levels and anthropometric measures of the offspring of normal and hypertensive parents, and found that children of hypertensive parents recorded a higher percentage of central body fat than the offspring of normotensive parents. De Visser and colleagues (1994) also postulated that body fat distributed around the torso region had an impact on primary hypertension, a theory confirmed by others (Gillum, 1987; Shear *et al.* 1987).

Numerous epidemiological studies have suggested that physical activity and/or fitness decreases blood pressure levels in hypertensive adults (Montoye, Metzner, Keller *et al.* 1972; Fagard, M'Buyamba, Staessen *et al.* 1985; Tipton, 1984; Hagberg, 1990). Although not always apparent, many intervention programmes have demonstrated that when physical activity is taken regularly and at an intensity of 40 to 60% of $\dot{V}O_2$ max, hypertensives can reduce blood pressure levels by approximately 10 mm Hg (Hagberg, 1990). Whether the same beneficial influences are seen in young people remains unproven. Whilst some studies have indicated decreased blood pressure is associated with increased levels of physical activity (Panico, Celentano, Krogh *et al.* 1987; Al-Hazzaa, Sulaiman, Al-Matar *et al.* 1994; Boreham *et al.* 1997), others have failed to confirm this relationship (Armstrong *et al.* 1991[a]; de Visser *et al.* 1994; Webber, Osganian, Feldman *et al.* 1996; Raitakari, Taimela, Porkka *et al.* 1997). During the Child and Adolescent Trial for Cardovascular Health (CATCH), a two and half year

intervention amongst more than 4000 young people did not yield any significant changes to blood pressure (Webber *et al.* 1996). An association between physical activity and BP levels would undoubtedly be advantageous because it implies that an active lifestyle could stem the development of hypertension. Moreover, it is unfortunate that the minimum level of activity required to lower BP levels has not been ascertained (Alpert and Wilmore, 1994). Many cross-sectional investigations have also identified a relationship between cardiorespiratory (CR) fitness and blood pressure levels in children (Fraser, Phillips and Harris, 1983; Hofman, Walter, Connelly *et al.* 1987; Tell and Vellar, 1988; Gutin, Basch, Shea *et al.* 1990). Despite such findings, data on young people are equivocal as many investigators have failed to find an association between physical fitness and BP (Fripp *et al.* 1985; Kwee and Wilmore, 1990; Armstrong *et al.* 1991[a]; Bazzano, Cunningham, Varrassi *et al.* 1992; Jenner, Vandongen and Beilin, 1992; Twisk, Kemper and van Mechelen, 2002). Hofman *et al.* (1987) investigated the relationship between the physical fitness and blood pressure levels of 2061 children (average age 9.1 years) and measured these variables at baseline and post one year. Although an association between both SBP and DBP levels and physical fitness was identified, some aspects of protocol should be questioned. For example, the Harvard Step Test was used to measure CR fitness but this might not be a sufficiently accurate method of determining CR fitness in the younger age group. The validity of the test is highly dependent on the correct measurement of pulse rate, yet no mention was made of the protocol adopted. If taken by the children themselves this would present a substantial source of error (Hofman *et al.* 1987).

The difficulty in arriving at a definitive conclusion with regards to the relationship between physical fitness and separate measures of SBP and DBP has been highlighted (Dwyer and Gibbons, 1994). In their studies of adults and children, Sallis, Patterson, Buono *et al.* (1988) and Tell and Vellar (1988) identified associations for both SBP and

DBP with fitness, whereas others could only link SBP to fitness levels (Fraser *et al.* 1983; Hofman and Walter, 1989; Dwyer and Gibbons; 1994). It has been suggested that SBP is more easily influenced by fitness than DBP, and although this is a distinct possibility it has not been confirmed (Dwyer and Gibbons, 1994). Changes in SBP are greater than changes in DBP during childhood and this suggests that SBP is more easily modified than DBP (Malina and Bouchard, 1991). In addition, it is extremely difficult to locate DBP in children, and a failure to measure it accurately means that an association might be missed (Dwyer and Gibbons, 1994).

Athough there is some evidence that increased levels of physical fitness and physical activity have a favourable effect on blood pressure levels in young people, the inconsistencies of findings indicate that data must be interpreted with caution. It could be that similar to the adult population, beneficial influences are only observed in young individuals with hypertension (Strong, Deckelbaum and Gidding, 1992; Baranowski, *et al.* 1992).

2.2.2 Obesity

Obesity refers to a condition of excess body fat or a state of elevated adiposity at which health problems are likely to prevail. It is a chronic condition that evolves over many years (Jebb and Moore, 1999). Confirmation of its presence include a body mass index (BMI) $\geq$ 30 kg m^{-2}, 130% of ideal weight, or summation of four skinfolds $\geq 95^{th}$ percentile (Armstrong and Davies, 1980; Flegal, 1993; Dietz, 1995; Fox, 1997; Williams *et al.* 2002). In Western society, the proportion of the population that are overweight increases through each decade of life until individuals reach approximately 60 years of age (Bouchard, 1997). According to Fox (1997), clinical obesity (BMI $\geq$ 30 kg m^{-2}) has increased significantly in Britain over the last two decades, from 6% to 15% in males and 8% to 16.5% in females. The Nutrition and Physical Activity Task Forces

(1995) estimated that on current evidence, these figures for obesity would rise to 18% in males and 24% in females by the year 2005. In an attempt to counter such increases, 'The Health of the Nation' (Bost, Primatesta and Dong, 1997) has set a target of 6% and 8% for males and females (aged 16-64 years) respectively, for the year 2005.

Obesity's association with CHD and related risk factors has been well established (de Visser *et al.* 1994; Stern, 1995; Kannel, D'Agostino and Cobb, 1996; Katzmarzyk, Gagnon, Leon *et al.* 2001; Tanaka, Togashi, Rankinen *et al.* 2002). Illnesses related to excessive body fat include CHD, diabetes mellitus, hypertension, arthritis, and kidney disease. However, despite increasing evidence relating these states of health to body fatness, opinions concerning the independent effect of obesity on CHD remain divided. Such disparity in opinion is probably as a consequence of obesity being a condition that enhances other risk factors such as hypercholesterolemia and hypertension, making absolute confirmation difficult (Voller and Strong, 1981; Pi-Sunyer, 1999). Although the majority of researchers concur that weight loss can lead to decreased incidence of hypertension and more favourable lipid profiles, no clinical trials have directly assessed the effects of weight loss on clinical end points such as CHD (Stern, 1995).

Data on the prevalence of obesity in European countries has been collated in the MONICA project (British Heart Foundation, 2000). This programme revealed that between 8% and 24% of males, and between 10% and 36% of females aged 35-64 years are obese. MONICA gave further notice of the severity of the problem indicating that obesity is increasing in all European countries for which data are currently available. The economic costs of obesity are phenomenal; for example, in the United States the direct costs of inactivity and obesity demands approximately 9.4% of the National health care monies (Colditz, 1999). In the United Kingdom, the treatment of obesity

costs the National Health Service more than £500 million a year (Crisp, Young, Bichard *et al.* 2001).

In addition to the problems in adulthood, and possibly more troubling, are conclusions that in the UK, there is an increased prevalence of overweight and obesity amongst children and adolescents (Chinn and Rona, 1994; British Heart Foundation, 1998; Institute of European Food Studies, 1999). A recent study reported that one in five nine-year-olds are overweight and one in 10 is obese; and the alarming fact is that these figures have almost doubled in the last decade (Rudolf, Sahota, Barth *et al.* 2001). Similar findings have been reported in the USA (Campaigne, Morrison, Schumann *et al.* 1994; Troiano, Flegal, Kuczmarski *et al* 1995; Freedman, Srinivasan, Valdez *et al.* 1997; Gortmaker, Peterson, Wiechen *et al.* 1999; Morkdad, Serdual, Dietz *et al.* 1999; Musaiger, Al-Ansari and Al-Mannai, 2000; Williams *et al.* 2002). Troiano and Flegal (1998) reported that whereas five percent of youngsters aged 12 to 17 years were identified as overweight between 1966 and 1970, this had risen to 11% and 12% for boys and girls respectively, between 1988 and 1994. In 1974, Wilmore and McNamara investigated the prevalence of CHD risk factors in children aged 8 to 12 years and determined that 13% of their sample was obese. A subsequent study by Wilmore and colleagues (1982) revealed that 14.9% of 13 to 15 year olds were obese, displaying greater than 25% total body fat. However, in the latter study the investigators did concede that the hydrostatic technique used to determine body fat levels could have led to an overestimation of values. In the interim period, Gilliam *et al* (1977) investigated the prevalence of CHD risk factors in children aged 7 to 12 years. They too opted for Wilmore and McNamara's (1974) standard for obesity (25% or greater) and found that an overall total of 10.6% of children were obese. Gilliam and co-workers (1977) similarly recognised the equivocal nature of their findings recognising that the final

value of 10.6% included pre-pubescent females with developing sex-specific fat (Gilliam *et al.* 1977). More recently, estimated body fatness values have been obtained for 11-16 year old British schoolchildren (Armstrong and Welsman, 1994[b]). According to the criteria detailed by the Royal College of Physicians, Armstrong and Welsman (1994[b]) found that an unacceptable 13.4% of boys and 9.7% of girls were 'overweight'. It is interesting to note Dietz's (1995) declaration that this increase in childhood obesity is more common in middle and upper socio-economic groups, despite the findings of others that increased body fat is more apparent in children and adolescents from underprivileged areas and lower social classes (Freeman, Weir, Whitehead *et al.* 1990; Guillame, Lapidus, Björntorp *et al.* 1997).

Despite such conclusions on childhood obesity, and the recognition that this is the most commonly witnessed CHD risk factor in young people (Gilliam *et al.* 1977), there remains no clear definition for the term (Rowland, 1990; Sallis, Chen and Castro, 1995). In his editorial on obesity, Bouchard (1997) referred to the ambiguity surrounding definitions for obesity and highlighted the need for an internationally accepted classification. Despite its limitations, body mass index (BMI) is the most commonly used index of obesity for both adults and young people; and is included in the majority of population and epidemiological studies (Martin and Ward, 1996; Guo and Chumlea, 1999; Baumgartner, Heymsfield and Roche, 1995; Williams *et al.* 2002; Eston, 2002). Body mass index is mass divided by stature squared (kg m^{-2}) and has been positively associated with many CHD risk factors (Martin and Ward, 1996; Cole, Bellizzi, Flegal *et al.* 2000). For adults, overweight is reported to occur at a BMI of 25 kg m^{-2} and over, while obesity is present at a BMI of 30 kg m^{-2} and over (World Health Organization, 1998). For the United Kingdom, current trends suggest that the average BMI for males and females will rise to 27.5 kg m^{-2} by the year 2005 (The Nutrition and Physical

Action Task Forces, 1995). The Health Survey for England, 1995 (Prescott-Clarke and Primatesta, 1997) was the fifth in a series of annual surveys commissioned by the Department of Health and evidence collated during this project indicated that about 59% of males and 50% of females of all ages were overweight or obese (BMI > 25 kg m^{-2}) (Bost *et al.* 1997). The survey also concluded that mean BMI and prevalence of obesity has continued to increase since 1993 (Bost *et al.* 1997).

For the younger population the problems surrounding a suitable definition are greater, and investigators continue to operate many different criteria and protocols to differentiate between normal and obese children (Wilmore and McNamara, 1974; Williams, Going, Lohman *et al.* 1992; Bar-Or and Baranowski, 1994; Dietz, 1995; Flegal, 1999; Freedman, Dietz, Srinivasan *et al.* 1999; Chinn and Rona, 2002; Smith and Rinderknecht, 2003). Such discrepancies in methodologies make comparisons across studies difficult. For example, Wilmore and McNamara (1974) adopted a standard of obesity of ≥ 25% body fat, whilst Epstein, Wing, Penner *et al* (1985) defined obesity as 20% above ideal body mass. More recently fatness levels at or above 25% and 30% for boys and girls respectively, have been offered as excessive values (Williams *et al.* 1992), whereas a BMI greater than the 85th percentile for sex and age was employed by Ewart, Young and Hagberg (1998). According to Freedman *et al.* (1999) the condition exists when body mass exceeds the 95th percentile for sex and age; whilst Owens, Gutin, Allison *et al.* (1999) used the criteria of the triceps skinfold exceeding the 85th percentile for sex, age and ethnicity. Adding to the confusion, Robinson (1993) declared that current cognitive limitations make it impossible to provide a clinically useful definition of obesity that is entirely based on measures of adiposity amongst younger individuals. There is obviously a need for appropriate reference standards in children and adolescents but progress is slow, hindered by a lack

of longitudinal studies (Hubbard, 1995). Despite the lack of clarification surrounding its definition, investigators continue to emphasise the importance of investigating the prevalence of obesity in the younger population. This is particularly important since overweight children and adolescents are at significantly greater risk of developing further CHD risk factors than individuals of normal mass (Freedman *et al.*1999).

Traditional methods of estimating body fatness include height-to-weight tables, however, such protocols fail to consider tissue distribution, and any extra weight for height could be derived from fat or muscle (Rowland, 1990). Body composition refers to the various tissues that contribute to the individual's total body mass, the primary ones being bone, muscle, fat and viscera. Many methods have been employed as measures of fatness or subcutaneous adiposity, including hydrostatic weighing, biolelectrical impedance, BMI, waist-to-hip ratios and skinfold thicknesses (Rowland, 1990; Kemper, 1995; Martin and Ward, 1996). The variability in determining body fatness in young people indicates that results should be interpreted cautiously. A widely used method for predicting subcutaneous adiposity involves measuring the thickness of a combination of selected skinfold (SKF) sites (Martin, Ross, Drinkwater *et al.* 1985). The rationale behind this form of measurement is the relationship that exists between fat located subcutaneously, and internal fat and body density. Typically these SKF measures are then entered into prediction equations to estimate whole body density. There are over one hundred population specific equations to predict whole body density from SKFs including the widely used Siri (1956) and Brozek, Grande, Andersen *et al.* (1963) formulae, however these equations as others, are valid only for individuals with similar characteristics e.g. sex, age, and activity level. Many are derived from adults and if employed for children and adolescents, large errors of estimation can occur (Boileau, Lohman and Slaughter 1985). When using such equations, the body is commonly

regarded as a two-component model (Martin and Ward, 1996), with the fat aspect having a constant density value of 0.90 $g \cdot ml^{-1}$ and the fat-free component having a constant density of 1.10 $g \cdot ml^{-1}$ (Siri, 1956; Malina and Bouchard, 1991). The growing body is, however, subject to changing composition, with children exhibiting a lower bone mineral content (5.4%) and higher water content (76.6%) than adults; consequently, if adult prediction equations are used, there will be an overestimation of childrens' percent fat (Lohman, 1986; Malina and Bouchard, 1991). Moreover, investigators have used a range of densities to develop such algorithms. Slaughter, Lohman, Boileau and colleagues (1988) recognised the need for equations that accounted for the transient relationship of SKF measures to fat-free mass density in the paediatric population and claimed that their modified equations overcame the previous limitations. The authors estimated percent fat using three methods: from density alone; from density and water; and from density, water and bone, and offered a number of predictive equations that considered the chemical immaturity of children. Janz, Nielsen, Cassady *et al.* (1993) cross-validated many of these equations and concluded that although promising, they were not entirely trustworthy. To avoid the problems of predictive equations, many researchers have been unwilling to convert to percent fat, electing to use the summation of SKF thicknesses as a measure of subcutaneous adiposity instead (Martin and Ward, 1996). Despite limitations in skinfold measurements, this method is recommended for the estimation of subcutaneous adiposity in young people (Roemmich, Clark, Weltman *et al.* 1997). The author of this study suggests that since the choice of skinfold sites could in itself be a limitation in adiposity assessment, data should be interpreted with caution.

The tracking and genetics of obesity are not completely understood. The Ten State Nutrition Survey revealed that the likelihood of childhood obesity was 80% if both

parents were obese, 40% if one parent was obese, and dropped to 20% if there was no incidence of obesity in first degree relatives (Garn and Clark, 1976). In their review of childhood and adolescent obesity, Hill and Melanson (1999) indicated a genetic contribution of anywhere between 25% and 70%, although the authors did suggest that this genetic component could be strongly influenced by environmental conditions. Several other researchers have similarly highlighted the ambiguity surrounding the aetiology of obesity, and state that there is a fair chance that excess fat in offspring is determined by a combination of both genotype and a shared environment (Rowland, 1990; Dietz, 1995; Katzmarzyk, Malina, Perusse *et al.* 2000). Although Bouchard (1997) suggested that it was more likely a specific genotype that caused obesity, he acknowledged that individuals of similar genetic characteristics could either become obese, or maintain a normal mass, and that lifestyle contributed to these possible scenarios (Bouchard and Blair, 1999).

A critical period of fat-cell development has been proposed and any overzealous feeding and/or lack of activity during this particular phase could magnify the risk of developing obesity (Donahue, Abbott, Bloom *et al.* 1987; Rowland, 1990). Confirmation of this theory would aid the battle against obesity as it suggests selected time slots during which increased efforts should be made. If adolescence is one such sensitive period for fat development, this would confirm the need to target this age group. Nevertheless, some researchers continue to question the existence of these important periods (Sallis *et al.* 1995).

Reporting on the findings of a 55-year study, Must, Jacques, Dallal and colleagues (1992) found that overweight adolescents tended to maintain this condition as adults, a theory supported by others (Abraham and Nordsieck, 1960; Stark, Atkins, Wolff *et al.*

1981; Serdula, Ivery, Coates *et al.* 1993; Guo, Roche, Chumlea *et al.* 1994; Must, 1996; Whitaker, Wright, Pepe *et al.* 1997; Kotani, Nishida, Yamashita *et al.* 1997; Seidell, 1999; Wright, Parker, Lamont *et al.* 2001). This finding was not confirmed in The Thousand Families Cohort Study (Wright *et al.* 2001). In this particular study, little tracking from childhood overweight to adulthood obesity was identified. However, consensus suggests that since the medical risks of excess body fat are believed to increase with age (Rowland, 1990), it seems judicious to suggest that high-risk groups within the younger population should be targeted. Recent evidence has indicated that an "incubation period" of 10 - 15 years is necessary before obesity has any detrimental effect on the body (Malina and Bouchard, 1991). Such a theory infers that every effort should be made during younger life to prevent the development of a high-risk physical profile during adulthood. Data gathered during the Muscatine study similarly signified that body mass, BMI and triceps skinfold thickness were carried through from childhood to adulthood (Clarke and Lauer, 1993). However, the authors were unable to ascertain why some obese children remained overweight as adults, yet others matured into normal weight individuals. The relationship between excessive bodyweight during childhood and obesity in later life was also investigated by Vanhala, Vanhala, Kumpusalo and colleagues (1998). The findings of this study intimated that not only did approximately 50% of obese children become obese adults, but also that obesity in later life was more harmful if the condition had originated during childhood. The investigators proposed that if obesity was sustained from childhood into adulthood, this would serve as a form of 'generator' for continued insulin resistance and lead to the clustering of hypertension and unfavourable metabolic conditions. The association between childhood obesity and adult cardiovascular mortality was examined in the Boyd Orr cohort (Gunnell, Frankel, Nanchanal *et al.* 1998). This 57-year follow-up study failed to ascertain whether BMI levels in childhood were associated with

overweight in adulthood. However, the study did indicate that although intervention programmes aimed at reducing weight in childhood are important, they might only influence health in later life if weight reduction strategies continue into adulthood. The conclusions of such studies indicate the need for further longitudinal research into childhood obesity as well as the importance of prevention and intervention programmes (Sallis *et al.* 1995; Boreham *et al.* 1997; Freedman *et al.* 1999; Fulton, McGuire, Caspersen *et al.* 2001). Although there is some evidence that successful treatment of overweight adolescents could lead to a 30% - 45% reduction in adult obesity (Griffith, Rivers and Hoinville (1985), the influence of nature over nurture remains to be established.

Several authors have confirmed the negative association between cardiorespiratory fitness and/or activity, and body fatness in adolescents (Beunen, Malina, Ostyn *et al.* 1983; Tell and Vellar, 1988; Kwee and Wilmore, 1990; Armstrong *et al.* 1991[a]; Cureton, Baumgartner and McManis, 1991; Hager, Tucker and Seljaas, 1995; Boreham *et al.* 1997; Guillame *et al.* 1997; Twisk *et al.* 1997; Twisk, Kemper, van Mechelen *et al.* 1998; Mota, Santos, Guerra *et al.* 2002), although the relatively small cohorts of some of these studies, along with their cross-sectional design, implies that findings should be considered with caution (Armstrong *et al.* 1991[a]). In addition, the inter-relationship between physical activity, physical fitness and body fatness implies that the cause - and - effect of obesity is not easily defined (Twisk, 2000).

The fitness, fatness, and CHD risk in Northern Irish adolescents was investigated by Boreham *et al.* (2001), with particular focus on the independence and relative strengths of associations. The authors reported that since the relationships between CHD risk factors and fatness were independent of fitness, intervention should focus upon weight

control. Bar-Or and Baranowski (1994) scrutinised the evidence linking body fatness to physical activity in adolescents and failed to identify consistent differences between the energy expenditure of obese and non-obese children. The investigators did not present definitive physical activity guidelines, but they did suggest that more than a twelve months' programme of endurance type activities was beneficial to reducing body fatness and that this yielded greater success when combined with a low-calorie diet. Epstein (1992) had previously articulated this view, whilst others have stipulated the need for an intervention programme that includes a focus on behaviour patterns (Rowland, 1990). Whatever the nature of the intervention, the possibility that childhood obesity is the single most important modifiable CHD risk indicator suggests that efforts should concentrate on preventing or reducing weight gain (Boreham *et al.* 2001).

Traditionally, health strategies for combating obesity have tended to concentrate on overall fatness, although in recent times more attention has been afforded to the relationship of fat or subcutaneous adiposity distribution with CHD. A number of investigators have postulated that abdominal or central body fat places individuals at increased risk of CHD and type 2 diabetes (Larsson, Svardsudd, Welin *et al.* 1984; Lapidus, Bengtsson, Larsson *et al.* 1984; Freedman, Burke, Harsha *et al.* 1985; Ducimetiere, Richard and Caubien, 1986; Stern and Haffner, 1986; Donahue, Abbott, Bloom *et al.* 1987; Maffeis, Pietrobelli, Grezzani *et al.* 2001; Teixeira, Sardinha, Going *et al.* 2001; Lakka, Lakka, Tuomilehto *et al.* 2002). Central body fat is thought to have stronger links with adult-onset obesity, as opposed to obesity that has transcended through from childhood into later life (Stern and Haffner, 1986; Donahue *et al.* 1987). Since males are more predisposed to abdominal fat than females, it is assumed that the former cohort is in greater danger (Gillum, 1987; Malina and Bouchard, 1991). As with other physical and physiological measures, the adolescent growth spurt plays an

important role in fat distribution. Stallones, Mueller and Christensen (1982) suggested that relative fat distribution was less important in predicting CHD risk factors in adolescents than it was for adults, however, additional knowledge would be useful as it indicates healthy and unhealthy profiles (Martin and Ward, 1996). Malina and Bouchard (1991) indicated that approximately 25% of the variation amongst individuals in fat distribution is genetically determined. Similarly, the work of de Visser *et al.* (1994) suggested that offspring of hypertensive parents were inclined to carry more central fat than children of two normotensive parents. In contrast to this, Bogin and Sullivan (1986) and Rona and Chinn (1987) suggested that socio-economic status was the primary mediator of fat distribution. Such discrepancy indicates more research is needed in this area. Although fat distribution in children has been investigated (Stallones *et al.* 1982; Bogin and Sullivan, 1986; Gillum, 1987; Rona and Chinn, 1987; de Visser *et al.* 1994; Cameron, Johnston, Koample *et al.* 1992; Rebato, Salces, Martin *et al.* 1998; Maffeis *et al.* 2001; Teixeira *et al.* 2001), differences according to sex and growth needs to be better understood. It is generally accepted that a healthy body mass in terms of quantity and distribution of fat should be encouraged throughout life.

2.2.3 Aerobic fitness

Despite being distinct concepts, physical fitness and physical activity are words that are often used interchangeably (Pate, Pratt, Blair *et al.* 1995). Physical fitness has been defined as "a set of attributes that people have or achieve" and that relates to the ability to perform physical activity (Caspersen, Powell and Christenson, 1985, p 128), it is considered an adaptive state (Malina, 1996). Physical activity on the other hand has been described as a complex set of behaviours that encompass "any bodily movement produced by skeletal muscles and results in energy expenditure" (Caspersen *et al.* 1985, p 126). The assessment of aerobic fitness and physical activity, has gained prominence

over recent years, primarily due to their supposed beneficial effect on CHD (Sallis *et al.* 1988; Tell and Vellar, 1988; Eaton, Lapane, Garber *et al.* 1995; Paffenbarger and Lee, 1996; Farrell, Kampert, Kohl, III *et al.* 1998; McMurray, Ainsworth, Harrell *et al.* 1998; Whaley, Kampert, Kohl III *et al.* 1999). The assessment of physical fitness is often preferred to physical activity as the former offers greater objectivity and less likelihood of misclassification. In addition, some researchers claim that aerobic fitness, and not physical activity is more closely related to CHD, hence efforts should be made to identify a threshold of daily physical activity necessary to elicit increased fitness levels in young people (Andersen and Haraldsdottir, 1995; Eaton *et al.* 1995; McMurray *et al.* 1998).

Although physical fitness is composed of several components, the component most strongly related to health is aerobic fitness. Whilst amongst adults the inverse relationship between aerobic fitness and CHD is well established, evidence in the younger population is equivocal. Such ambiguity is partly attributed to the influence of growth, maturation and body fatness (Tell and Vellar, 1988; Sallis *et al.* 1988; Malina and Bouchard, 1991; Ebbeling and Ward, 1992; Hager *et al.* 1995).

There is a perception that young peoples' fitness levels have declined over recent decades (Jopling, 1988; Strong, 1990; Dollman, Olds, Norton *et al.* 1999). Dollman and co-workers (1999) investigated the fitness and fatness characteristics of young Australians and found that the fitness levels of 10-11 year olds were lower in the 1990s compared to the 1980s. However, several investigators refute such claims maintaining that there is little evidence of a decline (Armstrong and Welsman, 1994[a]; Bar-Or and Malina, 1995; Rowland, 1996).

The most widely used index of aerobic fitness is maximal oxygen uptake ($\dot{V}O_2$ max). It indicates the efficiency of the cardiorespiratory and cardiovascular system to deliver oxygen to the working muscles (Åstrand and Rodahl, 1986; Rowland, 1990; Malina, 1996) and has been declared the preferred single indicator of health-related physical fitness status (Golden, 1991). Maximal oxygen uptake is assumed to have been reached when there is no further increase in $\dot{V}O_2$ and a plateau has been achieved despite an increased rate of exercise (Åstrand and Rodahl, 1986; Léger, 1996). However, since this plateau is not always witnessed in children, and indeed some adults, the highest attained $\dot{V}O_2$ is then referred to as peak $\dot{V}O_2$ ($P\dot{V}O_2$) (Armstrong and Welsman, 1994[a]). Despite the common usage of direct and indirect field and laboratory tests for the assessment of $\dot{V}O_2$ max, its validity as a reliable indicator of cardorespiratory and cardiovascular function in children and adolescents could be questioned (Rowland, 1990). But from our experience, with the correct motivation maximum aerobic power can be attained in the majority of young individuals (personal communication, Professor Bruce Davies, 2003).

According to Malina and Bouchard (1991), aerobic power increases linearly until about 13 years of age in females, and until about 16 years in males, however, not all investigators agree with this declaration. In the Amsterdam Growth Study, van Mechelen and Kemper (1995) reported that girls' $P\dot{V}O_2$ continued to increase until age 16 years; whereas others have confirmed that $P\dot{V}O_2$ continues to rise from 8 to 18 years in boys (Rutenfranz, Lange Andersen, Seliger *et al.* 1982; Cunningham, Paterson, Blimkie *et al.* 1984). Variations in sample size and selection, along with test protocol have contributed to such conflicting findings (Stewart, Brown, Hickey *et al.* 1995). It is accepted that body mass influences $\dot{V}O_2$ max, and to facilitate comparisons between individuals of dissimilar body mass, $\dot{V}O_2$ max values are often expressed relative to

body mass (Rowland, 1990; Armstrong and Welsman, 1994[a]). When values are presented in this way, there is further discrepancy amongst the literature. In their review of longitudinal studies, Armstrong and Welsman (1994[a]) found that when expressed in $ml \cdot kg^{-1} \cdot min^{-1}$, the $P\dot{V}O_2$ of boys from age 8 to 16 years did not change greatly across the age range, whereas girls' mass-related values decreased with age. Such differences between the sexes have been attributed to an increased accumulation of body fat in young females (Beunen and Malina, 1988). An earlier review by Krahenbuhl, Skinner and Kohrt (1985) had made similar conclusions. The relative difference between males and females aged 16 years has been reported to be as large as 50% (Beunen and Malina, 1988). Despite consensus favouring these conclusions, there is evidence to the contrary. Janz and Mahoney (1997) tracked the aerobic fitness of boys and girls through puberty and found that the magnitude of change in aerobic power when expressed relative to body mass did not differ greatly between the sexes. Recognising that their findings contradicted the conclusions of others, the authors suggested that the discrepancies might have occurred because their male cohort was yet to reach their aerobic fitness "spurt".

Since aerobic fitness is an easily measured risk indicator of CHD, it is important that acceptable standards of $\dot{V}O_2$ max are established. If youngsters are to be categorised, cut-off points are necessary, and if researchers are reluctant to use such criteria, they should refrain from labelling children as fit or unfit (Blair, 1992). A number of arbitrary thresholds have been proposed (Bell, Máček, Rutenfranz *et al.* 1986; Boreham *et al.* 1993) but experts remain undecided on optimal health levels with little known about the quantity and quality of exercise needed for well-being in children (Ebbeling and Ward, 1992). Despite this lack of consensus, some of the most commonly used thresholds were those reported by Bell *et al.* (1986). Bell and colleagues (1986) suggested a 'risk level'

of 35 $ml \cdot kg^{-1} \cdot min^{-1}$ for boys, and 30 $ml \cdot kg^{-1} \cdot min^{-1}$ for girls. The same authors also emphasised the importance of identifying 'health indicators' for children and proposed values of over 40 $ml \cdot kg^{-1} \cdot min^{-1}$ for boys, and 35 $ml \cdot kg^{-1} \cdot min^{-1}$ for girls. Although Bell *et al.*'s (1986) thresholds have proved popular in subsequent investigations of young people, since there is no empirical evidence to endorse their proposals authors have continued to adopt various criteria (Armstrong and van Mechelen, 1998). For example, in their study of teenagers' aerobic fitness, Boreham *et al.* (1993) based their thresholds of risk on the 25th centile score in the widely used 20 metre shuttle run test (20 MST). The 20 MST scores were converted to predicted $\dot{V}O_2$ max values using linear regression (Léger, Mercier, Gadoury, *et al.* 1988). This equated to 46.7 $ml \cdot kg^{-1} \cdot min^{-1}$ for boys, and 36.4 $ml \cdot kg^{-1} \cdot min^{-1}$ for girls.

A variety of field tests and quasi-laboratory tests have been designed to assess $\dot{V}O_2$ max (Anderson, 1992; Ebbeling and Ward, 1992) and a recent review by Armstrong and Welsman (1994[a]) scrutinised the value of these tests amongst the younger population. It is accepted that the most accurate method of determining $\dot{V}O_2$ max is the direct laboratory protocol and this is the gold standard against which other indirect protocols are validated (Léger, 1996; Cooley and McNaughton, 1999). Nevertheless, the test which is based upon on-line gas analysis, is expensive, time-consuming, requires trained personnel, and is generally inappropriate for large numbers in school settings (Eston and Brodie, 1985; Boreham, Paliczka, Nichols *et al.* 1990; Buono, Roby, Micale *et al.* 1991; Barnett, Chan and Bruce, 1993). Also, since the measurement of $\dot{V}O_2$ max necessitates exercising to exhaustion, many investigators have opted to avoid this protocol with children (Armstrong and Welsman, 1994[a]). Numerous field tests have been developed to predict $\dot{V}O_2$ max in children and adolescents, and these protocols offer improved reliability over ecological validity. Such tests include distance runs, step

tests and the 20 metre shuttle test (Jackson and Coleman, 1976; Léger *et al.* 1988; MacNaughton, Croft, Pennicott *et al.* 1990; Buono *et al.* 1991). The validity and reliability of these tests have been measured in a number of studies (Jackson and Coleman, 1976; McNaughton *et al.* 1990) with reliability correlation coefficients ranging from 0.47 to 0.90. Attempts have been made to improve the validity of such tests by using scores adjusted for body fatness, and using tests whose initial validity was confirmed on a similar cohort of children (Cureton *et al.* 1991; Docherty, 1996). Nevertheless, more research into the accuracy of prediction and validity of predictive tests is necessary in the younger population.

The 20 metre multistage fitness test, otherwise known as the 20 m shuttle run test or Pacer, (Léger *et al.* 1988; Vincent, Barker, Clarke *et al.* 1999), is favoured in numerous British schools and by many health-related fitness batteries (Council of Europe (Eurofit), 1988; Massicotte, 1990; Prudential Fitnessgram, 1994; McVeigh, Payne, and Scott, 1995). Such popularity indicates that despite its limitations as a predictor of aerobic fitness it is considered a worthwhile aerobic testing tool. The 20 metre multistage fitness test (20 MST) was initially developed for adults (Léger and Lambert, 1982), and later modified for children (Léger *et al.* 1988). The test is based upon the relationship between maximal running velocity and oxygen consumption (Boreham *et al.* 1990). The test protocol requires subjects to run back and forth between parallel lines set 20 m apart, with pace determined by means of an audiotape (Léger and Lambert, 1982; Léger *et al.* 1988). The original test used 2 minutes speed intervals but this was later modified to 1-minute stages (Léger *et al.* 1988; Léger and Gadoury, 1989). The test is incremental, hence it only necessitates maximal effort in the last minute or so of work, for this reason testers experience fewer problems with motivation compared to other field tests (Liu, Plowman and Looney, 1992; Vincent *et al.* 1999). It

has been accepted as a reliable and valid test of $\dot{V}O_2$ max in adults (Paliczka, Nichols and Boreham, 1987; Ramsbottom, Brewer and Williams, 1988; Léger and Gadoury, 1989). In the original study (Léger and Lambert, 1982) test-retest correlations of 0.95 were reported for adults, while a younger cohort produced a value of 0.89 (Léger *et al.* 1988). Further significant studies have reported validity coefficients for youths ranging from 0.54 to 0.90, whereas reliability coefficients of 0.82 to 0.93 have been observed (van Mechelen, Hlobil and Kemper, 1986; Boreham *et al.* 1990; Lui *et al.* 1992; Vincent *et al.* 1999; Wilkinson, Fallowfield and Myers, 1999). Despite these encouraging results some researchers continue to harbour reservations regarding its general use. Moreover, several investigators have recommended the inclusion of skinfold measures in regression equations if $\dot{V}O_2$ max is to be properly predicted from shuttle run performance (McVeigh *et al.* 1995). It is also worth noting that the reliability values offered by some studies indicate a relationship as opposed to an agreement between test and re-test scores (Bland and Altman, 1986). Anderson (1992) investigated the validity of the 20 MST as a predictor of aerobic capacity in young males aged 10 to 12 years. Although Anderson determined that the test was related to $\dot{V}O_2$ max (0.72), he found that the predicted values differed from the measured values. Anderson (1992) proposed that the effects of running economy, running efficiency and the utilisation of anaerobic energy sources, caused this discrepancy. The significance of anaerobic contribution to 20 MST performance was similarly highlighted by McVeigh *et al.* (1995). Despite such hesitancies in accepting the test as a true measure of peak $\dot{V}O_2$ in youngsters, several researchers have been encouraged by results. Many believe that if field tests are the only available options, the 20 MST should be the preferred indicator of aerobic fitness in youngsters (van Mechelen *et al.* 1986; Leger *et al.* 1988; Boreham *et al.* 1990; Liu *et al.* 1992; Mahar, Rowe, Parker *et al.* 1997).

2.2.4 Physical activity

Physical activity (PA) differs from physical fitness in that it is considered a 'behaviour' and not an 'attribute' (Caspersen *et al.* 1985). Malina (1996) made reference to its instability and intimated that this was caused by the transient attitudes of man towards participation in physical activity. There is concern that the average amount of daily PA undertaken by man has declined dramatically over the past century. This presents cause for concern since the human organism requires the impetus of exercise to promote a good state of health (Fox, 1997; Booth *et al.* 2000). Physical activity was once considered a secondary CHD risk factor (Armstrong and Davies, 1980), however, Melansson and Freedson (1996), reported that in recognition of its importance to general wellbeing, physical activity or more importantly the lack of it, has been elevated to a primary risk factor. Neverthelesss, despite reports that physical inactivity increases the incidence of at least 17 unhealthy conditions (Booth *et al.* 2000), not all investigators concur that inactivity is a risk factor of CHD (Kannel, Wilson and Blair, 1985; Andersen and Haraldsdottir, 1995). In adults, evidence tends to support the positive influence that activity has on health (Morris, Heady, Raffle *et al.* 1953[a]; Morris, Heady, Raffle *et al.* 1953[b]; Paffenbarger, Wing and Hyde, 1978; Davies, 1997; Puffer, 2001), but it is uncertain whether this relationship is as significant in the younger population (Baranowski, Bouchard, Bar-Or *et al.* 1992; Gutin and Owens, 1996; Rowland, 1996; Cavill, Biddle and Sallis, 2001). Moreover, the relevance of PA during childhood to health status in later life is ambiguous (Twisk *et al.* 2002). Nevertheless, the adoption of an active lifestyle is desirable; it has health benefits other than reducing the risk of CHD, including improved psychological wellbeing, and moral and social development (Steptoe and Butler, 1996). It also has a positive influence on other risk factors of CHD (Suter and Hawes, 1993; Andersen and Hippe, 1996).

Since there are grounds to suggest that physical activity patterns have a tendency to track from childhood into adulthood (Blair, 1992; McGinnis, 1992; Raitkari, Porkka, Räsänen *et al.* 1994; Kemper and van Mechelen, 1995), every effort should be made to evaluate, and if necessary to modify children's PA behaviours. The Framingham Offspring Study testified that habits nurtured in young adulthood precipitated blood lipid status in later life (Hubert, Eaker, Garrison *et al.* 1987). However, not all researchers concur with this opinion and although evidence is accumulating, it falls short of being conclusive. In his review of contemporary studies, Armstrong (1995) suggested that a definitive study on the tracking of PA habits from childhood through to adulthood was needed. Both Rowland (1991) and Aaron and Laporte (1997) have reported that there is little evidence to suggest a relationship between childhood activity, and childhood or adult health, an opinion previously articulated by Brill, Burkhalter, Kohl and colleagues (1989). This suggests a need to heed the more recent warning of Gutin and Owens (1996) that if future research fails to identify the health benefits of PA during adolescence, there could be a waste of resources, and limited monies would be better spent elsewhere. Kannel and collaborators (1985) also questioned the beneficial effects of PA on CHD and cited a number of reasons for this. The authors argued that the assessment of PA is often undertaken using unreliable and invalid questionnaires; and that relationships are often modest. They also referred to the inconsistency amongst studies; that healthy subjects were prone to PA, hence there is self-selection; that confounding variables remain a problem; and that evidence is inconsistent. However, although it is not possible to categorically state that inactivity during childhood and adolescence has an influence on long-term health, even the smallest of links could be important in health terms. Moreover, Gutin and Owens (1996) conceded that if one was convinced that there was no health benefit and therefore failed to encourage PA, the future health of a generation of children could be jeopardised. If it was substantiated

that PA levels during adolescence was predictive of adulthood levels, the relevance of activity interventions would gain better support (Sallis, 1995).

In 1980, Armstrong and Davies reviewed the prevalence of coronary heart disease risk factors in children and concluded that a criterion below which a young person was considered habitually inactive was yet to be established, a similar comment was made many years later by Thirlaway and Benton (1993). To date, although a number of researchers have attempted to compose guidelines for PA involvement (Pate *et al.* 1995; National Institute of Health, 1996; Pate, Trost and Williams, 1998), internationally recognised definitive data are yet to appear. In 1993, Sallis and colleagues assembled an International Consensus Conference on Physical Activity Guidelines for Adolescents (Sallis, Patrick and Long, 1994). Two important recommendations were made at this convention; that all adolescents should aim to engage in PA every day; and that they should participate in activities that last twenty minutes or more on at least three occasions per week. Despite these recommendations, significant others maintain that ambiguity prevails with regards to the level of activity necessary for optimum childhood health status (Pate, Long and Heath, 1994; Riddoch and Boreham, 1995) yet it is only through the identification of such physical activity standards that meaningful interventions can take place. The Centres for Disease Control and Prevention and the American College of Sports Medicine (Pate *et al.* 1995) recommended that all children and adults should aim to engage in at least 30 minutes or more of moderate - intensity physical activity on most, or better still, all days of the week. Pate and collaborators (1998) went further and claimed it was necessary for all young people to undertake moderate - intensity activity for one hour per day. They substantiated this by reporting that despite many youngsters participating in 30 minutes of moderate PA, obesity amongst the younger generation is continuing to increase (Chinn and Rona, 1994). It is

disheartening to note that in his review of children's cardiopulmonary fitness and PA patterns, Armstrong (1995) found that young people rarely experience even 10 minute periods of moderate to vigorous intensity, a finding supported by others (Buttriss, 2002). Armstrong and colleagues (1990[a]) had previously found that British teenagers had a low level of habitual physical activity and that they seldom participated in exercise that would benefit the cardiovascular system. Numerous others have claimed that physical activity declines through school age years (Ilmarinen and Rutenfranz, 1980; Riddoch, Mahoney, Murphy *et al.* 1991; Thirlaway and Benton, 1993; Pate, 1993; Bar-Or and Malina, 1995).

The type, quantity and quality of activity necessary for health benefits and a concomitant reduction in CHD potential remains equivocal (Jacobs, Ainsworth, Hartman *et al.* 1993; Riddoch and Boreham, 1995; Cavill *et al.* 2001). Where once it was considered that vigorous activities of sustained length were required (Cureton, 1987), there is some evidence that implies positive health returns regardless of mode, intensity, frequency and duration of exercise (Armstrong, 1989). The suggestion that PA elicits health benefits irrespective of intensity, frequency and duration is important, since several investigators have reported sex differences in activity levels. In his review of physical activity studies, Sallis (1993) concluded that boys were generally more active than girls and that the decline in activity levels seen with increasing age is greater in females. The observation that boys were more active than girls has been supported in more recent studies (van Mechelen, Twisk, Post *et al.* 2000; Guerra, Duarte and Mota, 2001; Mota *et al.* 2002; Trost, Pate, Sallis *et al.* 2002). In an investigation of Welsh children aged 11 to 16 years, the Welsh Youth Survey ascertained that 80% of 11-year old boys took part in vigorous activity outside school on two or more occasions per week, a figure that dropped to 70% for 16 year olds (Heartbeat Wales, 1986). For girls,

the corresponding figures were an alarming 63% and 33%. Thirlaway and Benton (1993) similarly investigated the physical activity levels of Welsh youngsters, focussing on West Glamorgan. They also found boys were generally more active than girls, particularly so at secondary school level, and that younger children were inclined to be more active than their older counterparts. The sex issue was in agreement with the findings of Duncan, Woodfield, Al-Nakeeb *et al.* (2002) who reported that 41% of British boys were classed as active compared to 24% of girls aged 11-14 years. The message is similar across Europe. In their investigation of German teenagers, Ilmarinen and Rutenfranz (1980) found that the activity levels of both sexes declined significantly from 14 years onwards. Kemper (1985) studied the habits of Dutch children and reported that the total time spent participating in activity declined gradually from the age of 12 to 13 years through 17 to 18 years. They also concluded that boys were more inclined to take on vigorous activity. In the USA, the National Children and Youth Fitness Study, Phase 1 (NCYFS-1) provided extensive data on the activity habits of U.S adolescents during the mid 1980s (Ross and Gilbert, 1985). The research similarly identified boys as being more active than girls, and that levels for both sexes declined during adolescence. According to the NCYFS-1, adolescents spend on average approximately one hour per day participating in moderate-to-vigorous activity, although it is accepted that the extremely high levels of activity of a small number of children would skew the data (Pate *et al.* 1994).

Evidence as to whether the younger generation is sufficiently active for optimal health remains inconclusive partly because the assessment of physical activity, especially amongst the paediatric population, is one of the most problematic tasks in epidemiological research (Trost *et al.* 2002). Paffenbarger, Blair, Lee *et al.* (1993, p 61) stated 'physical activity is so complex a life-element that its measurement and

assessment tend to be as highly complicated and difficult, as they are important'. Variability in protocols for assessing physical activity levels has led to an inconsistency in the findings. When self-report methods are employed 60-70% of children are considered sufficiently active for optimal health, however, when more objective methods are used, activity levels are significantly lower (Riddoch and Boreham, 1995; Trost *et al.* 2002). According to some (Melansson and Freedson, 1996), physical activity is a variable behaviour and therefore cannot easily be assessed, whereas others have treated it from a physiological perspective and have measured energy expenditure (Sallis *et al.* 1988; Boreham *et al.* 1993). Whilst such inconsistencies in the measurement of PA exist, exacerbated by the fact that there is no universally accepted "gold standard" to validate measures of PA, it is understandable that investigators disagree not only on its measurement, but also on the significance of any findings (Melansson and Freedson, 1996). There is no individual protocol that is deemed appropriate for all purposes, and if accurate data on the epidemiology of PA in young people is to be gained, each sample needs to be treated as a unique cohort (Melanson and Freedson, 1996). It is highly likely that the employment of a combination of assessment procedures is the best solution (Armstrong and van Mechelen, 1998).

Methods of physical activity assessment, include motion sensors, heart-rate monitoring, doubly-labelled water techniques, diaries, self-administered questionnaires, interviewer-administered questionnaires, recalls e.g., one-day, three-day and seven-day, direct observation, and accelerometers (Sallis, Buono and Roby, 1990; Armstrong, 1998; Riddoch *et al.* 1991; Kelly, 2000; Bouten, Wilhelme, van de Venne *et al.* 1996; Campbell, Katzmarzyk, Malina *et al.* 2000; Tell and Vellar, 1988; Linder, Durant and Mahoney, 1983; Wallace, McKenzie and Nader, 1985; Riddoch, 1990; Sallis *et al.* 1993; Trost *et al.* 2002). The most objective of these assessment measures are heart-rate

monitoring, observation, motion sensor monitoring and accelerometers (Melansson and Freedson, 1996; Armstrong and Welsman, 1997; Trost *et al.* 2002). However, despite the improved objectivity of these protocols; diaries, recalls, and questionnaires are the most favoured for larger studies as they do not pose as many practical and financial problems. Moreover, if during data collection children are required to wear heart-rate monitors or are aware that they are being observed, they might alter their normal behaviour. Admittedly, this limitation has been similarly cited for diary keeping.

If questionnaires are used to determine the physical activity levels of children, it is important that the normal daily routines of the particular cohort are considered and that allowances are made for the younger child's limited cognitive abilities. According to Montoye, Kemper, Saris *et al.* (1996) if consistency in the scoring and analysis of questionnaires is to be achieved, objective responses must be sought. Questionnaires should not over burden the child by requiring lengthy responses to too many questions. Ideally, queries should request information on transportation to and from school, participation in Physical Education lessons, break and lunchtime activities, extra-curricular activities, and evening and weekend pursuits (Montoye *et al.* 1996). Generally, children are unable to successfully estimate the duration and intensity of activities, but such information is paramount to our increased understanding of childrens' activity. A number of questionnaires have attempted to gather data on intensity of exercise by providing easily recognisable cues such as sweating or breathlessness (Heartbeat Wales, 1986; Riddoch, 1990). Montoye and collaborators (1996) suggested that children were better able to record strenuous activity, and if a protocol does lack sensitivity, this might result in unseen slight differences, which although small might be significant in health terms (Montoye *et al.* 1996). Bouchard *et*

al. (1983) claimed that despite their limitations, questionnaires are frequently able to differentiate between individuals of different energy expenditure habits.

Riddoch (1990) included a seven-day recall and lifestyle questionnaire in his investigation of the activity levels of Irish schoolchildren. The seven-day physical activity recall questionnaire was originally developed in 1979 for use in the Stanford Five City Project (Sallis, Haskell, Wood *et al.* 1985). Despite the claims of Baranowski, Dworkin, Cieslik *et al.* (1984) that diaries have been an improvement on retrospective questionnaires, this latter form of assessing physical activity habits is often deemed more appropriate than keeping a journal as it is less time consuming and does not influence normal activity. Nevertheless, there is an obvious limitation to a recall type question in that the period for which data are collected is assumed to be representative of all weeks. Riddoch (1990) claimed that since school children follow a set pattern of activities during a school week, seven days is an appropriate time period for evaluation. This finding was supported by others (Sallis *et al.* 1993). Interestingly, Sallis *et al.* (1993) also found that males were more reliable in their recall than their female peer group, possibly as a consequence of PA being more discernible to boys. Wallace *et al.* (1985) investigated the validity and reliability of a seven-day recall questionnaire with 11-13 year old boys. Unbeknown to the children, their activities were recorded by a trained observer every 15 minutes for an entire week. The recall questionnaire was completed on the day following the final day of activity. Immediate completion of the recall questionnaire is itself important as an increase in delay between the activity and the report will decrease reliability (Sallis, 1991). The authors determined that although children could only remember 46% of the mode of activity over a week, preliminary studies indicated adequate validity for the recall type of questionnaire in relation to direct observation. Wallace *et al.* (1985) added that both reliability and validity of recall

interviews improve with age, a view supported by others (Sallis, Buono, Roby *et al.* 1990; Sallis, 1991). It is difficult to assess the validity of questionnaires. Some investigators have attempted to validate the protocol against physical fitness, fatness, heart - rate, and diary type of protocol, but such approaches are of limited value as the potential for error in these methods is similarly large (Montoye *et al.* 1996). According to Riddoch (1990), when children are requested to recall activity there is a possibility of making two types of error. Firstly, children tend to over - estimate duration and intensity; and secondly, there is incomplete recall over longer periods of time. In his study Riddoch (1990) accepted that the questions posed related to how long children were involved in an activity, not to time spent being physically active. In Riddoch's (1990) defence, no attempt was made to interpret the data as a physiological measure of activity.

Pate (1993) reported that test-retest data from studies indicated much variation in the reliability of self-report measures (0.47 - 0.96). At first glance this would imply that the methods are untrustworthy, however, it might indicate the natural variation in habitual activity behaviour from one day to the next (Pate, 1993). It is worth remembering that despite the frequent use of recall questionnaires, information collated in this way must be viewed with caution; the data are subject to error and should be viewed as an estimation (Sallis, 1991). It is therefore essential that improvements continue to be made to the accuracy of existing methods (Melansson and Freedson, 1996) and that the protocols adopted in studies are socially acceptable, not laborious, minimally influence normal activity habits and provide information on intensity, frequency and duration (Armstrong and van Mechelen, 1998).

The inevitability of time restrictions and confounding variables means that it is unlikely that we will ever have direct evidence that exercise during childhood benefits the adult health profile (Rowland, 1996). If the true benefits of activity are to be identified, longitudinal data on various cohorts using identical protocol are needed. Nevertheless, cross-sectional studies remain important modes of data collection as they allow investigators to describe both the risk factor profile in a population and the relationship between these aspects (Andersen and Haraldsdottir, 1995).

2.2.5 Diet

Over-consumption is prevalent in Western society (Bergstrom, Hernell and Persson, 1993; Forbes, 1995), and as with other lifestyle patterns, children and adolescents tend to maintain their eating habits into later life (Voller and Strong, 1981; McGinnis, 1992). A well-balanced diet is one that provides adequate amounts of carbohydrate, fat, protein, minerals, vitamins and water (World Health Organization (WHO), 1990; American Heart Association (AHA), 2000). Adopting a high carbohydrate, low fat diet ensures an adequate supply of energy, as well as keeping levels of triglycerides (TG) and total cholesterol (TC) to a minimum. The amount of energy necessary to sustain a healthy state varies according to sex, age and activity and making specific recommendations for the general population is difficult. Since children and adolescents are growing individuals whose levels of activity vary greatly, so too will their energy requirements (Griffin, 1991; Malina and Bouchard, 1991). Adolescence in particular, is a period of rapid physical development, and growth itself is probably the best single bioassay of nutritional status (Forbes, 1995; Woteki and Filer, 1995). From early childhood to post adolescence, boys require more food than girls, which is to be expected since for this age range boys are generally taller and heavier than girls (Forbes, 1995; Post and Welten, 1995). Studies of adolescents have reported that daily

expenditure changes by 20 kcal for each kilogram of mass difference in females and by 17 $kcal \cdot kg^{-1}$ for males (Bandini, Schoeller, Cyr *et al.* 1990). Although governments of individual countries publish their own recommended daily amounts or allowances (RDAs) (Malina and Bouchard, 1991), it is worth considering that these values are better suited to suppliers of food as opposed to individuals (Griffin, 1991). Recommended daily amounts represent informed guesses with significant safety margins built in (Woteki and Filer, 1995).

The relationship between dietary fat and CHD has been widely explored (World Health Organization (WHO), 1990; Kuczmarski, Flegal, Campbell *et al.* 1994; Bray and Popkin, 1998; AHA, 2000). The Committee on Medical Aspects of Food Policy (COMA) (1991) recommended that total fat intake should provide no more than 35% of the total energy of the diet, whilst saturated fat should contribute no more than 11% (Department of Health (DOH), 1994). This was considered appropriate for individuals aged 5 years and above (DOH, 1994). A lower value of 30% has been proposed for Americans (Cheung, 1995; AHA, 2000) and by the World Health Organization (WHO) (1990). The WHO (1999) reported that for the UK population, fat intake accounted for 38% and 39% of the total daily energy for males and females respectively, though on a more positive note, the WHO (1999) did identify a decrease in fat intake in Northern and Western European countries. Despite some evidence that total fat intake has not changed greatly and even decreased over the last decade (Cheung, 1995; WHO, 1999), it has been suggested that total daily fat intake amongst young people is too high (Post and Welten, 1995; Fulton *et al.* 2001; Buttriss, 2002). In 1972, McGandy, Hall, Ford and co-workers investigated the dietary habits of 13 to 18 year old boys and found their diets to be high in total and saturated fats. Despite improved knowledge of healthy living, present day children and adolescents are similarly subject to excess fat in their

diet. Boreham *et al.* (1993) randomly selected a sample of Northern Irish children aged 12 to 15 years and found that fat intake provided approximately 40% of total daily energy. Boreham and colleagues (1993) also reported relatively low polyunsaturated to saturated fatty acid ratios, a finding previously observed in American adolescents (Witschi, Cooper, Ellison *et al.* 1990). The Amsterdam Growth Study examined the dietary habits of 200 males and females between the ages of 13 and 21 years, and similarly concluded that the daily food intake of teenagers and young adults was too high in total fat and too low in polyunsaturated fatty acids (Post and Welten, 1995). This can be partly explained by adolescents becoming more reliant on snacks and fast foods as sources of energy (Voller and Strong, 1981; McGinnis, 1992; Woteki and Filer, 1995). Such claims of elevated levels of saturated fat are especially disconcerting since adult surveys have reported direct relationships between fat, especially saturated fat, and unfavourable blood lipid profiles (Study Group of European Atherosclerosis Society, 1987; American Heart Association, 2000). Although there is a paucity of similar data on children, evidence gathered to date supports this notion (Turpeinen, Karvonen, Pekkarinen *et al.* 1979; Morrison, Larsen, Glatfelter *et al.* 1980; Kushi, Lew and Stare, 1985; Shea, Basch, Irigoyen *et al.* 1991). Nevertheless, there is some evidence to the contrary. The Amsterdam Growth Study failed to identify a relationship between increased fat intake and body fat and TC (Post and Welden, 1995), though this was partially attributed to the relatively low absolute values of these risk factors in the young cohort (Post and Welden, 1995). Suter and Hawes (1993) examined the relationship between risk factors of CHD in youths aged 10 to 15 years and reported that although saturated fat intake and dietary fat cholesterol was positively related to total cholesterol in boys, this was not significant ($r = 0.34$ and $r = 0.31$ $P > 0.05$). The authors intimated that the relatively low intake of saturated fat and cholesterol amongst

their population, coupled with differences in experimental protocol could have led to these findings.

The link between dietary habits and obesity in children has been established (McGloin, Livingstone, Greene *et al.* 2002), however, it is not known whether children and adolescents become overweight by consuming too many calories. Shah and Jeffrey (1991) reviewed eleven studies of childrens' eating habits and contrary to expectations, only three of these investigations reported high-energy intake amongst obese individuals. Nevertheless, since overweight individuals are inclined to underestimate their total calorific intake, conclusive evidence is difficult (Bandini *et al.* 1990; Brodney, McPherson, Carpenter *et al.* 2001). In addition to this, there are other methodological problems when assessing dietary patterns including day-to-day variations and accuracy of amounts recorded (Suter and Hawes, 1993).

Several investigators have sought to identify the most effective way of facilitating weight loss and hence decreasing the likelihood of CHD. Although the preferred intervention protocol is through a combination of diet and exercise (Fulton *et al.* 2001) there have been successful outcomes in diet alone interventions (Puska, Vartiainen, Pallonen *et al.* 1982; Becque, Katch and Rocchini, 1988). Becque *et al.* (1988) conducted an intervention programme on 36 obese adolescents. The cohort were randomised into three groups: (i) diet, (ii) diet and exercise, (iii) control. Following 20 weeks training, the authors reported a mean increase of 3.2 kg body mass in the control group; a mean decrease of 0.4 kg in the diet alone group; and a more beneficial mean decrease of 1.6 kg in the diet and exercise group. The combined diet and exercise group also posted the more favourable body fat percentage. Similar findings had been reported in previous work (Puska *et al.* 1982; Nader, Stone, Lytle *et al.* 1999).

Young people spend a considerable amount of their young lives in education, consequently amongst this particular population school-based programmes could be used to promote healthy eating. If such schemes are to succeed, governments need to be proactive in the implementation of school-based health strategies (Cheung, 1995; Fox, 1997; Biddle, Sallis and Cavill, 1998; National Health Forum, 2002). School meals account for 25%-40% of total daily energy intake (Woteki and Filer, 1995) it is therefore essential that these meals are nutritionally sound. Although school-based programmes would be worthwhile (Parcel, Simons-Morton, O'Hara *et al.* 1989), the 1980 Education Act absolved the Local Education Authorities of the UK from any legal obligation to meet specific nutritional standards in their school meals (Griffin, 1991). Hence, although the vast majority of schools do provide school meals, they are not obliged to do so. It is encouraging that in their Consultation Paper, 'Better Health, Better Wales'; the Welsh Office detailed the Welsh Assembly's intention to introduce minimum nutritional standards for school meals (Welsh Office, 1998). Several school-based health projects have been successfully implemented. For example, the Child and Adolescent Trial for Cardiovascular Health (CATCH) intervention (Luepker, Perry, McKinlay *et al.* 1996) involved 96 public elementary schools and included children from various ethnic and geographic backgrounds. CATCH successfully modified the fat content of school lunches, increased the amount of vigorous activity included in the PE programme, and improved the eating and activity habits of schoolchildren. The Planet Health Study (Gortmaker *et al.* 1999) was a similarly successful school-based programme. The aim of the project was to decrease obesity by increasing energy expenditure and promoting favourable dietary habits amongst 11 to 14 year olds (Gortmaker *et al.* 1999). Amongst intervention schools, obesity decreased from 23.6% to 20.3% in girls, whilst girls in the control schools showed an increase in obesity of 2.2%. For boys, a decrease in obesity was seen in both intervention and control groups

(Gortmaker *et al.* 1999). The outcomes from school-based intervention studies have been promising, however these programmes need to be implemented at national level if they are to prove worthwhile to society in general (Fulton *et al.* 2001).

2.2.6 Smoking

Smoking increases the risk of CHD. In the UK, approximately 20% of deaths from CHD in men and 17% of deaths from CHD in women are due to smoking (National Health Forum, in press). It is during the teenage years that this lifestyle habit takes hold, and even moderate smoking during adolescence and young adulthood could have damaging long-term consequences (Armstrong and Davies, 1980; Twisk *et al.* 1997; Boreham *et al.* 1997; Williams *et al.* 2002). According to the Office for National Statistics, 10% of 11-15 year olds were regular smokers in 2000 (Office for National Statistics, 2001). In the USA, the National Institute on Drug Abuse revealed that 64.4% of high school seniors reported having smoked cigarettes (Johnston, O'Malley and Bachman, 1991). Despite cigarette smoking being negatively associated with physical fitness and physical activity levels in young people (Raitakari, Leino, Räikkönen *et al.* 1995), there is some evidence that smoking does not detrimentally affect fitness levels in young people (Andersen, Henckel and Saltin, 1989).

2.2.7 Blood lipids and lipoproteins

Numerous epidemiologic investigations have indicated that coronary heart disease incidence is related to an unfavourable blood lipid profile. Total cholesterol (TC) is the most prominent member of the steroid family. Like triglyceride, it is composed of non-polar hydrophobic molecules. Cholesterol does not dissolve in blood and therefore must be combined with proteins to facilitate haematogenous transport the combinations thus formed are lipoproteins (Zubay, 1998). Lipoproteins are complex, soluble

macromolecules consisting of fat and protein; they serve to transport fat in the blood (Goldberg and Elliott, 1987). There are four main categories of lipoproteins and these are defined according to their density (a function of the lipid: protein ratio) (Goldberg and Elliott, 1987; Brewer, Gregg, Hoeg *et al.* 1988; Newsholme and Leech, 1992; Zubay, 1998). Chylomicrons are the largest of the lipoproteins and have the lowest density. Very low density lipoprotein-cholesterol (VLDL-C) have a lower lipid/protein ratio and are smaller than chylomicrons, but like chylomicrons they too contain a high proportion of apolipoprotein B (Apo B) (Zubay, 1998). Triglyceride is the principal lipid of both chylomicrons and VLDL-C (Goldberg and Elliott, 1987). Low density lipoprotein-cholesterol (LDL-C) has been shown to have a causal role in CHD because of its tendency to oxidise and enter the artery wall (Goldberg and Elliott, 1987; Brewer *et al.* 1988; Von Duvillard, 1997; Hickman, Briefel, Carroll *et al.* 1998; Zubay, 1998); it similarly contains apo B (Von Duvillard, 1997; Zubay, 1998; Lamarche, Lemieux and Despres, 1999). Since LDL-C is considered atherogenic, decreasing this lipoprotein could lessen the likelihood of developing CHD (Castelli, Doyle, Gordon *et al.* 1977; Gordon *et al.* 1977; National Heart, Lung and Blood Institute (NHLBI), 1984; Grundy, 1997). A decrease of 0.5 $mmol \cdot L^{-1}$ (22.3 $mg \cdot dl^{-1}$) (10.4%) in LDL-C has been associated with a 16% to 19% decrement in CHD risk (Lipid Research Clinics Programme, 1984). Conversely, high density lipoprotein-cholesterol (HDL-C) is recognised as antiatherogenic and has a strong, inverse relationship with CHD (NHLBI, 1984; Bush, Fried and Barrett-Connor, 1988; Rowland, 1990; Hickman *et al.* 1998; Zubay, 1998; Kokkinos and Fernhall, 1999). It is thought to facilitate the transport of cholesterol from peripheral tissues to the liver (Grundy and Denke, 1990; Von Duvillard, 1997) and is a stronger predictor of atherosclerosis than TC (Gordon *et al.* 1977; Goldberg and Elliott, 1987). A low concentration of HDL-C is considered to detrimentally affect the body's ability to remove cholesterol (NIH Consensus Panel, 1993; Von Duvillard, 1997;

Zubay, 1998), and below average values have been identified in approximately 40% of adult males suffering from CHD (Castelli *et al.* 1977). It has been reported that an increase in HDL-C independently accounts for a 2% decrease in CHD risk (NHLBI, 1984).

Although European wide estimates of death caused by high cholesterol levels are yet to be published (British Heart Foundation, 2000), it has long been accepted that total cholesterol plays a decisive role in the development of CHD (Lipid Research Clinics Program, 1984; Bush *et al.* 1988; Hickman *et al.* 1998; Zubay, 1998; Sharrett, Sorlie, Chambless *et al.* 1999). The risk is a continuous one, the greater the rise above 4.7 $mmol \cdot L^{-1}$, the greater the risk of developing CHD (Stamler, Wentworth and Neaton, 1986; Rowland, 1990). Total cholesterol levels of below 5.2 $mmol \cdot L^{-1}$ (200 $mg \cdot dl^{-1}$) are considered desirable for the adult population (American Heart Association Nutrition Committee, 1982; National Cholesterol Education Program, 1989). Reporting on the Framingham Study, Voller and Strong (1981) stated that males aged 30 to 39 years whose cholesterol levels were 6.7 $mmol \cdot L^{-1}$ or over, had a three times greater incidence of CHD than those with levels of 5.2 $mmol \cdot L^{-1}$ or less. It is generally agreed that young individuals recording values of 5.2 $mmol \cdot L^{-1}$ or over should be considered at risk of developing CHD in later life, whilst values of less than 4.9 $mmol \cdot L^{-1}$ are indicative of a healthy lipid profile (American Heart Foundation, 1979; Bell *et al.* 1986; Malina and Bouchard, 1991; NCEP, 1991; Bouziotas, Koutedakis, Shiner *et al.* 2001). According to Rowland (1990), for both boys and girls, the mean cholesterol value is about 4.14 $mmol \cdot L^{-1}$. Data from the Third National Health and Nutrition Examination Survey (NHANES III) was used to estimate mean and percentile distributions of serum TC in young people aged 4 to 19 years. Mean total cholesterol levels for 12 to 15 year old Americans was 4.16 ± 0.03 $mmol \cdot L^{-1}$ (mean $\pm$ SEM) confirming a decrease of 0.18

mmol·L^{-1} over the last thirty years (Hickman *et al.* 1998). A slightly higher value of 4.62 ± 0.75 mmol·L^{-1} (mean ± SD) was reported in British children aged 11 to 16 years (Armstrong *et al.* 1991[a]). In the 1970s, The Muscatine Study reported that 24% of 5,000 youngsters had a TC level greater than 5.2 mmol·L^{-1} (Lauer *et al.* 1975), whereas Gilliam and co-workers (1977) stated that 10.5% of their cohort had cholesterol values exceeding 5.2 mmol·L^{-1}. Wilmore and McNamara (1974) noted that in their group of 8 to 12 year old boys, 20% exceeded the same 5.2 mmol·L^{-1} cut-off point, although in a later investigation of older boys (13 to 15 year olds), Wilmore and colleagues (1982), reported that a slightly more acceptable 11% had exceeded the threshold. More recently, a study of Greek children aged 12 years, revealed that only 5% of the study cohort exceeded the same threshold point (Bouziotas *et al.* 2001). When screening for risk factors of chronic disease in children from 15 countries, Wynder and collaborators (1981) arbitrarily designated a lower cut-off point of 4.7 mmol·L^{-1} for TC. The authors found that Finnish children had the highest mean values for TC, whilst the lowest mean values were found in Nigerian, Greek and Italian children.

When measured directly, LDL-C values greater than 2.2 mmol·L^{-1} are considered a health risk for children and adolescents. Young individuals with calculated values of more than 3.8 mmol·L^{-1} are also at risk, whereas anything less than 1.8 mmol·L^{-1} is considered indicative of a healthy profile (Masopust, Máček, Rutenfranz *et al.* 1985; Bell *et al.* 1986; Bouziotas *et al.* 2001).). The NHANES III of young people reported a mean LDL-C level of 2.35 ± 0.05 mmol·L^{-1} (mean ± SEM) for adolescents aged 12 to 15 years (Hickman *et al.* 1998).

The National Cholesterol Education Program (1991) and the American Heart Association (Williams *et al.* 2002) recommended that for children, HDL-C values of

less than 0.9 mmol·L^{-1} are indicative of an adverse lipid profile, while any value greater than 1.3 mmol·L^{-1} is considered to reflect a healthy status (Bell *et al.* 1986; NCEP, 1991; Bouziotas *et al.* 2001). Cresanta, Srinivasan, Webber *et al.* (1984) reported that average HDL-C values for both sexes approximate 1.3 - 1.6 mmol·L^{-1} from ages 2 to 14 years and then decrease about 0.26 mmol·L^{-1} to adult levels. Data from the NHANES III revealed a mean HDL-C level of 1.29 ± 0.02 mmol·L^{-1} (mean ± SEM) for US adolescents (Hickman *et al.* 1998), while a smaller study of 11 to 16 year old British children reported a mean concentration of 1.43 mmol·L^{-1} ± 0.31 mmol·L^{-1} (mean ± SD) (Armstrong *et al.* 1991[a]). In children, HDL-C accounts for 3% of total cholesterol, whereas LDL-C accounts for 40%-60% of total cholesterol (Berenson and Epstein, 1983). Although changes in lipoprotein concentrations are known to occur during maturation, we are yet to ascertain why lipoprotein levels change more in 'high-risk' than 'low-risk' populations (Berenson, Srinivasan, Nicklas *et al.* 1988). In adults, HDL-C levels are higher in females than in males and this might account for the increased incidence of CHD in the latter.

The atherogenecity of total cholesterol depends upon the relative contribution of both low and high-density lipoproteins (Zubay, 1998), furthermore, researchers reporting on TC levels should recognise that changes to CHD risk could be attributed to changes in the ratio of the lipoproteins as opposed to a changes in TC (Gordon *et al.* 1977). Some researchers have claimed that the ratio of HDL-C to TC, and the ratio of LDL to HDL-C are more predictive of CHD than considering these variables independently (Bell *et al.* 1986; Rowland, 1990). In relation to HDL-C to TC, a ratio greater than 0.3 has been suggested as a health-indicator, whereas a ratio of anything less than 0.18 has been proposed to elevate CHD risk (Montoye, 1985). In their study of Danish 16 to 19 year olds, Andersen and co-workers (1989) reported a ratio in the range of 0.25-0.28 in both

sexes. This did not compare favourably with other industrialised countries. Alternatively, when TC to HDL-C is measured, a ratio of 4.0 is used as a cut-off point. Anything above this value is considered a health risk.

The exact role of hypertriglyceridemia in the development of atherosclerosis is equivocal and definitive data are sparse (Austin, 1989; NIH Consensus Development Panel, 1993; Harjai, 1999). Previously, several studies had identified a less significant correlation between triglycerides and CHD prevalence (Gordon *et al.* 1977; Castelli *et al.* 1977), but more recent investigations support the notion that excessive TG levels are an independent cardiac risk factor (Austin, 1989; Assmann, Schulte, Funke *et al.* 1998; Austin, 1999; Harjai, 1999; Rubins, 2000). For example, Gaziano, Hennekens, O'Donnell and co-workers (1997) concluded that fasting levels of TG are significant independent indicators of heart attack risk.

According to data collated in The Bogalusa Study, mean triglyceride levels are fairly stable at 0.62 - 0.68 $mmol \cdot L^{-1}$ until puberty, and during puberty values increase to 0.90 - 1.02 $mmol \cdot L^{-1}$ (Berenson and Epstein, 1983). A TG value of 1.7 $mmol \cdot L^{-1}$ or greater has been considered a health risk for all populations (Montoye, 1985, Bell *et al.* 1986; Bouziotas *et al.* 2001; National Cholesterol Education Program, 2002). Andersen *et al.* (1989) investigated the TG levels in older teenagers (16-19 years old) and found that concentrations averaged 0.82 $mmol \cdot L^{-1}$ for young males and a slightly higher 0.86 $mmol \cdot L^{-1}$ for young females. The 90^{th} percentile was reported at 1.18 $mmol \cdot L^{-1}$ and 1.30 $mmol \cdot L^{-1}$ for boys and girls respectively. These values were similar to data gathered on similar aged youngsters from other industrialised countries. In their study of 8 to 12 year old boys, Wilmore and McNamara (1974) identified 8.4% as having TG levels of over 1.13 $mmol \cdot L^{-1}$. A subsequent study by Wilmore *et al.* (1982) reported that 25% of

13 to 15 year old males exceeded this threshold. Gilliam and co-workers (1977) had previously identified 18% of their cohort in excess of 1.13 mmol·L^{-1}, whilst Lauer and colleagues (1975) reported 15% in excess of 1.58 mmol·L^{-1}. A recent study of Greek boys and girls found that only 1% of the cohort exceeded the 'at risk' cut-off point of 1.7 mmol·L^{-1}, whilst 23% of schoolchildren were considered 'borderline' risk (Bouziotas *et al.* 2001). The benefit of TG reduction remains mainly indirect. Modes of therapy including weight loss, reduced alcohol consumption and physical activity, all of which decrease triglyceride concentrations and might lead to decreased risk of CHD independent of the positive impact on TG levels (Harjai, 1999).

Concentrations of TC, LDL-C, HDL-C and TG have been tracked by many investigators (Laskarzewski, Morrison, de Groot *et al.* 1979; Lauer, Lee and Clarke, 1988; Cunnane, 1993; Twisk *et al.* 1995, Hickman *et al.* 1998; Nicklas, von Duvillard and Berenson, 2002). Total cholesterol levels whether high or low, tend to be sustained across a number of years (Laskazewski *et al.* 1979; Berenson, Srinivasan, Nicklas *et al.* 1988; Lauer *et al.* 1988; Barker, Osmond, Golding *et al.* 1989; Malina and Bouchard, 1991; Williams *et al.* 2002). Barker *et al.* (1989) reported that 40% of individuals measured at or above the 90th percentile for TC remained at that level 10 to 20 years later, a theory supported by the findings of the Muscatine Study (Lauer *et al.* 1988). Low density lipoprotein levels are similarly maintained over a period of time (Berenson *et al.* 1988; Lauer *et al.* 1988; Nicklas *et al.* 2002), with correlations of approximately 0.7 being cited for a period of 5 to 6 years (Malina and Bouchard, 1991). Laskazewski *et al.* (1979) concurred with the theory that levels tend to remain similar from 11 to 15 years of age, but reported that for their particular cohort of youngsters there was some decrease in LDL-C levels over the four year period of the study. High density lipoprotein and triglyceride levels are also thought to remain fairly stable over time

(Laskazewski *et al.* 1979), though correlations for TG are the lowest at 0.3 to 0.4 (Malina and Bouchard, 1991). More research is needed to confirm the relationship between childhood and adulthood lipid levels, nevertheless the suggestion that children with elevated levels tend to maintain these levels into later life (Bao, Sathanur, Srinivasan *et al.* 1996[a]), implies that this population should be monitored at regular intervals.

The associations between lipid and lipoprotein levels, and physical fitness and/or activity have been extensively explored, though information concerning young people remains equivocal (Armstrong and Simons-Morton, 1994). Confirmation or refutation of relationships are often confounded by the influence of body fatness, growth and sex (Máček, Rutenfranz, Lange Andersen *et al.* 1985; Sallis, Patterson, Buono *et al.* 1988; Armstrong *et al.* 1991[a]; Torfley, Batterham and Campbell *et al.* 1997), as well as the majority of studies being cross-sectional in design (Twisk, 2000). Because of these limitations, any findings should be viewed with caution. Wilmore and McNamara (1974) examined the relationship between adolescents' $\dot{V}O_2$ max and blood lipid status and failed to confirm significant relationships between these variables. Later studies have further explored the effect of training or activity status on total cholesterol concentrations in children and adolescents and have concluded that TC is largely unaffected by exercise stimulus (Thorland and Gilliam, 1981; Atomi, Kuroda, Asami *et al.* 1986; Tell and Vellar, 1988; Armstrong *et al.* 1990[b]; Kwee and Wilmore, 1990; Armstrong *et al.* 1991[a]; Al-Hazzaa *et al.* 1994). Despite this, others have reported that increased $\dot{V}O_2$ max has a favourable effect on TC, implying that this aspect merits further exploration (Wilmore *et al.* 1982; Máček *et al.* 1985; Hager *et al.* 1995).

Several investigators have failed to confirm significant relationships between HDL-C and $\dot{V}O_2$ max in young people (Sallis *et al.* 1988; Armstrong *et al.* 1990[b]; Kwee and Wilmore; 1990; Armstrong *et al.* 1991[a]; Dwyer and Gibbons, 1994; Twisk *et al.* 2002). Nevertheless, there is much evidence to the contrary (Bush *et al.* 1988; Stewart and Goldberg, 1992; NIH Consensus Development Panel, 1993). In the Oslo Youth Study a positive relationship was observed between the HDL-C status of adolescents and aerobic fitness; an association confirmed by others (Tell and Vellar, 1988; Atomi *et al.* 1986; Hofman and Walter, 1989; Stewart and Goldberg, 1992; NIH Consensus Development Panel, 1993; Bistritzer, Rosenzweig, Barr *et al.* 1995; Tolfrey *et al.* 1997). Intervention studies similarly yield conflicting findings. Rimmer and Looney (1997) conducted an experimental study on a cohort of adolescents to determine whether an aerobic fitness programme would alter TC and HDL-C levels. The authors found that changes in lipid and lipoprotein concentrations differed significantly by participant, and that individual genotype could have a confounding effect (Rimmer and Looney, 1997). The Child and Adolescent Trial for Cardiovascular Health (CATCH) (Webber *et al.* 1996) implemented a two and a half year health and fitness intervention on 4000 children and adolescents. There were no significant differences in TC and HDL-C levels post intervention. A shorter intervention programme also failed to confirm changes in HDL-C (Linder *et al.* 1983).

It has been suggested that particular physical activity traits could have a positive effect on the serum lipid profile of adults, and the possibility of this eventuality occurring in the younger population deserves consideration (Thorland and Gilliam, 1981; Rowland, 1996). Whereas several studies have failed to identify significant differences between the lipid profiles of active and sedentary youngsters (Máček *et al.* 1985; Armstrong *et al.* 1990[b]; Armstrong *et al.* 1991[a]), others have confirmed that active children have

higher HDL-C and lower TG levels (Linder *et al.* 1983; Tell and Vellar, 1988; Suter and Hawes, 1993; Raitkari *et al.* 1997). Although Thorland and Gilliam (1981) failed to identify significant differences between the lipid profiles of high and low active pre-adolescent males, they too reported differences in the ratio between the two lipids. Much of the discrepancy could be attributed to different protocols being used to estimate physical activity hence confounding the available evidence (Thorland and Gilliam, 1981; Armstrong and Simons-Morton, 1994; Montoye *et al.* 1996). Spanning a period of 15 years, The Amsterdam Growth and Health Study (Twisk *et al.* 1997; Twisk *et al.* 2002) failed to identify an association between physical activity levels and TC and HDL-C: TC ratio, although a positive relationship was reported between physical activity and development of HDL-C. In the Cardiovascular Risk Young Finns Study (Raitakari *et al.* 1994) adolescents were initially measured at 12 years, then at 18 years of age. Boys who remained active from 12 years through to 18 years exhibited lower TC: HDL-C ratios than their sedentary peers. However, no differences were observed when these measures were treated independently, furthermore there were no significant differences between the female groups (Raitakari *et al.* 1994). Raitakari and co-workers (1994) also investigated the relationship between physical activity and TG concentration. Although physical activity appeared to have a beneficial effect on triglyceride levels, after adjustment for insulin, the independent effect of physical activity on TG disappeared. This finding supported earlier reports that part of the effect of physical activity on TG might be mediated through insulin metabolism (Zimmett, Collins, Dowse *et al.* 1991). In conclusion, and as with adults (Bailey, Davies, Williams *et al.* 1998) there is some evidence that elevated levels of physical activity and fitness have a positive effect on lipid and lipoprotein status in children and adolescents. Despite several studies failing to identify a strong association between fitness and/or activity

and CHD risk factors in young people, efforts should be made to encourage an active lifestyle in this population.

Whereas apolipoprotein B (apo B) is associated with LDL-C, apolipoprotein A-1 (apo A-1) is linked with HDL-C (Goldberg and Elliot, 1987; Von Duvillard, 1997). Functionally, apo A-1 facilitates the action of lecithin cholesterol acetyl transferase (LCAT) whose enzyme converts free cholesterol into cholesterol esters. This process is essential as it enables excess unesterified cholesterol to be removed from lipoproteins and tissues (Von Duvillard, 1997). Apolipoprotein B is located on the surface coat of LDL-C and assists in its removal from the plasma by binding to LDL-C receptors on cells (Von Duvillard, 1997). It is considered by some to be a better predictor of CHD than LDL-C itself, especially when reference is made to the apo A-1 to apo B ratio (Clavel, Leavte, Javanel *et al.* 1997). Relatively little has been written on the apolipoprotein profiles of children, despite the notion that the concentrations of apo A-1 and apo B might be better indicators of future CHD (Berenson *et al.* 1988) and reports that children and adolescents from families with CHD have significantly higher levels of apo B (Glowinska, Urban, Koput, 2002). Nevertheless, Máček *et al.* (1985) proposed that in children, apo A values of lower than 1.4 $mmol \cdot L^{-1}$ should be considered a risk indicator, whereas values greater than 2.1 $mmol \cdot L^{-1}$, could be accepted as health indicators. For apo B, anything greater than 1.3 $mmol \cdot L^{-1}$ was proposed a risk, whilst a value of less than 0.8 $mmol \cdot L^{-1}$ was regarded as a health-indicator. In the Cardiovascular Risk in Young Finns Study, an inverse relationship was detected between physical activity levels and apo B concentrations in males; this was not true for females. No relationship was detected between physical activity and apo A-1 (Raitakari *et al.* 1997). When the same cohort was considered over a period of six years, active and inactive individuals posted similar apo A-1 and apo B levels (Raitkari *et al.* 1994).

Lipoprotein(a) (Lp(a)) is a distinctive lipoprotein complex in blood (Wild, Fortmann and Marcovina, 1997; Mackinnon and Hubinger, 1999). Although its composition is similar to that of LDL-C (Berg, 1963), it differs in that it contains the two proteins apo B 100 and apo (a), the latter of which is covalently linked to LDL-C by a disulfide bridge (Kronenburg, Steinmetz, Kostner *et al.* 1996) (Appendix 1). Whilst an Lp(a) level above 30 $mg \cdot dL^{-1}$ has been nominated as a threshold level for CHD risk in adults, it is also the threshold most frequently adopted for young people (Genzel-Boroviczény, Philipp, Kuhnle-Krahl *et al.* 1997; Chu, Makowski, Chang *et al.* 2000; Laskowska-Klita, Szymczak and Radomyska, 2001). Anything greater than 30 $mg \cdot dL^{-1}$ is believed to more than double the risk of developing CHD (Kronenburg *et al.* 1996). Population studies consistently show that serum Lp(a) levels are not normally distributed but are skewed towards low concentrations of less than 10 $mg \cdot dL^{-1}$ (Kronenburg *et al.* 1996; Mackinnon and Hubinger, 1999). This skewed distribution of Lp(a) is mostly attributed to variations in the apo (a) gene on chromosome 6 (Utermann, 1989). Only twenty percent of Caucasians are thought to have levels greater than 30 $mg \cdot dL^{-1}$ (Kronenburg *et al.* 1996).

There is accumulating evidence to suggest that high levels of lipoprotein(a) represent an independent risk for developing CHD and early myocardial infarction (Berg, Dahlen and Borreson, 1979; Kostner, Avogaro, Cozzalato *et al.* 1981; Sandkamp, Funke, Schulte *et al.* 1990; Scanu, 1992; Valentine, Grayburn, Vega *et al.* 1994; Kronenburg, *et al.* 1996). Nevertheless, it is the combined elevated levels of Lp(a) and LDL-C, and Lp(a) and fibrinogen, that are considered to have the greatest detrimental effect (Sveger, Flodmark, Nordborg *et al.* 2000; Cantin, Després, Lamarche *et al.* 2002). In a population of 175 schoolchildren aged 10 to 11 years, it was found that three of the children demonstrated combined elevated levels of Lp(a) and LDL-C, whereas 48 had

an isolated increase of Lp(a) concentration of some clinical importance (Sveger *et al.* 2000). Furthermore, in a study of 111 children, 23 had elevated levels of Lp(a) (>30 $mg \cdot dL^{-1}$), TC and LDL-C (Genzel-Boroviczény *et al.* 1997).

Serum Lp(a) is for the most part genetically determined by the apo (a) gene locus and remains fairly constant throughout life (Campbell, Tate, Lepre *et al.* 1992; Scanu, 1992; Clavel *et al.* 1997; Mackinnon and Hubinger, 1999). According to Boerwinkle, Leffert, Jingping *et al.* (1992), approximately 90% of an individual's Lp(a) level can be attributed to inherited genotype. Laskowska-Klita and colleagues (2001) investigated Lp(a) concentrations in hypercholesterolemic and normocholesterolemic children, and found that elevated Lp(a) levels, (> 30 $mg \cdot dL^{-1}$) were present in 45% of children with a positive family history of CHD. For children with no family history of CHD, the value was 29%. Although the findings of others (Kostner, Czinner, Pfeiffer *et al.* 1991; Srinivasan, Dahlen, Jarpa *et al.* 1991; Marquez, Mendoza, Carrasco *et al.* 1993; Vella and Jover, 1993; Wilcken, Wang, Greenwood *et al.* 1993; Glowinska *et al.* 2002; Cabrinety, Pisonero, Ajram *et al.* 2002) concur with the theory that Lp(a) levels are higher in groups with a family history of CHD, there are those who have reported otherwise (Okado, Sato, Yamazaki *et al.* 1995; Barth, Deckelbaum, Starc *et al.* 1999). Differences in methodological and analytical protocols are the most likely reason for the contradiction in findings. Lipoprotein(a) can be under or overestimated because of the size heterogenity of apo (a) (Marcovina, Albers, Gabel *et al.* 1995; Rocchini, 1999).

With the exception of niacin, lipid lowering drugs and diet are not thought to have a significant effect on Lp(a) concentration (Von Duvillard, 1997; Scanu, 1992; Mackinnon and Hubinger, 1999), however, some authors have claimed that physical activity and fitness could play an important role in lowering concentration (Van

Duvillard, 1997). Taimela, Viikari, Porkka *et al.* (1994) investigated the influence of physical activity on Lp(a) levels in Finnish young people (9-24 years) and confirmed a favourable inverse relationship i.e. that elevated levels of Lp(a) (> 25 $mg \cdot dL^{-1}$) were less apparent in the more active. MacAuley, McCrum, Stott *et al.* (1997) measured physical fitness using $\dot{V}O_2$ max estimated by extrapolation and identified a relationship between fitness and Lp(a) but for females only. Despite some scepticism regarding the association between physical activity and fitness, and Lp(a) levels, the possibility of an existence warrants further investigation.

There exists relatively little information about the distribution of Lp(a) in the paediatric population and internationally recognised reference values are yet to be established (Gozlan, Gross and Gruener, 1994). However, it could be argued that using Lp(a) levels to identify children at increased risk of CHD is ill-advised at the present time. There are limitations in immunoassay procedures and as yet, there is no effective treatment (Rocchini, 1999). Since values are not normally distributed, it is essential that large sample numbers be used in studies before any conclusions can be made (Kronenburg *et al.* 1996; Mackinnon and Hubinger, 1999).

2.3 RECENTLY IDENTIFIED CORONARY HEART DISEASE RISK FACTORS

Over the past two decades over 250 different variables have been identified as having an association with CHD (Choy, Mymin, Zhu *et al.* 2000). Established CHD risk factors such as smoking, obesity, hypercholesterolemia, hypertension and physical inactivity are thought to account for only a half to two thirds of CHD cases (Eikelboom, Lonn,

Genest *et al.* 1999) and in recent years a new group of potential CHD risk factors have emerged.

2.3.1 Homocyst(e)ine

Homocyst(e)ine (Hcy) is a sulphur-containing amino acid derived from methionine, an essential amino acid found in abundance in a typical Western diet (Malinow, Sexton, Averbuch *et al.* 1990; Fortin and Genest, 1995; Welch and Loscalzo, 1998; Malinow, Bostom and Krauss, 1999) (Appendix 1). Homocyst(e)ine is sometimes regarded as the by-product of the demethylation of methionine and it can be metabolised via two pathways (Fortin and Genest, 1995; Welch and Loscalzo, 1998; Eikelboom *et al.* 1999) (Appendix 1). If there is an excess of methionine, Hcy is directed to the transsulphuration pathway (Eikelboom *et al.* 1999), during which process, Hcy is metabolised to cystathionine by the action of cystathionine beta-synthase. For this to succeed, vitamin B_6 is required as a cofactor. The second pathway involves the remethylation of Hcy. This process requires methionine synthase, vitamin B_{12} as a cofactor, and methyltetrahydrofolate as a cosubstrate (Fortin and Genest, 1995; Eikelboom *et al.* 1999). The enzyme methylene tetrahydrofolate reductase and folic acid are essential contributors to this process. Since folate, vitamins B_6 and B_{12} are essential for the metabolism of Hcy to methionine, deficiencies in any one of these vitamins could lead to hyperhomocysteinemia (Cohen, Wilson, Chang *et al.* 1999), a condition of elevated homocyst(e)ine concentration. Such nutritional deficiencies might be the cause of most instances of moderate hyperhomocysteinemia (Eikelboom *et al.* 1999). Nevertheless ambiguity prevails with regards to whether a nutritional shortfall in any of these vitamins by itself can elevate Hcy levels, or whether this response is witnessed only in individuals with a genetic predisposition to hyperhomocysteinemia (Harjai, 1999).

As early as 1969, McCully proposed a relationship between elevated levels of Hcy and CHD (McCully, 1969; Clarke, 1998; Welch and Loscalzo, 1998). In recent studies, high levels of Hcy have been linked to endothelial damage, platelet activation and altered thrombus formation (Chambers, McGregor, Jean-Marie *et al.* 1998; Clarke, 1998; Welch and Lascalzo, 1998; Cohen *et al.* 1999; Eikelboom *et al.* 1999; Warsi, Hullin, Lewis *et al.* 2002). Nevertheless, conclusive evidence for a causal link between Hcy and CHD is sparse, and absolute confirmation of the mechanisms by which homocyst(e)ine affects atherosclerosis is yet to materialise (Stampfer, Malinow, Willett *et al.* 1992; Boushey, Beresford, Omenn *et al.* 1995; Fortin and Genest, 1995; Vermeulen, Stehauwer, Twisk *et al.* 2000).

The American Heart Association Science Advisory Panel proposed a normal range for fasting Hcy of 5 to 15 $\mu mol \cdot L^{-1}$ (Eikelboom *et al.* 1999; Malinow *et al.* 1999). Elevated levels, known as hyperhomocyst(e)inemia, have been accepted as 'modest' hyperhomocyst(e)inemia when values fall between 16 and 30 $\mu mol \cdot L^{-1}$, as 'intermediate' hyperhomocyst(e)inemia when values range from 31 to 100 $\mu mol \cdot L^{-1}$, and 'severe' hyperhomocyst(e)inemia when levels are greater than 100 $\mu mol \cdot L^{-1}$ (Malinow *et al.* 1999). However, a consensus has not been reached and researchers continue to employ different thresholds for normal and elevated values (Eikelboom *et al.* 1999). The association between Hcy concentration and disease is continuous. For every 5 $\mu mol \cdot L^{-1}$ increase in fasting Hcy, there is a 1.6 to 1.8 fold increased chance of developing CHD (Boushey *et al.* 1995). According to Chambers, McGregor, Jean-Marie *et al.* (1998) levels of only 12% above the upper limit of normal concentration (15 $\mu mol \cdot L^{-1}$), cause a three fold increased risk of myocardial infarction (MI).

Since the early and original work of McCully, relatively little has been reported on Hcy levels in children, though Reddy (1997) presented reference ranges for total Hcy in normal children aged 9.1 to 14.0 years of 8.3 ± 3.7 $\mu mol \cdot L^{-1}$ in boys and 8.3 ± 3.3 $\mu mol \cdot L^{-1}$ in girls. One of the most comprehensive studies of homocyst(e)ine in children was the Child and Adolescent Trial for Cardiovascular Health (CATCH) (Osganian, Stampfer, Spiegelman *et al.* 1999). The investigation reported that in adolescents aged 13 to 14 years where the 95th percentile was estimated at 8.5 $\mu mol \cdot L^{-1}$, boys registered significantly higher levels of homocyst(e)ine than girls (5.22 $\mu mol \cdot L^{-1}$ vs 4.84 $\mu mol \cdot L^{-1}$; $P \leq 0.001$). This finding supported the sex differences reported in adult studies (Fortin and Genest, 1995) but contradicted the findings in Norwegian children (Tonstad, Refsum, Siversten *et al.* 1996). Osganian *et al.* (1999) also investigated the associations between homocyst(e)ine and other CHD risk factors. A significant, but weak positive relationship was reported between homocysteine and SBP ($r = 0.08$; $P \leq 0.001$), and BMI ($r = 0.09$; $P \leq 0.001$) whereas no associations were identified with serum lipids and familial history of CHD. Conversely, an association between homocyst(e)ine and family history of cardiovascular disease was identified in Norwegian children (Tonstad, Refsum, Siversten *et al.* 1996), a finding supported by others (Greenlund, Srinivasan, Xu *et al.* 1999; Scott and Sutton, 1999; Laskowska-Klita *et al.* 2001). Tonstad *et al.* (1996) reported that children whose first degree male relatives had died prematurely of CHD (before age 55 years), had higher levels of homocyst(e)ine than offspring without such a history. Although Osganian *et al.* (1999) failed to support these claims they suggested that the discrepancies in results might have arisen from differences in family history measures. In addition, since levels of homocyst(e)ine in the male relatives included in Tonstad *et al.*'s study were unknown, a causative association between modest elevated levels of homocyst(e)ine and risk of disease remained unproven (Tonstad *et al.* 1996). To date, few studies have explored the possible associations

between physical activity and/or fitness, and homocyst(e)ine levels in young people, although for young male adults, it was reported that an increase in physical fitness led to a 15% decrease in Hcy levels (personal communication, Dr Damien Bailey and Professor Bruce Davies, 2003). Although more research is necessary, it is encouraging that for young people, an investigation by Gallistl, Sudi, Borkenstein *et al.* (2000), reported that a weight reduction programme including physical activity had a positive effect on the homocyst(e)ine levels of obese children.

Innocuous and inexpensive means of therapy such as small doses of folate and vitamins B_{12} and B_6 are reported as effective means of normalising Hcy levels (Malinow *et al.* 1990; Stampfer *et al.* 1992; Clarke, 1998; Welch and Loscalzo, 1998; Eikelboom *et al.* 1999; Bates, Mansoo, Gregory *et al.* 2002), implying that homocyst(e)inemia is an easily reversed risk factor (Malinow *et al.* 1990). Of the three forms of therapy, folate is regarded as the most effective, with dosages as low as 0.65 $mg \cdot d^{-1}$ producing positive returns (Harjai, 1999). In their review of folate intake, de Bree, Dusseldorp, Brouwer and co-workers (1997) reported that the average dietary folate intake for European men and women was 291 $\mu g \cdot d^{-1}$ and 247 $\mu g \cdot d^{-1}$ respectively. These levels met the most frequently recommended level cited for European countries of 200 $\mu g \cdot d^{-1}$ for both men and women, however, it has been suggested that for 'normal' plasma Hcy levels, a dietary folate intake of at least 350 $\mu g \cdot d^{-1}$ is necessary (de Bree *et al.* 1997). This suggests that many individuals fall short of the necessary dietary intake, and that the current recommendations for folate intake need addressing. A similar story was iterated in the United States where a substantial 80%-90% of the population had a dietary intake of less than 400 $\mu g \cdot d^{-1}$ (the recommended daily dosage). The national mean value (sexes combined) was 224 ± 2.8 $\mu g \cdot d^{-1}$ (mean $\pm$ SEM) (Subar, Block and James, 1989). In an attempt to address the problem of folate intake shortage, the US have added the vitamin

to its yeast products. Nonetheless, whether decreasing Hcy levels reduces cardiovascular morbidity and mortality remains unproven (Stampfer *et al.* 1992; Eikelboom *et al.* 1999; Harjai, 1999).

It is evident that further research is necessary to determine the exact contribution of genetics, diet and physical activity on Hcy levels in children and adults (Eikelboom *et al.* 1999). It could be that the relationship between CHD and elevated Hcy levels is both strong and independent of other risk factors. Nevertheless, inconsistent findings prevent total verification that elevated Hcy concentration will result in an increased CHD risk (Boushey *et al.* 1995; Eikelboom *et al.* 1999). Greater epidemiological evidence including longitudinal tracking of plasma levels is essential if we are to confirm any link between childhood and adulthood levels and to understand the influence that these values have on CHD.

2.3.2 Fibrinogen

Several studies have examined the relationship between plasma fibrinogen (Fg) levels and CHD (Lofmark, 1982; Chakrabarti, Hocking, Fearnnley *et al.* 1968; Meade, Chakrabarti, Haines *et al.* 1980; Kannel, Wolf, Castelli *et al.* 1987; Yarnell, Baker, Sweetnam *et al.* 1991; Krobot, Hense, Cremer *et al.* 1992; El-Sayed, 1996; Eriksson, Egberg, Wamala *et al.* 1999; Cantin, Despres, Lamarche *et al.* 2002). Whereas the majority of prospective studies have supported the notion that elevated plasma Fg concentration is a major risk factor of CHD (Ernst and Resch, 1993), others have refuted this position (Lofmark, 1982). The majority of studies related to the measurement of this aspect have centred on middle-aged and older males, with some attention afforded to the younger population (Bao, Srinivasan and Berenson, 1993; Sanchez- Bayle, Cocho, Baeza *et al.* 1993; Zahavi, Yaari, Salman *et al.* 1996; Mahon,

Cheatham, Kelsey *et al.* 1997; Poli, Tofler, Larson *et al.* 2000; Balagopal *et al.* 2002; Invitti, Guzzaloni, Gilardini *et al.* 2003).

Fibrinogen is the main coagulation protein in plasma (Poli *et al.* 2000) and is a major determinant of plasma viscosity; it is an acute phase reactant (Sanchez-Bayle *et al.* 1993; El-Sayed, 1996; Poli *et al.* 2000). Although there is continued uncertainty with regards to the pathophysiological mechanism through which elevated levels of Fg are associated with atherogenesis, it is probable that a high Fg concentration promotes platelet aggregation, encourages smooth muscle cell migration, proliferation, and increased blood viscosity (Meade, Mellows, Brozovic *et al.* 1986; El-Sayed, 1996). In addition to this, since fibrin binds to lipoprotein in the intima, this could stimulate accumulation of lipid in fibrous plaques (Smith and Thompson, 1994). Normal Fg values range from 150 to 350 $mg \cdot dL^{-1}$ (El-Sayed, 1996), although these values can be affected by various lifestyle and biological variables including age, sex, smoking, alcohol consumption, physical activity, body mass index, social class and disease (Lee, Smith, Lowe *et al.* 1990; Krobot *et al.* 1992; Elwood, Yarnell, Pickering *et al.* 1993; MacAuley *et al.* 1996; Pankow, Folsam, Province *et al.* 1998; Carroll, Cooke and Butterly, 2000). In the National Heart, Lung, and Blood Institute Family Heart Study, Pankow and colleagues (1998) found that 19% to 29% of the variance in plasma Fg concentration could be attributed to age, anthropometric indices, lifestyle, and metabolic factors. The Northwick Park Heart Study investigated Fg concentration in 1511 males aged 40 to 64 years (Meade *et al.* 1980). In the 4-year follow-up, it was found that 49 individuals had died, and 27 of these fatalities were the consequence of cardiovascular disease. A significant relationship was reported between fatal coronary events and Fg levels; this was independent of other risk factors. Similar findings have been reported for females (Eriksson *et al.* 1999; Kannel *et al.* 1996).

The effects of exercise and activity on fibrinogen levels and subsequent CHD risk have been difficult to ascertain, partly because of the contribution of confounding variables. Nevertheless, many investigators have explored this issue (Karp and Bell, 1974; Ohri, Chatterji, Dasg *et al.* 1983; Lee *et al.* 1990; de Paz, Lasierra, Villa *et al.* 1992; Ernst and Resch, 1993; Elwood *et al.* 1993; Ponjee, Jabssen and van Worson, 1993; El-Sayed and Davies, 1995; Koenig, Sund, Doring *et al.* 1997; Mahon *et al.* 1997; Carroll *et al.* 2000). The possibility of physical activity and/or fitness having a diminishing effect on Fg levels is attractive as this would provide us with another reason for promoting an active lifestyle, nevertheless, reports are conflicting. In 1974, Karp and Bell investigated the effect of moderate and maximal exercise on Fg concentration and reported no change; similar conclusions were described by El-Sayed and Davies (1995). The latter study did detect a significant decrease ($P < 0.05$) in the Fg concentration (6%) of young adults following 12 weeks endurance training, however this significance disappeared when data were corrected for haemoconcentration. The conclusions of El-Sayed and Davies (1995) supported those of de Paz *et al.* (1992) who also failed to confirm an association between Fg concentration and physical training. MacAuley *et al.*'s (1996) study of Northern Irish subjects aged 16 to 74 years similarly failed to observe a significant relationship between physical activity and Fg once adjustment was made for confounding variables. Interestingly, MacAuley and colleagues did confirm an association between Fg and physical fitness after accounting for age and smoking.

Despite several studies indicating little or no link between physical activity and/or fitness and fibrinogen levels, there is evidence to the contrary. For example, Stratton, Chandler, Schwartz *et al.* (1991) found endurance training significantly decreased Fg concentration in older but not younger males. Similarly, according to Ernst's (1993) review of the area, physical activity induces a seemingly small decrease (40 $mg \cdot dL^{-1}$) in

Fg levels. Although small, such a reduction might be of the utmost importance especially when considering the Northwick Park Heart Study's claim that a 10 $mg \cdot dL^{-1}$ difference in Fg concentration leads to a 15% decrease in the risk of developing CHD (Meade *et al.* 1986). The Scottish Heart Study investigated the Fg levels of 8824 individuals aged 40 to 59 years (Lee *et al.* 1990). It was found that inactive individuals had higher Fg levels than active individuals and that this continued to be the case after accounting for smoking. Curiously, when values were adjusted for both smoking and social class, the significant findings were lost (Lee *et al.* 1990). The Caerphilly Prospective Heart Study examined the relationship between physical activity and Fg, with cross-sectional evidence gained from 2398 males aged 50 to 64 years (Elwood *et al.* 1993). The data indicated that Fg concentration was lower by 24 $mg \cdot dL^{-1}$ in the most active men and it was postulated that the risk of developing CHD was reduced by 7% - 8% if regular activity was undertaken. A cross-sectional study by Carroll *et al.* (2000) examined the effects of leisure time physical activity on plasma Fg levels. The cohort comprised 635 non-smoking females (46.7 ± 7.7 years) and after adjusting for age and subcutaneous adiposity, increased activity was associated with decreased plasma Fg levels.

Whereas the majority of studies have reported a decrease or no change in Fg levels as a consequence of increased physical activity or fitness, to add to the confusion some authors have noted an increase (Ohri *et al.* 1983; Ponjee *et al.* 1993). A 13.07% increase in Fg concentration following activity was reported by Ohri *et al.* (1983), however, if the study had standardised for exercise-induced haemoconcentration, the increase might have lost its significance (El-Sayed, 1996). Ponjee *et al.* (1993) investigated the impact of nine months endurance training on Fg concentrations and similarly reported increased values.

There is a dearth of similar data for the younger population although a positive association between Fg and physical activity has been reported by some (MacAuley *et al.* 1996; Cook, Whincup, Miller *et al.* 1999). The Petah Tikva Project (Zahavi, Yaari, Salman *et al.* 1996) observed a low but significant relationship between sports activity and plasma Fg levels in 9 to 18 year olds ($r = 0.19, P < 0.01$, (boys); $r = 0.17$ $P < 0.05$, (girls)). Similarly, the Northern Ireland Health and Activity Survey, observed an inverse relationship ($P < 0.01$) between the physical fitness and Fg levels of older teenagers. However, the association diminished when correcting for confounding variables (MacAuley *et al.* 1996). No such relationship was reported by Mahon and colleagues (1997) in youngsters aged 10 to 16 years. Elevated levels of Fg have been reported in obese female adolescents (Balagopal *et al.* 2002) and since increased body fat levels are often associated with physical inactivity, this could imply that increased activity might play an important role in modulating Fg levels during childhood. The link between Fg levels and other CHD risk factors has also been investigated (Sanchez-Bayle *et al.* 1993). In a study of 2224 Spanish schoolchildren (2-18 years old) individuals with higher Fg concentrations displayed significantly higher TC, TG and VLDL-C levels (Sanchez-Bayle *et al.* 1993). Reports on the influence of physical activity and fitness on Fg are both diverse and confusing. Despite uncertainty regarding the potentiality of these variables on Fg levels (El-Sayed and Davies, 1995; El-Sayed, 1996) consensus suggests that an active lifestyle has a positive effect on CHD (Mahon *et al.* 1997).

2.3.3 C-reactive protein

C-reactive protein (CRP) is an acute phase reactant (Cohen *et al.* 1999) and an inflammatory marker. Although CRP has not been extensively explored, especially in populations such as women and children, evidence has indicated that an increased CRP level is associated with a concomitant increase in CHD risk (Ridker, Buring, Shih *et al.*

1998[a]; Ridker, Cushman, Stampfer *et al.* 1998[b]; Cohen *et al.* 1999). A plasma protein, C-reactive protein is released into the bloodstream as a result of active inflammation; since atherosclerosis is now being considered a low-grade inflammatory process (Ridker *et al.* 1998[a]), this supports the hypothesis that CRP is a molecular marker for underlying systemic atherosclerosis (Ridker *et al.* 1998[a]; Margaglione, Cappucci, Colaizzo *et al.* 2000). For example, individuals in the highest quartile of CRP levels might have a five-fold increased risk of suffering a MI (Cohen *et al.* 1999). Normally, CRP is present in small amounts with trauma and infection increasing its presence. In addition, age, BMI, adiposity, sex, physical inactivity and smoking are thought to affect CRP concentrations (Mendall, Patel, Ballam *et al.* 1996; Ridker *et al.* 1998[a]; Abramson and Vaccarino, 2002), many of which are modifiable. Furthermore, statin therapy is considered a means of lowering CRP (Bermudez and Ridker, 2002). Although more research is needed, in comparison to men, women tend to display higher median CRP levels (Ridker *et al.* 1998[a]).

Ridker and colleagues (1998[a]) have proposed that CRP concentrations reflect the magnitude of inflammatory reactions in the atherosclerotic vessels. Thus it is not the increased CRP concentration that poses the risk but the fact that it indicates atherosclerosis in the coronary arteries (Henrich, Schulte, Schönfield *et al.* 1995). If CRP levels do reflect the degree of atherosclerosis in important vessels, a higher concentration would be expected in individuals suffering from coronary artery related illnesses such as angina pectoris (de Maat, de Bart, Hennis *et al.* 1996; Ridker *et al.* 1998[b]). Ridker and colleagues (1998[b]) examined CRP concentration in apparently healthy males who later developed symptomatic peripheral arterial disease. The CRP levels of the cohort were compared against a control group who were free of disease. The median CRP values at baseline were significantly higher in the former group (1.34

$mg \cdot L^{-1}$ vs 0.99 $mg \cdot L^{-1}$), with males in the highest quartile of CRP at baseline considered to have a 2.2 times greater risk of future peripheral arterial disease than those in the lowest quartile (Ridker, 1998[b]). Ridker *et al.* (1998[a]) undertook a comparable study of women. Baseline CRP concentration was measured in 122 apparently healthy subjects who subsequently developed cardiovascular disease. As in the study by Ridker *et al.* (1998[b]), females who had suffered a cardiovascular event had higher baseline CRP levels than those who had not. The females with the higher baseline levels were thought to have a five-fold increased risk of suffering a cardiovascular event, and a seven-fold increased risk of suffering from a myocardial infarction or stroke (Ridker *et al.* 1998[a]). Not all studies have supported such findings, for example, Mendall *et al.*'s (1996) study found that the values for their apparently healthy subjects were similar to those reported by Haverkate, Thompson, Pyke *et al.* (1997) for individuals with stable angina pectoris.

With regard to the association between CRP and CHD some arbitrary thresholds have been proposed. A CHD risk value of > 33 $mg \cdot L^{-1}$ for adults aged 22–66 years was adopted by Margaglione *et al.* (2000), whereas values > 38 $mg \cdot L^{-1}$ for otherwise postmenopausal females and 15 $mg \cdot L^{-1}$ for otherwise healthy males have been suggested (de Maat *et al.* 1996). Sormunen, Kallio, Kilpi *et al.* (1999) determined a normal range for children of < 20 $mg \cdot L^{-1}$, whilst elevated values of CRP have been reported in overweight children (Ford, Galuska, Gillespie *et al.* 2001; Invitti *et al.* 2003). These latter findings concur with the observations of others that excess fat is a key promoter of chronic low-grade inflammation (Forouhi, Sattar and McKeigue, 2001).

To date, prospective data supports the notion that CRP is a molecular marker for CHD hence the area deserves attention. However, the derivation and interpretation of CRP in

young apparently healthy people with no significant atherosclerosis needs to be established. One could imagine a variety of inflammatory conditions raising plasma CRP not related to CHD.

2.3.4 Plasminogen activator inhibitor-1

Although investigators are yet to agree on the nature and extent of its association, elevated levels of plasminogen activator inhibitor-1 (PAI-1) have been linked with CHD (Meade *et al.* 1986; Hamsten, Wilman, de Faire *et al.* 1985; Hamsten, de Faire, Wallidus *et al.* 1987). Plasminogen activator inhibitor-1 is credited as an important regulatory component in fibrinolysis (Keijer, Linders, von Zonnereld *et al.*1991). Fibrin is an insoluble protein formed from fibrinogen by the action of the proteolytic enzyme thrombin (Kohler and Grant, 2000). The enzymatic breakdown of fibrin, or simply put, the dissolution of a blood clot, is called fibrinolysis; it is an effective protective mechanism against thrombus formation (Eliasson *et al.* 1994). The successful outcome of fibrinolysis is reliant upon the activities of tissue plasminogen activator (t-PA), urinary type plasminogen activator (u-PA), and plasmin (Eliasson *et al.* 1994; Kohler and Grant, 2000). Plasminogen is an inactive plasma enzyme and the precursor of plasmin, an active plasma enzyme that is capable of destroying fibrin as it is formed (Newsholme and Leech, 1991). The main inhibitors of fibrinolysis are PAI-1, a fast-acting inhibitor of t-PA and u-PA, and alpha$_2$antiplasmin (Kohler and Grant, 2000). Plasminogen activator inhibitor-1 is a linear glycoprotein consisting of 379 amino acids. It is produced by the vascular endothelium, but also exists in platelets. When platelets are stimulated by thrombin, PAI-1 is released on the platelet surface, and often to the detriment of well being, encourages preservation of a blood clot (Kohler and Grant, 2000).

Hamsten *et al.* (1987) studied 109 male patients, all of whom were below 45 years and had survived myocardial infarction. The investigation concurred with the findings of Hamsten and colleagues (1985) that low t-PA activity, mainly brought about by elevated levels of PAI-1, was common in post MI patients. These individuals were considered at greater risk of reinfarction. However, a later study of t-PA in survivors of MI failed to confirm a link between reinfarction and t-PA activity (Jansson, Nilsson and Johnson, 1991). The relationship between triglyceride (TG) levels and t-PA activity has also been investigated (Landin, Stigendal, Eriksson *et al.* 1990; Eliasson *et al.* 1994). Landin and co-workers (1990) reported both elevated PAI-1 and fibrinogen in middle-aged obese women with a high waist to hip ratio, but could not find an association between PAI-1 concentration and TG levels. This disagreed with the evidence of Hamsten *et al.* (1985) and Hamsten *et al.* (1987). Landin and colleagues (1990) proposed that such discrepancies could have occurred if the relationship observed in the earlier studies was weak, in addition, the range of TG levels in their own study was limited. Other advocates of the association between TG levels, PAI-1 and t-PA activity, include Eliasson and colleagues (1994). The authors studied fibrinolytic variables in a sample of 1558 individuals aged 25 to 64 years (Northern Sweden Monica Project). They found that high TG concentrations were determinants of t-PA activity, independent of degree and type of activity. Tissue plasminogen activator activity was negatively associated with BMI and TG levels, and that the relationships with PAI-1 activity was basically the reverse of t-PA, but stronger.

It has been hypothesized that PAI-1 levels can be modified, but conclusive evidence is scant. Stratton *et al.* (1991) examined the effects of six months intensive training on fibrinolytic variables in adult males. The subjects were divided into two groups, younger group (24 to 30 years), and an older group (60 to 82 years). Post-training, both

groups demonstrated similar increases in maximal aerobic power, though significant changes in fibrinolytic measures were only seen in the older cohort (Stratton *et al.* 1991). A 39% increase in t-PA activity, and a 52% decrease in PAI-1 was observed in the 60 to 82 year olds. It is uncertain why only the older group should demonstrate such differences, including a 13% decrease in FG concentration, but a larger subject cohort might have yielded different results (Stratton *et al.* 1991).

Svendsen, Hassager, Christiansen *et al.* (1996) assessed the short - and long - term effects of an energy restrictive diet with and without exercise. Although the addition of exercise had little effect on PAI-1 levels, a controlled diet did succeed in decreasing levels in overweight, post-menopausal women.

As yet there are very few data concerning PAI-1 levels in young people and whether such information would be advantageous to adult health status remains to be seen. However, an interrelationship between estimates of obesity with metabolic and haemostatic parameters including PAI-1 in obese children has been reported (Sudi, Gallistl, Payerl *et al.* 2001).

2.3.5 Endothelial dysfunction

Endothelial dysfunction is an impairment of endothelium-dependent vasodilation and an indicator of preclinical atherosclerosis (Anderson, 1999; Twisk, 2000; Schumacher, Seljeflot, Sommervoll *et al.* 2002; Spieker, Sudanon, Hurilmann *et al.* 2002). Even before atherosclerosis develops, hypercholesterolaemia impairs nitric oxide-mediated vasodilation, consequently, correction is imperative in children as it rapidly restores endothelial function to normal. Recent advances have enabled investigators to more easily determine this measure since it is now possible to determine the physiological

responses of blood vessels using non-invasive, high resolution ultrasound (Chambers *et al.* 1998). Methods of treating endothelial dysfunction include the use of antioxidants, drug therapy, and exercise (Hornig, Arakawa, Kohler *et al.* 1998; Quyyumi, 1998; Abbott, Harkness and Davies, 2002). Little has been written on children and adolescents, although a reduction or absence in flow-mediated dilation has been reported in children with familial hypercholesterolaemia (Celermajer, Sorensen, Gooch *et al.* 1992; Sorensen, Celermajer, Georgakopoulos *et al.* 1994; de Jongh, Lilien, Bakker *et al.* 2002). As treatments include exercise and antioxidants, this could be a reason for encouraging an active lifestyle, as well as ensuring an adequate supply of antioxidant vitamins in a young person's diet (Abbott *et al.* 2002).

Endothelial dysfunction is related to CHD but more research is required on the verification of treatment since many studies included small samples (Celermajer *et al.* 1992; Sorensen *et al.* 1994; Clarkson, Celermajer, Powe *et al.* 1997; Hornig *et al.* 1998).

2.3.6 Chlamydia pneumoniae

Chlamydia pneumoniae (C pneumoniae) is a common gram-negative bacillus and obligate intracellular parasite frequently identified in respiratory infection (Cohen *et al.* 1999). The majority of individuals are infected by C pneumoniae at some point and reinfection is common (Campbell, Kuo and Grayston, 1998). Chlamydia pneumoniae has been linked to CHD in adults and young people (Campbell *et al.* 1998; Sessa, Di-Petro, Santino *et al.* 1999; Wong, Dawkins and Ward, 1999; Hortovanyi, Illyes, Glasz *et al.* 2002) although a number of researchers dispute this association (Altman, Rouvier, Sazziota *et al.* 1999; Danesh, Whincup, Walker *et al.* 2000). In their investigation of patients with acute MI, Sessa *et al.* (1999) confirmed a link between infection by C

pneumoniae and CHD. Although high titres of C pneumoniae antibodies were related to an undesirable blood lipid profile, the investigators conceded additional research was necessary before absolute affirmation of this association could be made. Wong and co-workers (1999) used polymerase chain reaction (PCR) to detect C pneumoniae DNA in the mononuclear cell layers and platelets extracted from blood. Circulating C pneumoniae DNA was identified in 8.8% of the 669 males with coronary artery disease, compared with 2.9% of 135 males with normal arteries (odds ratio 3.2) (Wong *et al.* 1999). This was not apparent amongst the female cohort and the investigators proffered no reason for this. Several studies have refuted claims of a relationship between C pneumoniae and CHD (Danesh, Whincup, Levington *et al.* 2002). Danesh and colleagues. (2002) undertook a case-control analysis in a prospective cohort study and concluded that their findings, along with those from a meta-analysis of previous studies, dismissed the notion of any strong relationship between C pneumoniae IgA titres and IgG titres, and CHD. More research is necessary to confirm or refute any relationship between C pneumoniae and CHD, especially amongst the younger population (Wong *et al.* 1999; Danesh, Whincup, Levington *et al.* 2000). This can only be accomplished with the development of a valid and reliable serologic marker for C pneumoniae infection (Boman and Hammerschlag, 2002; Kolia and Fong, 2002).

2.4 SOCIO-ECONOMIC STATUS

The contribution of socio-economic factors to CHD has been widely explored. Traditional risk factors such as high blood pressure levels, high cholesterol levels, physical inactivity and obesity have received the majority of attention in children and

† Titre: describes the highest dilution at which the measured effect is detected.

‡ IgA and IgG: immunoglobins.

adolescents, although more recently identified risk factors have also been investigated (Cook *et al.* 1999; Cook, Mendall, Whincup *et al.* 2000). Closer scrutiny of this aspect is certainly needed, though recent evidence suggests that the direct influence of social circumstances during childhood on CHD in later life is probably quite small with little evidence of a consistent pattern (Gliksman, Kawachi, Hunter *et al.* 1995; van Lenthe, Boreham, Twisk *et al.* 2001; Dollman, Norton and Tucker, 2002).

Lapidus and Bengtsson (1986) completed a prospective study of middle-aged women and found that socio-economic factors were related to 12-year incidence of ischaemic heart disease and total mortality. A significant age specific association was identified between socio-economic status, according to husbands' occupation, and MI. Although a similar association was not reported between the socio-economic status (SES) of the women themselves and MI, it was reported that women with a lower education level had a significantly higher incidence of angina pectoris (Lapidus and Bengtsson, 1986). The British Regional Heart Study (Pocock, Shaper, Cook *et al.* 1980) examined the possible contribution of socio-economic factors on CHD, and found that towns containing predominantly manual workers had higher standardized mortality ratios (Shaper, 1984). In 1984, Shaper reviewed studies relating social class to CHD mortality and concluded that the evidence indicated the independence of social class and geography with respect to adult male mortality differences.

In young people, findings are equivocal. The prevalence of CHD risk factors in 8 to 11-year olds was investigated by Whincup, Cook, Adshead and co-workers (1996). The examined cohort was taken from geographic regions representing high and low adult cardiovascular mortality rates in England and Wales. The Welsh regions of Port Talbot and Rhondda represented two of the highest concentrations of adult cardiovascular

mortality. Children from high mortality towns posted higher blood pressure levels and ponderal index values, and were on average shorter than those from low mortality areas. However, TC concentration, post-load glucose measurements and waist-to-hip ratios did not differ. The failure to detect geographic differences in TC supported the conclusions of adult surveys by The British Regional Heart Study (Pocock *et al.* 1980).

It has been postulated that individuals of higher socio-economic status are more likely to engage in physical activity than those from lower socio-economic groups (Wold, Oeygard, Eder *et al.* 1994; Guillaume *et al.* 1997; Duncan *et al.*, 2002). Furthermore, current evidence suggests that children and adolescents from higher socio-economic groups are more inclined to maintain favourable activity levels into adult life (Wold and Hendry, 1998). Guillaume *et al.* (1997) measured CHD risk factors, including physical inactivity and obesity, in 6 to 12 year old children and found that the socio-economic conditions of families influenced childrens' exercise and television watching habits. The finding that children from families of better socio-economic conditions exercised more regularly, partly explained the higher prevalence of obesity in children from lower social classes (Guillaume *et al.* 1997).

Cheung (1995) similarly concluded that childhood obesity is directly related to socio-economic class and level of parental education. However, the authors failed to ascertain whether this association reflected differences in eating or exercise habits. Once again observations are inconsistent. Whereas Dietz (1995) declared that increased childhood obesity was more common in middle and upper socio-economic classes, others have discounted this view believing the condition to be more apparent in young persons from underprivileged areas and lower social classes (Bogin and Sullivan, 1986; Freeman *et al.* 1990; Gortmaker, Must, Perrin *et al.* 1993; Guillame *et al.* 1997). Rebato and co-

workers (1998) investigated fat distribution amongst an urban sample of males and females aged 4.5 to 19.5 years, with socio-economic status defined according to fathers' profession (Rebato *et al.* 1998). Central body fat distribution was higher in poorer socio-economic groups, but this was only evident in females. Bogin and Sullivan (1986) had reported higher central body fat values in both male and female Guatemalan children of lower socio-economic groups. The discrepancies amongst the findings could be attributed to the differences between developed and developing societies as well as researchers' interpretations of what constitutes high and low SES (Sobal and Stunkard, 1989).

Chinn and Rona (1994) reviewed data from the 1972, 1982 and 1990 Surveys of the National Study of Health and Growth. The information collated on Scottish and English children aged between 4.5 and 11.9 years was used to identify changes in stature, body mass, triceps skinfold thickness and mass-for-stature index. With the exception of English boys, greater increases in obesity related variables were observed from 1982 to 1990, than from 1972 to 1982. Much of this increase was attributed to increases in parental mass indices and a decrease in family size. Since the surveys did not identify consistent differences in trends according to social groups, the authors suggested the need for a 'population-based strategy' for decreasing overweight and obesity in children (Chinn and Rona, 1994).

In the Health Survey for England 1995 (Dong, Primatesta and Bost, 1997), higher blood pressure levels were observed among middle-aged and older men and women of lower social classes. The notion that young peoples' blood pressure levels vary according to their socio-economic background has also been investigated (de Swiet, 1992; Jenner *et al.* 1992; Whincup *et al.* 1996). Whincup *et al.* (1996) measured the blood pressure

levels of children aged 8 to 11 years, from areas of high and low adult cardiovascular mortality concluding that readings were higher in high mortality towns. The values were also higher than those recorded in ten year olds in the Brompton study (de Swiet, 1992). Although of significant importance it could not be concluded whether these latter differences were as a result of differences in protocol, or for sociological and/or geographical reasons. Jenner *et al.* (1992) had earlier investigated the relationship between SES and blood pressure levels in children (mean age 12.0 $\pm$ 0.4 years) and although independent relationships were identified in girls, the results conflicted with findings from other studies. Contradicting the findings of others, Jenner *et al.* (1992) determined that young females from a high SES area, and not those living in a low socio-economic area, had on average the highest blood pressure levels. Whincup and colleagues (1996) also examined waist to hip ratio, TC concentration and 30 minute post-load glucose measurements. Results were similar for both high and low mortality towns. In an investigation of children aged 10 to 11 years, Cook *et al.* (1999) failed to identify a relationship between social class and Fg levels. Results from the same cohort of children similarly indicated no association between CRP concentration and social class (Cook *et al.* 2000).

The evidence on the influence of SES on CHD risk in children and adolescents is equivocal with little reported regarding the influence of socio-economic status on recently identified risk factors. More studies are needed to improve our understanding of the relationship between CHD risk factors in the young and socio-economic status.

2.5 GLOBAL RISK

It has been established that a significant number of young people display at least one risk factor of CHD and as the number of risk factors in an individual's profile increases,

so too does the risk of developing coronary disease (Cunnane, 1993). Unfortunately, researchers have reported a clustering of risk factors in many children and adolescents (Wilmore and McNamara, 1972; Gilliam *et al.* 1977; Bao, Srinivasan, Wattigney *et al.* 1994; Boreham *et al.* 1997; Hardin *et al.* 1997; Kilkens, Gijtenbeek, Twisk *et al.* 1999; Nicklas *et al.* 2002).

Wilmore and McNamara (1972) investigated the prevalence of hypertension, obesity, elevated blood lipids, family history of MI, low physical activity and abnormal electrocardiograms in boys aged 8 to 12 years. Thirty-six percent exhibited no risk factors, 46% showed one risk factor, 14% exhibited two risk factors, 3% displayed three risk factors, and 1% had four risk factors. A similar analysis of 7 to 12 year olds was conducted by Gilliam *et al.* (1977). The authors reported that 62% of the 47 investigated exhibited at least one risk factor, whereas 20% displayed three risk factors, and 10% had four risk factors. One child exhibited five risk factors. In more recent years the prevalence of multiple risk factors in youths aged 18, 21 and 24 years, was investigated by Raitakari and co-workers (1995). A significant relationship was identified between the accumulation of risk habits such as smoking, alcoholic drinking, high-fat diet and physical inactivity, and the clustering of biological risk factors. Significantly, the study also established that multiple undesirable lifestyle habits have already clustered in young adulthood (Raitakari *et al.* 1995). In their cohort of adolescents, Boreham *et al.* (1997) found that 17.5% of boys aged 12 years, and 14.7% of girls, exhibited at least two CHD risk factors. The study also reported that by age 15 years, 16.7% of males and 22% of females displayed multiple risk factors. The prevalence of selected CHD risk factors was also investigated in a young Greek population and based on published criterion thresholds for CHD, 45% of boys and 50% of girls exhibited three or more risk factors (Bouziotas *et al.* 2001).

In contrast to the previous studies, no significant clustering ($P > 0.05$) of lifestyle risk factors was reported by Kilkens and collaborators (1999). However, clustering did exist at the individual level. For example, 17 adolescents displayed two lifestyle factors at age 13 years. The authors suggested that their results conflicted with those of others because their own cohort was smaller, younger, and from a different socio-economic background and education level (Kilkens *et al.* 1999). Mathematical algorithms have been constructed to calculate global risk in adults (Assmann, Schulte, van Eckardstein, 1996), but whether these would be suitable for a young population is yet to be established.

The aims of this thesis were to examine the prevalence of CHD risk factors in young people of differing socio-economic status and to establish to what extent CHD risk factors cluster in 12 to 13 year olds.

2.6 EXPERIMENTAL RATIONALE AND NULL HYPOTHESES (H_0)

The purpose of this research was:

- To compare coronary heart disease risk factors in 12 to 13 year boys and girls of differing socio-economic status.

- To compare coronary heart disease risk factors of 12 to 13 year old boys and girls.

- To establish the nature and extent of 'clustering' of coronary heart disease risk factors in 12 to 13 year old boys and girls.

- To identify any relationships between coronary heart disease risk factors in 12 to 13 year old boys and girls.

To test these research aims the following null hypotheses were proposed:

Null Hypothesis (1)

H_0: *There is no difference in the coronary heart disease risk factors of 12 to 13 year old boys and girls of differing socio-economic status.*

Null Hypothesis (2)

H_0: *There is no difference in the coronary heart disease risk factors of 12 to 13 year old boys and girls.*

Null Hypothesis (3)

H_0: *There is no evidence of the 'clustering' of coronary heart disease risk factors in 12 to 13 year old boys and girls.*

Null Hypothesis (4)

H_0: *There is no relationship between coronary heart disease risk factors in 12 to 13 year old boys and girls.*

CHAPTER 3

3.0 THEORETICAL AND METHODOLOGICAL BACKGROUND

3.1 SELECTION OF SUBJECTS

A cohort of Caucasian schoolchildren (n = 100 boys and 108 girls) from two schools (1 & 2) participated in this nationwide study. Year 8 pupils (12.0 ± 0.8 years) were considered appropriate for this investigation since they were comfortable with the routine of the school day; old enough to have established views and therefore patterns on food intake and activity; not subject to external examinations.

A complete list of Welsh comprehensive schools was provided by Estyn, Her Majesty's Inspectorate for Education and Training in Wales (Estyn, 2000). Information regarding each school's enrolment number and approximate nature of intake was similarly provided by Estyn. To avoid the logistical problems of visiting too many schools, single-sex schools were removed from the list. Two Welsh comprehensive schools (age range 11 to 18 years) were selected to represent educational institutions of differing socio-economic status (SES). Two reserve schools were also selected, and a pilot study was conducted at one of these latter schools.

The author approached the respective Head teachers for permission to conduct the project in their school. The aims and objectives of the project were explained to the Head teacher and other relevant teaching staff. It was stressed that the project would be an enjoyable and educational experience for all those involved, and that there would be

no disruption to the pupils' normal school timetables. It was explained that all testing would be integrated into the Physical Education programme and that comprehensive feedback would be given to all pupils and parents. Seasonal variations between school 1 and 2 were avoided (Thirlaway and Benton, 1993) since measurements were taken during the same season in both schools.

Once permission was granted from the respective Head teachers, Physical Education departments, Governors and Local Education Authorities, the Head teachers of both schools completed forms describing the nature of their schools' intake (Appendix 2), this was done according to criteria issued by Estyn (2000). School 1 had a total intake of 873 pupils, whilst school 2 had an intake of 943 pupils. In school 1, 75% of the pupils resided in rural areas, whilst the remaining 25% lived in a small town. In school 2, 25% of the pupils resided in rural areas, 50% lived in small towns, whilst the remaining 25% of pupils lived in the outer areas of a large town. According to Estyn's criteria, 100% of pupils in school 1 lived in a 'relatively prosperous' residential area, whilst 75% of the pupils in school 2 resided in an 'economically disadvantaged' area. In school 1, 0.2% of year 8 pupils were registered as being entitled to receive free school meals, whereas a substantially higher value of 12.6% was reported for school 2. Whereas the pupils in school 1 were considered 'advantaged' according to Estyn's criteria, school 2's intake were deemed 'disadvantaged'. The participants were bilingual (English-Welsh), and of mixed academic ability. The data derived from these forms, along with the evaluation of Census 2001 (National Statistics Online, 2003) allowed the author to differentiate between the schools according to socio-economic status. In Census 2001, the 'Indices of Deprivation 2000' (with rank 1 being the most deprived ward in Wales) gave the catchment area for school 1 a rank of 659 out of a total of 865 Welsh electoral divisions, while school 2's catchment area was given a rank of 114. From the above evidence,

school 1 was designated as being of a high socio-economic status, whilst school 2 represented low SES.

Informed written consent was sought from the pupils' parents or guardians. Parents or guardians were asked to obtain their child's agreement before consenting (Appendix 3). Both parties were requested to provide signatures. All measurements were undertaken in the subjects' respective schools. A total of sixty visits were made to the schools. Prior to participation, all protocols were explained fully, yet simply, to the participants. Reasons for the inclusion of the tests were also given. All pupils were assured anonymity, and informed that they were free to withdraw from the project at any time. All physiological and physical tests were conducted at the same relative time intervals and by the same tester, namely the author. An identical team of two medical doctors and five qualified phlebotomists were transported to both schools to conduct haematological tests. As with other measures, haematological tests were carried out at the same relative time intervals. The study protocol was approved by the University of Glamorgan Ethical Committee.

3.2 ANTHROPOMETRIC MEASUREMENTS

Protocols were similar to those recommended in the Anthropometric Standardization Reference Manual (Lohman, Roche and Martorell (eds), 1988). All physical and physiological measurements were completed between the times of 9.00 am and 11.30 am. Subjects were dressed in shorts and t-shirt, and were barefoot for all anthropometric measurements.

3.2.1 Stature

Equipment:

Portable stadiometer (Holtain Ltd, Crymych, UK)

Flexible tape measure

Protocol:

Standing height was measured using a portable stadiometer constructed by Holtain Ltd, Crymych, UK. Prior to each testing session, the accuracy of the stadiometer was verified with a tape measure. The measurement was taken as the maximum distance from the floor to the vertex of the head when standing barefoot and on a flat surface. Subjects were instructed to stand erect, heels together with the arms hanging loosely to the sides and palms facing inwards. The heels, buttocks, upper part of the back, and rear of the head were in contact with the wall. Using both hands, the author cupped the child's head along the mastoid processes. Pupils were instructed to look directly ahead, inhale deeply, and stand tall. To aid measurement the author applied gentle upward traction. The Brocca plane of the stadiometer was lowered to the most superior point of the subject's head whilst held in the Frankfort plane. Measurements were read to the nearest millimetre (Cameron, 1986; Gordon, Chumlea and Roche, 1988).

3.2.2 Body mass

Equipment:

Phillips electronic weighing scales (HP 5320)

Record sheet

Free weight of 5 kg mass

Protocol:

Body mass in air was calculated using electronic weighing scales (HP 5320). The weighing scales were calibrated prior to each testing session with a free weight of 5 kg mass. Readings were taken to the nearest tenth of a kilogram. All subjects stood barefoot with body mass evenly distributed between the feet. To obtain the most accurate readings weighing was conducted after voiding, thus minimising extraneous mass (Cameron, 1986; Gordon, Chumlea and Roche, 1988).

3.2.3 *Body mass index*

Protocol:

Body mass index (BMI) is regarded as an established method of determining overweight in children and adolescents (Cole, 1991; Himes and Dietz, 1994; Cole, Freeman and Preece, 1995, Teixeira, Sardinha, Going *et al.* 2001; Chinn and Rona, 2002; Mei, Grummer-Strawn, Pietrobelli *et al.* 2002). BMI was measured as the mass divided by stature squared (kg m^{-2}). To calculate BMI, body mass was measured in kg, and stature was converted from centimetres to metres (cm/100).

3.2.4 *Skinfold thicknesses*

Equipment:

Harpenden skinfold calipers (Holtain Ltd, Crymych, UK)

Water based marker pen

Siber-Heanger GPM Martin type anthropometric tape

Protocol:

Skinfold thickness were measured at four sites (Durnin and Rahaman, 1967) and on the right hand side of the body. Prior to any measurements being taken, specific skeletal and soft tissue landmarks were identified on the pupils using a water-based marker pen. Measurements were obtained at the triceps, biceps, subscapular and suprailiac sites using Harpenden skinfold calipers that exert a constant pressure of 10 gm^{-2}. In order to minimise error and ensure reliability, the protocol was repeated, with a third measurement taken if the first two measurements differed by more than 1.0 mm (Campaigne *et al.* 1994). For all measurements, skinfolds were raised using a pinching and rolling motion of the thumb and index finger of the left hand; this was done 1 cm above the marked site (Lohman *et al.* 1988). Skinfolds were held throughout the measurement and the calipers were positioned at right angles to the folds. Once the dial steadied, the reading was taken. Measurements were recorded to the nearest 0.1 mm. The majority of regression equations used to convert skinfold thicknesses to body fat percentage are based on adults. Considering the lack of accurate regression equations developed for young people, and since the pubertal status of the present cohort was not identified, the summation of the four skinfold thicknesses was considered an appropriate index of subcutaneous adiposity in the present study (Roemmich *et al.* 1997).

Triceps skinfold:

The subject's elbow was flexed at 90° and a point was marked at the mid acromiale-radiale line. A fold was raised 1 cm above the marked point on posterior side of the arm; the caliper was applied at the marked level.

Biceps skinfold:

A vertical fold was raised at the marked mid-acromiale-radiale line on the anterior side of arm. The caliper was applied 1 cm below the fingers.

Subscapular skinfold:

A fold was taken oblique to the inferior angle of the scapular in a direction running obliquely downward and laterally at an angle of about 45°. The caliper was applied 1 cm below the fingers.

Suprailiac Skinfold:

An oblique skinfold was raised at a position immediately superior to the anterior superior iliac spine. The caliper was applied 1 cm below the fingers.

3.2.5 Circumferences

Equipment:

Siber-Heanger GPM Martin type anthropometric tape

Protocol:

A flexible tape with millimetre markings was used to obtain girth measurements. Skin surfaces were not compressed, and there was no observable space between the skin and the tape during measuring. The tape loop was positioned perpendicular to the body segment being measured, and the cross-handed approach was employed. The pupils stood erect with feet together.

Hip girth

Measurements were taken at the level of the greatest posterior protuberance of the gluteals.

Waist girth

Measurements were taken at the narrowest part of the torso when observing from the anterior aspect. If difficulty arose in trying to locate the narrowest part, measurements were taken approximately halfway between the ribs and the iliac crest.

Waist to hip ratio

Waist to hip ratio (WHR) provided an index of relative fat distribution (Gillum, 1987; Sangi, Mueller, Harrist *et al.* 1992; Ledoux, Lambert, Reeder *et al.* 1997; Lurbe, Alvarez and Redon, 2001). Waist to hip ratio was measured as the waist girth divided by hip girth.

3.3 PHYSIOLOGICAL MEASUREMENTS

Subjects were dressed in shorts and t–shirt and wore suitable footwear.

3.3.1 Aerobic fitness

Equipment:

Tape recorder

C60 audiocassette for 20 metre Multistage Fitness Test (Brewer, Ramsbottom and Williams, 1998)

Cones

Stopwatch

Protocol:

The multistage fitness test or 20 metre shuttle test (20MST) initially developed for adults and later modified for children was used as a field test of aerobic fitness (Leger and Lambert, 1982; Leger *et al.* 1988). Results with specific populations have been favourable (van Mechelen *et al.* 1986; Boreham *et al.* 1990; Liu *et al.* 1992; Anderson, 1992; Barnett *et al.* 1993). Prior to the commencement of the test, assistants were assigned to record pupils' completed 20-m shuttles on a record sheet. Pupils ran back and forth between two parallel lines set 20 metres apart. The pace was set using a commercially available tape produced by the National Coaching Foundation that emitted a beep at the point pupils should be pivoting for the next line (Brewer *et al.* 1998). The speed of the tape player was confirmed prior to each data collection session. Running speed started at 8.5 $km \cdot hr^{-1}$ and increased by 0.5 $km \cdot hr^{-1}$ each minute, reaching 18.0 $km \cdot hr^{-1}$ at minute 20. Each level was announced on the tape, and a person trained in shuttle running accompanied the children throughout the test to help pacing. The pupils were told to keep up with the pacer until exhausted. If a pupil stopped or failed to get within approximately 2 m of an end line on two consecutive occasions, they were withdrawn. Consistent verbal encouragement was given throughout. Testing took place in the schools' sports halls. All pupils completed a prescribed warm up and warm down of light jogging and stretching. The main intention of this study was to investigate the relationship between physical fitness and CHD risk factors, and to compare across socio-economic status. The transformation of shuttle data to $\dot{V}O_2$ max is ill-advised (Andersen, 1992). For true maximal $\dot{V}O_2$ to be recorded, a plateau in $\dot{V}O_2$ must be reached yet few young people are motivated to reach this threshold. It was not the intention of the author to provide reference values for $\dot{V}O_2$ in adolescents, and it was felt that the 20 MST in the form of shuttle run scores, provided a simple and effective tool for assessing physical fitness in juveniles.

3.3.2 Blood pressure

Equipment:

Dinamap XL automatic blood pressure monitor

Pneumatic hose

Power cord

Cuffs

Protocol:

Blood pressure (BP) was measured indirectly using the Dinamap XL automatic blood pressure monitor (Critikron, Inc., Tampa, Fl.), which consisted of a compression bladder housed in a cuff. The Dinamap monitor has been validated in children (Park and Menard, 1987; Whincup *et al.* 1988). Before taking BP values, pupils sat for at least five minutes in a quiet environment (Tershakovec, Jawad, Stallings *et al.* 1998). Blood pressure was taken in the morning and the participant had not eaten or taken vigorous exercise during the 30 mins preceding the measurement. The pupils sat in chairs with a suitable support allowing the right arm to rest at a level that brought the antecubital fossa to approximately heart level. Pupils remained in a seated position throughout the BP measurements. The cuff was positioned over the brachial artery ensuring that the minimum cuff bladder width to arm circumference ratio of 40% as recommended by the American Heart Association was met (Whincup *et al.* 1996). The lower margin of the cuff was 2.5 cm above the antecubital space with the author able to insert two fingers between cuff and arm (Prescott-Clarke and Primatesta, 1997). Blood pressure was recorded three times, and the average of the second and third reading taken (Hagberg, Goldring, Ehsani *et al.* 1983; Prescott-Clarke and Primatesta, 1997; Harrell *et al.* 1998). Two minutes were given between measurements. The blood pressure measurements of

twenty pupils were compared with measurements taken from a random zero sphygmomanometer. There was no evidence of systematic drift (Whincup *et al.* 1988).

3.4 HAEMATOLOGICAL MEASUREMENTS

All haematological measurements took place in the respective schools' sports halls. Prior to testing, the author gave a simple explanation of why blood was being taken, how the procedure would be performed, and the level of discomfort the subjects might feel. The pupils were encouraged to ask questions, all of which were answered in a manner understandable to this particular age group. The school sports hall was divided into two areas by means of a large curtain. On one side of the hall, six separate areas were screened off to hold a chair, table and mat. On the other side of the curtain, pupils were instructed to remain seated for at least 30 minutes prior to testing and encouraged to relax by watching a video suitable for adolescent viewing.

Equipment

Trays

Tourniquets

21 gauge needles

Plastic holders

Vacutainer tubes: - 2 x SST, 1 EDTA and 1 sodium citrate per sample.

Medi-swabs

Cotton wool balls

Micropore tapes

Labels

Sharps containers

Rubber gloves

Freezer bags

Pipette

Permanent marker pens

Centrifuge appliance

Screens

Plastic aprons

3.4.1 Blood collection

Two medical doctors and five qualified phlebotomists, all of whom were experienced in paediatric sampling techniques, took blood samples from the pupils. The standard clinical procedures of the North Glamorgan NHS Trust (2001) were followed. These guidelines included health and safety procedures. Prior to each blood sampling session, a meeting was held at the respective schools during which the author confirmed that all medical doctors and phlebotomists were aware of the testing arrangements. As diet has been shown to exert a particular modulating effect on several blood-borne metabolites, samples were taken following an overnight fast. All pupils were given breakfast following their blood test. To control for biological variations, blood samples were collected between 8.30 am and 11.00 am (Reilly and Brooks, 1982). To control for plasma volume shifts, venous blood was sampled after the subject had assumed a seated position for at least 30 minutes (Pronk, 1993). To help allay anxieties, testing took place in the familiar surroundings of the respective schools' gymnasiums; children were approached in a confident and friendly manner; the procedure was again explained to individual pupils; and questions encouraged.

Pupils were asked whether they had received blood tests in the past and if so which arm was best for venepuncture. The hands of the operator were washed and gloves put on. The chosen arm was supported on an appropriate surface and a tourniquet was applied to the upper arm at mid-biceps brachii level, ensuring that the tourniquet could be easily released without compromising the needle's position. The arm was placed in a dependant position and when necessary the subject assisted the filling of a vein by clenching and unclenching the fist. The anticubital fossa was examined and the vein selected by palpitation or by sight. The median cubital vein was the first choice. The tourniquet was applied with the minimum constriction required to obtain a blood sample (Bachorik, 1982). The skin overlying the vein was cleansed with a medi-swab saturated with 70% v/v Isopropyl alcohol (Medi Swab, Smith and Nephew, UK) and allowed to dry. The green shank was removed from the operating end of the needle (Greiner Labortechnik Ltd, Glos.) and the needle inserted firmly into the vein from the direction in which the vein passed. The orifice of the needle pointed towards the operator. The required vacutainer tube (Becton Dickinson, Rutherford, NJ, USA) was passed onto the rubber-covered needle within the holder. Since firm pressure was applied at this juncture, counter pressure was needed to prevent the needle from being thrust further into the vein. The required amount of blood automatically passed into the tube and care was taken to ensure the blood had ceased flowing before the tube was removed. The tourniquet was released following removal of the last tube. Approximately 15 ml of venous blood was taken from the vena antecubitus. A cotton wool ball was placed over the point of needle entry into the skin, and as the needle was removed, the cotton wool ball was pressed firmly onto the puncture site. The subject was instructed to press the cotton wool ball onto the puncture site and to refrain from bending the arm. The needle was disposed of in the sharps disposal box. The cotton wool ball was held in position using micropore tape. All samples were labelled using a black waterproof marker pen.

Information included subject's name and number, date of birth, and school. Tubes were placed within a sealable plastic bag. Once the medical doctor and phlebotomist were satisfied that the subject was comfortable, the pupil was allowed to leave the medical centre. Following the blood test, the pupil was directed to sit in the canteen whilst the school nurse accompanied him/her during breakfast. In the event of a pupil fainting, both a team medical doctor and school nurse examined the pupil, and a letter was sent to the respective parent informing them of the incident. This occurred on two occasions.

Samples were analysed for total cholesterol (TC), triglycerides (TG), high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C), lipoprotein(a) (Lp(a)), glucose (Glu), homocysteine (Hcy), folate, vitamin B_{12}, and fibrinogen (Fg). For TC, TG, HDL-C, LDL-C, Lp(a), and glucose; blood samples were collected in ochre SST vacutainers, which included both clotting accelerator and separation gel. For Hcy, folate, and B_{12}, blood samples were collected in lavender EDTA vacutainers. Blood samples in SST and EDTA vacutainers were allowed to clot and then centrifuged at 3,500 rpm for 10 mins. Serum and plasma were extracted and placed into a vacutainer for subsequent analysis. For Fg, blood samples were collected in blue sodium citrate tubes. This tube was filled to the mark on the bottle. Depending on the haematological variable to be assayed, blood samples were transported immediately to the Biochemistry Departments of the Royal Glamorgan Hospital, Llantrisant, or Llandough Hospital, Penarth.

3.4.2 Blood analysis

3.4.2.1 Total cholesterol (TC)

Vitros CHOL Slides quantitatively measure cholesterol (CHOL) concentration in serum and plasma using the *Vitros* 950 System (Ortho-Clinical Diagnostics, Amersham, Bucks). The *Vitros* CHOL Slide is a dry, multilayered, analytical element coated on a polyester support. The method is based on an enzymatic protocol. A 10 µl drop of patient sample is deposited on the slide and is evenly distributed by the spreading layer to the underlying layers. The Triton X-100 (TX100) surfactant in the spreading layer aids in dissociating the cholesterol and cholesterol esters from lipoprotein complexes present in the sample. Hydrolysis of the cholesterol esters to cholesterol is catalysed by cholesterol ester hydrolase. Free cholesterol is then oxidized in the presence of cholesterol oxidase to form cholestenone and hydrogen peroxide. Finally, hydrogen peroxide oxidizes a leuco dye in the presence of peroxidase to generate a coloured dye. The density of dye formed is proportional to the cholesterol concentration present in the sample and is measured by reflectance spectrophotometry.

Wavelength:

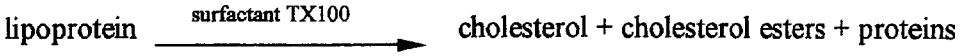
540 nm

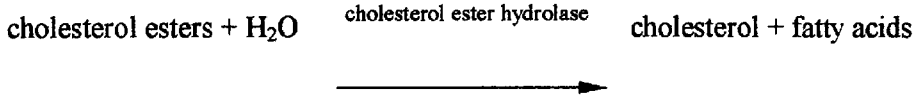
Assay time and temperature:

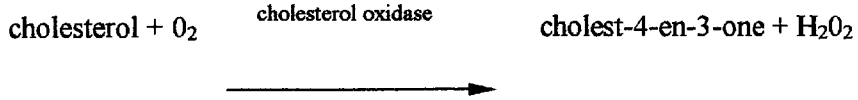
Approximately 5 minutes at 37°C

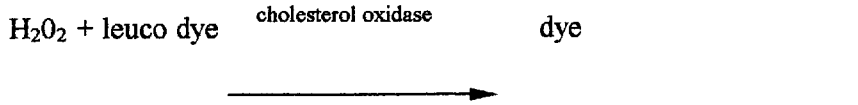
Reactive ingredients on the slide are Triton X-100; cholesterol oxidase; cholesterol ester hydrolase; peroxidase; and 2-4, 5-bis imidazole (leuco dye). Other ingredients include pigment, binder, buffer, surfactants, stabilizers, and cross-linking agent.

Reaction Sequence:









Calibration:

- when changing lot numbers of primary reagent packs
- when replacing system components
- when quality control results are repeatedly out of range
- when government regulations require

Reportable Range (Dynamic Range):

Conv. Units ($mg \cdot dL^{-1}$): 50.0-325.0

SI Units ($mmol \cdot L^{-1}$): 1.29-8.40

Alternate Units ($g \cdot L^{-1}$): 0.50-3.25

Laboratory analytical variance: 1.6%

3.4.2.2 High density lipoprotein-cholesterol (HDL-C)

The IL Test™ HDL-C Cholesterol allows a direct quantitative in *vitro* diagnostic determination of HDL-C in human serum and plasma using the ILAB™ 600 System (Instrumentation Laboratory Company, Lexington, MA, USA). The assay is a homogenous, direct method for measuring levels of HDL-C without the need to pre-treat. It is a two-reagent assay which uses the combination of a detergent to specifically solubilize the HDL-C particles in the sample, and a polyanion to assist in the selectivity by complexing the LDL-C, VLDL-C and chylomicron lipoproteins. The released HDL-C is then available to react with cholesterol esterase and cholesterol oxidase in the presence of chromogens to produce a blue colour complex. The amount of colour developed is measured on the IL system. High density lipoprotein-cholesterol concentration can be calculated by comparing the absorbance of the blue colour complex with the absorbance of the ILab HDL-C Calibrator .

Reactive ingredients of reagents are:

- HDL-C R1
- polyanion
- 4-aminoantipyrine
- HDL-C R2
- cholesterol oxidase
- cholesterol esterase
- perioxidase
- detergent
- N, N-bis (4-sulphobutyl)-m-toluidine-disodium (DSBmT)

Non-Reactive Ingredients:

MES buffer (pH 6.5), preservatives

Wavelength:600 nm

Calibration:

- when changing lot numbers of primary reagent packs
- when replacing system components
- when quality control results are repeatedly out of range
- when government regulations require

Linearity Range:

No rerun 1-150 $mg \cdot dL^{-1}$ (0.03-3.89 $mmol \cdot L^{-1}$)

With rerun 1-225 $mg \cdot dL^{-1}$ (0.03-5.83 $mmol \cdot L^{-1}$)

Laboratory analytical variance: 5.3%

3.4.2.3 Low density lipoprotein-cholesterol (LDL-C)

LDL-C was derived according to:

LDL-C ($mmol \cdot L^{-1}$) = total cholesterol – triglycerides / 2.2 - HDL-C

(Friedewald, Levy and Fredrickson, 1972)

3.4.2.4 Triglycerides (TG)

Vitros TRIG Slides quantitatively measure triglycerides (TRIG) concentration in serum and plasma using the Vitros 950 System (Ortho-Clinical Diagnostics, Amersham, Bucks). The Vitros TRIG Slide is a dry, multilayered, analytical element coated on a polyester support. The method is based on an enzymatic protocol. A 10 µl drop of

patient sample is deposited on the slide and is evenly distributed by the spreading layer to the underlying layers. The Triton X-100 (TX100) surfactant in the spreading layer aids in dissociating the triglycerides from lipoproteins complexes present in the sample. The TG molecules are then hydrolysed by lipase to yield glycerol and fatty acids. Glycerol diffuses to the reagent layer, where it is phosphorylated by glycerol kinase in the presence of adenosine triphosphate. In the presence of L-α- glycerol-phosphate oxidase, L-α-glycerophosphate is then oxidized to dihydroxyacetone phosphate and hydrogen peroxide. The final reaction involves the oxidation of a leuco dye by hydrogen peroxide, catalyzed by peroxidase, to produce a red coloured dye. The density of the dye formed is proportional to the TG concentration present in the sample and is measured by reflectance spectrophotometry.

Wavelength: 540 nm

Assay time and temperature:

Approximately 5 minutes at 37°C

Reactive ingredients on the slide are lipase; peroxidase; glycerol kinase; L-α-glycerol-phosphate oxidase; Triton X-100; and 2-4, 5-bis imidazole (leuco dye); and adenosine triphosphate. Other ingredients include pigment, binder, buffer, surfactants, stabilizers, scavenger, enzyme cofactors, dye solubizer, and cross-linking agent.

Reaction Sequence:

lipoprotein $\xrightarrow{\text{surfactant TX100}}$ triglycerides + proteins

triglycerides + H_2O $\xrightarrow{\text{lipase}}$ glycerol + fatty acids

glycerol + ATP $\xrightarrow{\text{glycerol kinase } MgCl_2}$ L-α-glycerophosphate + ADP

L-α-glycerophosphate +0_2 $\xrightarrow{\text{L-α-glycero-phosphate oxidase}}$ dihydroxyacetone phosphate + H_20_2

H_20_2 + leuco dye $\xrightarrow{\text{peroxidase}}$ dye + H_2O

Calibration:

- when changing lot numbers of primary reagent packs
- when replacing system components
- when quality control results are repeatedly out of range
- when government regulations require

Reportable Range (Dynamic Range):

Conv. Units ($mg \cdot dL^{-1}$): 10.0-525.0

SI Units ($mmol \cdot L^{-1}$): 0.11-5.93

Alternate Units ($g \cdot L^{-1}$): 0.10-5.25

Laboratory analytical variance: 2%

3.4.2.5 Lipoprotein(a) (Lp(a))

The measurement of Lp(a) in serum or plasma is complex. The variability in the size and structure of apo (a), homology (and thus potential cross-reactivity) with plasminogen, similarity to the apo B contained in LDL, and the appearance of apo (a) in both the free and lipoprotein-bound form, all contribute to this complexity (Kronenberg *et al.* 1996). Skewed distribution of Lp(a) levels towards the lower end (<10 $mg \cdot dl^{-1}$), together with wide individual variability, requires an assay with great sensitivity that is linear over a wide range to detect small differences between groups or across time. To confound matters further, the measurement of Lp(a) has not been standardised internationally, although several working groups have been established throughout the world to address this issue (Kronenberg *et al.* 1996).

In the present study, Lp(a) concentration was quantitatively measured using the Cobas Mira System. An anti-Lp(a) antibody coupled to latex particles forms high molecular weight immunocomplexes with Lp(a) antigen in the test sample. The resulting change in turbidity of the sample is detected photometrically and is directly proportional to the antigen concentration. These absorbance changes are converted into antigen concentration using a reference curve constructed with standards of known concentrations.

Wavelength: 550 nm

Reagents

1. Anti human : Lp(a) latex reagent (2 x 5 mls)

2. Lp(a) diluent (50 mls)
3. Lp(a) standard (6 x 0.5 mls)
4. Lp(a) control (2 x 0.5 mls)saline
5. 0.9% saline provided by the pharmacy department (Llandough Hospital)

Reagents 3 and 4 are lyophilised serum and are reconstituted at least 30 mins before testing.

The detection limit for Lp(a) was 8 $mg \cdot dl^{-1}$.

Laboratory analytical variance: 6%

3.4.2.6 Glucose (GLU)

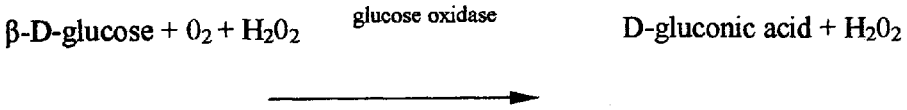
Vitros GLU Slides quantitatively measure glucose (GLU) concentrations in serum, plasma, urine, and cerebrospinal fluid using the *Vitros* 950 System (Ortho-Clinical Diagnostics, Amersham, Bucks). The *Vitros* GLU Slide is a dry, multi-layered, analytical element coated on a polyester support. A 10 µl drop of patient sample is deposited on the slide where the spreading layer promotes the uniform distribution of the sample and permits an even penetration of solute molecules into the underlying reagent layer. The oxidation of sample glucose is catalysed by glucose oxidase to form hydrogen peroxide and gluconate. An oxidative coupling catalysed by peroxidase in the presence of dye precursors to produce a red dye follows this reaction. The intensity of the dye is measured by reflected light.

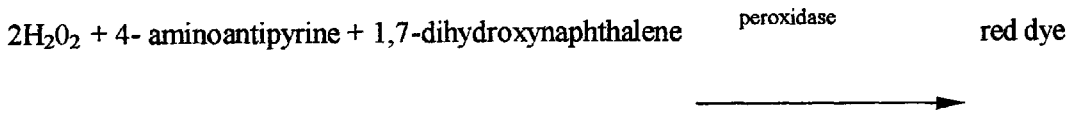
Wavelength: 540 nm

Assay time and temperature:

Approximately 5 minutes at 37°C

Reaction sequence:





Reactive ingredients included on the slide are glucose oxidase; peroxidase; 1,7-dihydroxynaphthalene (dye precursor); and 4-aminoantipyrine hydrochloride (dye precursor). Other ingredients include pigment, binders, buffer, surfactants, stabilizers, and cross-linking agent.

Calibration:

- when changing lot numbers of primary reagent packs
- when replacing system components
- when quality control results are repeatedly out of range
- when government regulations require

Reportable Range (Dynamic Range):

Conv. Units ($mg \cdot dL^{-1}$): 20.0-625.0

SI Units ($mmol \cdot L^{-1}$): 1.11-34.69

Alternate Units ($g \cdot L^{-1}$): 0.20-6.25

Laboratory analytical variance: 1%

3.4.2.7 Fibrinogen (Fg)

The IL Test™ Fibrinogen-C allows the quantitative determination of fibrinogen (Fg) in human plasma using IL Coagulation Systems (Instrumentation Laboratory Company, Lexington, MA, USA). The test uses an excess of thrombin to convert Fg to fibrin in diluted plasma. At high thrombin and low Fg concentration, the rate of reaction is a function of Fg concentration. The Fibrinogen-C kit consists of eight vials of Bovine thrombin (35 UNH/ml), bovine albumin, calcium chloride, buffer and filler and two vials of abnormal control plasma (human citrated plasma containing a reduced level of Fg). The ACL Futura Analyser measures Fg by turbidimetric (cogulometric) clot detection. The system measures and records the amount of time required for a plasma specimen to clot. Coagulation endpoint is detected by measuring change in optical density. Turbidimetric (cogulometric) clot detection is based on the principle that light passing through a medium as Fg is converted to fibrin and will be absorbed by the fibrin strands. Light transmitted through the plasma sample is monitored by a sensitive photodetector positioned 180° to the incident source. Light absorption increases as fibrin clot formation progresses. Consequently, light transmittance through the sample continuously decreases and is measured by the photodetector. The corresponding electrical signal output from the photodetector changes according to the detected light. The signal output is processed via software through a series of algorithms to determine the clot point.

Calibration:

- when changing lot numbers of primary reagent packs
- when replacing system components

- when quality control results are repeatedly out of range
- when government regulations require

Linearity Range:

80-700 $mg \cdot dl^{-1}$

Laboratory analytical variance: 1.6%

3.4.2.8 Folate

The Beyer Centaur® Folate assay allows a quantitative in *vitro* diagnostic determination of folate in human serum or plasma using the ADVIA Centaur® System (Bayer Corporation, USA). It is a competitive immunoassay using direct chemiluminescent technology. Folate in the subject sample competes with acridinium ester-labeled folate in the Lite Reagent for a limited amount of biotin-labeled folate binding protein. Biotin-labeled folate binding protein binds to avidin that is covalently linked to paramagnetic particles in the Solid Phase. In the Beyer Centaur® Folate assay the sample is pre-treated to release the folate from endogenous binding proteins in the sample.

The system automatically performs the following procedure:

- dispenses 150 µL of sample into a cuvette
- dispenses 50 µL of DTT/Releasing Agent
- dispenses 100 µL of Folate Binding Protein and 200 µL of Solid Phase, and incubates for 5.0 minutes at 37°C
- dispenses 100 µL of Lite Reagent and incubates for 2.5 minutes at 37°C
- separates, aspirates and washes the cuvettes with reagent water

- dispenses 300 µL each of Acid Reagent and Base Reagent to initiate the chemiluminescent reaction
- reports results

An inverse relationship exists between the amount of folate present and the amount of relative light units detected by the system.

Calibration:

A two-point calibration is used with the Beyer Centaur® Folate assay:

- when changing lot numbers of primary reagent packs
- when replacing system components
- when quality control results are repeatedly out of range
- when government regulations require

Sensitivity and Assay Range:

0.35-24 ng/ml (0.79-54.36 $nmol \cdot L^{-1}$)

Laboratory analytical variance: 4.8%

3.4.2.9 Vitamin B_{12} (VB_{12})

The ADVIA Centaur® VB_{12} System (Bayer Corporation, USA) allows a quantitative in *vitro* diagnostic determination of Vitamin B_{12} in human serum or plasma. It is a competitive immunoassay using direct chemiluminescent technology. VB_{12} from the subject sample competes with VB_{12} labelled with acridinium ester in the Lite Reagent for a limited amount of purified intrinsic factor, which is covalently linked to

paramagenetic particles in the Solid Phase to prevent rebinding after the Solid Phase is added to the sample.

The system automatically performs the following procedure:

- washes the ancilliary reagent probe with 100 µL of T3/T4/ VB_{12} Ancilliary Reagent
- dispenses 100 µL of sample into a cuvette
- dispenses 115 µL of DTT/Releasing Agent
- dispenses 200 µL of Solid Phase and incubates for 5.0 minutes at 37°C
- dispenses 200 µL of Lite Reagent and incubates for 2.5 minutes at 37°C
- separates, aspirates and washes the cuvettes with reagent water
- dispenses 300 µL each of Acid Reagent and Base Reagent to initiate the chemiluminescent reaction
- reports results

An inverse relationship exists between the amount of vitamin B_{12} present and the amount of relative light units detected by the system.

Calibration:

A two-point calibration is used with the ADVIA Centaur® B_{12} assay:

- when changing lot numbers of primary reagent packs
- when replacing system components
- when quality control results are repeatedly out of range
- when government regulations require

Sensitivity and Assay Range:

45-2000 pg/ml (33-1476 pmol·L^{-1})

Laboratory analytical variance: 1.6%

3.4.2.10 Homocyst(e)ine (Hcy)

The AxSYM® Homocysteine assay is a Fluorescence Polarization Immunoassay (FPIA) for the quantitative measurement of total Hcy in human serum or plasma on the Abbott AxSYM® system (Abbott Laboratories Abbott Park, IL, USA). The AxSYM Homocysteine assay is based on the Fluorescence Polarization Immunoassay (FPIA) technology. Bound homocysteine (oxidised form) is reduced to free homocysteine that is enzymatically converted to S-adenosyl-L-homocysteine. Homocysteine, mixed disulphide, and protein-bound forms of Hcy in the sample are reduced to form free homocysteine by the use of dithiothreitol.

The AxSYM Homocysteine reagents and sample are pipetted in the following sequence:
Sampling Centre

- Sample and all AxSYM Homocysteine Reagents required for one test are pipetted by the sampling probe into various wells of a Reaction Vessel (RV).
- Sample, Pretreatment Solution, Solution 4 (Line Diluent), and SAH Hydrolase are pipetted into one well of the RV to make up the predilution mixture.

The RV is immediately transferred into the Processing Centre. Further pipetting is done in the Processing Probe.

Processing Centre

An aliquot of the predilution mixture, Antibody, and Solution 4 (Line Diluent) are transferred to the cuvette of the RV. Tracer, Solution 4, and a second aliquot of the predilution mixture are transferred to the cuvette. SAH and labelled Fluorescein Tracer compete for the sites on the monoclonal antibody molecule. The FPIA optical assembly measures the intensity of polarized fluorescent light.

Calibration:

A six-point calibration is used with the Abbott AxSYM System assay:

- when changing lot numbers of primary reagent packs
- when replacing system components
- when quality control results are repeatedly out of range
- when government regulations require

Sensitivity: The sensitivity of the Abbott AxSYM System was calculated to be ≤ 0.8 $\mu mol \cdot L^{-1}$.

3.5 LIFESTYLE MEASUREMENTS

3.5.1 Health and socio-cultural questionnaire

A confidential health questionnaire (Appendix 3) with reply paid postage was forwarded to parents. The questionnaire established any family history of CHD and was used as a cross-check on medical suitability for pupil participation. A child was allowed to undergo testing only after completed questionnaires had been analysed by the author.

3.5.2 Smoking questionnaire

Smoking status was ascertained by means of a confidential questionnaire (Appendix 4).

3.5.3 Dietary intake questionnaire and seven-day diary

A confidential seven-day food diary, supplemented by a questionnaire (Appendix 5) was implemented for dietary assessment. Each pupil was given a copy of a food diary sheet in which they recorded everything they ate and drank over seven days. Data collection took place during the first week of March, 2002. To assist pupils in the completion of the diary the author conducted a 45 min training session. Participants were encouraged to continue with normal eating and drinking habits (Kemper and Van Mechelen, 1995) and were required to complete the diary and questionnaire in conjunction with their parents. Written instructions were provided to help pupils describe intake and estimate portion size. During the week of recording, the author was available by telephone to answer any queries. The completed questionnaires were scrutinized, and where necessary responses were checked via personal interviews with pupils. Pupils were asked to repeat this procedure twice over a period of time to ensure accuracy. The collated data were computer analysed by Health Options LTD (Health Options LTD., Love Lane, Cirencester, Gloucester, UK). Average daily calories from food, average daily calories from alcohol, average daily fibre, and percentage of total fat, saturated fat, carbohydrate and protein were calculated.

3.5.4 Physical activity questionnaire and seven-day recall

A confidential seven-day recall and supplementary physical activity questionnaire was distributed to all pupils (Appendix 6). Data collection took place during the second week of March, 2002. To aid pupils in the completion of the seven-day recall and questionnaire, the author conducted a 45 min training session. When necessary,

assistance was given to individual pupils to ensure comprehension of the questions posed. The author was on hand for any queries and concerns. The completed questionnaires were scrutinized and where necessary the author checked the responses via personal interview with pupils.

The method used for ascertaining the physical activity habits of youngsters in the present investigation was based on the work of Riddoch (1990) in the Northern Ireland Health and Fitness Survey. Montoye *et al.* (1996) remarked that the questionnaire was diligently designed and successfully implemented in a large study of adolescents. The author of the present study further determined the reliability of the questionnaire via a pilot study of children of similar age range, and from a comparable Welsh comprehensive school. The questionnaire was distributed twice to the same group of children 2 weeks apart. The majority of the children completed the questionnaire in less than 30 minutes and this was considered conducive to maintaining concentration (Riddoch, 1990; Harro and Riddoch, 2000). Validity was ensured by (i) obtaining additional information from Physical Education teachers (Saris, Binkhorst, Cranwinckel *et al.* 1980) (ii) the inclusion of repeat questions; (iii) requesting that questions be answered in both English and Welsh; (iv) ensuring that the children sat in groups of less than twenty and completed the questionnaires independently; (v) posing similar questions in both the seven-day recall and supplementary questionnaire, thus making certain that pupils responded truthfully.

To ensure that the methods of establishing physical activity were appropriate to the age group being studied, the author followed the guidelines suggested by various investigators:

- Recall was over a short time only ie previous week (Montoye *et al.* 1996)
- Questions referred to all days of the week, including weekends (Harro and Riddoch, 2000)
- Information on intensity, duration and frequency was sought (Montoye *et al.* 1996)
- The language was suitable for adolescents (Montoye *et al.* 1996; Harro and Riddoch, 2000; Guerra *et al.* 2001)
- A time frame based on the school day was used to improve accuracy (Riddoch *et al.* 1991; Montoye *et al.* 1996; Boreham *et al.* 1997; Twisk *et al.* 1999)
- Children were 'cued' by activities
- A minimum monitoring period of three days (Bar-Or, 1983; Gretebeck and Montoye, 1989)
- Questionnaires were administered by the author
- Determining energy expenditure from self-report measures was not sought (Armstrong and Welsman, 1997)
- Measurements did not interfere with normal activity patterns (Harro and Riddoch, 2000)

Questionnaires were available in both Welsh and English.

Post data analysis, the author re-visited the participating schools to give individual and group feedback on the study's findings. All pupils were given bilingual feedback proformas. The pupils were encouraged to ask questions, in confidence if necessary, and were informed that parents should contact the author if further clarification or advice was needed.

3.6 STATISTICAL METHODS

Data were analysed using a computerised statistical package (Minitab V.12, 1998).

To avoid a *type 2* error, the level of significance was set at $P \leq 0.05$. A *type 2* error occurs when the author accepts a false null hypothesis (H_0), moreover, anything that reduces the likelihood of a *type 2* error increases the power of a test (Vaughan, 1998). In this study, the author elected to protect against a *type 2* error since a failure to identify a real significant difference between means (*type 2* error) could lead to a missed opportunity for improved knowledge.

3.6.1 One way analysis of variance (ANOVA)

The Anderson-Darling test was applied to identify whether the residuals followed Gauss's curve of normal distribution (Munro, Jacobsen, Duffy *et al.* 2001). Homogenity of variance was assessed using Levene's test (Bryman and Cramer, 1996). For normally distributed data a simple one-way ANOVA was used to identify whether differences in CHD risk factors existed between schools and/or between sexes. The ANOVA produces a ratio value called F (F = average variance between groups divided between average variance within groups).

Null hypothesis (H_0) for F test: $\mu_1 = \mu_2 = \mu_3 = \mu_4 = 0$

Where:

$_1$ = boys in school 1

$_2$ = girls in school 1

$_3$ = boys in school 2

$_4$ = girls in school 2

Where residuals were not confirmed as normally distributed in the populations from which the samples were drawn, the researcher applied a Box and Cox test (Box and Cox, 1964). This procedure identified whether it was prudent for the data to be transformed thus allowing the application of an ANOVA.

The condition of equal residual is known as homoscedasticity. To ensure that the data being analysed were homoscedastic, the researcher correlated absolute residuals against the fits. If $P > 0.05$, it was concluded that the data were homoscedastic.

Where data could not be transformed in a meaningful manner, the researcher applied a non-parametric Kruskal-Wallis test which is the non-parametric analog of the ANOVA. The Kruskal-Wallis test produces a H value that approximates to the chi-square distribution.

The test statistic for H is:

$$[12 / N(N+1)] [\sum R_1^2 / n_1 + \sum R_2^2 / n_2 + \ldots\ldots \sum R_k^2 / n_k] - 3(N+1)$$

Where:

N = total of all subjects in all groups

n = subjects per group

k = number of groups

$_1$ = boys in school 1

$_2$ = girls in school 1

$_3$ = boys in school 2

$_4$ = girls in school 2

If statistically significant F ratios or H values were identified, a *post-hoc* Bonferroni-adjusted test was applied. The Bonferroni-adjusted test modifies the alpha level to account for more than one comparison being made. To obtain the appropriate level of significance, the assigned alpha level (0.05) was divided by the total number of comparisons between any two groups, in this case six. The result of this calculation ($0.05 / 6 = 0.0083$) was the designated significance level for comparing across groups. Bonferroni-adjusted t-tests for two independent means were used for significant F ratios.

Null hypothesis (H_0) for t test: $\mu_1 = \mu_2 = 0$

For significant H values, a Bonferroni-adjusted Mann-Whitney U test was applied. The Mann-Whitney test is the non-parametric analog of the independent t-test.

The formula for U_1 is: $U_1 = n_1 n_2 + (n_1 (n_1 + 1)) / 2 - \sum R_1$

The formula for U_2 is: $U_2 = n_1 n_2 - U_1$

Where:

n_1 and n_2 = number of subjects in each group

$\sum R_1$ = sum of the rankings for group 1.

3.6.2 Principal-components factor analysis with varimax orthogonal rotation

Principal-components factor analysis (PCA) allows for the conversion of a set of variables into a new set of variables that is an exact mathematical transformation of the original data (Dixon, 2001). The goal of PCA is to reduce the number of variables that the author has to handle (Bryman and Cramer, 1996). The extraction of components was made according to Kaiser's criterion. This extraction method retains for rotation those components that have an eigenvalue greater than 1 (Bryman and Cramer, 1996). The Kaiser citerion is the recommended method when the number of variables is less than 30 and the average communality is greater than 0.70 (Stevens, 1992). As this was the case in each of the PCA applied in the present study, Kaiser's citerion was adopted. A varimax-orthogonal rotation was performed on the retained components. The meaning of a component was determined by the variables that loaded most highly on it. Variables, that correlated less than 0.3 with a component were not considered (Bryman and Cramer, 1996, Dixon, 2001).

3.6.3 Multiple regression analysis

Multiple regression analysis (MRA) was used to assess the significance of selected independent variables (X) on selected dependent variables (Y). The prediction formula for multiple regression is represented by:

$$Y = a + b_1 X_1 + b_2 X_2 + b_3 X_3 \ldots\ldots + b_k X_k + e$$

Where:

Y = dependent variable

a = intercept

b = regression coefficients

X = independent variables

e = error term

k = number of independent variables

After selection of the dependent variable (Y), Y was correlated with all independent variables (X) to produce a correlation coefficient matrix. Only X variables that were significant ($P \leq 0.05$) with Y, were included in the MRA. If the Pearson's r between each pair of independent variables was in excess of 0.8, these X variables were suspected of multicollinearity and omitted from the MRA. Since for this study more than one X variable was always included in a prediction equation, it was necessary to standardise the units of measurement. The b values were converted to β–weights and these indicated which independent variable had the greatest impact on the selected dependent variable.

To assess whether data were homoscedastic the researcher correlated absolute residuals against the fits. If $P > 0.05$, it was concluded that the data were homoscedastic.

Example of a multiple regression *unstandardised* prediction equation in the present study:

SBP_p (mm Hg) = 74.7 + (0.378 x DBP (mm Hg)) + 0.723 x BMI (kg m^{-2}))

Where:

Systolic blood pressure (SBP_p) = dependent variable

74.7 = intercept

0.378 = regression coefficient for the first independent variable, diastolic blood pressure (DBP)

0.723 = regression coefficient for the second independent variable, body mass index (BMI)

Example of a multiple regression *standardised* prediction equation in the present study:

$SBP_p = -0.0007 + (0.337 \times DBP) + 0.255 \times BMI)$

Where:

Systolic blood pressure (SBP_p) = dependent variable

-0.0007 = intercept

0.337 = β–weight for the first independent variable, diastolic blood pressure (DBP)

0.255 = β–weight for the second independent variable, body mass index (BMI)

Thus indicating that as the β–weight for DBP is greater than that for BMI, DBP is the stronger predictor of SBP.

3.6.4 Effect size

The effect size serves as an index of meaningfulness (Cohen, 1988; Mullineaux, Bartlett and Bennett, 2001). Effect size was calculated in all cases where a test of equality of means (and medians) was established as statistically significant.

(i) ANOVA

For statistically significant F ratios

$$\omega^2 = [SS_{\text{BETWEEN GROUPS}} - ((k\text{-}1) \times MS_{\text{ERROR}})] / (SS_{\text{TOTAL}} + MS_{\text{ERROR}})$$

Where:

SS = sums of squares

MS = mean squares

k = number of means

(ii) Kruskal-Wallis

For statistically significant H values

$$\varphi = \sqrt{(\chi^2 / N)}$$

Where:

φ = A real product-moment correlation coefficient

χ^2 = Chi-square (compares $\geq$ 2 sets of nominal data that have been arranged into categories by frequency counts).

N = total sample size

(iii) t- test for two independent means (*post-hoc*)

$$ES = |\bar{x}_1 - \bar{x}_2| / s$$

Where:

$|\bar{x}_1 - \bar{x}_2|$ = difference between two means

s = pooled standard deviation

For the purposes of this study:

$ES \leq 0.2$ = small difference

$ES = 0.5$ = moderate difference

$ES \geq 0.8$ = large difference (Cohen, 1988)

3.6.5 Statistical power of the test

The statistical power of a test is the ability to correctly detect a difference or relationship if such a difference or relationship actually exists (Munro *et al.* 2001). It is the confidence placed in the rejection of the H_0 and an 80% level is regarded as an adequate level (Munro *et al.* 2001). Effect size and sample size (n), allowed the estimation of statistical power. To estimate statistical power the researcher referred to Cohen's (1988) Statistical Power Analysis tables,

where for:-

ANOVA

n (mean sample number) = $(n_1 + n_2 + n_3 + n_4) / 4$

Kruskal- Wallis

n (total sample number) = $n_1 + n_2 + n_3 + n_4$

t-test for two independent means

n (harmonic mean) = $(2\ n_1 n_2) / (n_1 + n_2)$

$_1$ = boys in school 1

$_2$ = girls in school 1

$_3$ = boys in school 2

$_4$ = girls in school 2

3.6.6 Confidence intervals

Presenting *P* values alone can lead to their being given more importance than they deserve (Gardner and Altman, 2000). Indeed, the *P* value should be regarded as a useful adjunct to the confidence interval (*CI*) (Gardner and Altman, 2000). A *CI* is a range, or interval of values within which the population parameter is estimated to fall based on the statistic and standard error (SE). The sample size affects the size of the standard error and this in turn affects the width of the *CI.* Increasing the sample size will reduce the width of the *CI.* Estimates of the confidence interval at the 95% were given for all statistically significant *post-hoc* findings based on the formula: 95% = $\bar{x} \pm 1.96$ (SE)

3.7 CRITERION THRESHOLDS FOR CORONARY HEART DISEASE RISK

Smoking

Smokers were defined as those who admitted to smoking one or more cigarettes a week (Bewley, Day and Ide, 1972; Bouziotas *et al.* 2001).

Waist-to-hip ratio

A WHR below 0.8 for females and 0.9 for males was arbitrarily defined as 'normal' (Bjorntorp, 1992; Reeder, Angel, Ledoux *et al.* 1992) and used as cut-off points in this study.

Body mass index

Overweight in schoolchildren (12 to 13 years) was defined as a body mass index (BMI) above the 95th percentile of combined data from five national surveys conducted between 1963 and 1994 (Health Examination Survey II and III, and National Health and Nutrition Examination Surveys I, II, and III) (Freedman *et al.* 1999). Chinn and Rona (2002) identified similar UK cut-off points. Boys with a BMI > 24.3 kg m^{-2} were defined as 'overweight', whilst girls who recorded > 25.6 kg m^{-2} were regarded as 'overweight'.

Body fat

Pupils were designated as 'high risk' if boys had a $\sum$ SKF $\geq$ 56.9 mm, and girls had a $\sum$ SKF $\geq$ 57.4 mm (Riddoch, 1990).

Blood pressure

If values for systolic (SBP) and diastolic (DBP) were 130 mm Hg or 85 mm Hg respectively, pupils were allocated to a 'high risk' group (Wynder *et al.* 1981; National Institutes of Health, 1987; Armstrong *et al.* 1991[a]).

Aerobic fitness

Pupils were designated as 'high risk' if boys scored $\leq$ 47 shuttles (10th percentile) in the 20 MST. The corresponding value for girls was $\leq$ 28 shuttles (Riddoch, 1990).

Physical activity

Individuals were classified as 'at risk' according to vigorous physical activity criteria (Riddoch, 1990). It is recommended that young people should participate in vigorous activity for 30 minutes per day (Pate, Trost, Dowda *et al.* 1999; Bouziotas *et al.* 2001).

Diet

Pupils were classified as 'at risk' according to their consumption of total fat and saturated fat. The dietary thresholds adopted in this investigation were > 30% total fat and >10% saturated fat (WHO, 1990; NCEP, 1991).

Total cholesterol

Individuals who recorded values of ≥ 5.2 mmol·L^{-1} were deemed to be 'at risk' of developing CHD.

High density lipoprotein

Scoolchildren were classified as 'at risk' when values were recorded ≤ 0.9 mmol·L^{-1}.

Low density lipoprotein

Individuals were deemed 'at risk' of developing CHD if values were ≥ 3.8 mmol·L^{-1}.

Triglycerides

Pupils were allocated to an 'at risk' group if values were ≥ 1.7 mmol·L^{-1}.

TC: HDL-C

Individuals were classified as 'at risk' if values were ≥ 4.0.

The haematological cut-off points identified above were assigned according to Montoye *et al.* (1985); Bell *et al.* (1986); NCEP, (1991); Boreham *et al.* (1997); Bouzioutas *et al.* (2001).

Fibrinogen

Pupils were allocated into an 'at risk' group if values were > 394 $mg \cdot dL^{-1}$ or the 90^{th} percentile according to Sanchez-Bayle *et al.* (1993).

Lipoprotein(a)

Individuals were classified as 'at risk' if values were ≥ 30 $mg \cdot dL^{-1}$ (Valentine *et al.* 1994; Mackinnon & Hubbinger, 1999; Sveger *et al.* 2000).

Homocyst(e)ine

Individuals were allocated into an 'at risk' group if values were ≥8.5 $\mu mol \cdot L^{-1}$ or the 95^{th} percentile according to Osganian *et al.* (1999).

3.8 PILOT STUDY (Appendix 7)

A group of twenty, Caucasian, year nine schoolchildren (age 13.0 ± 0.8 years) were randomly chosen to participate in the pilot study. All subjects attended the same Welsh medium, Local Education Authority maintained, comprehensive school. The residential area from which the schoolchildren were drawn was neither 'prosperous nor economically disadvantaged' (Estyn, 2000). Nine percent of the total intake of pupils were registered for free school meals. The pupils were of mixed academic ability.

The methodologies have been described previously. Reliability tests were performed on obesity measurements, blood pressure readings and the 20 metre shuttle test. The effectiveness of the food diary, and physical activity questionnaire and seven-day recall was also assessed.

To determine the reliability of data collection, the pilot group underwent assessment protocols twice (test-retest). The children were not informed that they were to be re-tested. In addition, the majority of the physical and physiological tests undertaken in the present investigation are included in the Eurofit test battery (Council of Europe, 1982; Klissouras and Tokmakidis, 1982), hence their protocols had been previously scrutinized for validity, reliability and objectivity.

To ensure consistency and accuracy, the author administered all test protocols. Tests were conducted on two separate occasions, seven days apart, at the same time of day and following the same sequential order.

Methods of establishing the reliability of experimental protocols include Pearson's correlation coefficient, intra-class coefficients, paired *t*-tests and repeated measures ANOVA. Bland and Altman (1986) argue that many of these traditional methods are measures of relationship as opposed to agreement. Recently, the 'Limits of Agreement' (LoA) method refined by Bland and Altman (1986), has been proposed as being a more appropriate method of assessing reliability. In the present investigation, a Bland-Altman 95% LoA plot was formed for each variable using a computerised statistical package (Minitab Inc., 1998). This procedure involved plotting the individual subject differences for the test and the retest on the Y-axis against the respective individual means for the test and retest on the X-axis (Bland and Altman, 1995). The direction and magnitude of the scatter around the zero line gave an estimation of systematic bias and random error, respectively. Plotting the absolute differences against the individual means and calculating the correlation coefficient confirmed homoscedasticity. Providing no relationship was identified ($r < 0.1$), the data were considered homoscedastic and the 95% LoA procedure was resumed. All variables measured in the pilot study were

confirmed as homoscedastic ($r < 0.1$). The Anderson-Darling normality test confirmed whether or not the differences between the test and retest for all variables were normally distributed. The P value for all measured variables was greater than 0.05 ($P > 0.05$), hence it was concluded that the exact boundaries for 95% LoA should lie between $\pm$ $1.96 \times s_{\Delta}$, where $\pm$ $1.96 \times s_{\Delta}$ is an index of random variation. A paired t test on the mean differences ($\bar{x}_{\Delta}$) was calculated to assess systematic bias.

The effectiveness of the proposed food diary and physical activity seven-day recall and questionnaire was also assessed. Following the pilot study, no amendments were considered necessary to the food diary; one question was reworded in the physical activity questionnaire.

To avoid inter-tester discrepancies, it was decided that the author would conduct all aspects of physical, physiological and lifestyle data collection.

CHAPTER 4

4.0 RESULTS

From November 2001 to March 2002, 100 boys and 108 girls, aged 12.0 ± 0.8 years were assessed for coronary heart disease (CHD) risk factors. The descriptive statistics for all physical, physiological, lifestyle and haematological variables are summarised in Tables 4.1 through 4.3. Absenteeism, mostly due to an influenza virus that struck both schools, along with schoolchildren refusing blood tests, accounted for the differences in sample size across variables. Sixty six percent of schoolchildren reported no vigorous activity over the previous seven days (Table 4.4). Girls' physical activity patterns caused greatest concern, since 83% in school 1, and 74.1% in school 2, failed to participate in any vigorous activity in the last week.

Coronary heart disease risk factors selected for this investigation included physical, physiological, lifestyle and haematological variables. Familial risk was also included as a risk factor. Socio-economic status (SES) was identified, but was not included as a risk factor during calculations. School 1 was designated as high socio-economic status (SES), whilst school 2 represented low SES. For all CHD risk factors measured in this study, standards were obtained from the most comprehensive data available for the age group investigated. Table 4.5 describes the population considered to be 'at risk' of future CHD according to the assigned criteria. When the findings from both schools were combined, 11.9% and 6.3% of schoolchildren exceeded the assigned criterion threshold for systolic blood pressure (SBP) and diastolic blood pressure (DBP), respectively. Poor fitness levels, as measured by the 20 MST, were reported in 29.5% of schoolchildren. For body mass index (BMI), 22.2% of boys and 12% of girls exceeded the elected criterion threshold. Fifty percent of girls, and 23.7% of boys,

recorded high waist to hip ratios (WHR). For summation of four skinfold thicknesses, 32.3% of the young population were considered to have excessive subcutaneous adiposity levels. The dietary habits of the majority of pupils were poor. Over 93% of the schoolchildren consumed diets that contained more than 10% saturated fat, with similar values being reported across the sexes and SES. For lipids and lipoproteins, the most worrying statistics were identified for total cholesterol (TC), the ratio of TC to high density lipoprotein cholesterol, and lipoprotein(a) (Lp(a)). In school 1, 9.1% of pupils exceeded the cut off point for TC, while 28.3% of pupils in school 2 recorded similar elevated levels. For TC: HDL-C, 18.8% of young people recorded values ≥ 4.0. High values of Lp(a) were identified in 26.8% of individuals. For new CHD risk factors, Fg concentration was high ($\geq$ 394 $mg \cdot dL^{-1}$) in many schoolchildren. In school 1, 60.8% of pupils exceeded the elected cut off point, while a slightly lower 46% recorded similar levels in school 2. Using the Hcy cut- off point of > 8.5 $\mu mol \cdot L^{-1}$, 21.4% of pupils in school 1, and 20.3% of pupils in school 2 exceeded this threshold.

Table 4.6 presents the frequency of single and multiple CHD risk factors in adolescents. Only two pupils were exempt from any CHD risk factor. More than one risk factor was exhibited in 83.9% of pupils in school 1, and 89.5% of schoolchildren in school 2. Three or more CHD risk factors were exhibited in 66.6% of schoolchildren. A disconcerting eight CHD risk factors were reported in three pupils, whilst an alarming nine risk factors were identified in two individuals.

In Tables 4.7 through 4.9, parametric and non-parametric tests indicate significant differences ($P \leq 0.05$) according to socio-economic status (schools) and sex. For SES (ie according to school), statistically significant differences ($P \leq 0.05$) were detected in SBP (boys only), TC (boys only), LDL-C (boys only), Fg (boys only), and folate (boys

and girls). Significant differences ($P \leq 0.05$) were also identified according to sex, for SBP, DBP, aerobic fitness, WHR, $\sum$ SKF, daily calorie intake, TC, LDL-C, Fg and folate concentration. The results from Bonferroni adjusted post hoc tests are presented in Tables 4.10 through 4.13.

A useful adjunct to the P value in estimating the practical significance of a test result is the effect size. Both the effect size and statistical power of the tests are presented in Tables 4.14 through 4.16.

A number of statistically significant relationships ($P \leq 0.05$) were identified in a correlation coefficient matrix (Appendix 8).

The results of the principal components factor analysis (PCA) are presented in Tables 4.17 - 4.20. As expected, the findings indicated significant clustering of lipids and lipoproteins; homocyst(e)ine, folate and vitamin B_{12}; and, total and saturated fat.

Multiple regression analysis (MRA) was conducted to evaluate the relationship between one or more independent variables (IV) with a dependent variable (DV). The CHD risk factors selected as DVs were, blood pressure, aerobic fitness, body mass index, summation of four skinfold sites, total cholesterol, high density lipoprotein cholesterol, low density lipoprotein cholesterol, triglyceride, lipoprotein(a), fibrinogen, and homocyst(e)ine. By examining the direction and magnitude of the β weights assigned to each IV in the standardised regression equations, the author was able to determine which CHD risk factor was the strongest predictor. Final non-standardised and standardised regression equations, including only those variables that made a significant contribution to explained variance in the dependent variable, are presented in Tables

4.21 through 4.48. Multiple regression analysis revealed that BMI was a strong predictor of SBP and DBP. When central and peripheral body fat were treated independently, the primary mediator of SBP was central fat, whilst peripheral fat was the strongest predictor of DBP. The ratio of TC to HDL-C, had the greatest effect on all blood lipids and lipoproteins. A surprising feature was the importance of vitamin B_{12} to Lp(a). Body mass index explained the largest proportion of variation in Fg; and as expected, the main predictors of homocyst(e)ine were vitamin B_{12} and folate. The primary mediator of performance in the 20 MST was the skinfold thickness at four sites. Much of the evidence gathered in the multiple regression analysis supported the findings of the principal components factor analysis.

Table 4.1 Means and standard deviations ($\bar{x} \pm s$) of physical and physiological variables

		School 1		School 2	
Variable	**Units**	**Male (n=61)**	**Female (n =69)**	**Male (n =90)**	**Female (n =91)**
Age	yrs	12.0±0.7	12.0±0.8	12.0±0.9	12.0±0.8
Stature	m	1.54±0.08	1.55±0.07	1.54±0.09	1.54±0.06
Body mass	kg	51.1±12.9	51.9±10.6	50.4±15.3	51.6±11.6
Σ SKF	mm	43.7±22.5(56)*	49.5±19.6(61)*	44.9±29.1(77)*	56.9±23.7(84)*
Waist	cm	70.30±10.97	65.72±8.92	71.26±11.6	65.96±9.26
Hips	cm	86.04±9.23	87.57±8.5	86.0±10.61	86.18±10.03
WHR		0.8±0.1	0.8±0.1	0.8±0.1	0.8±0.1
BMI	kg m^{-2}	21.2±4.4	21.6±3.6	21.2±5.0	21.6±4.2
SBP	mm Hg	111 ±13 (56)*	113±12(65)*	117±12 (83)*	116±11 (84)*
DBP	mm Hg	63±9 (56)*	70 ±9 (65)*	64±10 (83)*	70±12(84)*
Aerobic fitness	shuttles	54±21 (60)*	39 ±14 (67)*	59±20(87)*	41±14

Where $n \neq$ denoted number, actual sample number is presented in brackets.

Table 4.2 Means and standard deviations ($\bar{x} \pm s$) of haematological variables

		School 1		School 2	
Variable	**Units**	**Male (n =36)**	**Female (n =41)**	**Male (n =64)**	**Female (n =67)**
Glu	$mmol \cdot L^{-1}$	5.04±0.34	5.08±0.33	4.92±0.41	4.90±0.46
TC	$mmol \cdot L^{-1}$	4.19±0.71	4.37±0.59	4.73±0.81	4.68±0.71
HDL-C	$mmol \cdot L^{-1}$	1.36±0.34	1.44±0.32	1.43±0.28	1.52±0.36
LDL-C	$mmol \cdot L^{-1}$	2.39±0.62	2.49±0.48	2.83±0.70	2.68±0.59
TC: HDL C		3.26±0.93	3.15±0.70	3.43±0.90	3.21±0.79
TG	$mmol \cdot L^{-1}$	0.98±0.45	0.98±0.46	1.04±0.52	1.05±0.37
Lp (a)	$mg \cdot dL^{-1}$	24.9±27.7(35)*	34.5±45.8	27.9±34.3(63)*	24.7±30.3(66)*
Lp (a)	$mg \cdot dL^{-1}$	Median = <8	Median = <8	Median = <8	Median = <8
Fg	$mg \cdot dL^{-1}$	437.7±96.9(33)*	393.3±81.4	381.5±57.3(55)*	386.5±74.3(58)*
Hcy	$\mu mol \cdot L^{-1}$	7.06±2.47(35)*	6.5±1.64(38)*	6.8±1.94(61)*	7.92±4.13(64)*
Folate	$ng\ mL^{-1}$	9.58±4.05	11.1±4.14	13.16±5.77(60)*	8.38±2.90(66)*
B_{12}	$pmol \cdot L^{-1}$	424.7±104.7(35)*	502.5±220.2	435.4±142.0(60)*	428.7±157.7(64)*

* Where $n \neq$ denoted number, actual sample number is presented in brackets.
† Because Lp(a) values are non-continuous,over a broad range, and highly skewed towards lower end, results are also presented as medians.
‡ The detection limit for Lp(a) was 8 $mg \cdot dL^{-1}$. Values < 8 $mg \cdot dL^{-1}$were coded as 7.9 $mg \cdot dL^{-1}$ for statistical analysis.

Table 4.3 Means and standard deviations ($\bar{x} \pm s$) of dietary variables

		School 1		School 2	
Variable	**Units**	**Male (n =32)**	**Female (n =36)**	**Male (n =52)**	**Female (n =45)**
Total fat	%	33.4±3.3	34.4±3.1	35.5±4.6	34.8±4.1
Saturated fat	%	13.0±2.0	13.9±2.1	14±2.5	14.1±2.6
CHO	%	51.2±4.4	51.0±4.3	49.5±4.7	50.4±4.8
Protein	%	15.4±2.9	14.6±2.7	14.8±3.03	14.9±3.5
Fibre	grams per day	13.9±3.2	11.9±2.8	11.3±3.3	12.5±3.5
Daily average food intake	kcals	1904±330	1645±246	1823±385	1723±313

Table 4.4: Results from physical activity questionnaire and seven day recall

Experienced no vigorous activity during last 7 days			
	Boys (%)	Girls (%)	Both (%)
School 1	57.5	83.0	71.3
School 2	48.1	74.1	61.8
Both	52.2	78.1	66
Normally active (greater intensity than walking) during break time			
	Boys (%)	Girls (%)	Both (%)
School 1	25.0	0	11.5
School 2	44.2	0	20.9
Both	35.9	0	16.8
Normally active (greater intensity than walking) during lunchtime			
	Boys (%)	Girls (%)	Both (%)
School 1	50	0	23.0
School 2	71.2	12.1	40.0
Both	62.0	6.6	32.5
Number of pupils who enjoy physical activity			
	Boys (%)	Girls (%)	Both (%)
School 1	100	91.5	95.4
School 2	100	93.1	96.4
Both	100	92.4	95.9
Number of pupils whose parents do not participate in regular activity (neither parent)			
			Both (%)
School 1			25.3
School 2			36.4
Both			31.5
Motorised transport to school			
			Both (%)
School 1			97.7
School 2			91.8
Both			94.4

Table 4.5 Percentage of schoolchildren with CHD risk factors

Risk factors	School 1 (%)	School 2 (%)	Both schools (%)
SBP (≥130mm Hg)	10.7	12.8	11.9
DBP (≥ 85 mm Hg)	5.0	7.3	6.3
SBP and DBP (≥130/85 mm Hg)	1.7	3.1	2.5
BMI			
> 24.3 kg m^{-2} (m)	21.3	23.1	22.2
> 25.6 kg m^{-2} (f)	13	11	12
WHR			
≥ 0.9 (m)	19.7	26.4	23.7
≥ 0.8 (f)	47.8	51.7	50
SKF			
≥ 56.9 mm (m)	28.6	23.1	25.4
≥ 57.4 mm (f)	31.2	44.1	38.7
FIT			
≤ 47 shuttles (m)	36.7	35.7	36.1
≤ 28 shuttles (f)	28.4	19.8	23.4
TOT FAT (> 30%)	85.3	89.5	87.7
SAT FAT (> 10%)	91.2	94.7	93.3
TC (≥ 5.2 mmol·L^{-1})	9.1	28.3	18.7
HDL-C (≤ 0.9 mmol·L^{-1})	1.3	0.8	1
LDL-C (≥ 3.8 mmol·L^{-1})	0	6.1	3
TG (≥ 1.7 mmol·L^{-1})	7.8	11.5	10.1
TC: HDL-C (≥ 4.0)	19.5	18.3	18.8
Lp (a) (> 30mg·dL^{-1})	31.6	24	26.8
Hcy (> 8.5 µmol·L^{-1})	21.4	20.3	21
Fg (≥ 394 mg·dL^{-1})	60.8	46	51.9

Table 4.6 Percentage of schoolchildren in the different risk factor clusters

No. risk factors	School 1 (%) (High SES)	School 2 (%) (Low SES)
0	0	2.4
1	16.1	8.1
2	18.4	21.8
3	19.5	19.4
4	21.8	17.7
5	13.8	12.1
6	5.7	10.5
7	3.5	4.8
8	1.2	1.6
9	0	1.6
10	0	0
11	0	0

CHD risk factors included in this table are obesity measures; PA/FIT; hypertension; elevated levels of TC, LDL-C, TC: HDL-C, TG, Lp(a), Fg, Hcy, saturated fat; low levels of HDL-C and familial risk of CHD.

Table 4.7 Differences in physical and physiological variables according to sex and socio-economic status.

Variable	Units	*F*-ratio	*H*-value	*ES*	*Power*	*P*
Σ SKF†	mm		24.33	0.30	>99.5%	0.000†
WHR†			60.86	0.44	>99.5%	0.000†
BMI†	kg m^{-2}		3.91			0.271
SBP†	mm Hg		11.97	0.20	82%	0.008†
DBP†	mm Hg		24.88	0.30	>99.5%	0.000†
Aerobic Fitness	shuttles	24.95		0.190	84%	0.000*

*$F_{3,302}(0.05) = 2.60$

†$H_{3,} = 7.81$

Note where differences were identified as being statistically significant, the estimated effect size and statistical power are presented.

Table 4.8 Differences in dietary variables according to sex and socio-economic status.

Variable	Units	*F*-ratio	*H*-value	*ES*	*Power*	*P*
Total fat†	%		4.50			0.212
Sat fat†	%		4.50			0.214
Daily average intake*	kcals	4.29		0.056	7%	0.006*

*$F_{3,161)}(0.05) = 2.60$

†$H_3(0.05) = 7.81$

Note where differences were identified as being statistically significant, the estimated effect size and statistical power are presented.

Table 4.9 Differences in haematological variables according to sex and socio-economic status.

Variable	Units	*F*-ratio	*H*-value	*ES*	*Power*	*P*
TC*	$mmol \cdot L^{-1}$	5.81		0.065	11.6%	0.001*
HDL-C (√)*	$mmol \cdot L^{-1}$	2.16				0.094
LDL-C*	$mmol \cdot L^{-1}$	4.90		0.054	8%	0.003*
TC: HDL-C			3.54			0.315
TG	$mmol \cdot L^{-1}$		2.69			0.443
Glu	$mmol \cdot L^{-1}$	2.23				0.086
Lp (a)	$mg \cdot dL^{-1}$		0.74			0.863
Fg (log_e)†	$mg \cdot dL^{-1}$	3.70		0.155	37%	0.013†
Hcy	$\mu mol \cdot L^{-1}$		3.52			0.319
Folate (log_e)‡	$ng \cdot mL^{-1}$	13.44		0.042	40%	0.000‡
B_{12}	$pmol \cdot L^{-1}$		3.34			0.343

*$F_{3,204}$ (0.05) = 2.60; †$F_{3,183}$ (0.05) = 2.60; ‡$F_{3,199}$ (0.05) = 2.60

H_3 (0.05) = 7.81

Note where differences were identified as being statistically significant, the estimated effect size and statistical power are presented.

Table 4.10 Results of post hoc test (*t*-test for two independent means) to determine specific mean differences in physical and physiological variables

Variable	Units	Group	*t*-ratio	*df*	*P*
Aerobic fitness	shuttles	School 1(m) School 2(m)	-1.33	145	0.19
		School 1(f) School 2(f)	-0.98	156	0.33
		School 1(m) School 1(f)	4.84	125	0.0000*
		School 2(m) School 2(f)	6.90	176	0.0000*
		School 1(m) School 2(f)	4.54	149	0.0000*
		School 2(m) School 1(f)	7.03	152	0.0000*

* Significant using Bonferroni's adjustment $P \leq 0.0083$

Table 4.11 Results of post hoc test (*t*-test for two independent means) to determine specific mean differences in dietary variables.

Variable	Units	Group	*t*-ratio	*df*	*P*
Average daily food intake	kcals	School 1(m) School 2(m)	0.99	82	0.33
		School 1(f) School 2(f)	-1.23	79	0.22
		School 1(m) School 1(f)	3.70	66	0.0004*
		School 2(m) School 2(f)	1.39	95	0.17
		School 1(m) School 2(f)	2.45	75	0.017
		School 2(m) School 1(f)	2.45	86	0.016

* Significant using Bonferroni's adjustment significant at $P \leq 0.0083$

Table 4.12 Results of post hoc test (*t*-test for two independent means) to determine specific mean differences in haematological variables

Variable	Units	Group	*t*-ratio	*df*	*P*
TC	$mmol \cdot L^{-1}$	School 1(m)	-3.32	98	0.0013*
		School 2(m)			
		School 1(f)	-2.31	106	0.023
		School 2(f)			
		School 1(m)	-1.23	75	0.22
		School 1(f)			
		School 2(m)	0.40	129	0.69
		School 2(f)			
		School 1(m)	-3.31	101	0.0013*
		School 2(f)			
		School 2(m)	2.44	103	0.016
		School 1(f)			
LDL-C	$mmol \cdot L^{-1}$	School 1(m)	-3.09	98	0.0026*
		School 2(m)			
		School 1(f)	-1.77	106	0.079
		School 2(f)			
		School 1(m)	-0.73	75	0.47
		School 1(f)			
		School 2(m)	1.32	129	0.19
		School 2(f)			
		School 1(m)	-2.29	101	0.024
		School 2(f)			
		School 2(m)	2.75	103	0.0069*
		School 1(f)			
Fibrinogen	$mg \cdot dL^{-1}$	School 1(m)	3.42	86	0.0009*
		School 2(m)			
		School 1(f)	0.43	97	0.67
		School 2(f)			
		School 1(m)	2.14	72	0.036
		School 1(f)			
		School 2(m)	-0.40	111	0.69
		School 2(f)			
		School 1(m)	2.82	89	0.0059*
		School 2(f)			
		School 2(m)	-0.84	94	0.41
		School 1(f)			
Folate	$ng \cdot mL^{-1}$	School 1(m)	-3.26	94	0.0015*
		School 2(m)			
		School 1(f)	4.00	105	0.0001*
		School 2(f)			
		School 1(m)	-1.62	75	0.11
		School 1(f)			
		School 2(m)	5.95	124	0.0000*
		School 2(f)			
		School 1(m)	1.73	100	0.086
		School 2(f)			
		School 2(m)	1.96	99	0.53
		School 1(f)			

* Significant using Bonferroni's adjustment $P \leq 0.0083$

Table 4.13 Results of post hoc test (Mann-Whitney (*U*)) to determine specific mean differences in physical and physiological variables.

Variable	Units	Group	Point estimate (median)	95% *CI* (median)	*U*	*P*
Σ SKF	mm	School 1(m) School 2(m)	1.20	-3.80, 6.80	3854.0	0.6437
		School 1(f) School 2(f)	-6.20	-13.00, 0.80	4017.0	0.0811
		School 1(m) School 1(f)	6.80	0.20, 14.20	3969.0	0.0438
		School 2(m) School 2(f)	15.00	8.80, 20.80	8049.0	0.0000*
		School 1(m) School 2(f)	13.20	6.40, 20.20	6762.0	0.0004*
		School 2(m) School 1(f)	8.40	2.41, 15.39	4868.5	0.0070*
WHR		School 1(m) School 2(m)	-0.00	-0.315, 0.0100	4351.0	0. 31
		School 1(f) School 2(f)	0.00	0.00001, 0.00001	5355.0	0.4450
		School 1(m) School 1(f)	-0.10	-0.09999, -0.00001	3574.5	0.0000*
		School 2(m) School 2(f)	-0.10	-0.10000, -0.00001	6443.0	0.0000*
		School 1(m) School 2(f)	-0.10	-0.10000, -0.00002	5962.0	0.0002*
		School 2(m) School 1(f)	-0.10	-0.10000, -0.10000	3834.0	0.0000*
SBP	mm Hg	School 1(m) School 2(m)	-6.00	-10.00, -2.00	3236.0	0.0033*
		School 1(f) School 2(f)	-3.00	-6.00, 1.00	4472.0	0.1231
		School 1(m) School 1(f)	3.00	-1.00, 7.00	4242.5	0.1496
		School 2(m) School 2(f)	-1.00	-4.00, 3.00	6922.0	0.6689
		School 1(m) School 2(f)	-6.00	-9.00, -2.00	3281.5	0.0046*
		School 2(m) School 1(f)	-3.00	-7.00, 0.00	4377.5	0.0727
DBP	mm Hg	School 1(m) School 2(m)	-1.00	-4.001, 1.998	3727.5	0.4092
		School 1(f) School 2(f)	0.00	-2.998, 3.990	4917.5	0.8722
		School 1(m) School 1(f)	7.00	3.000, 10.001	4678.0	0.0002*
		School 2(m) School 2(f)	5.00	2.000, 7.999	8070.5	0.0012*
		School 1(m) School 2(f)	6.00	2.999, 10.001	6755.0	0.0004*
		School 2(m) School 1(f)	5.00	2.000, 9.001	5761.0	0.0004*

* Significant using Bonferroni's adjustment significant at $P \leq 0.0083$

Table 4.14 Effect size and statistical power of post hoc tests (*t*-test for two independent means) for physical and physiological variables

Variable	Units	Group	95% *CI*	*ES*	*Power*
Aerobic fitness	shuttles	School 1(m) School 1(f)	9.1, 21.7	0.860	>99.5%
		School 2(m) School 2(f)	12.7, 22.8	1.035	>99.5%
		School 1(m) School 2(f)	7.5,18.9	0.75	>99.5%
		School 2(m) School 1(f)	14.4, 25.6	1.143	>99.5%

* Significant using Bonferroni's adjustment $P \leq 0.0083$

Table 4.15 Effect size and statistical power of post hoc tests (*t*-test for two independent means) for dietary variables

Variable	**Units**	**Group**	**95% *CI***	***ES***	***Power***
Average daily food intake	kcals	School 1(m) School 1(f)	119, 400	0.896	90%

* Significant using Bonferroni's adjustment $P \leq 0.0083$

Table 4.16 Effect size and statistical power of post hoc tests (*t*-test for two independent means) for haematological variables

Variable	Units	Group	95% *CI*	*ES*	*Power*
TC	$mmol \cdot L^{-1}$	School 1(m) School 2(m)	-0.86, 0.22	0.692	90%
		School 1(m) School 2(f)	-0.78, -0.194	0.683	89%
LDL-C	$mmol \cdot L^{-1}$	School 1(m) School 2(m)	-0.71, -0.155	0.645	86%
		School 2(m) School 1(f)	0.096, 0.587	0.552	74%
Fibrinogen	$mg \cdot dL^{-1}$	School 1(m) School 2(m)	24, 88.8	0.754	93%
		School 1(m) School 2(f)	15, 87.2	0.62	81%
Folate	$ng \cdot mL^{-1}$	School 1(m) School 2(m)	-5.75, -1.40	0.689	89%
		School 1(f) School 2(f)	1.37, 4.07	0.793	98%
		School 2(m) School 2(f)	3.19, 6.37	1.06	>99.5%

* Significant using Bonferroni's adjustment $P \leq 0.0083$

Table 4.17 Principal component factor analysis of physical and physiological CHD risk factors

Variable	Units	1	2	Communality
Σ SKF	mm	0.59	0.66	0.79
WHR			0.80	0.71
BMI	$kg\ m^{-2}$	0.63	0.64	0.80
SBP	mm Hg	0.71		0.51
DBP	mm Hg	0.83		0.70
Aerobic fitness	shuttles	-0.49	-0.54	0.54
Variance		2.252	1.792	4.043
% Variance		37.5	29.9	67.4

- Criterion used for deciding which components to exclude = Kaiser
- Only loadings of 0.30 or greater are included

Table 4.18 Principal component factor analysis of dietary CHD risk factors

		Factor		
Variable	**Units**	**1**	**2**	**Communality**
Total fat	%	0.96		0.92
Sat fat	%	0.96		0.92
Daily average food intake	kcals		1.00	1.00
Variance		1.831	1.003	2.834
% Variance		61%	33.4	94.5

- Criterion used for deciding which components to exclude = Kaiser
- Only loadings of 0.30 or greater are included

Table 4.19 Principal component factor analysis of haematological CHD risk factors

		Factor				
Variable	**Units**	**1**	**2**	**3**	**4**	**Communality**
Glu	$mmol \cdot L^{-1}$				0.64	0.44
TC	$mmol \cdot L$		0.96			0.95
HDL-C	$mmol \cdot L^{-1}$	0.85	0.33			0.88
LDL-C	$mmol \cdot L^{-1}$	-0.31	0.90			0.91
TC: HDL- C		-0.90	0.35			0.94
TG	$mmol \cdot L^{-1}$	-0.79				0.69
Lp(a)	$mg \cdot dL^{-1}$		0.50	0.32	-0.30	0.50
Fg	$mg \cdot dL^{-1}$				-0.73	0.55
Hcy	$\mu mol \cdot L^{-1}$			-0.81		0.67
Folate	$ng \cdot mL^{-1}$			0.73		0.54
B_{12}	$pmol \cdot L^{-1}$			0.50	-0.30	0.37
Variance		2.344	2.321	1.548	1.224	7.44
% Variance		21.3	21.1	14.1	11.1	67.6

- Criterion used for deciding which components to exclude = Kaiser
- Only loadings of 0.30 or greater are included

Table 4.20 Principal component factor analysis of all CHD risk factors

	Factor								
Variable	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**Communality**
Glu							0.73		0.58
TC		0.94							0.94
HDL-C			0.80						0.84
LDL-C		0.91							0.91
TC:HDL		0.46	-0.83						0.94
TG			-0.74						0.72
Lp(a)		0.51	0.40						0.61
Fg							-0.62		0.43
Hcy					0.83				0.72
Folate	-0.48				-0.53			-0.32	0.69
B_{12}					-0.62				0.53
Σ SKF	0.79								0.77
WHR							-0.37	-0.68	0.70
BMI	0.74								0.78
SBP						0.89			0.81
DBP	0.36					0.66		0.35	0.73
Aerobic Fitness	-0.85								0.76
Tot fat				0.93					0.89
Sat fat				0.94					0.90
Average calorie daily intake								-0.72	0.64
Variance	2.517	2.414	2.290	1.971	1.513	1.494	1.398	1.308	14.904
% Variance	12.6	12.1	11.5	9.9	7.6	7.5	7.0	6.5	74.5

- Criterion used for deciding which components to exclude = Kaiser
- Only loadings of 0.30 or greater are included.

Table 4.21 Final unstandardised predictive equation and data summary for the prediction of systolic blood pressure (SBP_p(mm Hg)) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in SBP.

$$SBP_p \text{ (mm Hg)} = 74.7 + (0.378 \times \text{DBP (mm Hg)}) + 0.723 \times \text{BMI (kg m}^{-2}))$$

Predictor	Units	Unstandardised Coefficient	*t* ratio	*P*
Constant		74.65	14.86	0.000
DBP	mm Hg	0.37779	4.51	0.000
BMI	kgm^{-2}	0.7229	3.42	0.001
$s_{YX} = \pm$ 10. 2 mm Hg			R^2_{adj} = 26.3%	$F_{2,182}$= 33.86

Table 4.22: Final standardised predictive equation and data summary for the prediction of systolic blood pressure (SBP_p(mm Hg)) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in SBP.

$$SBP_p = -0.0007 + (0.337 \times \text{DBP}) + 0.255 \times \text{BMI}))$$

Predictor	Standardised Coefficient (β)	*t* ratio	*P*
Constant	-0.00068	-0.01	0.991
DBP	0.33712	4.51	0.000
BMI	0.25468	3.42	0.001
$s_{YX} = \pm$ 0.858		R^2_{adj} = 26.3%	$F_{2,182}$= 33.86

Table 4.23: Final unstandardised predictive equation and data summary for the prediction of diastolic blood pressure (DBP_p(mm Hg)) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in DBP.

$$DBP_p \text{ (mm Hg)} = 38.9 + 1.34 \times \text{BMI (kg m}^{-2}\text{)}$$

Predictor	Units	Unstandardised Coefficient	*t* ratio	*P*
Constant		38.926	11.56	0.000
BMI	kg m^{-2}	1.3432	8.52	0.000
$s_{YX} = \pm$ 9.0 mm Hg			R^2_{adj} =26.3%	$F_{1,183}$= 72.64

Table 4.24: Final standardised predictive equation and data summary for the prediction of diastolic blood pressure (DBP_p(mm Hg)) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in DBP.

$$DBP_p = -0.0014 + (0.530 \times \text{BMI})$$

Predictor	Standardised Coefficient (β)	*t* ratio	*P*
Constant	-0.00142	-0.02	0.982
BMI	0.53035	8.52	0.000
$s_{YX} = \pm$ 0.848		R^2_{adj}=28.0%	$F_{1,183}$= 72.64

Table 4.25: Final unstandardised predictive equation and data summary for the prediction of systolic blood pressure (SBP_p(mm Hg)) from skinfold measurements in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in SBP.

$$SBP_p \text{ (mm Hg)} = 109 + (0.276 \times \text{SUPRA/SUB (mm)})$$

Predictor	Units	Unstandardised Coefficient	*t* ratio	*P*
Constant		108.568	68.89	0.000
SUPRA/SUB	mm	0.27636	4.89	0.000
$s_{YX} = \pm 11.2$mm Hg			$R^2_{adj} = 11.1\%$	$F_{1,183} = 23.88$

Table 4.26: Final standardised predictive equation and data summary for the prediction of systolic blood pressure (SBP_p(mm Hg)) from skinfold measurements in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in SBP.

$$SBP_p = -0.0013 + (0.338 \times \text{SUPRA/SUB})$$

Predictor	Standardised Coefficient (β)	*t* ratio	*P*
Constant	-0.00125	-0.02	0.986
SUPRA/SUB	0.33816	4.89	0.000
$s_{YX} = \pm 0.943$		$R^2_{adj} = 11.1\%$	$F_{1,183} = 23.88$

Table 4.27 : Final unstandardised predictive equation and data summary for the prediction of diastolic blood pressure (DBP_p(mm Hg)) from skinfold measurements in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in DBP.

$$DBP_p \text{ (mm Hg)} = 55.8 + (0.448 \times \text{TRI/BIC (mm)})$$

Predictor	Units	Unstandardised Coefficient	*t* ratio	*P*
Constant		55.760	32.45	0.000
TRI/BIC	mm	0.44759	7.15	0.000
$s_{YX} = \pm 9.4$mm Hg			$R^2_{adj} = 21.5\%$	$F_{1,182} = 51.10$

Table 4.28: Final standardised predictive equation and data summary for the prediction of diastolic blood pressure (DBP_p(mm Hg)) from skinfold measurements in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in DBP.

$$DBP_p = -0.0070 + (0.466 \times \text{TRI/BIC})$$

Predictor	Standardised Coefficient (β)	*t* ratio	*P*
Constant	-0.00698	-0.11	0.915
TRI/BIC	0.46550	7.15	0.000
$s_{YX} = \pm 0.885$		$R^2_{adj} = 21.5\%$	$F_{1,182} = 51.10$

Table 4.29: Final unstandardised predictive equation and data summary for the prediction of 20 metre shuttle test ($20MST_p$(shuttles)) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in 20MST.

$$20MST_p \text{ (shuttles)} = 75.3 - (0.505 \times \Sigma \text{ SKF(mm)})$$

Predictor	Units	Unstandardised Coefficient	*t* ratio	*P*
Constant		75.308	29.51	0.000
Σ SKF	mm	-0.50508	-10.69	0.000
s_{YX}= ± 15.5 (16) shuttles		R^2_{adj}= 38%	$F_{1,184}$= 114.36	

Table 4.30: Final standardised predictive equation and data summary for the prediction of 20 metre shuttle test ($20MST_p$(shuttles)) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in 20MST.

$$20MST_p = -0.0128 - (0.642 \times \Sigma \text{ SKF})$$

Predictor	Standardised Coefficient (β)	*t* ratio	*P*
Constant	-0.01276	-0.22	0.825
Σ SKF	-0.64163	-10.69	0.000
s_{YX}= ±0.787	R^2_{adj}= 38%	$F_{1,184}$= 114.36	

Table 4.31: Final unstandardised predictive equation and data summary for the prediction of body mass density (BMI_p(kg m^{-2})) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution (P<0.05) to explained variance in BMI.

BMI_p (kg m^{-2}) = 12.2 + (0.107 × Σ SKF (mm)) + (6.24 × WHR (ratio)) - (0.0282 ×20MST (shuttles))

Predictor	Units	Unstandardised Coefficient	*t* ratio	*P*
Constant		12.208	5.59	0.000
Σ SKF	mm	0.106817	10.72	0.000
WHR		6.241	2.31	0.022
20MST	Shuttles	-0.02816	-2.37	0.019
s_{YX}= ±2.48kg m^{-2}		R^2_{adj}= 60.3%	$F_{3,182}$= 94.73	

Table 4.32: Final standardised predictive equation and data summary for the prediction of body mass density (BMI_p(kg m^{-2})) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution (P<0.05) to explained variance in BMI.

BMI_p = 0.0126 + (0.636 × Σ SKF) + (0.104 x WHR) - (0.132 × 20MST)

Predictor	Standardised Coefficient (β)	*t* ratio	*P*
Constant	-0.01256	-0.29	0.773
Σ SKF	0.63578	10.72	0.000
WHR	0.10386	2.31	0.022
20MST	-0.13195	-2.37	0.019
s_{YX}= ±0.593	R^2_{adj}= 60.3%	$F_{3,182}$= 94.73	

Table 4.33: Final unstandardised predictive equation and data summary for the prediction of summation of 4 skinfold sites (Σ SKF_p (mm)) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in Σ SKF.

Σ SKF_p (mm) = -15.8 + (8.92 × TG mmol·L^{-1})+ (3.45 × BMI (kg m^{-2})) - (0.330 × 20MST (shuttles))

Predictor	Units	Unstandardised Coefficient	*t* ratio	*P*
Constant		-15.793	-1.78	0.076
TG	mmol·L^{-1}	8.920	3.71	0.000
BMI	kg m^{-2}	3.4539	10.71	0.000
20MST	shuttles	-0.32977	-5.23	0.000
s_{YX}= ±14.0mm		R^2_{adj}= 66.2%	$F_{3,182}$= 121.74	

Table 4.34: Final standardised predictive equation and data summary for the prediction of summation of 4 skinfold sites (Σ SKF_p (mm)) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in Σ SKF.

Σ SKF_p = -0.0043 + (0.161 × TG + (0.58 × BMI) - (0.260 × 20MST)

Predictor	Standardised Coefficient (β)	*t* ratio	*P*
Constant	-0.00430	-0.10	0.917
TG	0.16143	3.71	0.000
BMI	0.58029	10.71	0.000
20MST	-0.25959	-5.23	0.000
s_{YX}= ±0.561	R^2_{adj}= 66.2%	$F_{3,182}$= 121.74	

Table 4.35: Final unstandardised predictive equation and data summary for the prediction of total cholesterol (TC_p(mmol·L^{-1})) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution (P<0.05) to explained variance in TC.

TC_p (mmol·L^{-1}) = -2.87 + (1.09 × TC:HDL-C (ratio)) + (2.65 × HDL-C (mmol·L^{-1}))

Predictor	Units	Unstandardised Coefficient	*t* ratio	*P*
Constant		-2.8729	-11.77	0.000
HDL-C	mmol·L^{-1}	2.65459	27.43	0.000
TC:HDL-C	ratio	1.09247	29.03	0.000
s_{YX}= ±0.31mmol·L^{-1}		R^2_{adj}= 83.5%	$F_{2,184}$= 470.08	

Table 4.36: Final standardised predictive equation and data summary for the prediction of total cholesterol (TC_p(mmol·L^{-1})) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution (P<0.05) to explained variance in TC.

TC_p = -0.0000 + (1.22 × TC:HDL-C) + (1.15 × HDL-C)

Predictor	Standardised Coefficient (β)	*t* ratio	*P*
Constant	-2.8729	-0.00	1.000
HDL-C	1.15046	27.43	0.000
TC:HDL-C	1.21727	29.03	0.000
s_{YX}= ±0.407	R^2_{adj}= 83.5%	$F_{2,184}$= 470.08	

Table 4.37: Final unstandardised predictive equation and data summary for the prediction of high density lipoprotein-cholesterol (HDL-C_p($mmol \cdot L^{-1}$)) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in HDL-C.

$$HDL\text{-}C_p\ (mmol \cdot L^{-1}) = 2.75 - (0.266 \times TC{:}HDL\text{-}C(ratio)) - (0.553 \times WHR\ (ratio))$$

Predictor	Units	Unstandardised Coefficient	*t* ratio	*P*
Constant		2.7517	14.05	0.000
TC:HDL-C	ratio	-0.26624	-13.06	0.000
WHR	ratio	-0.5528	-2.23	0.027
$s_{YX}= \pm 0.23 mmol \cdot L^{-1}$		$R^2_{adj}=50.2\%$	$F_{2,184}= 94.80$	

Table 4.38: Final standardised predictive equation and data summary for the prediction of high density lipoprotein-cholesterol (HDL-C_p ($mmol \cdot L^{-1}$)) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in HDL-C.

$$HDL\text{-}C_p = -0.0000 - (0.685 \times TC{:}HDL\text{-}C) - (0.117 \times WHR)$$

Predictor	Standardised Coefficient (β)	*t* ratio	*P*
Constant	-0.00000	-0.00	0.000
TC:HDL-C	-0.68451	-13.06	0.000
WHR	-0.11675	-2.23	0.027
$s_{YX}= \pm 0.706$	$R^2_{adj}=50.2\%$	$F_{2,184}= 94.80$	

Table 4.39: Final unstandardised predictive equation and data summary for the prediction of low density lipoprotein-cholesterol (LDL-C_p(mmol·L^{-1})) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution (P<0.05) to explained variance in LDL-C.

$$\text{LDL-C}_p\ (\text{mmol}\cdot\text{L}^{-1}) = 1.04 + (0.453 \times \text{TC:HDL-C (ratio)}) + (0.000366 \times \text{Lp(a)}\ (\text{mg}\cdot\text{dL}^{-1}))$$

Predictor	Units	Unstandardised Coefficient	*t* ratio	*P*
Constant		1.04	7.24	0.000
TC:HDL-C	ratio	0.45349	10.74	0.000
Lp(a)	mg·dL^{-1}	0.0003661	3.65	0.000
s_{YX}= ±0.48mmol·L^{-1}		R^2_{adj}= 41.4%	$F_{2,182}$=65.97	

Table 4.40: Final standardised predictive equation and data summary for the prediction of low density lipoprotein-cholesterol (LDL-C_p(mmol·L^{-1})) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution (P<0.05) to explained variance in LDL-C.

$$\text{LDL-C}_p = -0.0091 + (0.602 \times \text{TC:HDL-C}) + (0.204 \times \text{Lp(a)})$$

Predictor	Standardised Coefficient (β)	*t* ratio	*P*
Constant	-0.00912	-0.16	0.870
TC:HDL-C	0.60194	10.74	0.000
Lp(a)	0.20352	3.65	0.000
s_{YX}= ±0.756	R^2_{adj}= 41.4%	$F_{2,182}$=65.97	

Table 4.41: Final unstandardised predictive equation and data summary for the prediction of triglycerides (TG_p(mmol·L^{-1})) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution (P<0.05) to explained variance in TG.

$$TG_p \text{ (mmol·L}^{-1}) = -0.310 + (0.348 \times \text{TC:HDL-C (ratio)}) + (0.00367 \times \Sigma \text{ SKF (mm)})$$

Predictor	Units	Unstandardised Coefficient	*t* ratio	*P*
Constant		-0.30953	-3.41	0.001
TC:HDL-C	ratio	0.34841	12.64	0.000
Σ SKF	mm	0.0036704	3.91	0.000
s_{XY}=0.30mmol·L^{-1}		R^2_{adj}=54.8%	$F_{2,184}$=113.72	

Table 4.42: Final standardised predictive equation and data summary for the prediction of triglycerides (TG_p(mmol·L^{-1})) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution (P<0.05) to explained variance in TG.

$$TG_p = -0.0000 + (0.655 \times \text{TC:HDL-C}) + (0.203 \times \Sigma \text{ SKF})$$

Predictor	Standardised Coefficient (β)	*t* ratio	*P*
Constant	-0.00000	-0.00	1.00
TC:HDL-C	0.65533	12.64	0.000
Σ SKF	0.20283	3.91	0.000
s_{XY}=0.672	R^2_{adj}=54.8%	$F_{2,184}$=113.72	

Table 4.43: Final unstandardised predictive equation and data summary for the prediction of lipoprotein(a) ($Lp(a)_p(mg \cdot dL^{-1})$) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in Lp(a).

$$Lp(a)_p\ (mg \cdot dL^{-1}) = 43.1 + (0.521 \times B_{12}\ (pmol \cdot L^{-1}))$$

Predictor	Units	Unstandardised Coefficient	*t* ratio	*P*
Constant		43.05	0.57	0.566
B_{12}	$pmol \cdot L^{-1}$	0.5206	3.30	0.001
$s_{xy} = \pm 342\ mg \cdot L^{-1}$		$R^2_{adj} = 5.3\%$	$F_{1,175} = 10.90$	

Table 4.44: Final standardised predictive equation and data summary for the prediction of lipoprotein(a) ($Lp(a)_p(mg \cdot dL^{-1})$) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in Lp(a).

$$Lp(a)_p = -0.0170 + (0.239 \times B_{12})$$

Predictor	Standardised Coefficient (β)	*t* ratio	*P*
Constant	-0.01703	-0.24	0.814
B_{12}	0.23886	3.30	0.001
$s_{xy} = \pm 0.962$	$R^2_{adj} = 5.3\%$	$F_{1,175} = 10.90$	

Table 4.45: Final unstandardised predictive equation and data summary for the prediction of fibrinogen ($FG_p(mg \cdot dL^{-1})$) in schoolchildren aged 12-13years. Includes only those terms that make a statistically significant contribution ($P<0.05$) to explained variance in FG.

$$FG_p\ (mg \cdot dL^{-1}) = 263 + (6.34 \times BMI(kg\ m^{-2}))$$

Predictor	Units	Unstandardised Coefficient	*t* ratio	*P*
Constant		263.50	8.22	0.000
BMI	$kg\ m^{-2}$	6.338	4.16	0.000
$s_{YX} = \pm 76.56 mg \cdot dL^{-1}$		$R^2_{adj} = 8.7\%$	$F_{1,170} = 17.27$	

Table 4.46: Final standardised predictive equation and data summary for the prediction of fibrinogen ($FG_p(mg \cdot dL^{-1})$) in schoolchildren aged 12-13years. Includes only those terms that make a statistically significant contribution ($P<0.05$) to explained variance in FG.

$$FG_p = 0.0208 + (0.332 \times BMI)$$

Predictor	Standardised Coefficient (β)	*t* ratio	*P*
Constant	0.0208	0.28	0.776
BMI	0.33161	4.16	0.000
$s_{YX} = \pm 0.956$	$R^2_{adj} = 8.7\%$	$F_{1,170} = 17.27$	

Table 4.47: Final unstandardised predictive equation and data summary for the prediction of homocyst(e)ine (Hcy_p ($\mu mol \cdot L^{-1}$)) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in Hcy.

$$Hcy_p\,(\mu mol \cdot L^{-1}) = 10.7 - (0.205 \times Folate\,(ng \cdot mL^{-1})) - (0.003331 \times B_{12}\,(pmol \cdot L^{-1}))$$

Predictor	Units	Unstandardised Coefficient	*t* ratio	*P*
Constant		10.7	14.93	0.000
Folate	$ng \cdot mL^{-1}$	-0.205	-4.65	0.000
B_{12}	$pmol \cdot L^{-1}$	-0.00331	-2.55	0.012
$s_{YX} = \pm 2.73 (\mu mol \cdot L^{-1})$		$R^2_{adj} = 15.0\%$	$F_{2,168} = 16.05$	

Table 4.48: Final standardised predictive equation and data summary for the prediction of homocyst(e)ine (Hcy_p ($\mu mol \cdot L^{-1}$)) in schoolchildren aged 12-13years. Includes only those terms that made a statistically significant contribution ($P<0.05$) to explained variance in Hcy.

$$\Sigma\, Hcy_p = 10.7 - (0.205 \times Folate) - (0.003331 \times B_{12})$$

Predictor	Standardised Coefficient (β)	*t* ratio	*P*
Constant	0.01610	0.22	0.823
Folate	-0.33347	-4.65	0.000
B_{12}	-0.18560	-2.55	0.012
$s_{YX} = \pm 0.937$	$R^2_{adj} = 15.0\%$	$F_{2,168} = 16.05$	

CHAPTER 5

5.0 DISCUSSION

The present study of schoolchildren aged 12 to 13 years of differing socio-economic status (SES) presents some interesting and novel information on a range of coronary heart disease (CHD) risk factors. The main findings of this investigation are that all pupils (100%) in school 1 (high SES) exhibit at least one CHD risk factor, whilst only three (97.6%) pupils in school 2 (low SES) are exempt of the selected risk factors. Although the possibility that the schoolchildren in the present epidemiological study were not representative of their subgroups must be considered, the findings here support previous indications that CHD risk factors are present in young people. Whether these risk factors contribute significantly to CHD in later life remains controversial. Only through well-designed, longitudinal investigations such as The Amsterdam Growth Study (Kemper, 1995) will this ambiguity be resolved.

5.1 CRITERION THRESHOLDS FOR CORONARY HEART DISEASE RISK

Published and widely used criterion thresholds for coronary heart disease risk were assigned to all physical, physiological, haematological and lifestyle parameters. The percentage of schoolchildren that attained or exceeded published criterion thresholds for CHD risk factors are identified and presented in Table 4.5. The findings of this study were compared with those of previous studies that had used similar cut-off points. In choosing the criterion thresholds, the author considered whether there was sufficient similarity in subjects' characteristics; including age, sex, race, sample number, and methodology.

5.1.1 Physiological and physical variables

5.1.1.1 Blood pressure

In school 1, 10.7% of pupils recorded systolic blood pressure (SBP) ≥ 130 mm Hg, whereas a slightly higher 12.8% of pupils from school 2 presented with similar values. For diastolic blood pressure (DBP), 5% of pupils from the higher SES school recorded DBP ≥ 85 mm Hg; whilst 7.3% of pupils in school 2 met or exceeded this cut-off point. Combined elevated levels of SBP and DBP were recorded in 1.7% and 3.1% of schoolchildren in school 1 and school 2, respectively. When the findings of both schools were combined, 11.9% and 6.3% of schoolchildren exceeded the assigned criterion threshold for SBP and DBP respectively (Wynder *et al.* 1981; National Institutes of Health, 1987; Armstrong *et al.* 1991[a]). These findings do not compare favourably with those of Armstrong *et al.* (1991[a]). In their study of 107 British schoolchildren aged 12 to 13 years, only two of the females exceeded Wynder's diastolic cut-off point, while none of the children exceeded the systolic cut-off point.

Although blood pressure (BP) measurements in this study were based on three consecutive measurements taken several minutes apart, BP is a labile variable and clearly the current protocol had limitations as a measure of 'usual' BP. However, whilst differences in protocol could similarly cause discrepancies in findings, the knowledge that many consider Wynder's arbitrarily designated cut-off points of ≥ 130 mm Hg (SBP) and ≥ 85 mm Hg (DBP) as too high for children aged 12 to 13 years (Wynder *et al.* 1981) implies that the present study's findings are a matter of concern. Although it appears that hypertension could begin during childhood and adolescence (Daniels, 1997), there is no direct evidence that elevated blood pressure early in life plays a part in CHD in adulthood. Permanent essential hypertension in children is rare (Morgenstern, 2002). However, the possibility of such a lifetime association implies

that preventive strategies should be aimed at the younger population. Individuals such as those recognised as having elevated hypertension need to be targeted, especially if the BP risk is only one of several CHD risk indicators identified.

5.1.1.2 Aerobic fitness

Although a recent study reported that the fitness levels of present-day schoolchildren were significantly lower than those described in the 1980s (Dollman *et al.* 1999), to date the evidence is inconclusive. Researchers are yet to agree on optimal levels of aerobic fitness for children and adolescents, partly because the outcome measure 'coronary health' cannot be measured by mortality data. For the purposes of this study, the author used the 10^{th} percentile for aerobic fitness according to Riddoch (1990). The study of Northern Irish children and adolescents recorded the fitness levels of a sufficiently large sample of similarly aged schoolchildren, and used an identical protocol. In school 1, 36.7% of boys were at the 10^{th} percentile for the 20 MST ($\leq$ 47 shuttles) (Riddoch, 1990). In school 2, a slightly lower 35.7% of boys recorded $\leq$ 47 shuttles. Twenty eight percent of girls in school 1 scored $\leq$ 28 shuttles (10^{th} percentile), whilst for girls in school 2, the value was a lower 19.8%. The combined values of both schools produced a value of 29.5% for pupils falling into the 10^{th} percentile for aerobic fitness. The disclosure that despite their more favourable PA habits, more boys than girls were at the 10^{th} percentile might indicate that Riddoch's (1990) criterion thresholds for aerobic fitness were inappropriate for this cohort. The author did not convert shuttle scores to predicted $\dot{V}O_2$ max. This informed decision meant that comparisons with previous data were limited.

According to Riddoch's (1990) criterion thresholds, many Welsh adolescents have poor fitness levels regardless of socio-economic status or sex. However, the author is mindful

that one must exercise caution when adopting such arbitrary cut-off points. The revelation that no significant differences in fitness levels were identified according to SES might be indicative of a convergence of lifestyle habits such as activity and dietary patterns. Many children and adolescents, regardless of SES, have access to televisions and computers, are transported to and from school, and are subject to similar time constraints in the PE curriculum.

5.1.1.3 Anthropometric measures

The prospect of parents outliving their children because of an epidemic of obesity afflicting the younger generation is a real one. There is a need to determine the severity of this problem and to undertake preventative and remedial strategies amongst young people. Overweight and obese young people are at considerable risk for increased prevalence of several CHD risk factors, both now, and in later life (Dietz, 1998; Freedman *et al.* 1999).

Whilst gross obesity in a young person might be obvious, total misclassification often occurs and there is a need for objective indices (Himes and Bouchard, 1989). In the present study, three anthropometric measures contributed to the author's conclusions on overweight and obesity levels in the schoolchildren studied. Such an approach proved to be more indicative of an individual's body fatness levels, and facilitated comparisons with previous work.

5.1.1.4 Body mass index

In children and adolescents, body mass index (BMI) changes significantly with age. It is for this reason that young peoples' BMI needs to be considered using age-related reference curves. A cut-off point for BMI was assigned at the 95th percentile for 12 to

13 year olds according to the recommendations of Freedman *et al.* (1999) and Chinn and Rona (2002). Boys who recorded a BMI > 24.3 kg m^{-2} were defined as 'overweight', whilst girls who recorded > 25.6 kg m^{-2} were reported as 'overweight'. The author was mindful that since BMI does not differentiate between muscle and fat, and is also influenced by natural growth and biological development, findings had to be treated cautiously. However, these percentiles seemed to be reasonable definitions for overweight in children and adolescents. The 95th percentile according to the NHANES cohorts is a frequently used cut-off point (Freedman *et al.* 1999; Hakeem, 2001; Williams *et al.* 2002; Chinn and Rona, 2002), especially when used alongside alternative anthropometric parameters such as skinfold thicknesses.

In school 1, 21.3% of the boys recorded BMI > 24.3 kg m^{-2}, whilst 23.1% of the boys in school 2 posted a BMI value > 24.3 kg m^{-2}. For girls, 13% of pupils from a higher SES recorded BMI > 25.6 kg m^{-2}, whilst 11% of pupils in school 2 met or exceeded this cut-off point. When the findings of both schools were combined, 22.2% of the boys and 12% of the girls exceeded the assigned criterion threshold. As there is no single BMI definition adopted by all international communities, comparing the present cohort's data with those from other studies is difficult. However, since the present investigation adopted the cut-off points of the Bogalusa Heart Study it was realistic to make comparisons with this particular investigation (Freedman *et al.* 1999). Between 11.6% and 13% of boys, and 9% and 10.7% of girls in the Bogalusa Study exceeded the 95th percentile, these values were lower, especially in boys, than those collected in the present study. Although allegations that the US has the greatest prevalence of childhood obesity in the Western world could be refuted considering these recent findings, the significant difference in the respective studies' sample sizes could have influenced results.

5.1.1.5 Waist to hip ratio

Waist to hip ratio (WHR) has been used as a crude estimate of body fat distribution, and increased abdominal fat deposits indicated by a high WHR are postulated to increase the risk of CHD (Gillum, 1987). Hip circumference is an indicator of adipose tissue over the hips and buttocks, whereas waist circumference indicates adipose tissue in the waist and abdominal region. A WHR below 0.8 for females and 0.9 for males has been arbitrarily defined as 'normal' (Reeder *et al.* 1992; Bjorntorp, 1992). The author concedes that this ratio has limited use in the young population (Mueller and Malina, 1987), however, since the measure was used as an adjunct to other obesity indices, it was considered that the cut-off points could be adopted as crude estimates of body fat distribution. In school 1, 19.7% of boys recorded WHR $\geq$ 0.9, whilst 26.4% of boys in school 2 posted similar results. For girls, 47.8% of pupils from the higher SES school recorded WHR $\geq$ 0.8, whilst 51.7% of pupils in school 2 met or exceeded this cut-off point. When the findings of both schools were combined, 23.7% of the boys and 50% of the girls exceeded the assigned criterion threshold. Although WHR is often used as an index of obesity in young people, it should be emphasised that a lack of standardisation in its measurement implies that comparing across studies is ill advised. Hip measurement is particularly susceptible to variations (Ledoux *et al.* 1997) and authors differ in their choice of cut-off points. The relatively high percentages recorded for girls could be indicative of excess subcutaneous adiposity in this particular population, resulting from high levels of physical inactivity and a diet laden in saturated fat. However, it could also indicate the limitations of this measure of body fatness in young people and imply that the girls' cut-off point of 0.8 is too harsh.

5.1.1.6 Summation of skinfold thicknesses measured at four sites

Subcutaneous adiposity was also estimated from the summation of four skinfold sites ($\sum$ SKF). Subcutaneous fat levels change significantly during growth and maturation, hence, as with other anthropometric CHD risk factors in young people, skinfold thickness is best considered using age-related reference curves. For the purposes of this study, the 90th percentile for 12 to 13 year olds according to Riddoch (1990) was used as a cut off point. In school 1, 31.2% of girls recorded $\sum$ SKF $\geq$ 57.4 mm, in school 2 (lower SES), 44.1% of girls reported similar levels. For boys, 28.6% of pupils from the higher SES school recorded $\sum$ SKF $\geq$ 56.9 mm, whilst 23.1% of pupils in school 2 met or exceeded this cut-off point. When the findings of both schools were combined, 32.3% of pupils exceeded the assigned threshold.

Difficulties arise when trying to compare skinfold thicknesses across studies since researchers have differed in their choice of skinfold sites, their decision to convert their skinfold measures to % body fat, as well as cut-off points. Nevertheless, although the Northern Ireland cohort had lower body fat levels than those from many other countries (Riddoch, 1990), it is apparent from the number of schoolchildren, particularly girls, that are in the 90th percentile for body fatness according to Riddoch (1990), that obesity could be a problem amongst young people in Wales.

From the combined results of all anthropometric measures the author concluded that many Welsh schoolchildren, particularly girls, were overweight or obese. This finding was irrespective of socio-economic status. The similarity in adiposity levels regardless of SES could be attributed to the convergence of lifestyle habits, including undesirable eating patterns and physical inactivity tendencies over recent years.

5.1.2 Lifestyle variables

The inevitable subjectivity of collecting lifestyle data such as physical activity and dietary patterns, implies that it is difficult to make conclusive judgements. However, several cautious observations regarding 12 to 13 year olds lifestyles could be made from the data gathered.

5.1.2.1 Physical activity

Pupils were classified as partaking in vigorous physical activity (PA) based on criterion set by Riddoch (1990) (Appendix 6). Vigorous PA was defined according to rate of breathlessness. Despite recommendations that children and adolescents should experience vigorous PA for at least 30 minutes per day (Pate *et al.* 1999; Bouziotas *et al.* 2001), 71.3% of pupils in school 1, and 61.8% of pupils in school 2 had not participated in any vigorous PA over the last seven days. This included pupils accounting for Physical Education lessons. When the findings of both schools were combined, 66% of pupils had failed to participate in vigorous PA in the last week. For both schools, girls exhibited the worst physical activity habits, with 83% of girls in school 1, and 74.1% in school 2 reporting no vigorous PA in the past seven days. The current findings confirm previous research on young British populations (Heartbeat Wales, 1986; Riddoch, 1990; Thirlaway and Benton, 1993; Duncan *et al.* 2002). Moreover, the sex differences were highlighted in the normal activity patterns exhibited during the schools' break and lunch time. Whereas 35.9% of boys took part in PA of greater intensity than walking during break time, no girls reported such activity (both schools combined). Schools have been applauded for providing 'breakfast clubs' and 'tuck shops', yet by doing this they have inadvertently encouraged young people to become physically inactive during their 15-20 minute morning recess. During lunchtime, 62% of boys took part in PA of greater intensity than walking, whereas a

very low 6.6% of girls reported such activity (both schools combined). Interestingly, for girls, this was 0% for school 1, and 12.1% for school 2. The differences between schools were similar for the boys, and for both break and lunchtime. A more extensive extra-curricular programme, facilitated by a healthier staff to pupil ratio in school 2, could have contributed to these differences.

Physical activity is accepted as an important component of a healthy lifestyle. Despite this, and considering the recommendations of others (Pate *et al.* 1999; Bouziotas *et al.* 2001), the findings of the present study support long-standing concerns that young people do not experience sufficient daily PA. This finding is consistent with that of Buttriss (2002) who reported that between 40% and 69% of children in Britain are largely inactive. On a positive note, 96% of pupils in this study enjoyed PA. The present investigation's disclosure that female adolescents are less active than their male counterparts was not surprising and concurred with the findings of others (Ross and Gilbert, 1985; Kemper *et al.* 1985; Heartbeat Wales, 1986; Williams, 1988; Armstrong *et al.* 1990[a]; Thirlaway and Benton, 1993; Raitakari *et al.* 1997; Guerra *et al.* 2001).

Low activity levels during adolescence might be attributed to a number of reasons. According to the National Heart Forum, 30% of young people go to school by car (Little, 2002). In the present study over 90% of pupils (school 1 and school 2) were taken to school via motorized transport. This high statistic, despite many schoolchildren living in close proximity to school 2, could be partially attributed to the safety concerns of parents.

Although a number of adolescents in the present study participated in many of their physical activities outside of school hours, Physical Education (PE) lessons provide an

important potential source of PA. The frequency, content, and structure of PE lessons are of paramount importance, yet school sport and PE has been squeezed and neglected in recent years. As both the Westminster Government and the Welsh Assembly Government focus on improving academic standards, many Head teachers have enforced a gradual decrease in the amount of curriculum time allocated to PE. England and Wales have the lowest amount of PE time in secondary school in the European Union (Little, 2002). If Physical Educationalists are to maximize the opportunities for improving and promoting physical activity habits in schoolchildren, schools must be encouraged by a proactive government to allocate at least two hours of curriculum time for PE each week (Welsh Assembly Government, 2002). In turn, Physical Education teachers need to ensure that lessons contain sufficient vigorous activity.

The relationships between parental activity and the activity of their offspring highlight the power of parent modelling (Sallis *et al.*1988; Moore, Lombardi, White *et al.* 1991). In the present study, 25.3% of parents from school 1, and 36.4% of parents from school 2 did not participate in regular exercise. The improved statistics for school 1 could be partly explained by the farming lifestyle of many of this community's population.

Finally, technological advances have led to an increase in the amount of time young people spend watching television and playing or working on computers. The prevention and management of chronic diseases in the younger population and subsequent adulthood, necessitates educating young people to adopt more healthy lifestyles.

The implication that PA habits during childhood and adolescence might influence adult PA indicates that the younger population should be targeted (Blair, 1992; McGinnis, 1992; Raitkari *et al.* 1994; Kemper and van Mechelen, 1995). Although the findings of

the present study support the notion that many children and adolescents are not sufficiently active, it is accepted that the assessment of PA, particularly amongst younger people is extremely problematic. Despite considering the normal daily routine of the young cohort and seeking objective responses in the recall questionnaire, the present findings relied on the somewhat limited cognitive abilities of 12 to 13 year olds. Furthermore, the current assessment protocol did not make allowances for spontaneous PA despite a young person's tendency to exercise sporadically. The author of the present investigation concedes that these PA findings are 'estimations of true' (Sallis, 1991) but is confident that such data were useful in the compilation of a CHD risk factor profile.

5.1.2.2 Diet

A cut-off point of > 30% total fat and > 10% saturated fat was used in the present study (WHO, 1990; NCEP, 1991; Williams *et al.* 2002). An alarming 87.7% of pupils exceeded the cut-off point for total fat consumed, whilst a similarly unfavourable 93.3% exceeded the recommended amount of saturated fat. The statistics were similar irrespective of socio-economic status and infers a narrowing of dietary habits in modern society.

Despite present society's increased knowledge of what constitutes a well-balanced diet, an investigation of the dietary habits of Texan schoolchildren (9 to 17 years) conducted more than a decade ago reported similar results (McPherson, Nichaman, Kohl *et al.* 1990). Among all subjects, 83% of schoolchildren studied consumed diets containing more than 30% of calories from total fat, whilst 93% of the children consumed diets that contained greater than 10% saturated fat. Similar adverse results were disclosed in The National Diet and Nutrition Survey (Gregory, Lowe, Bates *et al.* 2000) which showed

that 92% of children had intakes of saturated fat that exceeded the recommendations of the Committee on Medical Aspects of Food Policy (COMA) (1994). COMA recommended that saturated fat should contribute no more than 11% of total dietary energy.

The results of the present findings compared unfavourably with those reported by Bouziotas *et al.* (2001) for 12-year-old Greek children. In the Greek study, 15.5% of children exceeded 30% for total fat intake, whilst 41% exceeded the 10% cut-off point for saturated fat. Despite suggestions that the dietary patterns across Europe are converging (BHF, 2000), this finding supports the notion that fat intake in the Mediterranean countries remains lower than in Western European countries. It appears that British schoolchildren remain partial to a proatherogenic diet.

According to the National Diet and Nutrition Survey (Gregory *et al.* 2000) a pattern of 'modern malnutrition' has been revealed amongst British children of low socio-economic status (SES). Such a condition refers to a diet reliant upon chips, cakes, biscuits and salty snacks as energy providers. The present study indicated that this was a problem common to many schoolchildren irrespective of SES.

Although they are not obliged to do so, schools provide 25% to 40% of a child's energy intake (Woteki and Filer, 1995). Families on low incomes entrust schools to provide their children with a healthy diet, and it is estimated that 30% of children do not go home to a cooked meal (Sodexho, 2000). Younger children cannot be expected to make educated choices regarding consuming a balanced diet, the onus therefore lies on influential parties such as schools. However, the quality of school meals, whether free or paid for, is variable across the UK. Despite a moral responsibility to encourage

healthy lifestyles amongst their charges, Local Education Authorities sanction school canteens to maximize profits. Sadly, even Physical Education departments are encouraged to install vending machines providing snacks and drinks in an attempt to boost ailing funds. There must be statutory provision under the Education Acts. It is imperative that the government introduces minimum national nutritional standards for school meals, especially since family demographics intimate that in the near future more meals will be consumed outside the family home (Woteki and Filer, 1995).

5.1.2.3 Smoking

None of the schoolchildren reported smoking one or more cigarettes a week (Bewley *et al.* 1972; Bouziotas *et al.* 2001). This is lower than the statistics presented by the National Heart Foundation (Little, 2002) who reported that one in ten, 11 to 15 year olds smoke regularly. A reticence to admit to smoking despite assured confidentiality, could have led to the present study's null findings. Nevertheless if this study's findings were indicative of smoking status amongst 12 to 13 year olds, it is a welcome statistic.

5.1.3 Haematological variables

The collection of blood samples in a young population requires substantial planning and administration. Furthermore, the acceptability of the procedure to young people, parents and teachers, might significantly affect response rates and possibly the value of any findings. Overall the response rates in the present study were good, the greatest problem arose from absenteeism as a result of an influenza virus that affected both schools during the time of testing. The socio-economic status of the schools did not influence response rates.

5.1.3.1 Total cholesterol

Schoolchildren who recorded total cholesterol (TC) values of $\geq$ 5.2 mmol·L^{-1} were deemed to be 'at risk' (Montoye *et al.* 1985; Bell *et al.* 1986; NCEP, 1991; Boreham *et al.* 1997; Bouziotas *et al.* 2001). In school 1, 9.1% of pupils recorded TC $\geq$ 5.2 mmol·L^{-1}, whilst a disconcerting 28.3% of pupils from school 2 (lower SES) posted a level $\geq$ 5.2 mmol·L^{-1}. It should however be noted that comparing total cholesterol levels might be problematic (Blank, Hoeg, Kroll *et al.* 1986) especially since some of the higher TC levels could be explained by higher HDL-C values. When the findings of both schools were combined, 21.2% of pupils exceeded the assigned criterion threshold. These results are higher than those recorded by Bouziotas *et al.* (2001) in their study of Greek children aged 12 years. Five percent of schoolchildren exceeded the cut-off point in the Greek study, supporting the theory that the cardioprotective Mediterranean diet of more fish, fruit and vegetables, but less meat, might benefit the lipid profiles of young people.

5.1.3.2 Low density lipoprotein

Recent prospective studies revealed that the presence of low density lipoprotein (LDL-C) was associated with a threefold increase in the risk of developing CHD (Lamarche *et al.* 1999). A value $\geq$ 3.8 mmol·L^{-1} represented a risk of developing CHD in later life (Montoye *et al.* 1985; Bell *et al.* 1986; NCEP, 1991; Boreham *et al.* 1997; Bouziotas *et al.* 2001). Whilst no pupils in school 1 met or exceeded the 'at risk' threshold, a slightly higher 6.1% reached the cut-off point in school 2. When the findings of both schools were combined, 3% of pupils exceeded the assigned cut-off point. The current investigation's findings compare favourably with the results of a similar study of Greek children (Bouziotas *et al.* 2001) where elevated LDL-C levels were reported in 6.5% of the population. Interestingly, this finding does not support the theory that children from

Mediterranean countries have superior blood lipid profiles to young people in more Western European countries.

The concentration of TC and LDL-C provides an indirect index of exposure to a proatherogenic diet and physical inactivity, confounded by genetic factors. Since no statistically significant differences in relation to SES were identified amongst the dietary and activity patterns of schoolchildren, this might suggest that the SES differences in TC and LDL-C, can be attributed to genetic influences (Kwiterovich, 1995). Although familial history of dyslipidemia was sought in this investigation, using parents as sources of such information is debatable.

5.1.3.3 High density lipoprotein

High density lipoprotein cholesterol (HDL-C) has a strong, inverse relationship with CHD, and low concentrations could detrimentally affect the body's ability to remove cholesterol from the tissues. If values for HDL-C were ≤ 0.9 mmol·L^{-1}, individuals were considered at risk of developing CHD in later life (NCEP, 1991, Williams *et al.* 2002). Only 1.3% of schoolchildren in school 1, and 0.8% of pupils in school 2 recorded values ≤ 0.9 mmol·L^{-1}, giving a combined statistic of 1% for both schools. These values are similar to those reported by Bouziotas *et al.* (2001) for 12-year-old Greek children. In the earlier study, 1.5% of children exceeded the same 'at risk' cut-off point. Both investigations reported that girls only recorded ≤ 0.9 mmol·L^{-1}. Since boys in both studies were more physically active and physically fit than their female counterparts, this supports previous claims that there is a positive and significant relationship between HDL-C and aerobic fitness in young people.

5.1.3.4 TC: HDL-C

It is claimed that the ratio of TC to HDL-C is a strong predictor of CHD, and a risk factor that tracks through from adolescence to adulthood (Bao *et al.* 1996[a]; Bao Srinivasan, Berenson *et al.* 1996[b]). Using a cut-off point of 4.0 for TC: HDL-C, 19.5% of the schoolchildren in school 1 exceeded this cut off point, whilst in school 2 a slightly lower 18.3% reached the 'at risk of developing CHD' criterion threshold (Bell *et al.* 1986; Boreham *et al.* 1997). When the findings of both schools were combined, 18.8% of pupils exceeded the assigned cut-off point. The individual or combined effects of physical inactivity, a high saturated fat diet and genetic factors could have contributed to these high ratios.

5.1.3.5 Triglycerides

Triglyceride (TG) has emerged as a significant independent CHD risk factor (Assmann, Schulte, Funke *et al.* 1998). A value of ≥ 1.7 mmol·L^{-1} represented a risk of developing CHD in later life (Montoye *et al.* 1985; Bell, Macek, Rutenfranz *et al.* 1986; NCEP, 1991; Boreham *et al.* 1997; Bouziotas *et al.* 2001; NCEP, 2002). In school 1, 7.8% of adolescents recorded levels at or above 1.7 mmol·L^{-1}; in school 2, 11.5% recorded similar results. When the findings of both schools were combined, 10.1% of pupils exceeded the assigned cut-off point. These results do not compare favourably with those of Greek schoolchildren aged 12 years in whom a low 0.5% reached the assigned threshold (Bouziotas *et al.* 2001). Such differences in the number of schoolchildren recording elevated levels of TG supports the benefits of a Mediterranean diet. However, the poor levels of activity reported in the present study could similarly have been influential.

5.1.3.6 Lipoprotein(a)

The measurement of Lipoprotein(a) (Lp(a)) is a complex issue and there is a need for an internationally accepted protocol that will yield comparable results across all populations. As with other recently identified risk indicators of CHD, the lack of consistent methodologies and reference values for young people implies that we are not yet certain what constitutes 'normal' or 'at risk' levels for this particular population. Considering the inherent difficulties in measuring the physical, physiological and lifestyle characteristics of young people, age and sex-specific percentiles would be best. However, a cut-off point for Lp(a) that is frequently adopted for young people is the 'at risk' value associated with adults of $\geq$ 30 mg·dL^{-1}. Schulpis, Karikas, Gavirili *et al.* (2001) reported a 95th percentile of 29.1 mg·dL^{-1} for their Greek cohort (6 to 14 years). Since young Greeks have posted some of the lowest ever reported Lp(a) levels, the author of the present study opted to use the similar and widely used criterion threshold of $\geq$ 30 mg·dL^{-1}. At present any findings obtained with Lp(a) must be interpreted cautiously, especially in children and adolescents. As with other studies, the distribution of Lp(a) levels among adolescents was skewed towards the lower end, non-continuous, and over a broad range (Srinivasan *et al.* 1991; Taimela, Viikari, Porkka *et al.* 1994; Barth *et al.* 1999). This would have accounted for the large standard deviation values observed. In the present study 31.6% of adolescents in school 1 recorded 'at risk' values of $\geq$ 30 mg·dL^{-1}, whilst a slightly lower 24% recorded $\geq$ 30 mg·dL^{-1} in school 2 (Valentine *et al.* 1994; Mackinnon & Hubbinger, 1999; Sveger, Flodmark, Nordborg *et al.* 2000). Forty nine percent of schoolchildren recorded levels below 8 mg·dL^{-1} (lowest detection point). When the findings of both schools were combined, 26.8% of pupils exceeded the assigned criterion threshold. The findings of the current investigation are not dissimilar to those of Genzel-Boroviczeny *et al.* (1997). Genzel-Boroviczeny and colleagues (1997) reported elevated levels ($\geq$ 30 mg·dL^{-1}) in 20% of children.

Elevated levels of both Lp(a) and LDL-C are considered to have the greatest detrimental influence on health status (Sveger *et al.* 2000). In the present study, 3.4% of adolescents demonstrated this unfavourable condition. This was higher than the value identified by Sveger and colleagues (2000) who reported combined elevated levels of Lp(a) and LDL-C in 1.7% of schoolchildren aged 10 to 11 years with family history of CHD. Although it is debatable whether changes in lifestyle patterns and drug treatment, with the exception of niacin, have a significant effect on Lp(a) levels, the combined detrimental influence of Lp(a) and LDL-C can be reduced by lowering the modifiable LDL-C (Maher, Brown, Marcovina *et al.* 1995).

Lipid related observations indicate the need to target young people with intervention programmes that improve dietary and physical activity patterns.

5.1.3.7 Fibrinogen

Although it is considered an important building block in laying down the foundation for the development of atherosclerosis, relatively little has been written on fibrinogen (Fg) levels in young people. The 90th percentile according to Sanchez-Bayle *et al.* (1993) was chosen as a cut-off point. 'At risk' values were > 394 mg·dL^{-1} or the 90th percentile according to (Sanchez-Bayle *et al.* 1993). An alarming 60.8% of pupils in school 1 were assigned to the 90th percentile, whilst a slightly improved 46% recorded levels > 394 mg·dL^{-1} in school 2. The results of this study compared poorly with those of Sanchez-Bayle *et al.* (1993) where only 11.7% had an Fg level > 394 mg·dL^{-1}, and supports the notion that elevated Fg levels are evident in young people (Bao *et al.* 1993; Sanchez-Bayle *et al.* 1993; Mahon *et al.* 1997; Invitti *et al.* 2003).

Despite the worrying findings of the present study, the knowledge that intra-individual variability exists in Fg levels implies that a single measurement is inadequate for the prediction of CHD risk. Although the current findings provide valuable reference data for adolescents, the evidence regarding Fg levels in young people and future CHD risk remains inconclusive and further investigation is required.

5.1.3.8 Homocyst(e)ine

There is scant information on homocyst(e)ine (Hcy) distribution in children and adolescents. In the present study, 21.4% of the pupils in school 1 exceeded the 95th percentile according to Osganian *et al.* (1999) (> 8.5 $\mu mol \cdot L^{-1}$). In school 2, a slightly lower 20.3% of pupils recorded > 8.5 $\mu mol \cdot L^{-1}$. These statistics are substantially higher than those reported in the CATCH cohort (Osganian *et al.* 1999). Osganian *et al.* (1999) reported that only 3.3% of children (aged 13 to 14 years) who took multivitamins exceeded the 95th percentile, whilst a slightly higher 5.7% was reported amongst non-users. Dietary analysis in the present study revealed that of those pupils who exceeded the 95th percentile, 7.9% took multivitamins. Overall, 14.9% of pupils in the < 95th percentile regularly took multivitamins. This finding suggested that multivitamin intake could help maintain normal levels of homocyst(e)ine.

Methodological differences in Hcy measurements, particularly clotting time, could have contributed to the disparity in findings between the current investigation and that of previous studies. Moreover, many researchers fail to calculate or report statistical power, but if the power is too low, findings are meaningless. Cohen (1988) has inferred a minimum power of 0.8 (80%) for rejection of the null hypothesis in the behavioural sciences. The author of the present study ensured adequate power of the test and was confident in the statistical findings presented in this study. It is accepted that puberty

might influence homocyst(e)ine levels through increased muscle mass and hormonal activity, consequently the levels reported here might differ significantly to what might be reported for the same population in later life. Longitudinal studies are essential if any just conclusions are to be made regarding the possible influence that elevated Hcy levels in adolescence has on CHD risk during adulthood. Moreover, if Hcy levels are high in children, it is highly likely that the parents will have similarly elevated, if not higher levels. Recent advances in assay procedures have led to the relatively easy measurement of Hcy, furthermore, the treatment of elevated levels via folate and vitamins B_{12} and B_6 is inexpensive. This suggests that as a haematological measurement of CHD, homocyst(e)ine is appealing.

5.2 CLUSTERING OF CORONARY HEART DISEASE RISK FACTORS

The present data give evidence of the development of multiple CHD risk factors at an early age (Table 4.6), furthermore a possible genetic transference of some risk factors cannot be dismissed. As the number of risk factors in an individual's profile increases, so too does the likelihood of developing CHD (Cunnane, 1993; Raitakari *et al.* 1994). Cooley and McNaughton (1999) suggest that 40% of children aged 12 years have at least one major risk factor of CHD. Unfortunately, the current study confirms the supposition that many children and adolescents exhibit more than one of these indicators of CHD (Wilmore and McNamara, 1972; Gilliam *et al.* 1977; Bao *et al.* 1994; Boreham *et al.* 1997; Hardin *et al.* 1997; Twisk *et al.* 1999).

The physical, physiological, haematological and lifestyle risk factors for all pupils were calculated. Individuals from families with a history of CHD, hyperlipidemia, or hypertension in first-degree relatives, were allocated an additional risk factor

(Bergstrom, Hernell, Persson *et al.* 1995). Although familial history of CHD was included, it should be recognised that parents could have experienced difficulty in accurately recalling family history, especially of the deceased. In addition to this, although some parents did offer information with respect to a family history of dyslipidemia, the likelihood of parents being tested for blood lipids was small. Nevertheless, although familial history could be regarded as a surrogate measure of CHD, there might be genetic and lifestyle reasons for the familial aggregation of an elevated risk profile (Bao *et al.* 1993). The investigation of this risk factor is by itself warranted.

In school 1, 83.9% of pupils exhibited more than one risk factor. In school 2, which had the additional risk factor of pupils being of low SES not included in this calculation, 89.5% displayed more than one risk factor. When familial risk was removed, this value was 73.6% and 76.6% for school 1 and 2 respectively. In school 1, 65.5% of schoolchildren exhibited at least three risk factors, whilst for school 2, 67.7% displayed the same number. In school 1, 24.1% of pupils exhibited at least five CHD risk factors, for school 2 this statistic was 30.6%. An alarming eight risk factors were reported in three pupils, whilst two pupils exhibited nine CHD risk indicators. Both individuals were males, and both attended school 2. For schoolchildren exhibiting multiple risk factors, the need for counselling over a protracted period was obvious.

The comparison of results across studies is particularly difficult since definitions of clustering are based on contrasting sets of CHD risk factors and criterion thresholds. However, since the present study supports the theory that the clustering of risk factors does occur in young people, there is a clear and undeniable need for intervention in this particular population. The monitoring of multiple CHD risk indicators in young people

might be more important than focussing on individual factors (Bao *et al.* 1994). Previous work has shown that CHD risk factors accumulate in the offspring of families of low socio-economic status (Bergstrom *et al.* 1996). However, since multiple CHD risk factors were apparent in the pupils of both schools, this indicates the need to focus on the health of all adolescents, regardless of SES. Although some CHD risk factors are not considered modifiable, the vast majority of these indicators can be influenced. The multifactorial nature of coronary heart disease suggests that all modifiable risk indicators should be afforded attention.

5.3 SOCIO-ECONOMIC AND SEX DIFFERENCES IN CORONARY HEART DISEASE RISK FACTORS

The overall aim of this study was to ascertain whether there was a difference in the CHD risk status of young people according to socio-economic status. In the present study, school 1 represented high socio-economic status (SES), whilst school 2 represented low SES. It has been claimed that efforts to combat CHD should be targeted towards the economically disadvantaged as this is the population associated with greatest incidence of CHD related morbidity and mortality (Gliksman *et al.*1995). It has also been postulated that children and adolescents from lower socio-economic backgrounds are less likely to engage in lifetime physical activity (Wold, Oeygard, Eder *et al.* 1994). However, whether or not SES reflects differences in CHD risk factors including current activity levels and food choices remains to be elucidated.

5.3.1 Physical and physiological parameters (Table 4.7)

5.3.1.1 Blood pressure

For systolic blood pressure (SBP), significant differences ($P \leq 0.05$) were identified between the boys from both schools, and between the boys from school 1 and girls from school 2. Sex differences were prominent in the results for diastolic blood pressure (DBP). Significant differences ($P \leq 0.05$) were reported between the boys and girls from both schools, and between the boys and girls across socio-economic status.

In accordance with others (He, Horlick, Fedun *et al.* 2002), significant differences ($P \leq 0.05$) relating to sex were evident. Since these results did not always favour the boys, to state that these differences were the result of the superior fitness and reduced levels of body fatness of boys would be presumptuous.

Results obtained from this study fail to support the conclusions of Jenner and co-workers (1992) that SES influences BP. However, neither do they concur with the observations of Dwyer, Coonan, Worsley *et al.* (1980) and Gillum, Prineas, Gomez-Marin *et al.* (1985) that SES does not affect BP in young people. The lack of any consistent significant differences according to SES in the present study have been reported by others (Freeman *et al.* 1990; van Lenthe *et al.* 2001).

It is noteworthy that the BP levels recorded here are higher than those reported for other children and adolescents (Armstrong *et al.* 1991[a]; Bouzioutas *et al.* 2001) but similar to those described elsewhere (Jenner *et al.* 1992; Twisk *et al.* 1999). The range of BP values reported in different cohorts probably reflects the differences in measurement protocols. For example, the electronic instruments used in the present and previous studies (Park and Menard, 1987; de Visser *et al.* 1994; Prescott-Clarke and Primatesta,

1997; Guerra *et al.* 2001), were convenient, but might record different measurements than those made by a sphygmomanometer (Williams *et al.* 2002; Morgenstern, 2002). Moreover, differences in maturation age and environmental conditions could have proved influential.

5.3.1.2 Aerobic fitness

The significant differences ($P \leq 0.05$) observed in the aerobic fitness of boys and girls were anticipated and were in agreement with others (Riddoch, 1990; Armstrong *et al.* 1990[b]; Armstrong *et al.* 1991[b]). Boys outscored girls within and between schools. Increased subcutaneous adiposity, less muscle mass, less haemoglobin concentration, and decreased stroke volume were the probable causes of poorer fitness levels in girls (Armstrong and Welsman, 1994[a]), however, the possibility that girls were less motivated to perform maximally cannot be dismissed. Although significant differences were observed between schools, this was sex and not SES led. Unlike some previous reports, (Guillaume *et al.* 1997; Wold and Hendry, 1998), the present study failed to identify any significant differences in physical activity or fitness levels according to SES, a finding that was in keeping with the observations of others (Dwyer *et al.* 1980; Riddoch, 1990; Freeman *et al.* 1990; Batty and Leon, 2002). Different methods for establishing socio-economic status could have contributed to the discrepancies in findings, but a narrowing of aerobic fitness levels according to SES could also be the cause. Although the young people investigated in this study attended schools in very different socio-economic areas, the content and delivery of the PE curriculum, including sporting facilities available, was similar for both schools. Since many children depend on school for their activity experiences, it is hardly surprising that the difference in fitness levels with respect to SES, were negligible at this early stage of life. In terms of the null hypothesis, power values for all *t*-test analyses of aerobic fitness were very high

at > 99.5% (α = 0.05). These were considerably higher than the conventional 80% minimum required for rejection (Cohen, 1988).

Mean aerobic fitness for this cohort was lower than that recorded for Dutch, Northern Irish, and Scottish adolescents (van Mechelen *et al.* 1986; Riddoch, 1990; Twisk *et al.* 1999; McVeigh *et al.* 1995;). Since young people exhibit a broad range of fitness scores, it is likely that several pupils had low levels. Although the current data might support the opinion that young people are not as physically fit as they were in past decades (Strong, 1990), the author concurs that the evidence is far from conclusive (Rowland, 2002). Despite the author's attempt to limit comparisons to studies that have employed the same protocol; different environmental conditions, time of year, motivational factors, and efficiency of anaerobic energy sources could have influenced findings.

5.3.1.3 Anthropometric measures

For the purposes of this discussion, anthropometric measures (WHR, BMI, $\sum$ SKF) were considered together.

For waist to hip ratio, sex differences were notable. Significant differences ($P \leq 0.05$) occurred between the boys and girls of both schools and between the sexes across socio-economic status. Boys recorded higher WHR in all school and sex permutations, an observation reported by others (Gillum, 1987). This was to be expected since from puberty males' body fat is more centrally distributed than that for females (Malina and Bouchard, 1991; Sangi *et al.*1992), a pattern of fat that is considered to increase the risk of CHD. As expected, significant differences ($P \leq 0.05$) were reported between the sexes for summation of skinfold thicknesses. Subcutaneous adiposity is more evident in

females than in males, especially during adolescence, a theory confirmed in this study. Mean significant differences ($P \leq 0.05$) were identified between the boys and girls of both schools, and between the boys of school 1 and girls of school 2. No significant differences ($P > 0.05$) were reported within sex but between SES, for either WHR or skinfold thickness (SKF). Irrespective of permutation, no significant differences ($P > 0.05$) were observed for body mass index (BMI). The lack of significant differences in obesity measures according to socio-economic status is in disagreement with the findings of some (Bogin and Sullivan, 1986; Freeman *et al.* 1990; Guillame *et al.* 1997; Evans, Newton, Ruta *et al.* 2000; Burke, Beilin, Dunbar *et al.* 2001; Hakeem, 2001; Moore, Howell, Treiber *et al.* 2002). Although there is some evidence that body fatness is influenced by SES, this evidence is far from compelling (Dwyer *et al.* 1980; Rebato *et al.* 1998; de Spiegelaere, Dramaix, Hennart *et al.* 1998; Troiano and Flegal, 1998; Batty and Leon, 2002).

It is interesting to note that the mean sum of skinfold thicknesses ($\sum$ SKF) in the present study is higher than that recorded for similar age groups in Northern Ireland (Riddoch, 1990; Twisk *et al.* 1999) and the Netherlands (van Mechelen and Kemper, 1995), this was true for both boys and girls. Northern Irish children and adolescents had also compared favourably against those in other countries (Riddoch, 1990). As mentioned previously, BMI did not differ within the context of socio-economic status (school) or sex. The lack of significant difference in BMI according to SES was in contrast to the observations of Moore and colleagues (2002) for 8 to 15 year olds. Mean BMI for the present study's cohort was 21.4 ± 4.3 kg m^{-2}. As with SKF, this value does not compare favourably with adolescents in Northern Ireland (Riddoch, 1990) or for those in The Amsterdam Growth Study (van Mechelen and Kemper, 1995). Since there was a concomitant increase in $\sum$ SKF in the present study, the possibility that the greater BMI

here was caused by increased bone and muscle mass could be dismissed, the probable cause being increased subcutaneous adiposity.

If comparisons are to be made across studies, standardised procedures for measuring adiposity must be followed. Although researchers do their utmost to maintain this uniformity, variability in methods occur and this could partly explain the discrepancies in findings. However, since the author was mindful of the importance of adhering to standardised procedures, and that the observation of unfavourable body fatness levels was consistent across all measures of adiposity, it is probable that the present findings reflected greater adiposity amongst Welsh adolescents irrespective of SES or sex. Such high levels of adiposity could be attributed to the effects of lifestyle and genetic factors.

5.3.2 Lifestyle variables (Table 4.8)

5.3.2.1 Diet

The findings indicate a significant difference between the average daily calorie intake of boys and girls in school 1 ($P \leq 0.05$), but no other differences were identified for this variable. In considering the H_0, a power value of 90% was identified. The difference in average total calorific intake between the sexes was to be expected since after two years of age, estimated energy requirements become greater in males than in females (Malina and Bouchard, 1991). The increased activity levels of boys, a finding confirmed in the present study, was the probable cause of this sex difference. It is unlikely that the increased energy requirement of boys is caused by the greater muscle mass of males, as there is no sex difference in the energy requirement per unit of fat-free mass (Malina and Bouchard, 1991).

There was no evidence of significant differences in either total fat or saturated fat intake, between sex or across SES. The conclusions of this study are in disagreement with the findings of Freeman *et al.* (1990) who reported a poorer diet amongst schoolchildren (15 to 16 years old) in an underprivileged area. In this study, mean fat intake for girls was 34.6 ± 3.6%, whilst for boys the figure was 34.5 ± 4%. Saturated fat intake was reported as 14 ± 2.4% and 13.5 ± 2.3% for girls and boys, respectively. Similar results were reported in American adolescents (McPherson *et al.* 1990) supporting the opinion that too many of Western society's young people are meeting energy requirements through a high-fat diet. However, if children and adolescents are advised to consume a lower-fat diet, careful attention should be given to the type of foods recommended to replace the energy provided from fat.

5.3.3 Haematological variables (Table 4.9)

5.3.3.1 Lipids, lipoproteins and glucose

For total cholesterol (TC), significant differences ($P \leq 0.05$) were identified between the boys from school 1 and school 2, but not between the girls from the respective schools. For the statistically significant difference elicited between the male cohorts, a power value of 90% ($\alpha = 0.05$) was identified. Differences were also reported between the boys from school 1 and the girls from school 2 (power value of 89% ($\alpha = 0.05$)). Several significant differences were also revealed for low-density lipoprotein-cholesterol (LDL-C). Differences occurred between the boys from both schools, and the boys from school 2 and girls from school 1. In considering the null hypothesis, an acceptable power value of 86% was identified in the former *t*-test. However, the power value of 74% achieved in the latter analysis was below the minimum 80% conventionally required for rejection of the H_0.

No significant differences ($P > 0.05$) were identified according to SES or sex for, high-density lipoprotein-cholesterol (HDL-C), TC: HDL-C, triglygerides (TG), or glucose (Glu). This was in contrast to the observations of Malina (1995) who reported that TC and TG levels did vary according to SES amongst Latin American children. Overall, despite the identification of some differences, the findings from the current investigation indicated that the concentrations of established haematological CHD risk factors were similar irrespective of SES, a conclusion made by others (Berenson *et al.* 1980; Dwyer *et al.* 1980; Freeman *et al.* 1990; Cook *et al.* 1996). It is evident that more work of a longitudinal nature is needed. Levels of haematological parameters are highly variable within a population and for small differences to become statistically significant, large sample numbers are necessary. However, the author of the present study was mindful of this and accounted for both effect size and the power of the test outcome.

Slightly more favourable haematological values have been reported for American, Norwegian, and in particular Greek adolescents (Berenson *et al.* 1980; Tell and Vellar, 1988; Hickman *et al.* 1998; Bouziotas *et al.* 2001). However, the present cohort's results were similar, and at times better, than those previously reported for British, Australian and Northern Irish young people (Armstrong *et al.* 1991[a]; Dwyer and Gibbons, 1994; Boreham *et al.* 1997).

5.3.3.2 Lipoprotein(a)

A great deal of controversy surrounds the role of Lp(a) as a risk factor of CHD, not least so because of the discrepancies in its measurement. In this cohort of Welsh schoolchildren no significant differences ($P > 0.05$) were identified with respect to sex or SES. The lack of significant difference between the sexes has been reported elsewhere (Taimela *et al.* 1994; Genzel-Boroviczény *et al.* 1997; Schulpis *et al.* 2001).

There is no conclusive evidence regarding the influence of socio-economic status on Lp(a) in either adults or the younger population. Although the findings here would indicate that SES has no effect on Lp(a) concentration in adolescents, only through longitudinal studies could this relation be totally dismissed.

Mean Lp(a) concentrations in this study are considerably higher than those reported for other populations (Gozlan, Gross and Gruener, 1994; Schulpis *et al.* 2001), and was particularly true for girls in school 1. If, as postulated, Lp(a) levels remain essentially the same from childhood to adulthood (Schulpis *et al.* 2001) the present findings suggest that the Welsh population, regardless of SES, are at a high risk of developing CHD because of genetically determined elevated levels of Lp(a).

From the limited research available, it would seem that Lp(a) concentrations are similar in young people, irrespective of sex or socio-economic status. As with other recently established CHD risk factors, the value of screening for lipoprotein(a) is debatable. This is particularly true for Lp(a) since difficulties inherent in its measurement can lead to spurious data. Although studies such as the one undertaken here provide valuable insight into the biological CHD risk profiles of adolescents, only through large-scale longitudinal studies can the true relevance of Lp(a) screening be determined.

5.3.3.3 Fibrinogen

Fibrinogen (Fg) is widely recognised as an independent risk factor for CHD. With respect to SES, a significant difference was reported between the fibrinogen levels in boys, although, perhaps contrary to expectations, boys in school 1 (higher SES) reported a higher mean value than the boys in school 2. When considering the H_0, a power value of 93% ($\alpha = 0.05$) was identified. Despite the boys in school 1 recording significantly

higher Fg levels than the girls in school 2 (power value of 81% ($\alpha = 0.05$)), differences according to sex were not consistent across all permutations. Whilst a number of paediatric studies have failed to report significant differences in Fg concentration within the context of SES (Cook *et al.* 1999; Batty and Leon, 2002), adults from a lower socio-economic background have been shown to record higher Fg levels than their higher SES counterparts (Lee *et al.* 1990; Brunner, Smith, Marmot *et al.* 1996). Paediatric research in this area is limited and it might be that subtle differences have been missed.

The similarity in Fg concentrations across the sexes is in line with some recent publications (Bao *et al.* 1993; Fu and Nair, 1998; Volpi, Lucidi, Bolli *et al.* 1998) but is in disagreement with others who have found higher Fg levels in females (Sanchez-Bayle *et al.* 1993; Mahon *et al.* 1997; Cook, Whincup, Miller *et al.* 1999; Invitti *et al.* 2003). The higher levels recorded for females in some studies could be caused by the increased clottability of Fg in females (Lee *et al.* 1990). Although not relevant to this study, some authors have also reported a positive linear relationship between the use of oral contraceptives or pregnancy, and Fg levels (Balleisen, Bailey, Epping *et al.* 1985; Krobot *et al.* 1992). Sex differences might have been more apparent in an older cohort.

The comparison of mean levels of Fg across studies is difficult since methodological procedures can differ, however, it is inherently interesting to make cautious comparisons. The mean levels of Fg in the current cohort of schoolchildren are higher than those previously reported for similar cohorts (Sanchez-Bayle *et al.* 1993; Mahon *et al.* 1997; Cook *et al.* 1999). Although the values recorded here were also higher than those reported for a group of obese Italian children aged 6 to 18 years (Invitti *et al.* 2003), age is known to affect Fg values. The lack of a single standardised assay and the presence of intra-individual variability in Fg levels implies that the stratification of

CHD risk cannot be accurately made with a single Fg determination (Rosenson, Tangney and Hafner, 1994; Harjai, 1999). As with many other recently established CHD risk factors few studies of this nature have been conducted and more extensive research is required.

5.3.3.4 Homocyst(e)ine

Elevated homocyst(e)ine is an independent risk factor of atherosclerosis (McCully, 1969) but information on its presence and subsequent relevance in young people, is scant. In this study, no significant differences ($P > 0.05$) were identified according to sex or SES for homocyst(e)ine. In contrast to this, the Child and Adolescent Trial for Cardiovascular Health (CATCH) (Osganian *et al.* 1999) reported a significantly higher mean concentration in boys than girls. Although the large cohort investigated by Osganian *et al.* (1999), must give credence to the study's findings, several other investigations have failed to report sex differences (Tonstad *et al.* 1996; Reddy, 1997) suggesting that the area warrants further exploration.

The lack of significant differences with respect to Hcy and according to SES, is in agreement with the findings of some (Batty and Leon, 2002), but not others (Tonstad *et al.* 1996). As with many other recently identified CHD risk factors, research is in its infancy. The mean levels for Hcy in the present study were higher than corresponding mean levels reported by others (Tonstad *et al.* 1996; Osganian *et al.* 1999), although the data did compare favourably with the only set of available reference values (Reddy, 1997). Despite the mean Hcy levels being lower than published reference ranges, the relatively small samples investigated by Reddy (1997) could weaken its validity as a reference source. Typically, as with other recently established CHD risk indicators,

further clarification of the relevance of elevated homocyst(e)ine in early years is needed.

5.3.3.5 Folate

Folate levels appear to coincide with SES. A significant difference ($P \leq 0.05$) in folate concentration was identified between schools for both boys and girls. In terms of the H_0, power values of 89% and 98% ($\alpha = 0.05$) were identified; both higher than the conventional minimum required. Since the pupils' diets and vitamin intake were similar irrespective of SES, this observed difference could not be explained. With respect to sex, a significant difference was reported between the boys and girls from school 2 (power value > 99.5% ($\alpha = 0.05$)), but for no other permutation. Sex differences were not consistent, therefore the current findings could not support Osganian *et al.*'s (1999) claims that males have significantly higher folate levels than females.

The mean levels for folate in this study were lower than those reported by CATCH (Osganian *et al.* 1999). However, in contrast to the present investigation, CATCH measured non-fasting folate levels, this in itself could have caused the contrariety in findings.

5.3.3.6 Vitamin B_{12}

No significant differences ($P > 0.05$) were identified according to SES or sex for vitamin B_{12}. The lack of significant differences within the context of sex was in contrast to the findings of CATCH. Osganian *et al.* (1999) reported higher levels of vitamin B_{12} in US females when compared to males. An analysis of the dietary habits of schoolchildren in the present study did not reveal any sex differences and supported the lack of differences in vitamin B_{12} levels.

Mean vitamin B_{12} levels in this study were considerably higher than those reported by Osganian *et al.* (1999). Since the increased values here did not result in the Welsh schoolchildren comparing favourably with the CATCH cohort with respect to Hcy levels, one could question the notion that vitamin B_{12} reduces Hcy in young people.

In the present study, as in previous research (van Lenthe *et al.* 2001), no clear pattern of differences in CHD risk factors was found according to socio-economic status. Making comparisons with other studies is difficult since researchers employ different methodological procedures for determining CHD risk factor levels, and different criteria for ascertaining socio-economic status. Nevertheless, the observations made in the current investigation encourage the author to conclude that the lifestyle patterns and subsequent prevalence of CHD risk factors of young people from differing socio-economic backgrounds have converged in recent years.

5.4 CORONARY HEART DISEASE RISK FACTOR ASSOCIATIONS

A number of statistically significant ($P \leq 0.05$) relationships were generated in a matrix of correlation coefficients (Appendix 8). Many of these values could not be entered into the multiple regression analysis as they exhibited multicollinearity ($r \geq 0.80$). Furthermore, backward elimination during multiple regression calculation resulted in only those variables contributing significantly to explained variance of the dependent variable being included in the predictive equation. Since these correlation coefficients were nevertheless statistically significant, the author deemed it necessary to make comment.

Although no relationship was identified between HDL-C and LDL-C, blood lipids (TC, HDL-C, LDL-C, TC: HDL-C, TG) were, as expected, significantly interrelated. Lipoprotein(a) was also significantly related to TC and LDL-C and supported the idea that Lp(a) measurements should form part of a CHD lipid risk profile. This study failed to confirm an association between Fg and WHR, however, significant relationships were identified between Fg and other body fat measures. The findings of the present study confirmed the outcomes from previous investigations, and intimated that environmental and genetic factors that control obesity might also affect Fg levels (Krobot *et al.* 1992; Bao *et al.* 1993; MacAuley *et al.* 1996; Zahavi *et al.* 1996; Sudi *et al.* 2001; Balagopal *et al.* 2002).

A low, but significant inverse relationship was identified between SBP and HDL-C concentration, indicating that as in adults, increased HDL-C is associated with lower CHD risk in young people. Elevated blood pressure is often the consequence of overweight and obesity, a theory supported in the present findings. With the exception of WHR, the data revealed significant, positive relationships between systolic blood pressure and body fat indices. Diastolic blood pressure was similarly significantly related to all body fat measures, although again this was with the exception of WHR. The results of this study could indicate a weakness in WHR as an obesity measure in adolescents. The strong relationship identified between BP and obesity indices in young people was in agreement with others and suggested that body size and obesity could play a part in the aetiology of primary hypertension (Fripp *et al.* 1985; Jenner *et al.* 1992; Boreham *et al.* 1993; Moussa, Skaik, Selwanes *et al.*, 1994; Stewart *et al.* 1995; Guerra *et al.* 2001; Smith and Rinderknecht, 2003). Elevated DBP was also significantly related to increasing levels of TG and TC: HDL-C, and decreasing levels of HDL-C. These findings, along with the observation that lower levels of aerobic

fitness were associated with elevated DBP, indicated the importance of including DBP as a CHD risk factor. The findings in this study were in agreement with the suggestions of previous investigations that a superior aerobic fitness and BP profile, might be due to lower body fat levels (Tell and Vellar, 1988).

Several studies have shown a strong association between body fatness and blood lipid levels (Bar-Or and Barnaowski, 1994; Twisk, 2000), however, in the present study, the relationships were inconsistent. As expected, body fatness was significantly and positively correlated with TG, TC: HDL-C, and Fg, whilst a significant, inverse relationship was observed with HDL-C. However, since there was little evidence of an association between obesity levels and TC, LDL-C or Lp(a), the findings in this study were inconclusive. As reviewed by Armstrong and Simons-Morton (1994) and Twisk (2000), aerobic fitness had a favourable influence on TG, HDL-C, and TC: HDL-C. However, contrary to expectations, no significant relationship was identified between aerobic fitness and TC or LDL-C concentration in this study. Furthermore, no significant association was observed with Lp(a), suggesting that Lp(a) levels are for the most part genetically determined. The inconsistencies of the present data support previous claims that although there is some evidence that aerobic fitness has a beneficial effect on blood lipid levels, the evidence remains ambiguous.

5.5 PRINCIPAL COMPONENT FACTOR ANALYSIS

As stated previously, it was evident that many pupils exhibited more than one CHD risk factor. Principal component factor analysis (PCA) enabled the author to identify which risk factors appeared to form a group or cluster, and was considered a useful adjunct to other statistical procedures used in this study. Although the inclusion of PCA as an

analytical tool in paediatric epidemiological studies is not common, it has been used previously (Twisk *et al.* 1999).

The clustering of blood lipid and lipoprotein variables was evident (Tables 4.19 and 4.20). This finding supported the interrelationships observed in the correlation coefficient matrix (Appendix 8). As expected, homocyst(e)ine, folate and vitamin B_{12} tended to group together. The addition of Lp(a) in Component 3 (Table 4.19) was surprising but could be dismissed with its relatively low loading (0.32). However, the relationship was again highlighted in Component 4 (Table 4.19), suggesting that more research is needed into the association between Lp(a) and vitamin B_{12} levels.

There can be little doubt that obesity measures tend to group together (Table 4.17) and the evidence gathered during PCA supports observations in the correlation coefficient matrix. As anticipated, the cluster of risk factors in Component 1 reveal how blood pressure levels correlate in the same direction as body fat levels, whilst aerobic fitness correlates in the opposite direction. The inclusion of folate in Component 1 (Table 4.20) was surprising and could not be explained in the present study. Moreover, despite its statistical significance, there might be no physiological relevance to this finding. The clustering of total and saturated fat (Tables 4.18 and 4.20) was not surprising, neither was the observation that systolic and diastolic blood pressure were grouped together. However, the composition of Component 7 (Table 4.20), which included glucose, fibrinogen and WHR was unexpected. Although statistically significant, this finding might not be physiologically relevant and needs further investigation.

5.6 MULTIPLE REGRESSION ANALYSIS OF CORONARY HEART DISEASE RISK FACTORS

The multiple regression analysis in this study revealed that body mass index was the strongest predictor of increased blood pressure. Although BMI was significant in explaining the variation in BP levels (Tables 4.21-4.24), the author is mindful of the limitations associated with the BMI measure and recommends that the findings should be treated cautiously. It might be that the schoolchildren were assigned to a certain BMI range because of a low fat/high muscle ratio, and not a high fat/low muscle ratio. Although additional measures of body fatness (WHR and $\sum$ SKF) were not strong determinants of blood pressure levels, significant relationships had been identified between SKF measurements and BP levels in the correlation coefficient matrix (Appendix 8), whilst PCA similarly revealed clustering of these variables. The significant accumulative effect of all the body fat to BP relations implied that overweight young people should be targeted for a CHD risk reduction intervention.

The impact of fat distribution on blood pressure has been recognized (Lapidus *et al.* 1984; Gillum, 1987; Shear *et al.* 1987; Ledoux *et al.* 1997; Katzmarzyk, Malina, Song *et al.* 1999) although for children and adolescents it is still a matter of controversy (Brambilla, Manzoni, Sironi *et al.* 1994; Moussa *et al.* 1994). When central (subscapular and suprailiac skinfold sites) and peripheral (triceps and biceps skinfold sites) adiposity measures were considered independently, central fat was the primary mediator of systolic blood pressure (Table 4.25), whilst peripheral fat had the greatest effect on diastolic blood pressure (Table 4.28). Nevertheless, the strength of these associations could be deemed small based on R^2 (adj). The findings here were in accord with previous reports on children and adolescents, and concurred with the role of fat distribution in adult chronic disease (Stallones, Mueller and Christensen, 1982; Gillum,

1987; de Visser *et al.* 1994; Daniels, Morrison, Sprecher *et al.* 1999; Maffeis, Pietrobelli, Grezzani *et al.* 2001; Lurbe *et al.* 2001). Moreover, the successful identification of adiposity indices associated with BP in young people could facilitate the improved identification of those individuals at risk of hypertension. The present study did not support previous findings that sex affected the relationship between BP and central fat in young people (He *et al.* 2002). Waist to hip ratio has also been used as an index of fat distribution (Gillum, 1987; Moussa *et al.* 1994) and since the present study did not identify a significant contribution from this measure, the present findings regarding the influence of fat distribution on BP levels could be deemed inconclusive. The effect of fat distribution on blood pressure in young people is still a matter of controversy and a more accurate index of fat distribution is necessary if its true impact on BP is to be ascertained (Moussa *et al.* 1994). For improved clarification of the mechanisms involved in the relationships between BP measures and adiposity distribution, future studies should include the measurement of the visceral fat depot and sexual maturity.

The impact of body fat levels on aerobic fitness is well established. The present finding that the summation of skinfold thickness measured at four sites is the best predictor of 20 MST performance (Tables 4.29 and 4.30), similarly highlighted by PCA, confirms the results of previous studies (Tucker and Bagwell, 1991; Baranowski *et al.* 1992; Bar-Or and Baranowski, 1994; Moussa *et al.* 1994; Hager *et al.* 1995;Twisk *et al.* 1997; Bouziotas *et al.* 2001).

Several researchers have dismissed the relevance of BMI as an obesity index, however, in the present study, both the summation of skinfold thickness measured at four sites, and waist to hip ratio, were strong predictors of BMI (Table 4.31). This finding revealed

close and significant interrelationships between the obesity measures and supported the value of BMI in epidemiological studies. As expected, BMI (positive relationship) and 20MST performance (inverse relationship) were significant in explaining the variation in the summation of skinfold thickness measured at four sites (Table 4.32). However, triglyceride concentration was also a strong predictor of body fat and supported the earlier observation that there was a strong interrelationship between these variables, confirming its importance as a CHD risk factor

The interrelationships between haematological variables were expected and had been revealed in both the correlation coefficient matrix and PCA. With the exception of total cholesterol (for which it was the second best predictor), TC:HDL-C explained the largest proportion of variation in all blood lipids (Tables 4.35 through 4.42). The next best predictor for HDL-C was WHR. The negative significant association between HDL-C and WHR supported previous evidence that higher HDL-C levels are observed in leaner individuals (Epstein, Kuller, Wing *et al.* 1989; National Institute of Health, 1993; Dwyer and Gibbons, 1994; Dietz, 1995; Twisk *et al.* 1995; Wedderkopp, 2002). The ratio of total cholesterol to high density lipoprotein cholesterol (TC: HDL-C) was the primary mediator of low-density lipoprotein. The interrelationships amongst lipids and lipoproteins is unsurprising. In children, LDL-C accounts for 40-60% of TC (Berenson and Epstein, 1983), and whilst the relationship between LDL-C and HDL-C is not fully understood, the reverse cholesterol transport mechanism is postulated as a possible theory (Von Duvillard, 1997) (Appendix 1). The second most important predictor of LDL-C was lipoprotein(a). The significant contribution made by Lp(a) to LDL-C supported previous claims regarding the strength of association between these haematological variables (Maher *et al.* 1995; Genzel-Boroviczény *et al.* 1997; Mackinnon and Hubinger, 1999; Chu *et al.* 2000, Sveger *et al.* 2000). The summation of

skinfold thickness measured at four sites was the second strongest predictor of triglyceride concentration. The positive and significant association between body fat and TG confirmed the findings of others (National Institute of Health, 1993; Dwyer and Gibbons, 1994; Harjai, 1999; Sothern, Despinasse, Brown *et al.* 2000; Wedderkopp, 2002). Surprisingly, vitamin B_{12} was the most important factor in Lp(a) (Table 4.43 through 4.44), and as levels of B_{12} increased, so too did Lp(a) concentration. This finding was very interesting and had not, to the author's knowledge, been observed in any other study. Humans obtain vitamin B_{12} exclusively from animal dietary sources such as meat, milk, and eggs (Brewster, 1989). Although ω-3 fatty acids have had some success in lowering Lp(a) levels (Scanu and Scandiani, 1991), the significant lowering of Lp(a) through low-fat diets has proven unsuccessful (Beil, Terres, Orgass *et al.* 1991). Hence the author could not confirm a link between a high saturated fat diet, which could lead to concomitant increases in B_{12}, and increased levels of Lp(a). Despite its statistical significance in this study, ultimately, B_{12} was only responsible for 5.3% of the variation in Lp(a) and it is conceivable that this finding was not physiologically significant. Nevertheless, PCA had similarly confirmed a relation between these parameters adding to the relevance of this finding.

The strongest predictor of fibrinogen concentration was body mass index (Tables 4.45 and 4.46). This finding supported fibrinogen's significant relationship with obesity levels reported in the correlation coefficient matrix and PCA. Although methods for lowering fibrinogen levels have not been confirmed, the indirect treatment of elevated levels via diet management and exercise could be promising.

In the present study, the main contributors to homocyst(e)ine concentration were vitamins B_{12} and folate (Tables 4.47 and 4.48). These findings were expected since the

associations between homocyst(e)ine and vitamins B_{12} and folate are well-known (Malinow *et al.* 1990; Stampfer *et al.* 1992; Clarke, 1998; Welch and Lascalzo, 1998; Eikelboom *et al.*1999; Bates *et al.* 2002) and had been confirmed in other analytical procedures in this study. If reducing Hcy levels decreases the risk of cardiovascular morbidity and mortality, the present findings supported the positive contribution that vitamins B_{12} and folate could make to Hcy concentration.

In conclusion, this study of Welsh schoolchildren aged 12 to 13 years of differing socio-economic status, reveals a high incidence of CHD risk factors in young people. These risk factors might be related to lifestyle or genetic factors. Moreover, it also gives evidence of the development of multiple risk factors from a young age. Previous research had suggested that risk factors were more prevalent in young people of low SES (Gliksman *et al.* 1995) however, this was not confirmed in the present study. The data here indicate that young individuals exhibit risk factors regardless of socio-economic background. However, it is accepted that SES differences might become more apparent in later life as environmental factors become more influential. Although physical inactivity and overweight are more prevalent in girls, CHD risk factors are present in both sexes. The current study also confirms that recently established risk factors are present from a young age.

If the findings of the present study are to be heeded, the primary prevention of CHD needs to be directed towards young people irrespective of socio-economic derivation. Clearly, young people in whom multiple risk factors are already present need to be targeted with intervention studies, as do female adolescents. Health education, primarily

the responsibility of schools and parents, needs to be directed towards improving diets and increasing the amount of activity undertaken by children and adolescents.

The results from this study contribute to the knowledge and understanding of CHD risk factors in young people. In particular, the information on recently established risk factors provides welcome benchmark data for future research. These findings should act as a caveat against the dangers of ignoring evidence that the origins of CHD have often begun in youth.

CHAPTER 6

6.0 REALISATION OF AIMS

Examination of the null hypothesis in the present study required the realisation of four experimental objectives presented at the end of Chapter 2. These were:

- To compare coronary heart disease risk factors in 12 to 13 year old boys and girls of differing socio-economic status.

- To compare coronary heart disease risk factors of 12 to 13 year old boys and girls.

- To establish the nature and extent of 'clustering' of coronary heart disease risk factors in 12 to 13 year old boys and girls.

- To identify any relationships between coronary heart disease risk factors in 12 to 13 year old boys and girls.

6.1 TESTING THE NULL HYPOTHESES (H_0)

The following section considers the four null hypotheses that were proposed at the outset of this study:

Null Hypothesis (1)

H_0: *There is no difference in the coronary heart disease risk factors of 12 to 13 year old boys and girls of differing socio-economic status.*

For all coronary heart disease risk factors, relatively few significant differences ($P \leq 0.05$) were identified. Overall, there is sufficient evidence to accept the null hypothesis.

Null Hypothesis (2)

H_0: *There is no difference in the coronary heart disease risk factors of 12 to 13 year old boys and girls.*

For physical and physiological variables, several significant differences ($P \leq 0.05$) were identified between boys and girls. Overall, there is sufficient evidence to reject the null hypothesis.

For dietary and haematological variables, relatively few significant differences ($P \leq 0.05$) were identified between boys and girls. Overall, there is sufficient evidence to accept the null hypothesis.

Null Hypothesis (3)

H_0: There is no evidence of the 'clustering' of coronary heart disease risk factors in 12 to 13 year old boys and girls.

Since 66.6% of schoolchildren exhibited three or more coronary heart disease risk factors, overall, there is sufficient evidence to reject the null hypothesis.

Null Hypothesis (4)

H_0: There is no relationship between coronary heart disease risk factors in 12 to 13 year old boys and girls.

Several significant relationships ($P \leq 0.05$) were identified between coronary heart disease risk factors, and overall, there is sufficient evidence to reject the null hypothesis.

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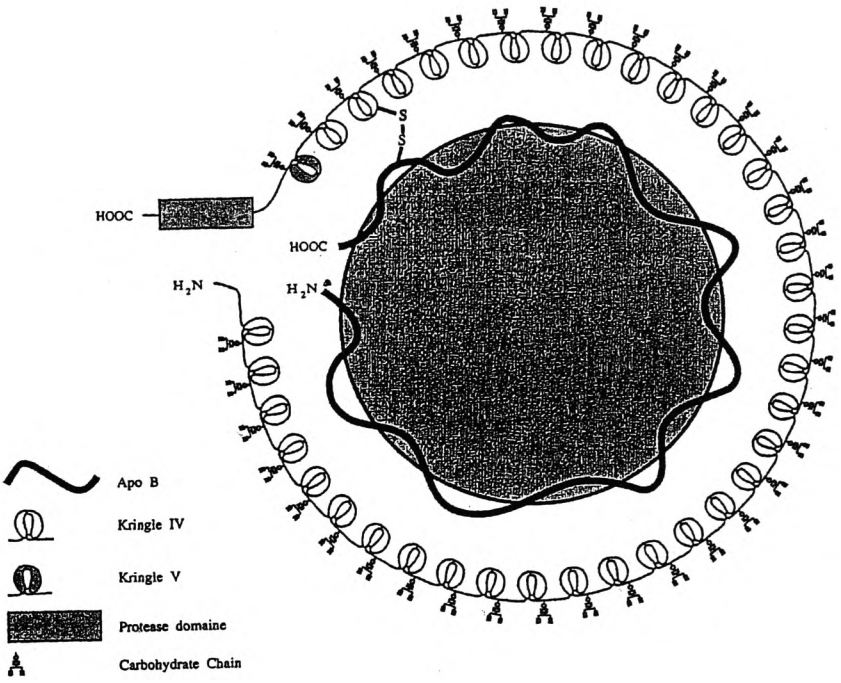
APPENDIX 1

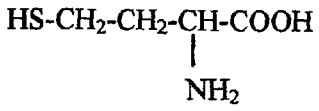
Schematic model of Lp(a)



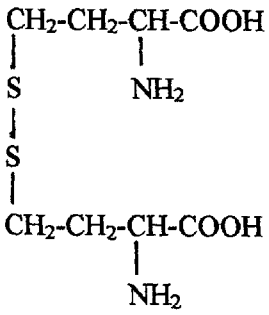
The core LDL particle is attached via a disulfide bridge to the highly polymorphic glycoprotein apo(a). Apo(a) consists of an inactive protease domain, a kringle V domain, and a varying number of kringle IV domains.

From: Utermann, G. (1989) The mysteries of lipoprotein(a). *Science*. Vol. 246, pp 904-910.

Molecular species of homocysteine

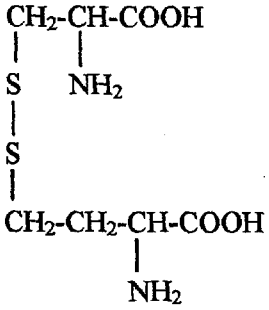


Homocysteine



Homocysteine-homocysteine disulfide

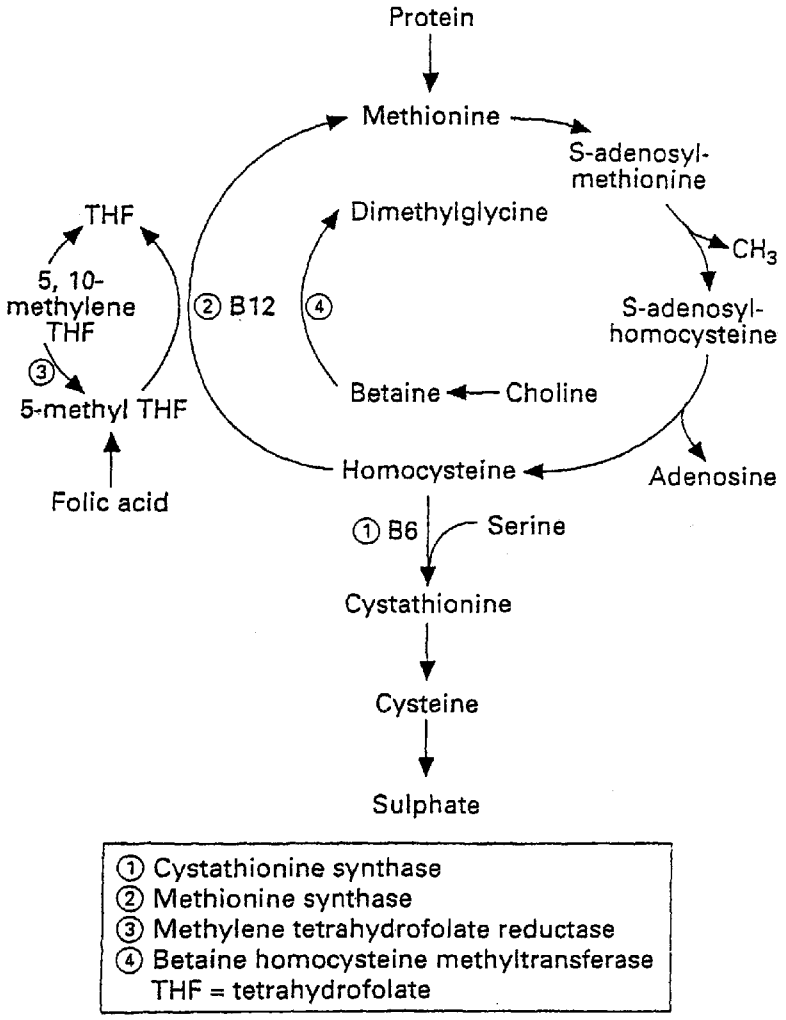
(Homocystine)



Cysteine-homocysteine disulfide

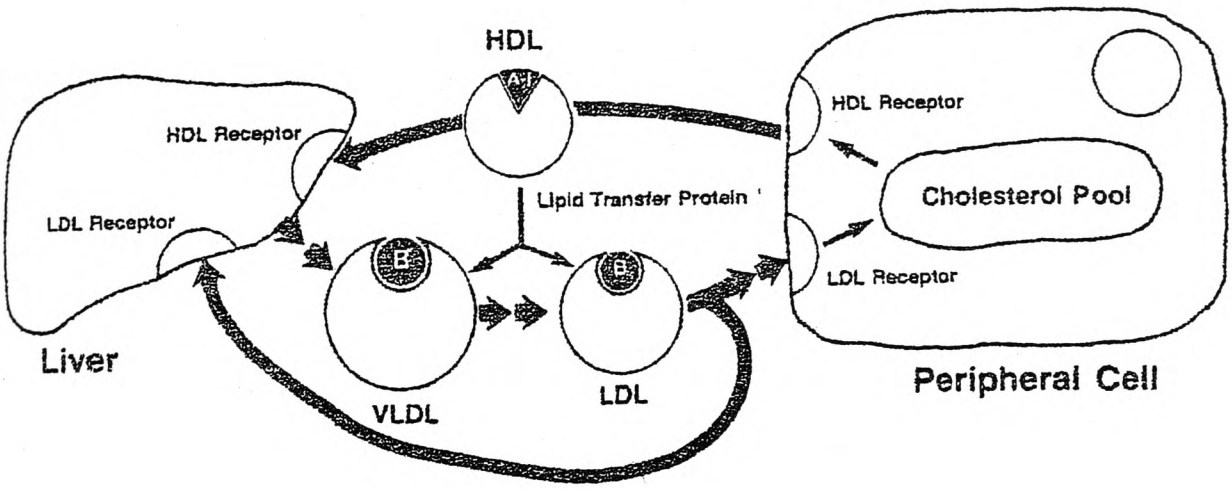
From: Malinow, M.R., Bostom, A.G., Krauss, R.M. (1999) Homocyst(e)ine, diet and cardiovascular diseases. A statement for healthcare professionals from the nutrition committee. American Heart Association. *Circulation*. Vol. 99, pp179.

Transsulphuration and remethylation of homocyst(e)ine



Montalescot, G. (1996) Genes, green and homocysteine. *Heart.* Vol. 76, pp 103-104.

Schematic overview of reverse cholesterol transport



VLDL secreted by the liver is ultimately converted to LDL, the major cholesterol carrying lipoproteins in plasma. LDL interact with the high-affinity LDL receptors on the liver and peripheral cells. It has been proposed that the excess cholesterol in peripheral cells is removed and transported to the liver by HDL. HDL facilitate the removal of cholesterol from the cell by interaction with the putative HDL receptor. Cholesterol within HDL may be directly transported back to the liver, where HDL interact with the hepatic HDL receptor, or the cholesterol may be exchanged into VLDLD or LDL and transported back to the liver via the apo B containing lipoproteins. From: Brewer, H.B., Gregg, R.E., Hoeg, J.M., Fojo, S.S. (1988) Apolipoproteins and lipoproteins in human plasma: an overview. *Clinical Chemistry.* 34/8 (B), B6.

APPENDIX 2

AROLWG CADW'N IACH A HEINI PLANT YSGOL CYMRU

HEALTH AND FITNESS SURVEY OF WELSH SCHOOLCHILDREN

I'w chwblhau gan bennaeth yr ysgol – Diolch yn fawr
For completion by the head of the school - Many thanks

1. **Enw'r ysgol:**
Name of school

2. **Cyfeiriad yr ysgol:**
Address of school

3. **Math o ysgol:**
Type of School

4. **Ystod oedran y disgyblion:**
Age range of children

5. **Nifer y disgyblion:**
Number of children

6. **Nifer y disgyblion ym mlwyddyn 8:** Merched= *Girls* Bechgyn= *Boys*
Number of pupils in year 8

Yng nghwestiynau 7 a 8 rhowch dic yn y bylchau perthnasol. Rhaid i'r canrannau ddod i gyfanswm o 100%.

7.

Mathau o ardaloedd y daw'r *disgyblion ohonynt* ***Types of areas from which pupils are drawn***	Canrannau yn fras Approximate percentages				
	100%	75%	50%	25%	0%
Gwledig *Rural*					
Tref(i) bach *Small town(s)*					
Ardaloedd allanol tref/dinas fawr *Outer areas of large town/city*					
Ardaloedd mewnol tref/dinas fawr *Inner areas of a large town/city*					

8.

Ffyniant yr ardal breswyl y daw'r disgyblion ohonynt *Prosperity of the residential Area*	Canrannau yn fras Approximate percentages				
	100%	75%	50%	25%	0%
Cymharol ffyniannus *Relatively prosperous*					
Heb fod yn ffyniannus nac o dan anfantais economaidd *Neither prosperous nor economically disadvantaged*					
Ardal o dan anfantais economaidd *Economically disadvantaged Area*					

9.

Natur y disgyblion a dderbynir yn fras ***Approximate nature of the intake***	
Breintiedig *Advantaged*	
Difreintiedig *Disadvantaged*	
Heb fod yn freintiedig nac yn ddifreintiedig *Neither advantaged nor disadvantaged*	
Grwpiau breintiedig a difreintiedig amlwg iawn *Both sizeable advantaged and disadvantaged groups*	

10.

Gallu'r disgyblion a dderbynnir yn fras ***Approximate ability of the intake***	
Ystod gallu lawn *Full range of ability*	
Llawer o ddisgyblion galluog ac ychydig o ddisgyblion llai galluog *Many able and few less able pupils*	
Llawer o ddisgyblion llai galluog ac ychydig o ddisgyblion galluog *Many less able and few able pupils*	

11.

Canran y disgyblion yn fras sydd wedi'u cofrestru fel rhai a hawl i gael prydau bwyd ysgol am ddim *Approximate percentage of pupils registered as being entitled to receive free school meals*	
Canran disgyblion BL 8 sydd wedi'u cofrestru fel rhai a hawl i gael prydau bwyd ysgol am ddim *Approximate percentage of Yr 8 pupils registered as being entitled to receive free school meals*	

APPENDIX 3

HEALTH AND FITNESS SURVEY OF WELSH SCHOOLCHILDREN

A PROJECT UNDERTAKEN BY THE UNIVERSITY OF GLAMORGAN

Dear parent or guardian,

A team from the University of Glamorgan, Pontypridd, is undertaking a national survey of the health and fitness levels of Welsh schoolchildren, a project that has been approved by the University of Glamorgan Medical Ethical Committee. The aim of the project is to help schools educate children in healthy living.

A number of health problems related to coronary heart disease originate during childhood, and many of these problems e.g. high blood pressure, high cholesterol and obesity, are amenable to change. It would greatly benefit the present and future health status of Welsh children if we were able to propose preventive strategies that could be undertaken during school time. Health-related exercise units within the Physical Education programme would be ideally suited to this purpose.

For this to happen we need to conduct a series of health and fitness measurements on schoolchildren. Ysgol Gyfun X is one of four schools randomly selected to participate in this important study. Since Year 8 pupils are needed for this survey we have enclosed for your attention, a consent form detailing the intended tests, as well as queries regarding your child's ability to perform the tests safely. All measurements will be carried out in a safe environment and by qualified personnel. Blood sampling will be carried out by a GP and qualified phlebotomist (specialist in taking blood), and will involve taking a small sample of blood once only.

We sincerely hope that you will allow your child to participate in this survey, but please ensure that they too are happy to be included. The testing promises to be enjoyable and educational for each individual pupil.

Yours faithfully,

Non Eleri Thomas MA (Senior Lecturer at UWIC/Independent Inspector of Secondary Schools)
Professor Bruce Davies (Head of Applied Sciences at University of Glamorgan)
Dr Julien Baker (Senior Lecturer at University of Glamorgan)

PLEASE RETURN THE FOLLOWING CONSENT FORM TO THE PHYSICAL EDUCATION DEPARTMENT BY MONDAY, 15TH OCTOBER.

For any further information please contact Non Eleri Thomas on 07980519279 or nevans@uwic.ac.uk

HEALTH AND FITNESS SURVEY OF WELSH SCHOOLCHILDREN

HEALTH AND FITNESS CONSENT FORM

To be completed by parent or guardian. ***CONFIDENTIAL***

Pupil's name:

Pupil's date of birth:

Pupil's school:

Please answer the following questions if you agree to your child taking part in the survey.
For each question **circle** the appropriate answer.

1. Has your child ever suffered from any illness or disease that may affect his or her ability to take part in exercise?

YES / NO

If "YES", please give details.

2. Has your child ever complained of chest pain, wheeziness, headaches or dizziness during or after exercise?

YES / NO

If "YES", please give details.

3. Are you aware of any complaint (e.g. joint soreness) which may prevent your child taking part in normal exercise?

YES / NO

If "YES", please give details.

4. Is your child receiving any medication or medical treatment at present?

YES / NO

If "YES", please give details

5 Has your child ever been in hospital?

YES / NO

If "YES", please give details.

6. Is your child recovering from a viral complaint (such as 'flu') at present?

YES / NO

If "YES", please give details.

7. Has any member of your child's immediate family been treated for, or suspected to have had, any of these conditions? Please identify their relationship to your child (father, mother, grandparent etc).

Tick as appropriate.

a) heart disease
b) diabetes
c) stroke
d) high blood pressure
e) high cholesterol

Please **circle** as appropriate.

I **AGREE / DO NOT** agree to my child .....(name) taking part in the health and fitness survey, the nature of which has been explained to me in this letter.
I understand that my child will participate in the following tests and that all test results will remain confidential at the University of Glamorgan.

Weight and height
Skinfold thickness (body fat)
20metre shuttle run (Bleep test) (fitness)
Blood pressure
Blood analysis (cholesterol)
Activity Questionnaire
Dietary Questionnaire

Signature.....(parent)

Signature.....(pupil)

PLEASE RETURN THIS CONSENT FORM TO THE PHYSICAL EDUCATION DEPARTMENT BY MONDAY, 15TH OCTOBER. TESTING CANNOT BEGIN UNTIL ALL FORMS HAVE BEEN RETURNED.

PLEASE ENSURE THAT YOU HAVE ANSWERED ALL QUESTIONS.

Thank you.

APPENDIX 4

LIFESTYLE QUESTIONNAIRE

Girl or Boy:

School:

Have you ever smoked cigarettes? YES/NO

Do you smoke presently? YES/NO

Cigarettes ……..a day

At what age did you start smoking? ……..years

Have you ever drunk alcohol? YES/NO

Do you drink presently? YES/NO

At what age did you start drinking? ……..years

APPENDIX 5

Questionnaire

01842

1 Surname

First names

Mr/Mrs/Miss/Ms/other

2 Address

Post Code

3 Age Years

4 Sex Male Female

5 Weight

Height

6 How often do you exercise?

- Less than once a week
- Once a week
- 2 to 3 times a week
- More than 3 times a week

7 What type of exercise do you usually take part in?

8 Which of the following do you eat regularly? (at least once a week)

Meat	Fruit
Fish	Vegetables
Eggs	Cereals
Dairy produce	Bread

9 When you eat meat, do you trim the fat off before eating? (If you are vegetarian please answer "yes")

Yes No

10 When you eat meat/poultry, do you remove the skin before eating? (If you are vegetarian please answer "yes")

11 When you have omelettes or scrambled eggs how many eggs do you use?

1 egg 2 eggs More than 2

12 What type of cheese do you eat most often?

- Full fat (stilton, gorgonzola, brie etc.)
- Low fat (cottage cheese, edam etc)
- Cheddar type (cheddar, cheshire etc)
- Soft, cream or processed cheese

13 What type of milk do you normally use?

- Cow's full cream
- Cow's semi-skimmed
- Cow's skimmed
- Condensed milk

Other type

14 Do you normally eat?

- Butter
- Margarine
- Low fat spread
- Very low fat spread
- No spread

15 How is your food normally cooked?

- Grilled
- Fried
- Roasted
- Boiled/steamed
- Microwaved

16 Which of the following is your food normally cooked in?

Lard/dripping	Butter
Margarine	No fat used

Vegetable oil, specify type

17 Is the bread you normally eat?

White	Brown
Wholemeal/granary	

Other type

18 What type of salad dressing would you normally use?

Oil/vinaigrette	Mayonnaise
Salad cream	
Low calorie salad cream/ vinaigrette/mayonnaise	

Other type

19 Do you normally drink?

Black tea	White tea
Black coffee	White coffee

Other drinks

Number of spoons of sugar per cup

20 Which of the following do you normally drink?

- Fruit squashes
- Sugar free/low sugar squash
- Fruit juices (fresh/long life)
- Carbonated drinks e.g. lemonade, cola etc.
- Low calorie carbonated drinks or mixers

21 Do you normally add salt to your food?

- In cooking
- At the table
- Do not add salt
- Use salt substitute

22 How often do you eat 'fast'/convenience/ take-away foods either in a restaurant or at home?

- 0-2 times per week
- 3-5 times per week
- More than 5 times per week

23 Do you regularly take any dietary supplements?

Multi-vitamins & minerals, brand

Others

24 Do you suffer from any illness/condition for which you are currently receiving treatment or requires you to modify your diet?

Please specify

25 Are you currently taking any medicines or tablets?

Personal food and drink record

Meals	*Monday*	*Tuesday*	*Wednesday*	*Thursday*	*Friday*	*Saturday*	*Sunday*
Breakfast							
Lunch							
Dinner							
Snacks							
Drinks							

APPENDIX 6

HOLIADUR YMARFER CORFF

Prif bwrpas yr holiadur yw i ddarganfod faint o ymarfer corff rydych yn tueddu gwneud mewn wythnos. Mae'r gwybodaeth yn hollol gyfrinachol ond mae'n holl bwysig eich bod yn ateb yn onest ac yn gywir.

Diolch yn fawr am eich cydweithrediad.

ENW LLAWN:

DOSBARTH:

Ticiwch (√)un ateb:

- Ysgol Bro Myrddin ( )
- Ysgol Gyfun Rhydfelen ( )

- Bachgen ( )
- Merch ( )

Ticiwch (√)UN ateb yn unig.

1. Sut yr ydych fel arfer yn teithio i'r ysgol?
1. Bws, car, tren ayb.
2. Beic
3.Cerdded

How do you normally travel to school?

1. Bus, car, train etc
2. Bicycle
3. Walk

2. Sut yr ydych fel arfer yn teithio adref o'r ysgol?
1. Bws, car, tren ayb
2. Beic
3. Cerdded

How do you normally travel home from school?

1. Bus, car, train etc
2. Bicycle
3. Walk

3. Tua faint o amser mae'n cymryd i gyrraedd ysgol ar ol gadael eich cartref?
1. **Llai na 5 munud**
2. **5-15 munud**
3. **15-30 munud**
4. **30 munud – 1 awr**
5. **Mwy na 1 awr**

How long does it normally take you to travel to school after leaving your home?
1. Less than 5 minutes
2. 5-15 minutes
3. 15-30 minutes
4. 30 minutes – 1hour
5. More than 1 hour

4. Pa mor aml yr ydych yn byr eich anadl yn ystod eich gwersi addysg gorfforol a gemau?
1. Dwi ddim yn cymryd rhan yn y gwersi
2. Bron byth
3. Weithiau
4. Eitha' aml
5. Trwy'r amser

During your PE and games lessons, how often do you get out of breath?
1. I don't do PE or games
2. Hardly ever
3. Occasionally
4. Quite often
5. Always

5. Beth yr ydych yn tueddu gwneud yn ystod amser egwyl?
1. Eistedd lawr (sgwrsio, darllen, gwaith ysgol)
2. Aros yn yr unfan neu cerdded
3. Rhedeg o gwmpas yn chwarae gemau

What do you normally do at morning break?
1. Sit down (talking, reading, doing schoolwork)
2. Stand or walk around
3. Run around playing games

6. Beth yr ydych yn tueddu gwneud yn ystod yr awr ginio?
1. Eistedd i lawr (sgwrsio, darllen, gwaith ysgol)
2. Aros yn yr unfan neu cerdded
3. Rhedeg o gwmpas yn chwarae gemau

What do you normally do at lunch time?
1. Sit down (talking, reading, doing schoolwork)
2. Stand or walk around
3. Run around playing games

7. Pa mor aml yr ydych yn aros ar ol ysgol i gnweud chwaraeon?
1. Byth
2. 1 - 2 waith yr wythnos
3. 3 –4 gwaith yr wythnos
4. 5 gwaith yr wythnos

How many days per week do you stay behind at school for sports?
1. None
2. Once or twice a week
3. 3-4 times a week
4. 5 times a week

8. Sawl noson yr wythnos yr ydych yn cymryd rhan mewn chwaraeon neu ymarfer corff?
1. Dim
2. 1-2 waith yr wythnos
3. 3-5 gwaith yr wythnos
4. 6 neu 7 gwaith yr wythnos

How many evenings per week do you take part in sports or other physical activities?
1. None
2. Once or twice a week
3. 3-5 times a week
4. 6 or 7 times a week

9. Ar y foment, a ydych chi'n cymryd rhan mewn chwaraeon neu urhyw ymarfer corff arall dros y penwythnos?
1. Ydw
2. Nac ydw

At the moment, do you take part in sports or other physical activities at the weekend?
1. Yes
2. No

10. Pa mor egniol ydych chi yn ystod y gwyliau i gymharu ag yn ystod tymor ysgol?
1.Llai egniol
2.Tua'r un peth
3. Mwy egniol

During school holidays, how active are you, compared to during term time?
1. Less active
2. About the same
3. More active

Ticiwch (√) **UN** ateb yn unig:

12. Ydy atebion cwestiwn 11. yn adlewyrchu eich patrwm arferol o ymarfer?
1. Rwy'n tueddu bod yn llai egniol
2. Tua'r un peth
3. Rwy'n tueddu bod yn fwy egniol

Is your answer to the last question typical of your exercise pattern at this time of year?
1. I am usually less active
2. About the same
3. I am usually more active

13. Yn gyffredinol, a ydych yn mwynhau ymarfer corff ?

1. **Ydw**
2. **Nac ydw**

Os 'YDW' esboniwch:

....

Os 'NAC YDW' esboniwch:

....

Generally speaking, do you enjoy physical activity?

1. Yes

2. No

If 'YES' say why you like it:

... ...

If 'NO' say why you do not like it:

... ...

14. Yn gyffredinol, a ydych yn mwynhau gwersi addysg gorfforol a chwaraeon?

1. **Ydw**
2. **Nac ydw**

Os 'YDW' esboniwch

....

Os 'NAC YDW' esboniwch

....

Generally speaking, do you enjoy Physical Education and games lessons?

1.Yes

2.No

If 'YES' say why you like it:

... ...

If 'NO' say why you do not like it:

... ...

15. Pa UN o'r canlynol sydd yn eich disgrifio orau?

1. **Mae fy amser hamdden yn tueddu i gael ei lenwi gan gweithgareddau fel gwylio teledu, darllen, siarad a ffrindiau ayb, yn hytrach na phethau egniol**

NEU

2. **Weithiau (1 neu 2 waith yr wythnos) byddaf yn cymryd rhan mewn gweithgaredd egniol (ee nofio, beicio, loncian) yn ystod fy amser hamdden**

NEU

3. **Byddaf yn gwneud gweithgareddau egniol yn eithaf aml (4-6 gwaith yr wythnos) yn ystod fy amser hamdden**

NEU

4. **Byddaf yn gwneud rhywbeth egniol yn aml iawn yn ystod fy amser hamdden (7 neu mwy yr wythnos)**

Which ONE of the following statements describes you best?

1. *All or most of my free time is spent doing things that involve little physical effort (eg watching TV, reading, talking to friends)*

OR

2. *I occasionally (once or twice a week) do things in my free time that involve physical effort (eg swimming, cycling, jogging)*

OR

3. *I quite often (4-6 times a week) do things in my free time that involve some physical effort*

OR

4. *I very often (7 or more times a week) do things in my free time that involve some physical effort*

16. Isod gwelir restr o bobl, rydym am gwybod os ydynt fel arfer yn gwneud ymarfer corff.

Ticiwch (√) os ydynt yn ymarfer o leiaf dwy waith yr wythnos?

1. **Tad**
2. **Mam**
3. **Brawd hyn**
4. **Chwaer hyn**
5. **Ffrind gorau**

Below is a list of people, we want to know whether they normally take regular exercise.

Tick (√) alongside if that person exercises at least twice a week.

1. Father
2. Mother
3. Elder sister
4. Elder brother
5. Best friend

17. Yn ystod y 7 **diwrnod ddiwethaf** faint o weithgareddau sydd wedi eich gwneud yn **fyr eich anadl**?

Llenwch y tabl isod. Os na wnaethoch unrhyw ymarfer ewch ymlaen i'r rhan nesaf.

	Pa mor aml? Sawl gwaith yn y 7 diwrnod ddiwethaf?	Pa mor hir? Beth oedd hyd y weithgaredd? Awr munudau	Pa mor anodd? A oeddech yn fyr eich anadl? Byth 1 Ychydig 2 neu Llawer 3
Cerdded tuag at ag i ffwrdd o'r ysgol	☐☐	☐ ☐☐	☐
Beicio tuag at ag i ffwrdd o'r ysgol	☐☐	☐ ☐☐	☐
Gweithgareddau amser **egwyl** ee • pel-rwyd • hoci • pel-droed • rygbi • eraill (enwch)	....	....	....
Gweithgareddau awr **ginio** (gan gynnwys clybiau ac ymarferion) ee • pel-rwyd • hoci • pel-droed • rygbi • gymnasteg • eraill (enwch)	....	....	....
Gwersi addysg gorfforol a chwaraeon ee • gymnasteg • gemau • dawns • iechyd a fitrwydd • eraill (enwch)	....	....	....

	Pa mor aml? Sawl gwaith yn y 7 diwrnod ddiwethaf?	Pa mor hir? Beth oedd hyd y weithgaredd? Awr munudau	Pa mor anodd? A oeddech yn fyr eich anadl? Byth **1** Ychydig **2** neu Llawer **3**
Ymarferion a chlybiau **ar ol** ysgol ee • pel-rwyd • hoci • pel-droed • rygbi • gymnasteg • eraill (enwch)	....	....	....
Gemau ysgol (enwch)	....	....	....
Gweithgareddau nos (enwch)	....	....	....
Gweithgareddau penwythnos (enwch)	....	....	....
Gweithgareddau eraill (enwch)	....	....	....

17. We want to know about all the exercise you have taken **that made you get out of breath in the last 7 days**

In the table below, fill in each section as accurately as you can. If you took no exercise in any of the sections, do not write in those boxes, go straight on to the next section.

	How Often? How many times in the last 7 days?	How Long? How many hours and minutes did each session last? Hours Minutes	How Hard? Did you get out of breath? Never 1 A little 2 or A lot 3
Travel to and from school (walking)	☐☐	☐ ☐☐	☐
Travel to and from school (cycling)	.....	.....	.....
Morning break activities eg • netball • hockey • football • rugby • others (name)	....	....	....
Lunch time activities (including clubs and practices) eg • netball • hockey • football • rugby • gymnastics • others (name)	....	....	....
PE and games lessons eg • gymnastics • games • dance • health and fitness • others (name)	....	....	....

	How Often? How many times in the last 7 days?	How Long? How many hours and minutes did each session last? Hours Minutes	How Hard? Did you get out of breath? Never **1** A little **2** or A lot **3**
After school clubs and practices eg • netball • hockey • football • rugby • gymnastics • others (name) …… …… …… ……	…… …… …… …… …… …… …… …… ……	…… …… …… …… …… …… …… …… ……	…… …… …… …… …… …… …… …… ……
School matches (name) …… …… …… …… ……	…… …… …… …… ……	…… …… …… …… ……	…… …… …… …… ……
Evening activities (name) …… …… …… …… ……	…… …… …… …… ……	…… …… …… …… ……	…… …… …… …… ……
Weekend activities (name) …… …… …… ……	…… …… …… ……	…… …… …… ……	…… …… …… ……
Any other activities (name) …… …… ……	…… …… ……	…… …… ……	…… …… ……

APPENDIX 7

PILOT STUDY

The raw data for aerobic fitness, blood pressure and anthropometric variables test, re-test were entered into a computerised statistical package (Minitab V.12, 1998). All variables were identified as being homescedastic ($r < \pm 0.1$) therefore the 95% LoA was resumed. The P values for all variables, as obtained from the Anderson-Darling normality test, was greater than 0.05 ($P > 0.05$), hence the data was accepted as normally distributed.

20 MST

The correlation coefficient of absolute difference and mean: $r = -0.086$

Table 8.1 A paired *t*-test of the 20 MST test, re-test

Variable	N	Mean	St Dev	SE Mean	*T*	*P*
Diff	20	0.00	2.62	0.585	0.00	1.00

Reliability estimates are given as $\bar{x}_{\Delta} \pm (1.96 \times s_{\Delta}) = 0.00 \pm (1.96 \times 2.616) = 0.00 \pm 5.13$. Therefore the 95% LoA for this test lie between –5.13 and + 5.13 (2dp).

SBP

The correlation coefficient of absolute difference and mean: $r = 0.045$

Table 8.2 A paired *t*-test of the SBP test, re-test

Variable	N	Mean	St Dev	SE Mean	*T*	*P*
Diff	20	-0.0150	2.323	0.519	-0.29	0.776

Reliability estimates are given as $\bar{x}_{\Delta} \pm (1.96 \times s_{\Delta}) = -0.0150 \pm (1.96 \times 2.323) =$ -0.150 ± 4.55. Therefore the 95% LoA for this test lie between –4.70 and + 4.39 (2dp).

DBP

The correlation coefficient of absolute difference and mean: $r = 0.017$

Table 8.3 A paired *t*-test of the DBP test, re-test

Variable	N	Mean	St Dev	SE Mean	*T*	*P*
Diff	20	0.050	2.585	0.578	0.09	0.932

Reliability estimates are given as $\bar{x}_{\Delta} \pm (1.96 \times s_{\Delta}) = 0.050 \pm (1.96 \times 2.585) =$ 0.050 ± 5.07. Therefore the 95% LoA for this test lie between –5.01 and + 5.11 (2dp).

Biceps skinfold

The correlation coefficient of absolute difference and mean: $r = -0.084$

Table 8.4 A paired *t*-test of the biceps skinfold test, re-test

Variable	N	Mean	St Dev	SE Mean	*T*	*P*
Diff	20	0.0035	0.2866	0.0641	0.05	0.957

Reliability estimates are given as $\bar{x}_{\Delta} \pm (1.96 \times s_{\Delta}) = 0.0035 \pm (1.96 \times 0.2866) =$ 0.0035 ± 0.56. Therefore the 95% LoA for this test lie between –0.59 and + 0.60 (2dp).

Triceps skinfold

The correlation coefficient of absolute difference and mean: $r = 0.089$

Table 8.5 A paired *t*-test of the triceps skinfold test, re-test

Variable	N	Mean	St Dev	SE Mean	*T*	*P*
Diff	20	-0.005	0.2448	0.0547	-0.09	0.928

Reliability estimates are given as $\bar{x}_{\Delta} \pm (1.96 \times s_{\Delta}) = -0.005 \pm (1.96 \times 0.2448) =$ -0.005 ± 0.48. Therefore the 95% LoA for this test lie between –0.55 and + 0.45 (2dp).

Subscapular skinfold

The correlation coefficient of absolute difference and mean: $r = 0.063$

Table 8.6 A paired *t*-test of the subscapular test, re-test

Variable	N	Mean	St Dev	SE Mean	*T*	*P*
Diff	20	0.0025	0.2533	0.0566	0.04	0.965

Reliability estimates are given as $\bar{x}_{\Delta} \pm (1.96 \times s_{\Delta}) = 0.0025 \pm (1.96 \times 0.2533) =$ 0.0025 ± 0.50. Therefore the 95% LoA for this test lie between –0.48 and + 0.53 (2dp).

Suprailiac skinfold

The correlation coefficient of absolute difference and mean: $r = -0.061$

Table 8.7 A paired *t*-test of the suprailiac test, re-test

Variable	N	Mean	St Dev	SE Mean	*T*	*P*
Diff	20	0.0025	0. 335	0.0749	0.03	0.955

Reliability estimates are given as $\bar{x}_{\Delta} \pm (1.96 \times s_{\Delta}) = 0.0025 \pm (1.96 \times 0.335) =$ 0.0025 ± 0.57. Therefore the 95% LoA for this test lie between –0.57 and + 0.57 (2dp).

APPENDIX 8

Table 8.8 Correlation coefficient matrix for all CHD risk factors

	Glu	TC	TG	HDL-C	LDL-C	TC:HDL	Fg	Hcy
TC	0.086 0.243							
TG	-0.034 0.642	0.356 **0.000**						
HDL-C	0.112 0.128	0.295 **0.000**	-0.459 **0.000**					
LDL-C	0.058 0.434	0.924 **0.000**	0.338 **0.000**	-0.016 0.823				
TC:HDL	-0.033 0.651	0.409 **0.000**	0.718 **0.000**	-0.703 **0.000**	0.617 **0.000**			
Fg	-0.133 0.082	-0.011 0.891	-0.009 0.908	-0.146 0.056	0.061 0.429	0.109 0.155		
Hcy	-0.038 0.610	-0.005 0.948	0.014 0.854	-0.015 0.837	-0.004 0.956	-0.011 0.883	0.037 0.640	
Folate	-0.013 0.865	0.019 0.798	-0.011 0.884	0.020 0.792	0.017 0.822	0.007 0.927	0.021 0.782	-0.354 **0.000**
B12	-0.010 0.890	0.040 0.599	-0.024 0.747	0.022 0.771	0.044 0.555	0.001 0.993	0.074 0.338	-0.229 **0.003**
Lp (a)	-0.048 0.515	0.246 **0.001**	-0.019 0.796	0.135 0.067	0.230 **0.002**	0.040 0.590	0.022 0.772	-0.138 0.067
SBP	0.046 0.537	-0.094 0.202	0.067 0.368	-0.188 **0.010**	-0.038 0.611	0.120 0.103	0.048 0.535	0.135 0.072
DBP	-0.047 0.521	-0.013 0.863	0.203 **0.006**	-0.217 **0.003**	0.028 0.705	0.167 **0.023**	0.083 0.279	0.086 0.252
∑ SKF	-0.125 0.088	0.086 0.241	0.405 **0.000**	-0.264 **0.000**	0.107 0.145	0.309 **0.000**	0.212 **0.005**	0.051 0.496
Triceps	-0.175 **0.016**	0.218 **0.003**	0.354 **0.000**	-0.141 **0.054**	0.216 **0.003**	0.289 **0.000**	0.057 0.460	0.090 0.232
Biceps	0.048 0.517	-0.081 0.269	0.292 **0.000**	-0.264 **0.000**	-0.054 0.468	0.204 **0.005**	0.212 **0.005**	-0.029 0.704

Table 8.8 (cont) Correlation coefficient matrix for all CHD risk factors

	Glu	TC	TG	HDL-C	LDL-C	TC:HDL	Fg	Hcy
Supra	-0.118	0.086	0.355	-0.220	0.100	0.263	0.176	0.034
	0.109	0.244	**0.000**	**0.002**	0.174	**0.000**	**0.021**	0.648
Sub	-0.117	0.089	0.407	-0.292	0.125	0.332	0.253	0.067
	0.110	0.228	**0.000**	**0.000**	0.089	**0.000**	**0.001**	0.375
BMI	-0.115	-0.011	0.316	-0.339	0.059	0.310	0.304	0.070
	0.117	0.877	**0.000**	**0.000**	0.426	**0.000**	**0.000**	0.351
WHR	-0.161	-0.043	0.240	-0.225	-0.010	0.158	0.128	0.040
	0.027	0.560	**0.001**	**0.002**	0.893	**0.030**	0.095	0.593
20 MST	0.074	0.008	-0.218	0.150	0.003	-0.146	-0.111	-0.129
	0.316	0.908	**0.003**	**0.041**	0.971	**0.047**	0.149	0.087
Total fat	-0.032	0.119	-0.006	0.115	0.083	-0.040	-0.069	0.142
	0.706	0.153	0.939	0.168	0.321	0.630	0.434	0.098
Sat fat	0.032	0.153	0.012	0.150	0.101	-0.033	-0.096	0.067
	0.705	0.067	0.884	0.072	0.226	0.698	0.273	0.437
Av daily calories	0.098	-0.048	-0.173	0.080	-0.039	-0.128	-0.045	-0.026
	0.239	0.569	**0.038**	0.338	0.638	0.124	0.606	0.763

Cell contents: Correlation
P value

* Bold face type is significant ($P \leq 0.05$)

Table 8.8 (cont) Correlation coefficient matrix for all CHD risk factors

	Folate	B_{12}	Lp(a)	SBP	DBP	∑ SKF	Triceps	Biceps
B_{12}	0.142 0.058							
Lp(a)	0.155 **0.037**	0.242 **0.001**						
SBP	0.083 0.267	-0.123 0.102	-0.020 0.786					
DBP	-0.141 0.058	-0.027 0.724	-0.099 0.183	0.474 **0.000**				
∑ SKF	-0.165 **0.026**	-0.142 0.058	-0.043 0.560	0.359 **0.000**	0.486 **0.000**			
Triceps	-0.143 **0.054**	-0.138 0.065	-0.071 0.339	0.314 **0.000**	0.423 **0.000**	0.842 **0.000**		
Biceps	-0.138 0.064	-0.031 0.682	0.015 0.843	0.195 **0.008**	0.342 **0.000**	0.743 **0.000**	0.365 **0.000**	
Supra	-0.161 **0.030**	-0.167 **0.026**	-0.049 0.504	0.306 **0.000**	0.394 **0.000**	0.930 **0.000**	0.749 **0.000**	0.649 **0.000**
Sub	-0.144 **0.052**	-0.143 0.056	-0.023 0.759	0.347 **0.000**	0.417 **0.000**	0.929 **0.000**	0.736 **0.000**	0.658 **0.000**
BMI	-0.160 **0.031**	-0.169 **0.024**	0.004 0.954	0.436 **0.000**	0.533 **0.000**	0.786 **0.000**	0.603 **0.000**	0.573 **0.000**
WHR	0.032 0.664	-0.094 0.209	-0.007 0.929	0.062 0.405	-0.016 0.827	0.245 **0.001**	0.135 0.065	0.183 **0.012**
20 MST	0.283 **0.000**	0.088 0.243	0.016 0.833	-0.121 0.102	-0.404 **0.000**	-0.619 **0.000**	-0.474 **0.000**	-0.571 **0.000**
Total fat	-0.163 **0.054**	-0.267 **0.002**	-0.149 0.076	-0.027 0.751	-0.053 0.526	0.081 0.334	0.154 0.064	0.024 0.776
Sat fat	-0.089 0.292	-0.169 **0.048**	-0.150 0.074	-0.025 0.763	-0.092 0.277	0.040 0.636	0.120 0.151	0.006 0.940
Av daily calories	0.121 0.153	-0.038 0.662	-0.080 0.343	-0.035 0.674	-0.164 **0.051**	-0.197 **0.018**	-0.172 **0.038**	-0.199 **0.016**

Table 8.8 (cont) Correlation coefficient matrix for all CHD risk factors

	Supra	Sub	BMI	WHR	20MST	Total fat	Sat fat	Av daily calories
Sub	0.857 **0.000**							
BMI	0.668 **0.000**	0.788 **0.000**						
WHR	0.251 **0.001**	0.370 **0.000**	0.275 **0.000**					
20MST	-0.522 **0.000**	-0.573 **0.000**	-0.554 **0.000**	-0.093 0.205				
Total fat	0.083 0.323	0.015 0.857	-0.031 0.715	-0.001 0.987	-0.124 0.136			
Sat fat	0.044 0.601	-0.033 0.690	-0.114 0.173	-0.086 0.304	-0.109 0.191	0.831 **0.000**		
Av daily calories	-0.144 0.083	-0.174 **0.037**	-0.185 **0.026**	0.088 0.290	0.136 0.103	-0.078 0.352	-0.015 0.861	

Cell contents: Correlation
P value

* Bold face type is significant ($P \leq 0.05$)