Relationships between modifiable lifestyle factors and indicators of cardiovascular risk

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Author's Declaration

I declare that the work contained in this thesis is original, and I am the author of this thesis. All the design, analyses and data processing were carried out by myself. I have also personally carried out all the measurements and collected and analysed the data for the observational study. I have done all the work under the supervision of Dr Catherine R Hankey and Professor Mike EJ Lean.

Mehdi Akbartabar Toori

Supervisor's Declaration

I certify that the work reported in this thesis has been performed by Mehdi Akbartabar Toori and that during the period of study he has fulfilled the conditions of the ordinances and regulations governing the degree of Doctor of Philosophy.

List of Contents

List of Tables

List of figures

List of Publications

Papers

Akbartabartoori M, Lean MEJ, Hankey CR (2005). Relationships between cigarette smoking, body size and body shape. *International Journal of Obesity*, 29, 236-243.

Akbartabartoori M, Lean MEJ, Hankey CR (2006). Smoking combined with overweight or obesity markedly elevates cardiovascular risk factors. *European Journal of Cardiovascular prevention and rehabilitation*. In press

Abstracts

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Akbartabartoori M, Lean MEJ, Hankey CR (2004). Relationship between cigarette smoking and body mass index and shape. 13th European congress on Obesity, 26-29th May, Prague. *International Journal of Obesity*, 28 (suppl 1): S57.

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Akbartabartoori M, Lean MEJ, Hankey CR (2005). Relationships between HDL-C and non-HDL-C concentrations with anthropometric measurements and lifestyle factors. 14th European Congress on Obesity, 1-4th June, Athens. Obesity Reviews. 6 (supple1): 104.

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List of Abbreviations

ANOVA	Analysis of Variance
AT-LPL	Adipose Tissue- Lipoprotein Lipase
BMI	Body Mass Index
BAT	Brown Adipose Tissue
CDC	Centres for Disease Control
CI	Confidence Interval
CO	Carbon Monoxide
CRP	C-reactive Protein
CV	Coefficient of variation
CVD	Cardiovascular Disease
CHD	Coronary Heart Disease
DLW	Double Labelled Water
EI/BMR	Energy Intake/ Basal Metabolic Rate
EPIC	European Prospective Investigation into Cancer and Nutrition
FFM	Fat Free Mass
FFQ	Food Frequency Questionnaire
FM	Fat Mass
GHQ	General Health Questionnaire
HC	Hip Circumference
HDL-C	High Density Lipoprotein (cholesterol)
INTERMAP	International Study of Macro- and Micro-nutrients and Blood Pressure
IL-6	Interleukine-6

IPAQ	International Physical Activity Questionnaire
LDL_C	Low Density Lipoprotein (cholesterol)
MONICA	Monitoring Cardiovascular Disease
MRI	Magnetic Resonance Image
MET	Metabolic Equivalent
NHANES III	Third National Health and Nutrition Examination Survey
Non-HDL-C	Non- High Density Lipoprotein (cholesterol)
NRT	Nicotine Replacement Therapy
REE	Resting Energy Expenditure
RMR	Resting Metabolic Rate
SHS	Scottish Health Survey
SPSS	Statistical Package for Social Sciences
TFEQ-R	Tree Factor Eating Questionnaire Revised 18 Items
TNF- α	Tumor Necrosis Factor- α
VLDL	Very Low density Lipoprotein
VO^2	Volume of Oxygen
WC	Waist Circumference
WHO	World Health Organization
WHR	Waist –Hip- Ratio

Summary

The global demographic, socio-economic and technological changes linked with lifestyle modifications are widely considered to be the underlying cause of the increased prevalence of CVD and other non-communicable disease worldwide. Understanding the role of the lifestyle factors in associations with these problems is important for treatment and prevention. The aims of the present thesis were:

1) To evaluate the associations between some lifestyle factors, body weight and shape, and CVD risk factors.

2) To determine the combination association between lifestyle factors and body weight and CVD risk factors.

3) To evaluate the effects of a smoking cessation program on energy balance

To achieve these aims a secondary analysis of Scottish Health Survey (SHS) 1998 and an observational study have been carried out.

The relationships between smoking status and body size and shape have been examined using the SHS data from those aged 16-74 years. After adjustment for some confounding factors, BMI was lower in current smokers and higher in ex-smokers ($p<0.001$) when compared to non-smokers in the survey population as a whole. However, examination of age categories showed no such differences in BMI between current smokers and non-smokers in men aged 16-24 years or women aged below 55 years. In the age band 16-24 years, prevalence of cigarette smoking was highest at 51% (men) and 43% (women) in obese subjects and lowest at 35% (men) and 33% (women) in people with BMI 25-30 kg/m^2. For women current smokers, mean WC and WHR were higher and HC was lower compared to non-smokers ($p<0.001$). In

men only HC was lower in current smokers compared with non-smokers for the entire sample ($p<0.001$). In women smoking was linked to the development of central adiposity. The gender-related central adiposity of men is not further increased by smoking, but a lower HC could point to reduced muscle mass.

The independent and combined associations of smoking, overweight and obesity with CVD risk factors: total cholesterol, HDL-C, non-HDL-C, CRP and fibrinogen were defined. In multivariable analysis BMI and smoking were the most important factors for the CVD risk factors. Smoking was independently associated with higher CRP and fibrinogen concentrations in both sexes, and lower HDL-C and higher non-HDL-C in females ($p<0.001$). Overweight or obesity were independently associated with higher CRP, total cholesterol, non-HDL-C and lower HDL-C in both sexes, and higher fibrinogen in females ($p<0.001$). Overweight or obese current smokers had higher CRP and fibrinogen and lower HDL-C concentrations compared to the reference group of never-smokers with BMI below 25 kg/m^2 ($p<0.001$). Obese current smokers had the highest mean value and OR for the risk factors across the categories, particularly for lower HDL-C (OR= 11) and elevated CRP (OR= 9) ($p<0.001$).

The associations between current recommended physical activity levels (5×30 moderate and 3×20 vigorous) with BMI showed that these levels of activity were associated with lower BMI and risk of obesity. Moderate activity (5×30) in combination with vigorous activity (3×20) was associated with lower central obesity independent of BMI. Vigorous activity alone has more limited value.

Separate and joint associations of physical activity and BMI with CVD risk factors, GHQ12 and predicted CHD risk have been evaluated in another study. Obesity was independently associated with higher OR for elevated cholesterol, CRP, systolic blood pressure, non-HDL-C and lower HDL-C ($p<0.001$), and with greater predicted CHD risk compared to BMI < 25 kg/m^2. Regular physical activity reduced the OR of lower HDL-C and higher CRP, and average predicted 10 year CHD risk in obese subjects, but did not eliminate the higher risk of the measured CVD risk factors in this group. The OR of these two risk factors was still high, when compared with those who were inactive with BMI <25 kg/m^2 ($p<0.001$). Those who reported being physically active had improved GHQ scores in all BMI categories ($p<0.001$).

The association between some lifestyle factors and dietary habits showed that those in the youngest age group, current smokers, inactive people and those from lower social class were more likely to have unhealthy dietary habits and in contrast, older adults, non-smokers, active subjects and people from higher social class were more likely to achieve the recommended dietary targets. Overweight and obese subjects reported consuming less energy dense foods and sugar compared with those of a BMI <25 kg/m^2. This study also showed that some healthy lifestyles were associated with unhealthy dietary patterns.

In an observational study, the effects of a smoking cessation programme using nicotine replacement therapy (NRT) on body weight and shape, dietary patterns and physical activity in free-living subjects were studied. Fifty-five subjects were recruited at the baseline and for the subsequent follow-up sessions, 32, 21 and 18 subjects attended at week 7, 12 and 18 respectively. Mean weight gain among subjects who completed the 18 weeks of the study was 2.9 kg (median 3.6 kg, $p<0.01$) (males gained 1.1 kg (median 2.7 kg), females gained 3.8 kg (median 4.3 kg, $p<0.01$)). Mean WC of females at week 18 increased 4.1 cm compared with

baseline ($p < 0.01$). In this study total energy intake among participants were lower than their actual requirements. The overall quality of diet of the participants was poor and did not changed significantly during the study. Reported physical activity was unchanged from baseline tp week 18.

In conclusion, smoking was associated with a lower BMI in the sample as a whole, but not for the youngest age group. Smoking cessation was associated with weight gain. Smoking and obesity were the two major risk factors, which showed the strongest associations with the CVD risk factors, and their combination exaggerated CVD risk factors. Achievement of currently recommended physical activity levels were associated with lower BMI and prevalence of obesity, and a smaller WC and WHR. However, approximately 50% of active subjects were overweight and obese. These levels of activity were associated with lower CVD risk factors, however the joint associations of physical activity and BMI showed that obese active people still had higher CVD risk factors than inactive people with BMI < 25 kg/m^2. Smoking and inactivity were two major modifiable behaviours that showed the strongest associations with unhealthy dietary habits. Smoking cessation was associated with increased body weight and WC within weeks of cessation, particularly in females with NRT. Attrition rates were high and effective weight maintenance strategies may improve this.

Chapter 1: Introduction

Overweight and obesity representing the disease-process of excess body fat accumulation, are now major public health problems in both developed and developing countries. The prevalence of obesity is increasing globally and in this century more people will die from adverse effects of over nutrition than of starvation. Most of the increase in the prevalence of overweight and obesity has happened during last decade, among European countries, the UK showed the greatest increase (Rossner, 2002). In England about 45% of men and 32% of women, in Scotland about 43% of men and 32% of women are overweight (defined by BMI 25 to 30 kg/m^2) an additional 17% of men and 21% of women in England and about 20% of men and 22% of women in Scotland are obese (defined by BMI ≥30 kg/m^2) (Shaw *et al.*, 2000). The determinants of weight gain and obesity have proved to be multifactorial and are affected by a combination of both genetic and environmental factors. Over the past decades the lifestyle of many people in the world, particularly in developed countries, has changed and recent increases in the prevalence of obesity worldwide are suggested to be caused by an environment that promotes inactivity, excessive food intake and unhealthy lifestyle.

Obesity as reflected by raised BMI is a major risk factor for many chronic diseases, which include diabetes, hypertension, dyslipidaemia, cardiovascular disease, sleep apnea, musculoskeletal disorders and some cancers. The total disease burden is difficult to estimate accurately, but probably accounts for 4-8 % of total health care budgets (Seidell, 1995; Colditz, 1999). Overweight and obesity increase the risk of death from all causes (Rossner, 2002). BMI is correlated with total body fat (Lean *et al.*, 1996), but waist circumference has a generally stronger correlation with total body fat and additionally reflects the more metabolically active intra-abdominal fat mass (Han *et al.*, 1997). Central or abdominal fat accumulation, indicated by high waist circumference is an independent risk factor for coronary

heart disease. In England around 28% of men and 20% of women have a WHR ≥ 0.95 and ≥ 0.85 respectively (Petersen & Rayner, 2002).

In industrialised countries, cigarette smoking is another major cause of preventable disease. Approximately 45 million Americans and more than 1.2 billion people worldwide continue to use tobacco. In the Scottish Health Survey 1998, 34% of Scottish men and 32% of women aged 16-74 reported that they smoked cigarettes (Shaw *et al.*, 2000). Tobacco causes many chronic diseases such as coronary heart disease, cancer, emphysema, bronchitis, and respiratory infections in both men and women. Smoking is widely recognized as the most significant modifiable risk factor for a number of diverse health outcomes, including respiratory cancers, heart disease and stroke. Mortality from smoking-related diseases in women is increasing worldwide and it has been suggested that women may be more sensitive than men to the health hazards of smoking (Prescott *et al.*, 1998). Smoking increases the risk of Coronary Heart Disease (CHD). It is estimated that about 20% of deaths from CHD in men and 17% of deaths from CHD in women are due to smoking. Cigarette smoking in both adults and teenagers is more prevalent in Scotland and Northern Ireland than in England or Wales (Petersen & Rayner, 2002).

Anthropometric measurements and biological factors differ between smokers and non-smokers. These differences may be due to smoke components, different behaviours or lifestyles in smokers and non-smokers. Nicotine addiction is an extremely complex process that involves biological, psychological, behavioural and cultural factors. Body weight is one of the factors that influences smoking and that is influenced by smoking. Smoking and obesity are prevalent health risks, each of which has important effects on morbidity. People who both smoke and are obese may be at particularly high risk for cardiovascular disease

(CVD) and need to tackle both of these issues. The association of body mass index and mortality is typically J-shaped (Meyer *et al.*, 2002) or U-shaped (Willett *et al.*, 1999) with increased death rates among both the leanest and the heaviest persons. The association can be affected by some confounding factors like smoking. It is said that the increased risk associated with leanness may be due to smoking and its related illness (Albanes *et al.*, 1987; Willett *et al.*, 1999). On the other side, the mortality risk among obese smokers increased two or four fold in comparison with obese and normal-weight never smokers respectively (Meyer *et al.*, 2002). Therefore both obesity and smoking threaten life expectancy separately and combination of them may make it worse. It has been shown in the middle-aged adults who were obese and smoked lost seven more years of life than normal weight smokers (Peeters *et al.*, 2003).

Despite these powerful adverse effects of smoking, many smokers refuse to quit smoking. One of the many possible reasons is to avoid unwanted weight gain. Nowadays, given overweight and obesity are major public health problems, many researchers and public health authorities are trying to inform and warn people about the adverse effects of overweight and obesity. Therefore there is a great concern that these efforts may indirectly lead to a greater use of smoking as a means of weight control and discourage smokers from quitting (Wee *et al.*, 2001). Thus, the study of relationships between smoking and body weight, especially, understanding the factors, which will influence on this relationship during both smoking and cessation, are valuable, and may guide public health policy.

In nutritional epidemiology, the effects of nutrients, foods and food groups on health cannot be considered in isolation, on the contrary, the effects of inter-related lifestyle factors such as smoking, physical activity, diet and alcohol consumption should be considered in combination

with socio-economic factors. Both smoking and increased body weight are risk factors for many chronic diseases such as cardiovascular diseases, diabetes etc, the understanding as to how these two risk factors are related to each other is an important issue, which may assist efforts to tackle both of these risk factors effectively.

1.1. Current smoking and body weight

Many studies have shown that there is a negative relationship between current smoking and body weight. Albanes et al investigated the effect of cigarette smoking on body weight in a 12103 subjects aged 19-74 in the US in 1976 and 1980(Albanes *et al.*, 1987). These findings revealed smokers were leaner than non-smokers, and duration of smoking, but not smoking intensity, had a greater effect on body weight. An analysis of 1911 monozygotic male twin pairs (Eisen *et al.*, 1993) showed that cigarette consumption was associated with significantly reduced body weight, whereas alcohol consumption did not significantly change body weight. Flegal et al also found the same result in the United States in which a study of current smokers, both male and female, had the lowest age-adjusted prevalence of overweight and lowest BMI of all groups aged 35 or older (Flegal *et al.*, 1995). Among urban Chinese adults smoking was inversely associated with overweight in both genders (Hu *et al.*, 2002).

The association between smoking and relative body weight may differ among subgroups within one population. Strauss and Mir studied 1132 adolescents aged 12-18 years who were enrolled in the NHANES III study; they found that there were no differences in body weight and BMI among smokers and non-smokers (Strauss & Mir, 2001). Hispanic teenage girls who were smokers reported significantly higher BMI than non-smokers whereas there were no

significant differences among Caucasians and African-Americans teenage girls (Baer Wilson & Nietert, 2002).

Molarius et al examined the association between smoking and reported body weight among 42 populations in 69000 men and women aged 35-64, who participated the WHO MONICA project (Molarius *et al.*, 1997). This study showed that regular smokers had significantly lower median BMI in 20 (men) and 30 (women) out of 42 populations, and in some populations there was no association between smoking and body weight. The inverse relationship was stronger among women than among men. Ex-smokers had significantly higher BMI than never smokers only in 10 out of 42 populations among men, whereas among women there was not a consistent pattern. Based on the findings, authors suggested that the magnitude of the inverse association between smoking and body weight might be related to the prevalence of smoking in the population.

It has been shown that age is an important modifying factor of the association between smoking and BMI (Marti *et al.*, 1989). The inverse relation between smoking and relative body weight tends to be stronger in older than younger subjects. This may be because of longer duration of smoking in elders.

In terms of dose of cigarette smoking, some studies have shown that there is a curvilinear or U-shaped relationship between smoking and body weight that is, those who smoked 10- 20 (5-20) cigarettes per day were the leanest group, whereas heavy smokers, those who smoke more than 20 cigarettes per day were the heaviest. In other words there is a positive relationship between heavy smoking and body weight. The Minnesota Lipid Research Clinical data showed there was a U-shaped association between smoking and body weight in which, those

who smoked 15-29 cigarettes a day were the lightest group (Jacobs & Gottenborg, 1981). Albanes et al reported moderate smokers (6-20 cigarettes per day) were leaner than either light smokers (1-6) or heavy smokers (more than 20) (Albanes *et al.*, 1987). To assess the association of heavy smoking and increased body weight, Istvan et al analysed the baseline data of 891589 participants in a prospective study (Istvan *et al.*, 1992). They observed that heavy smokers, and those who smoked two or more packs cigarettes per day, were more overweight than those who smoked 10-20 cigarettes per day.

In the WHO MONICA project a U-shaped relationship between BMI and number of cigarette smoked was found only among women (Molarius *et al.*, 1997). Oh and Seo analysed the medical records of 400 male patients aged 20-76 who visited a university hospital in Korea (Oh & Seo, 2001). They showed that there was a significant quadratic relationship between smoking intensity and BMI. The plot of this association was slightly U-shaped, but it was not particularly remarkable. This study also showed heavy smokers, who consumed more alcohol, had higher BMI. In other words smoking intensity and alcohol consumption had a complicated interactive relationship with BMI.

It has been suggested that the historically negative association between smoking and body weight might be changing to a positive one. Marti et al (Marti *et al.*, 1989) evaluated the interrelation between smoking and body weight and its change over time by using data from Finnish population in 1982 and 1987. The main finding of the study was that the generally accepted negative association between smoking and body weight disappeared in Finnish men and decreased in Finnish women during 1982-1987. In 1987 a significant positive association between smoking and weight was observed in all younger men and also both younger men and women showed a positive association between smoking and WHR. Individual health

behaviours with increased intensity of unhealthy habits such as high intake of saturated fat, high consumption of alcohol and less exercise especially among younger men might be the most likely explanation for the results.

In an investigation of 6751 African American and White seventh-grade students Klesges et al, reported that daily smokers had significantly higher BMI than did non-smokers (Klesges *et al.*, 1998a). The authors expressed that the association might be explained in two plausible ways. Firstly, smoking is an ineffective weight-control strategy in adolescents and, secondly, heavier adolescents start to smoke in an effort to lose weight. In this study smoking, weight and height were self-reported which might be a matter for error.

Socio-economic status is also a potential confounder in the relationship of smoking and body weight (Molarius *et al.*, 1997). It has been shown that lower education is associated with higher body mass index (Molarius *et al.*, 2000).

Molarius and Seidell (Molarius & Seidell, 1997) in a study using data from the Monitoring Project on Cardiovascular Disease Risk Factors in the Netherlands from 1987 to 1991 observed that the association between smoking and relative body weight differed by level of education. Among men, low educated heavy smokers weighed significantly less and highly educated heavy smokers weighed significantly more than never smokers. Ex-smokers weighed significantly more than never smokers at high and medium level of education whereas there was no difference in BMI between never and ex-smokers at low educational levels. Among women, smokers had a lower BMI than never smokers, but it was significant at low education level. Female poorly educated ex-smokers weighed less than never smokers. In this study lifestyle factors such as alcohol consumption, fat intake, physical activity, which were associated with BMI, and factors related to smoking did not explain the variation in the

association by education. The authors also mentioned that among men they observed the abundant use of alcohol among heavy smokers at higher educational levels, which might have contributed to their higher BMI in comparison with never smokers.

1.1.1. Summary

Based on these studies it can be concluded that although smoking lowers body weight, the associations between smoking and body weight vary considerably among populations, subgroups, different socio-economic and different time periods, which should be considered in any research in this area.

1.2. Smoking, central adiposity and metabolic abnormalities

In general, except some for some differences between subgroups and some population inconsistencies, smokers are leaner in comparison with non-smokers. However, some studies have shown that smokers may have more central obesity than never smokers. Smoking is reported to be positively related to greater abdominal fat in young and middle-aged persons. Past and current smoking habits are positively associated with abdominal fat (indicated by WHR) and waist circumference) in older men, but not in older women (Visser *et al.*, 1999b).
A study of 1122 men aged 19-102 years pointed out that WHR in smokers was significantly higher than in nonsmokers and that this association was dose respondent (Shimokata *et al.*, 1989). Seidell et al studied the association of smoking habits and body fat distribution in 512 European men aged 38 years from six different towns during 1988-1989 (Seidell *et al.*, 1991). They found smoking habits were not related to body mass index, but heavy smokers had higher waist circumferences and higher WHR than never smokers. It has been concluded that

smoking is independently related to fat distribution. Lissner et al in a representative sample of Swedish women showed that smokers had a significantly lower BMI, but higher WHR than non-smokers (Lissner *et al.*, 1992). The result after further adjustment for BMI indicated that smokers had more upper body fat than nonsmokers with similar body mass. Although Swedish Women who stopped smoking gained weight, they did not experience the degree of upper-body fat deposition that generally accompanies weight gain.

Wareham et al in a population-based cohort study with a sample of 1122 aged 49-65 years found overall obesity as indicated by BMI was lower in current smokers, but they had higher central adiposity as indicated by WHR (Wareham *et al.*, 1996). Particular patterns of alcohol consumption and physical inactivity were associated with smoking. Jee et al reported that "Paradox A" which is defined as a group of subjects with the lowest BMI and the highest WHR, is more prevalent among smokers than non-smokers (Jee *et al.*, 2002). They studied the association of paradox A and smokers among subjects who participated in the Korean Nationwide Health Examination Survey (1998). Their results indicated the risk of paradox A among smokers was 2.1 fold higher for men and 2.5 fold higher for women than non-smokers. The authors suggested that cigarette smoking is possibly to be associated with diabetes mellitus through paradox A because smokers generally have lower BMI, but higher levels of central obesity.

It has been reported that smoking acutely impairs glucose tolerance and insulin sensitivity, and enhances serum cholesterol and triglycerid concentrations (Frati *et al.*, 1996). The results of the study on long-term users of nicotine containing chewing gum showed that nicotine was associated with insulin resistance and metabolic abnormalities linked with the insulin resistance syndromes. Therefore, long-term use of nicotine replacement therapy should be

limited (Eliasson *et al.*, 1996). In another study Eliasson et al examined the association of the various aspects of insulin resistance among healthy non-obese male smokers and non-smokers (Eliasson *et al.*, 1997). The study demonstrated that smokers who were insulin resistant, had some disorders related to insulin resistance syndromes such as lower HDL-cholesterol and higher fasting triglycerides. They also exhibited lipid intolerance and impaired elimination of triglycerides from a mixed meal even after fasting normotriglyceridemia. Higher levels of abdominal fat and increasing catecholamines as a result of increased sympathetic nervous system activity in smokers may be possible reasons for insulin resistance in smokers. Catecholamines are potent insulin-antagonistic hormones that have long term effects on cellular synthesis of insulin-regulated proteins (Eliasson *et al.*, 1997).

The association of cigarette smoking and HbA1c, as a marker of long-term glucose homeostasis, in a large population-based study (EPIC-NORFOLK study) revealed that mean HbA1c concentrations were highest in current smokers in comparison with never and ex-smokers (Sargeant *et al.*, 2001). This study showed that smoking has long-term effects that may lead to increased risk of diabetes and its complications such as cardiovascular disease. In this study women who were current smokers had higher mean WHR compared to non-smokers. It is said that cigarette smoking is an independent and modifiable risk factor for type 2 diabetes (Uchimoto *et al.*, 1999; Manson *et al.*, 2000; Wannamethee *et al.*, 2001). Several reasons may explain this association, which include, increased blood glucose level, impaired insulin sensitivity, dyslipidemia, increased abdominal fat, free radical oxidative damage and oxidative stress and toxic effects of nicotine on the pancreas. However, Wareham et al suggested that there is not likely to be a causal relationship between smoking and insulin resistance (Wareham *et al.*, 1996). In addition, Perry et al in the British regional heart study of middle-aged men reported that although after adjusting for age and BMI current smoking was

associated with a 50% increase in the risk of diabetes compared with those who had never smoked (Perry *et al.*, 1995). However, multivariate analysis indicated that this association was no longer significant after adjustment for physical activity, prevalence of coronary heart disease, alcohol intake, blood pressure, HDL-c, heart rate and uric acid.

1.2.1. Summary

In general, it seems smoking may be an important environmental risk factor for insulin resistance syndromes which may lead to increasing cardiovascular mortality and morbidity. The mechanism of the positive association between smoking and fat distribution is not clear. Possible mechanisms may be differences in serum hormone levels between smokers and nonsmokers such as sex hormones, or some other possible confounding factors such as alcohol intake and stress level (Seidell *et al.*, 1991).

1.3. Weight concern and smoking

Generally, it was accepted that smokers weigh less than age-matched non-smokers, and most smokers gain weight after smoking cessation due to an increase in food intake, and a decrease in energy expenditure or both of them. Many believe that the prospect of weight gain discourages smokers from quitting because many studies have shown that smoking cessation causes weight gain. Numerous studies have suggested that there is an association between concern about body weight and higher prevalence of smoking particularly among younger adults and adolescents.

A study on a cross-sectional sample of 16862 children 9-14 year of age, showed that among both girls and boys contemplation of smoking was positively related to weight concerns and

experimentation seemed to be positively related to weight control behaviours (Tomeo *et al.*, 1999).

Pirie et al reported that concerns about potential weight gain were more common among young women, within average age of 19.2 years, than in men (Pirie *et al.*, 1991). Wee et al, studied the relationship between weight control efforts and smoking cessation behaviours among 17213 adults in the United States (Wee *et al.*, 2001). The results showed that weight control efforts after adjustment for sociodemographic factors and BMI were significantly associated with current smoking. In this study among adults trying to lose weight, those younger than 30 years were more likely to smoke currently, whereas older adults were as likely or even less likely to smoke than subjects not trying to control weight. These results were independent of sex and BMI. This study suggested that younger adults may use smoking as a means of weight control and public health efforts should pay more attention to this group and aim to inform them about the health effects of smoking and healthy weight control habits.

Weight-related behaviors and conditions, which include dieting behaviors, eating disorders and disordered eating behaviors, are prevalent among adolescents particularly among girls. In a study of 6728 adolescents in grade 5 to 12 (Neumark-Sztainer & Hannan, 2000), it has been found almost half of the girls reported that they had at some point been on a diet compared to a fifth of the boys. In this study significant association were found between dieting and disordered eating and low self-esteem, high levels of depression, suicidal ideation, and high levels of stress. There was also a significant and direct association between tobacco use and both dieting and disordered eating among girls but not among boys. In a prospective study the association of dieting frequency and smoking initiation was tested among middle school girls

and boys. The results showed that only among girls, dieting in early adolescence increased the risk of becoming a smoker.

Although there may be several mechanisms linking dieting and smoking, higher weight concerns among females and psychological or physiological factors such as stress, food deprivation and age at menarche are plausible reasons for the association (Austin & Gortmaker, 2001). In this study obesity unexpectedly was related to a reduced risk of smoking initiation among girls and also there was no significant sex difference in dieting frequency at baseline. Body image attitudes may be important factors in female smokers. Ben-Tovim and Walker measured body attitudes in Australian women such as feelings of fatness attractiveness, strength and fitness and the importance of weight (Ben-Tovim & Walker, 1991). They showed that women smokers feel less attractive than never smokers, but they did not consider themselves to be less strong and fit than non-smokers. King et al (King *et al.*, 2000) found the same result that women smokers may be more dissatisfied with their bodies than women in general (King *et al.*, 2000). They, however, found that women smokers perceived themselves as heavier than the control, non-smoking group.

It is said that concern about weight gain after smoking cessation is a huge barrier for giving up smoking and that many weight concerned individuals attempt to quit, but terminate their efforts to cease smoking very early. In the other words, the rate of successful smoking cessation and maintenance among weight-concerned smokers is very low. It has been shown that women with strong concerns about their weight are less likely to engage in a smoking cessation attempt, and therefore special efforts will be needed to overcome this barrier to quitting (Brouwer & Pomerleau, 2000).

It has been suggested that adolescents may use tobacco as a form of weight control, but there is scant information available to draw any meaningful conclusions. Strauss et al have demonstrated that the prevalence of smoking was increased two-fold among normal weight adolescent girls who have tried to lose weight, whereas there was no increased risk of smoking among overweight girls or boys trying to lose weight (Strauss & Mir, 2001). In contrast, overweight boys who were trying to lose weight were less likely to smoke than those who were not trying to lose weight. In this study the prevalence of smoking in general was similar in the normal and overweight. These results show that it is possible that their dieting behaviors lead to increased prevalence of smoking among adolescents. Fear of fatness, which is common during female adolescence, may encourage teenage girls to use harmful slimming strategies. In a study of 420 Irish schoolgirls aged 15 years reported that 59% of subjects wanted to be slimmer and 68% had previously tried to lose weight. They reported using unhealthy weight control practices such as fasting and smoking (Ryan *et al.*, 1998). Voorhees et al conducted a comprehensive study on data from a cohort of girls aged 9-10 years, the study participants also were followed until aged 18-19 years to understand what childhood and early adolescent factors predict daily smoking at young adult age (Voorhees *et al.*, 2002). The results revealed that apart from family, social environment, and behavioral factors, for both white and black girls, weight-related variables such as a higher drive for thinness at ages 11-12 were significant predictors for smoking at ages of 18-19 years.

1.3.1. Summary

These studies show that there is a widespread and strong concern about body weight at early age, particularly in women. This issue may be one to explain why individuals starting smoking, deny a desire to quit especially among women and teenagers. Nevertheless there are

some unanswered questions in this field e.g. to what extent does obesity encourage people to start smoking and why have some studies shown that normal-weight people have significant, but unnecessary weight concerns.

1.4. Smoking and dietary habits

Both smoking and poor dietary habits are important risk factors for many diseases, especially chronic diseases. A study on 932 high school pupils aged 12-15 years showed smoking was associated with consumption of less fresh foods and greater consumption of fatty foods (Coulson *et al.*, 1997).

A cross sectional study of 3430 teenage 16-17 years who were participants in the 1970 Longitudinal Birth Cohort in Britain revealed that dietary habits of smokers, particularly girls, differed from those of non-smokers. Smokers consumed significantly more chips, alcoholic beverages and coffee and less puddings, fruit, fruit juice and breakfast cereals. Female smokers consumed significantly less vegetables than non-smokers (Crawley & While, 1995). In another study using a sub-sample of this cohort it has been shown parental smoking habits has a negative effect on dietary habits of their offspring. Teenagers who lived with a parent who smoked had similar dietary patterns to teenagers who themselves smoked (Crawley & While, 1996).

A meta-analysis on fifty-one published nutritional surveys from 15 different countries with 47250 non-smokers and 35870 smokers has shown that smokers have unhealthy patterns of nutrient intake compared with non-smokers. On average smokers reported significantly higher intakes of energy, total fat, saturated fat, cholesterol and alcohol and lower intake of

polyunsaturated fat, fibre, vitamin C, E and beta- carotene than non-smokers (Dallongeville *et al.*, 1998). Strauss and Mir reported that there were no differences in caloric or fat intake among adolescent smokers and non-smokers, but that smokers reported eating less fruit and vegetables and consuming more alcohol compared to non-smokers (Strauss & Mir, 2001). The quantity of alcohol consumption in smokers was more than five times as much as that in non-smokers (odds ratio 5.28, (3.82-7.28)).

The association of smoking and food intake has been studied among Caucasian, African-American, and Hispanic female teenagers in grades 9-12, who participated in the Youth Risk Behavior Survey. The results showed Caucasian teenage smokers consumed significantly less milk, fruit, fruit juice, and vegetable and Hispanic teenage smokers consumed significantly less fruit juice in compare with non-smokers. African-American teenage smokers tend to consume higher levels of these foods, but these data do not reach statistical significance (Baer Wilson & Nietert, 2002).

It has been suggested that current smokers because of their different personality and psycho-social variables such as peer group and parental attitudes, especially the younger smokers, are not interested in health promotion in general, and as a result they try to resist adopting health behaviors which may be leading to poorer dietary knowledge and practice with a less healthy lifestyle.

Grunbereg pointed out in both human and rats, nicotine administration or cigarette smoking is followed by a decreased consumption of sweet foods (Grunberg, 1982). Hall et al evaluated changes in food intake after quitting smoking on ninety-five subjects (Hall *et al.*, 1989). The results indicated that significant increases in energy, sucrose and fat intake 2 weeks after the

quit date. Abstainers gained over 9 lb by 26 weeks post-quit and this increased weight was predicted by increased energy intake only in women. Bowen et al suggested that serotonin-enhancing substances such as tryptophan and a high carbohydrate diet could be used in smoking cessation programs to improve quit rates (Bowen *et al.*, 1991). A link between glucoregulation and cigarette craving has been proposed, and also suggested that a desire to smoke may in part represent a mislabeling of sensations arising from a physiological need for carbohydrates (West *et al.*, 1990; West *et al.*, 1999). Some studies showed dextrose supplementation (West & Willis, 1998), increased carbohydrate intake (Helmers & Young, 1998) and glucose tablets (West *et al.*, 1999) were effective in reducing the desire to smoke and may be a useful and simple aid to giving up smoking. In contrast, Harakas and Foulds could not confirm these earlier findings (Harakas & Foulds, 2002). Their study showed 12 g oral glucose did not affect tobacco-craving symptoms, however the author mentioned it might be because of the different research design. It also has been suggested that glucose might decrease desire to smoke only in more dependent, heavy smokers or in smokers who have been smoking for many years (Harakas & Foulds, 2002).

Although it is said that smoking or nicotine exposure is an anorectic agent, there is no clear evidence of reduced eating in smokers. Therefore energy intake is not significantly influenced by smoking or nicotine administration. However there may be a transient increase in eating over a short time following smoking cessation which may decrease transiently after relapse (Perkins, 1993).

Tobacco smoke contains many oxidants and free radicals that can cause oxidative damage to lipids, proteins, DNA, carbohydrates and other biomolecules. In smokers the oxidative

damage can be as a result of both the direct effect of oxidants in cigarette smoke and the consequences of lower antioxidant nutrition status associated with smoking (Kim *et al.*, 2003).

Woodward et al compared health knowledge, behavior and lifestyles between 4896 smokers and 4595 nonsmokers by using data from the Scottish Heart Study (Woodward *et al.*, 1994). Their findings revealed that smokers had poorer dietary knowledge and practice than nonsmokers. Based on their results smoking was associated with several cardiovascular and poor health risk factors such as low HDL-cholesterol, low intake of antioxidant vitamins and fibre, high serum total cholesterol (among women) triglycerides and fibrinogen values, high intake of salt, butter, whole milk, dietary cholesterol and alcohol. In the Multiple Risk Factor Intervention Trial (Stamler *et al.*, 1997) smokers at baseline also had more unfavorable dietary patterns than those of nonsmokers. Smokers reported consuming more: energy, alcoholic beverages, meals away from home, energy from fat, and dietary cholesterol, and less low fat dairy products and fruit and vegetables than nonsmokers. The greater the number of cigarettes smoked per day, the poorest the diet quality. This study also showed special intervention during the trial years 1-6 led to change in diet composition for those who stopped smoking from unfavorable to almost favorable.

A Study on 459 French men, aged 20-60 years, also showed that heavy smokers had poorer dietary patterns than nonsmokers. Smokers tended to consume more total energy, and less fruit and vegetables resulting in lower vitamins C, E and carotene intakes compared to nonsmokers. Cigarette smoking was inversely associated with plasma vitamin C and beta-carotene independent of alcohol and dietary intake, but not to vitamin E concentration (Marangon *et al.*, 1998).

Wei et al investigated the associations between smoking and serum concentrations and dietary intakes of some antioxidants in the adults US population aged 17- 50 years (Wei *et al.*, 2001). They found that smokers compared with nonsmokers, had significantly lower dietary intake of vitamin C and beta-carotene, and also there were an inverse relationship between serum levels and cotinine levels for vitamins C and E and beta-carotene. Teenage smokers aged 15-17 years with a short smoking history showed evidence of lower intake of daily energy and ascorbic acid accompanied by oxidative damage (Kim *et al.*, 2003). Weight gain after smoking cessation may be due to increased energy intake as a result of increasing intake of sweet taste carbohydrate such as sucrose, and fat (Hall *et al.*, 1989).

1.4.1 Summary

In conclusion, generally smokers have a more unhealthy diet and eat more fat, saturated fat, alcohol, and chips and eat less fresh vegetables and fruits. There is an inconsistency between the energy intake of smokers and ex-smokers with their actual energy needs. Because of the appetite suppressing effect of nicotine smokers should consume lower amount of energy than nonsmokers, but most epidemiological studies have shown that smokers eat equal or more than nonsmokers. Information about the dietary patterns during smoking cessation and during the maintenance of cessation is lacking.

1.5. Smoking, appetite and energy expenditure

Weight gain or weight loss is normally due to the change in energy balance involving energy expenditure through physical activity or resting metabolic rate, energy intake or both. Nicotine, which is the main component of tobacco, is primarily responsible for the effects of smoking on body weight.

Perkins et al (Perkins *et al.*, 1992) found that nicotine may not acutely suppress appetite in fasting smokers and suggested that other possible actions of nicotine or smoking may result in a lower body weight. They argued the general belief that smoking decreases body weight by suppressing appetite (Perkins, 1992a). He noted that changes in eating due to smoking happens around the set point, on which body weight is regulated. In other words, smoking cessation increases eating only until a new, higher body weight set point is reached whereas relapse, and perhaps reinitiation of smoking, decreases eating only until a lower set point is reached. Cabanac and Frankham reported that cigarette smoking and nicotine lowers the body weight set point and they suggested that weight gain after cessation might be due to readjustment of body weight set point as a result of the removal of nicotine from the body and the influence of nicotine on palatability of food is mediated through the body set point (Cabanac & Frankham, 2002).

Perkins et al (Perkins *et al.*, 1994) in a controlled nicotine intake via tobacco smoking study on 20 male and female smokers showed that neither usual cigarette smoking, low nicotine cigarette or sham smoking had acute effects on hunger or eating in male or female smokers (Perkins *et al.*, 1994). In spite of an increase in food intake over days or weeks following smoking cessation, many cross-sectional studies in humans have shown no difference or even increase in eating among smokers compared with non-smokers (these have already been mentioned in smoking and the dietary habits section), whereas it has been commonly accepted by the general population that smoking has a suppressive effect on appetite. Many animal studies have shown that nicotine has an anorectic effect and decreases food intake. A study of systemic nicotine administration on rats during a 7-day period showed that food intake in both sexes declined and that the reduction was due to significant reduction of meal sizes without

changing meal numbers (Blaha *et al.*, 1998). The systemic infusion of nicotine in male rats increased hypophagia with an increase lateral hypothalamic area dopamine and serotonin, while nicotine cessation decreased both neurotransmitters and was associated with hyperphagia (Miyata *et al.*, 1999). Miyata et al in another experimental model using rats demonstrated that at infusion of nicotine for 7 days significantly decreased body weight and food intake through decreasing in meal size without compensatory increase of meal number (Miyata *et al.*, 2001). In contrast, nicotine cessation led to hyperphagia resulting in an increase in body weight via an increase in meal size. It has also been shown that the nicotine increases the duration of the estrous cycle and the intermeal interval. They concluded these metabolic effects of nicotine might be partly related to an activation of hypothalamic serotonergic system.

Leptin is a hormone produced by the adipocyte which appears to play an important role in changing body weight by regulating appetite and energy expenditure. As both nicotine and leptin affect appetite and energy expenditure it has been suggested there may be a link between them. Hodge et al demonstrated a link between the action of nicotine on body weight and leptin, suggesting that smoking via nicotinic mechanisms may modify the sensitivity of leptin receptors (Hodge *et al.*, 1997). In this study, which was carried out in male smokers and nonsmokers from three different populations, smokers had lower levels of serum leptin independent of BMI, WC and diabetes status. Donahue at al reported the same results from a cross-sectional study among 422 different ethnic groups, and showed cigarette smoking was inversely related to leptin concentrations (Donahue *et al.*, 1999). Mantzoros et al found a negative and independent association between cigarette smoking and leptin concentrations (Mantzoros *et al.*, 1998). A study on 54 male smokers and non-smokers and 19 long-term nicotine gum uses revealed that plasma leptin concentrations were higher in smokers and other

long-term users of nicotine than in non-smokers, Smoking cessation for a further 8 weeks increased the leptin levels. They concluded that an indirect effect of insulin resistance and relative hyperinsulinaemia in the smokers and nicotine users was more likely to cause the higher leptin levels (Eliasson & Smith, 1999). Nicklas et al based on the results of their study pointed out that higher plasma leptin may be one of the mechanisms for a lower body weight in smokers compared to non-smokers (Nicklas *et al.*, 1999). Larsson and Ahren reported that circulating leptin levels were not different between smokers and non-smokers in non-obese postmenopausal women (Larsson & Ahren, 1999). The results of this study also indicate that smoking per se does not affect leptin expression or secretion. In an animal study an infusion of nicotine for 7 days decreased food intake (a significant decrease in meal size) and as a consequence body weight decreased. During nicotine infusion plasma leptin concentrations were significantly lower in the nicotine group than in the control group and after the infusion period there was no difference between the two groups. This study suggested that leptin was not involved in the anorectic effect of nicotine (Miyata & Meguid, 2000). Sanigorski et al (Sanigorski *et al.*, 2002) in an experimental study on lean and obese Psammomys obesus showed continuous nicotine infusion significantly lowered body weight and food intake in both lean and obese P. obesus. These changes occurred in the presence of significantly reduced plasma leptin concentrations in lean P. obesus whereas in obese animals, leptin levels remained unchanged. This study demonstrated that nicotine's effects on food intake appear to be independent of the leptin signaling pathway. They hypothesized that nicotine's effects on food intake are mediated through the central nervous system by affecting a number of neurotransmitters involved in energy homeostasis and indirectly activating the sympathetic nervous system. The associations of smoking and leptin seem to differ in different populations and depends on experimental protocol.

It is said that smokers eat the same amount as or more than non-or ex- smoker, while they weigh less. Based on energy balance regulations smokers must expend more energy than non or ex-smokers if this perception is correct (Perkins, 1993). A review of the metabolic effects of smoking suggested that the effect of cigarette smoking on reducing body weight is more likely to be due to increasing whole body metabolism rather than decreasing calorie intake or increasing physical activity (Perkins, 1992b). Resting Metabolic Rate (RMR) that is the energy expended for maintenance of major body function during a period of complete rest and controlled environment constitutes 60-75 percent of daily energy expenditure (Poehlman, 1989). Therefore a small or transient change in RMR could affect energy balance and therefore weight gain. Perkins noted most studies indicate smoking and nicotine exerts no chronic effect on RMR, but that there is an acute effect due to smoking for which the magnitude of the effect is variable among studies (Perkins, 1992b). Smoking also produces a moderate increase and significant increase in RMR (<10%) that lasts approximately 30 minutes after each cigarette. Thus acute metabolic effects of smoking are probably not significant beyond 30 minutes after smoking. Kimm et al demonstrated that young women aged 18-21 years who smoked cigarettes had a higher 24h REE (68 k cal/day) even after controlling for differences in body size (Kimm *et al.*, 2001).

It is said that the amount of energy attributable to an acute effect of smoking at rest is less than the amount of energy, which is needed for usual rate of weight gain after smoking cessation. In a free-living situation smoking is accompanied by a variety of activities such as daily work and physical activity that themselves can acutely influence metabolic rate. Hofstetter et al (Hofstetter *et al.*, 1986), using a metabolic chamber showed cigarette smoking in conjunction with casual activity during metabolic measurement increased 24-hour energy expenditure by approximately 10 percent. In another study, Perkins et al (Perkins *et al.*, 1989) demonstrated

that the excess energy expenditure attributable to nicotine was more than twice as great during exercise as during rest (Perkins *et al.*, 1989). Even male smokers who were more aerobically fit and were more physically active, but with longer smoking histories had greater thermic response to nicotine (Perkins & Sexton, 1995).

Audrain et al (Audrain *et al.*, 1995) investigated the metabolic effects of cigarette smoking in 20 normal weight and 20 obese female smokers over 2-day periods. The results of this investigation indicated that Resting Energy Expenditure (REE) increased in both obese and normal weight subjects, but the amount and duration of the increase in REE was higher in normal weight than obese smokers. REE after smoking in normal weight participants increased significantly and remained higher during 30 minutes after smoking whereas, in obese subjects REE increased significantly only for the first 10 minutes after smoking and dropped slightly below baseline between 20- 30 minutes post smoking. The average REE 30 minutes after smoking in normal weight smokers was twice as much as the REE in obese subjects (133 vs. 66 kcal/day). The authors suggested body fat possibly has an impact on nicotine's effect on REE through the autonomic nervous system. Therefore obese smokers might have a lower sympathetic nervous system response than normal weight smokers. It is also mentioned that obese smokers might need a higher dose of nicotine to induce a given response because of having a higher body mass. Walker et al observed that body fatness was inversely correlated with REE in 40 male smokers with generally normal weight (Walker *et al.*, 1993). This negative association in another study was only marginally significant (Perkins & Sexton, 1995). It has been shown that low sympathetic nervous system activity was associated with weight gain and development of central adiposity in men (Tataranni *et al.*, 1997).

Yoshida et al (Yoshida *et al.*, 1989; Yoshida *et al.*, 1990; Yoshida *et al.*, 1994) in experimental models demonstrated that cigarette smoke and nicotine increased norepinephrine turnover (as an indicator of sympathetic nervous system activity), thermogenesis in brown adipose tissue and resting metabolic rate in obese mice which led to the loss of body weight. There is an inverse relationship between sympathetic activity and percent body fat, and also an inverse relationship between sympathetic activity and energy intake (Bray, 2000). Walker and Kane (Walker & Kane, 2002) studied the effects of nicotine on resting energy expenditure and plasma catecholamine in normal and overweight male smokers. In this study 32 fasted smokers (13 normal weight and 19 overweight) were given two reference yield cigarettes (0.16 mg nicotine) and two high yield cigarettes (1.7 mg nicotine each). The results showed that there was no thermic response or catecholamine release when subjects consumed low yield cigarettes. Normal weight men demonstrated a significant (7.2% REE) thermic response to smoking high yield cigarettes whereas overweight subjects showed no significant response to that. Plasma norepinephrine increased by 49% only in normal weight subjects in response to high yield cigarette as well. These findings suggested that BMI interacts with the thermic effect of nicotine and neuroendocrine function in male smokers. Because nicotine is alkaline and slightly lipophilic, the higher level of body fat in obese smokers blunts its thermic effects.

1.5.1 Summary

In animal studies nicotine suppresses appetite and increases hypophagia via lateral hypothalamic neurotransmitters in brain and as a consequence energy intake decreases specially decrease meal size. In epidemiological studies smokers eat the same as or even more than non-smokers. Cigarette smoking or nicotine increases resting metabolic rate through increasing sympathetic nervous system activity and increasing thermogenesis in BAT. It

seems that light physical activity, typically carried by free-living people, increases the metabolic effects of nicotine. Obese people have a lower response to the metabolic effects of nicotine. Therefore obese smokers may lose less weight than normal-weight smokers. Based on the studies mentioned, nicotine has an acute effect on RMR that lasts 30 minutes after smoking and the long-term or chronic effects of nicotine on RMR still require clarification and therefore further research.

1.6. Smoking cessation and weight gain

Weight gain is one of the most common consequences of smoking cessation. Perkins et al (Perkins *et al.*, 1990) showed that energy balance changed significantly during periods of normal smoking, smoking cessation and resumption of smoking in a prospective study in 7 female during three weeks. During abstinence resting metabolic rate decreased while energy intake increased and vice versa during resumption of smoking.

The amount of weight gain among females is higher than males after quitting (Williamson *et al.*, 1991). The mean weight gain attributable to the cessation of smoking, after adjusting for age, race, level of education, alcohol use, illnesses related to change in weight, base-line weight, and physical activity, was 2.8 kg in men and 3.8 kg in women using data from the NHANES cohort (1971-1984) (Williamson *et al.*, 1991). Additionally, 9.8 percent of men and 13.4 percent of women who stopped smoking over 10 years gained more than 13 kg. This study also suggests the amount of weight gain after smoking cessation is nearly the same of the amount of weight loss during smoking. In the Nurse's Health Study the average weight gain attributable to smoking cessation after 2 years follow-up study between 1986-1988 was about 2.4 kg in middle-aged women (Kawachi *et al.*, 1996). In this study moderate intensity

level of physical activity attenuated the amount of post-cessation weight gain. The findings of Lung Health Study (1986-1994), which was based on a cohort of 5887 male and female smokers in the United State and Canada, showed the average weight gain after 5 years smoking cessation was 8.7 kg for women and 7.6 kg for men. Sixty percent of this weight gain occurred during the first year of cessation (O'Hara *et al.*, 1998). It should be noticed that this study had some limitations such as the selection of heavy smokers and smokers with some degree of lung dysfunction at baseline, which might in itself favour greater weight gains (Thun & Colditz, 1998).

Hall et al noted that the rate of weight gain during smoking cessation is high in the first weeks or months and plateaus after 6 months (Hall *et al.*, 1986). In contrast with these findings, results from the Lung Health Study showed weight gained in the first year of cessation not only did not decline in the subsequent years but also encouraged additional weight gain in the 5 years follow up.

In the monozygotic male twin study (Eisen *et al.*, 1993), it has been demonstrated that the percentage of BMI greater than 27.8 (used as a measure of clinically significant obesity in this paper) in ex-smokers was 33% higher than their current smoking siblings and they had 1.8 times increased risk of the obesity by comparison with heavy smokers.

A study of the influence of smoking cessation on the prevalence of overweight in the United States revealed that smoking cessation might be associated with a small increase in the prevalence of overweight. In this study, which was based on a national sample of 5247 adults 35 years of age or older in the third National Health and Nutrition Survey, people who quit smoking within previous 10 years had gained significantly more weight than those who never

smoked. Average weight gain due to smoking cessation was 4.4 kg for men and 5.0 kg for women and also 16 percent of male ex-smokers and 21 percent of female ex-smokers gained greater than 15 kg. However, this study because of using the respondent's reports of their smoking history and past weight might be subject to error (Flegal *et al.*, 1995). Women, who stopped smoking in the UK during a 26-year period, had the largest increase in mean BMI (Owen-Smith & Hannaford, 1999). Some studies have shown that weight gain after smoking cessation explain only a small percentage of overall prevalence of overweight or obesity in the population (Simmons *et al.*, 1996; Burke *et al.*, 2000).

Although smoking cessation is accompanied by weight gain particularly during the short term around cessation, it is still a matter of debate whether the increased body weight remains over time or if it would be the same as never smokers after a long duration of smoking cessation. Chen et al examined the relationship of body weight and smoking cessation among 1633 adults. In this study, even though ex-smokers had the highest BMI in comparison to non and current smokers, BMI decreased with increasing years of cessation after 2 years (Chen *et al.*, 1993). In a cross-sectional analysis, among light and moderate smokers there was no clear trend in mean BMI or proportion of large BMI according to years of cessation and these groups gained weight up to the level of never smokers. Among heavy smokers, ≥25 cigarettes a day, those who quit 2-4 years ago had greater weight gain than never smokers. However, in this group also there was a decreasing trend in the mean BMI with increasing years of cessation (Mizoue *et al.*, 1998). In contrast Williamson et al (Williamson *et al.*, 1991) showed that the relative risk major weight gain among quitters was high regardless of duration of smoking.

1.6.1. Nicotine replacement therapy

It has been suggested that removal of nicotine from the body is the factor most responsible for weight gain after smoking cessation. Thus, replacement of nicotine during abstinence by gum, transdermal patch or intranasal spray might reduce or prevent weight gain. Emont and Cummings reported an inverse correlation between weight gain and number of pieces of nicotine gum chewed per day during one-month smoking cessation in heavier smokers (Emont & Cummings, 1987). This study showed that nicotine replacement might help prevent weight gain following smoking cessation, especially for the more dependent smokers. Gross et al conducted a study using active nicotine or placebo gum on 40 clinic volunteers for 10 weeks (Gross *et al.*, 1989). They found active nicotine gum resulted in a 50% reduction in cessation-related weight gain. However, their 23 weeks follow up showed that 10-week use of nicotine gum delayed rather than prevented eventual weight gain. In a study by using placebo, 2mg or 4mg of nicotine gum after 90 days post cessation the gum users gained 3.7, 2.1 and 1.7 kg respectively (Doherty *et al.*, 1996). In connection with this study, Nordstrom et al examined the effects of nicotine gum on weight change for 1 year after cessation among the 92 participants who had ceased smoking (Nordstrom *et al.*, 1999). They found that the weight gain was dependent on the dose of the nicotine replacement. Those who replaced a higher proportion of their pre-cessation cotinine level during the gum therapy period gained less weight than those who replaced less cotinine. The researchers suggested that further study is needed to clarify whether sufficiently high levels of nicotine replacement can help to prevent cessation- related weight gain completely. Transdermal nicotine replacement therapy may attenuate post cessation weight gain. Dale and his colleagues (Dale *et al.*, 1998) demonstrated that transdermal nicotine patch decreases weight gain only during the period of use.

The potential factors that may be responsible for weight gain after smoking cessation were listed by Talcott et al (Talcott *et al.*, 1995). These factors include increasing the intake of foods that are high in fat and sugar, increasing alcohol consumption, decreasing metabolic rate and energy expenditure. The study of mechanisms that contribute to weight gain suggested that smoking cessation was associated with significant changes in adipose cell metabolism in which the adipose tissue lipoprotein lipase (AT-LPL) activity increases. This increase in LPL activity may contribute to the increase in body weight associated with smoking cessation (Ferrara *et al.*, 2001).

Talcott and his colleagues (Talcott *et al.*, 1995) in an intensive program on 332 military recruits of which 86 subjects were smokers who quit during 6 weeks of basic training showed that post cessation weight gain can be eliminated under constant supervision with an ideal treatment environment. In this study factors such as numbers of cigarettes smoked per day, duration of smoking and fear of weight gain were not associated with post cessation weight gain. However this study was conducted in a very restricted situation, that is not applicable to the free-living. A randomised controlled trial on 281 healthy, sedentary female smokers showed that vigorous exercise facilitates short and long-term smoking cessation in women when combined with a cognitive-behavioural smoking cessation program. Vigorous exercise improves exercise capacity and delays weight gain following smoking cessation (Marcus *et al.*, 1999). Stamler et al (Stamler *et al.*, 1997) in the Multiple Risk Factor Intervention Trial showed that long-term intervention with nutritional counseling can reduce the amount of weight gain after smoking cessation. However, in spite of decreasing the energy intake in this clinical trial during smoking cessation weight gain continued, but the amount of weight gain in the special intervention group (1.7 kg) was less than that in the usual care group (3 kg) and

heavy smokers (≥30 cigarettes per day) gained more weight than those who smoked less than 30 cigarettes per day in both groups.

1.6.2. Summary

The literature suggests that one of the major deterrents to smoking cessation is a gain in body weight. However, the amount of weight gain, intensity of weight gain and duration of weight gain differs between studies. The associations of weight gain after quitting and the intensity of smoking and duration of smoking before quitting are not clear. There are not enough studies that have examined the long-term effects of smoking cessation, and in comprehensive programs for the prevention of weight gain. Hence, understanding the factors that contribute to this weight gain after stopping smoking in different populations may maximise the success of long-term smoking cessation.

1.7. Aims of the thesis:

1) To evaluate the associations between some lifestyle factors, body weight and shape, and cardiovascular (CVD) risk factors.

2) To determine the combination association between lifestyle factors and body weight and shape and CVD risk factors.

3) To evaluate the effects of a smoking cessation program on body weight and shape, dietary habits and physical activity.

1.7.1. Research questions

RQ 1) what is the association between smoking status (current smoker, ex-smoker and non-smoker) and BMI and body shape (WC, HC, WHR)?

RQ 2) what is the independent and combined association between smoking and BMI with CVD risk factors?

RQ 3) what is the association between the currently recommended physical activity levels with BMI and body shape?

RQ 4) what is the independent and combined association between currently recommended physical activity levels and BMI with CVD risk factors?

RQ 5) what is the association between dietary habits and other lifestyle factors?

RQ 6) how do smoking cessation programes using NRT affect body weight and shape, dietary habits and physical activity?

1.7.2. Setting and study designs

The SHS is a cross sectional survey that was carried out in Scotland by the joint health survey unit of the national center for social research and Department of epidemiology and public health of University of College London. The aim of the survey was to provide a comprehensive picture of the health of the Scottish population, their health risk factors and monitor progress towards health targets. The second SHS, which was carried out in 1998 recruited 9047 persons aged 16-74 years old.

Secondary analysis of Scottish Health Survey (SHS) 1998 has answered aims 1-2. A smoking cessation programme running in East Kilbride. This observational study of subjects who are participating in the programme has answered thesis aim 3.

Chapter 2: Methods

This thesis is divided into two sections. The first section, which covers the main part of the thesis, reports the relationships between smoking, physical activity and diet in a secondary analysis of the Scottish Health Survey (SHS) database 1998. The second part of this thesis reports, in chapter 7, an observational study of subjects who were participating in a smoking cessation programme. In this chapter the principal methodological aspects of both of these studies will be explained.

2.1. Scottish Health Survey

The Scottish Health Survey (SHS) is a cross sectional survey that was designed to provide a comprehensive picture of the health of the Scottish population and to document the prevalence of risk factors for cardiovascular and other diseases, as well as monitoring progress towards health targets. The first SHS was carried out in 1995 and the second in 1998. The survey was commissioned by the Scottish Executive Health Department and carried out by the joint health surveys unit of the National Centre for Social Research and the Department of Epidemiology and Public Health at University College London. Full details of the survey methods have been published elsewhere (Shaw *et al.*, 2000). For the purpose of this study some major parts of this survey will be explained briefly.

2.1.1. Survey design and sampling

The survey was designed to provide a nationally representative sample of the population of Scotland aged between 2 and 74 years and living in private households. This thesis reports the analysis of data from adults aged 16-74 years.

The survey used a stratified, multi-stage random sample method of 312 postcode sectors from a total of 936 in Scotland by level of deprivation. A letter was sent to each sampled address to

inform residents that their household had been selected for inclusion in the survey, and request their participation before the first visit was arranged. At each residential address up to three households were selected randomly by the interviewer using specially designed random number tables attached to each address record form. Within each household, one person aged 16-74 years was randomly selected to be included in the survey. Interviewing began in April 1998, and was carried out over a 13 month period, and to avoid any seasonal differences in health, lifestyle, and accidents, the survey fieldwork was distributed evenly over this period. Computer assisted personal interviewing was used for the interview in the subjects home. Interviewers collected information on health related topics including general health, cardiovascular problems, physical activity, smoking and alcohol consumption, socio-demographic information, and height and weight were measured. After the first interview, and if the individual agreed to continue the survey then, nurses collected information on prescribed drugs and vitamin supplements, further anthropometric measurements (waist, hip and demi-span), blood pressure measurements, and took saliva and blood samples. All interviewers and nurses were fully trained for their jobs before the survey and their work was monitored. Ethical approval was obtained from the Research Ethics Committees for all area health boards in Scotland.

From 312 postcode sectors 15288 addresses were selected randomly in which 11836 households were eligible and 9047 adult persons aged 16-74 years were interviewed (response rate 76 percent). The nurses visited 7455 adults and of those 6178 gave a blood sample (response rate 52 percent), however a further almost 250 subjects agreed to give blood sample, but for some reasons blood sample was not obtained. Therefore the actual response rate for blood sample was 55 percent. The response rate for height measurements was 73%, for weight measurements was 71%, and for blood pressure was 62%. The characteristics of all

those who refused to give blood sample were not available in the SHS database to compare with the characteristics of those who gave blood sample. However, data from those how had valid BMI, but refused to give blood sample were available. The analysis of these data showed that in total and in both men and women the mean BMI between those who gave blood sample and those who refused was not significantly different, but women obese subjects were slightly more likely not to give blood sample.

2.1.2. Potential bias in SHS

Bias can be a major problem in an epidemiological study. It can be defined as a systematic deviation from the truth, which means the study produces an incorrect conclusion, either about the existence of an association or about its strength, and in a cross-sectional study bias can also result in a false estimate of prevalence of disease or health indicators (Silman & Macfarlane, 2002). The major source of biases can be either the study subjects or the process of information gathering and these issues should be considered in the design and conduct of the study as such problems cannot be solved by analysis.

Response rate in a population survey is an important factor affecting both the accuracy and precision of the prevalence estimate of risk factors and associations. A low response rate in a survey may cause inaccurate and imprecise of population estimators (Tolonen *et al.*, 2005). There is no exact cut-off point for the minimum response rate to be acceptable in a survey; however, if there is no significant difference for variable of interest between respondents and non-respondents then a low response rate is acceptable. If non-respondents are considerably different from respondents, then this can cause severe bias in the outcome even with a high response rate. Many studies have shown that respondents and non-respondent are different in terms of age, socio-economic and demographic status, lifestyles and other health-related

behaviours (Van Loon *et al.*, 2003; Tolonen *et al.*, 2005). Overall, non-respondents are more likely to be young, men, non-married, lower socio-economic status, practice more unhealthy behaviours and have poorer health status than the respondents (Van Loon *et al.*, 2003; Tolonen *et al.*, 2006). However, it is possible that in a cross-sectional study those with a disease are more likely to respond than those who are healthy (Silman & Macfarlane, 2002). In spite of some of the differences between respondents and non-respondents, some studies showed that these differences did not cause a major bias in the relationships between variables examined (Van Loon *et al.*, 2003; Boshuizen *et al.*, 2006). Tolonen (Tolonen *et al.*, 2005) and Jones (Jones, 1996) have suggested that response rates of 70% to 90% can be acceptable, depends on the likelihood of non-respondents having very different characteristics to the respondents. For postal surveys response rates rarely reach these levels. Interpretations of survey results must always be cautious, and never imply causality.

In SHS, the overall response, as mentioned before, is in a generally acceptable range, however the response rate provide for providing a blood sample was lower. Due to some features of the sampling design in SHS the sample of individuals selected did not have equal chances of selection and there were also different response rates by region, age, and sex. These inequalities of selection and non-response bias by region, age and sex at the first stage (interview) were weighted to match the age and sex distribution of the mid-1998 population estimates for Scotland. The weighted sample size for adults was close to the unweighted and it did not change any of the reported survey estimates. As the SHS comprised a number of multi-stage surveys (initials interview, nurse visit, and blood sample) the proportion of drop out was higher in the final stage. For the present study, in the first chapter, weighted data were used, however the results were almost identical using unweighted data, so these were used in all subsequent chapters.

It is either impossible or impracticable to perform an unbiased study, however it is possible to minimize the potential bias, with regard to available recourses, with a careful design and conduct of a study (Silman & Macfarlane, 2002).

2.1.3. Variables

2.1.3.1 Social class

Social class was based on the Register General's Standard Occupation Classification using the current or last occupation of the informant or the current or last occupation of the chief income earner within informant's household in different categories: professional, intermediate, skilled (non-manual), skilled (manual), partly skilled, and unskilled (Shaw *et al.*, 2000). For the social classification of the chief income earner, the details of the current or last occupation were recorded, for 96% of the total sample.

There are different methods for assessing socioeconomic status, however the most accepted method in UK is based on OPCS. This method may have some limitations specially when unemployment is high. In this study education levels has not been used as a confounding factor because it is believed that higher social class is strongly associated with greater education levels. Higher education normally leads to a higher occupation and better income that resulting a higher social class.

2.1.3.2. Cigarette smoking

In SHS information about cigarette smoking for adults aged 18-74 was collected by interview and for those aged 16 and 17 by means of a self-completion questionnaire. Cotinine, which is

a metabolite of nicotine in the body, was also measured in saliva in this survey. Cotinine is one of the biological markers of current smoking and has a half-life of 16-20 hours in the body. A salivary level of equal or over 15 ng/ml was defined as the cut-off for detecting current smokers in this survey. The half-life of this metabolite in the body means current smokers whose last cigarette was smoked more than a day ago cannot be detected.

In this survey 34% of men and 32% of women were self-reported current cigarette smokers. Apart from cigarette smoking, a further 4% of men smoked only pipes or cigars and not cigarettes. For the purpose of our study of tobacco smokers we excluded subjects who smoked pipes or cigars. Cigarette smoking status was classified as follows: regular cigarette smokers, those who said they smoked cigarettes at all at the time of the interview; ex-smokers, those who smoked cigarettes regularly in the past but not currently; and non-smokers: those who had never smoked cigarettes regularly and were not current smokers.

Saliva cotinine level, as an objective measurement, was used for validation of self–reported smoking behaviour. After cross-checking with cotinine levels, it was realised that there was some under-reporting of smoking behaviour. Under-reporting in men aged 25-74 years was between 2-4% and in women in this age group was 1-3%. The highest prevalence of under-reporting was seen in the age group 16-24 years for both genders (11 % in men and 7% in women). The self reported smoking was justified and used due to the limited amount of under-reporting and absence of over reporting.

2.1.3.3. Physical activity

Physical activity is a complex multidimensional form of human behavior that has shown beneficial effects on health. To evaluate the association between physical activity and health, an accurate method is needed for assessment of the different types and total amount of physical activity. Physical activity defined as 'any bodily movement produced by the contraction of skeletal muscles resulting in caloric expenditure' (Caspersen *et al.*, 1985). Most of the health benefits from physical activity are as a result of regular habitual physical activity performed over a period of time (Sjostrom *et al.*, 2004) and mainly involves legs. Total habitual physical activity usually comprised occupational physical activity, transport or moving from place to place, household domain, gardening, and leisure time and recreational physical activity. Apart from these types of physical activity, the frequency, the duration, and the intensity of each types of activity are important in assessment of total physical activity.

There are three types of physical activity assessment methods, which are subjective methods (activity diaries, questionnaires), objective methods (pedometers, accelerometers, heart rate monitoring) and criterion methods (doubly labeled water (DLW), indirect calorimetry and direct obsevation) (Vanhees *et al.*, 2005). Criterion methods are the most reliable and measurements from other methods should be validated against them. The DLW method is the most useful method for measuring long-term energy expenditure in free-living subjects. The basis of this method is the oral administration of two stable isotopes ^{2}H and ^{18}O as water (^{2}H $2^{18}O$). The rate of distribution of ^{2}H reflects water output whereas the rate of disappearance of ^{18}O reflects water out put plus carbon dioxide production rate, from which total energy expenditure can be calculated. This method is expensive and needs complicated analytical procedures, therefore it is not suitable for epidemiological analysis (Schoeller *et al.*, 1986; Vanhees *et al.*, 2005). It does not differentiate different types of activity, but provides an integrated figure over a 2-4 week period.

A variety of mentioned methods have been used to evaluate physical activity. The use of all of these methods, except for questionnaires, may be time-consuming and expensive. The most widely used methodological tool in assessing physical activity levels in population studies is the activity questionnaire, which is practical, applicable and accurate relative to other methods. Questionnaires are often the only feasible method of assessing habitual physical activity in large populations. Therefore the advantages of the self-reported measures are their ability to collect data from a large number of people at low cost and the fact that normally it does not change the behaviour of the subjects under study. It is possible to assess different types of physical activity simultaneously.

Physical activity questionnaires have been used in numerous studies and are closely related to the results obtained by some validated methods. A variety of physical activity questionnaires are available, many of which only identify leisure time physical activity or work activity.

In the SHS, physical activity was estimated by a questionnaire. The physical activity module is originally based on a major national study of activity carried out in 1990, the Allied Dunbar National Fitness Survey. The questionnaire used in this survey was the most widely used and respected physical activity questionnaire available in the UK, having been adopted by the Health Education Authority and Sports Council of England. The questionnaire asked about the frequency, duration and intensity of four major types of activity (activity at home, walks at least 15 minutes or more, sport and exercise, and activity at work) in the four weeks before the interview. These activities were then summed up to calculate estimated total physical activity, which was divided into five categories based on different levels of physical activity recommendations. Categories were: inactive, low activity, at least 30 minutes moderate activity on at least 5 days a week, at least 20 minutes vigorous activity on at least 3 days a

week, and 30 minutes moderate activity on 5 days a week plus 20 minutes vigorous activity on 3 days a week. These 5 categories were collapsed into 3 main categories: active, those who reached at least one of the two guideline levels (either 3 occasions of twenty minutes vigorous activity per week or 5 occasions of moderate activity per week or both); less active: those were not active enough to meet either guideline level but were active on at least one day a week; and inactive, those respondents who reported less than one day per week of moderate or vigorous activity of at least 20 minutes duration.

2.1.3.4. Dietary assessment

Dietary assessment is essential for investigating diet-health relationships, identifying high risk population groups, formulating food and nutrition policies, selecting appropriate nutrition intervention and for the monitoring of nutritional programs (Buzzard, 1994). There are different methods that are used to collect qualitative or quantitative information about food consumption at the national, household or individual levels. There is no single direct method to provide a true picture of dietary habits (Westerterp & Goris, 2002) and it has been shown that common methods have some errors to determine the usual intake, and the nature and magnitude of the errors depend on both the dietary data collection methodology and the subjects of the study (Beaton, 1994).

Food consumption at the national level is most frequently determined by using a food balance sheet. These sheets provide information on national per capita food availability, but give no information on food consumption at the individual level. Household food consumption methods measure food and beverages available for consumption by a household family group or institution during a specific time period (Gibson, 1990). There are two main approaches to

individual dietary assessment; prospective and retrospective. Prospective methods such as weighed intake or estimated food records involve collecting or recording current diet while retrospective methods like 24-hour-recall, food frequency questionnaire and diet history requires subjects to recall either recent or past diet.

There are some advantages and disadvantages in these methods. The main advantages of retrospective methods are that they are quick and cheap, they need low subject motivation and lower literacy than prospective methods. The main disadvantage of retrospective methods is that they rely on memory and this is a major problem to remember the accurate frequency of consumption and food portion size especially in elderly and children (Nelson & Bingham, 1997). Prospective methods provide a direct measure of current diet, the length of recording can be varied to suit study needs and daily variation can be described. Being expensive, time-consuming and requiring respondent skills and literacy are the main limitations of prospective methods (Nelson & Bingham, 1997).

2.1.3.4.1. 24 hour recall

In this method the respondent is asked by a trained interviewer to recall the actual food and drinks consumed during the previous 24 hours. Details of all food and beverage consumed including cooking methods, brand names, and vitamin and mineral supplementations are recorded (Gibson, 1990). The main advantages of this method are speed and ease of administration and this allows large number or subjects to be interviewed. For these reasons 24 hour recall is commonly used in some large-scale studies. Other strengths of this approach are low respondent burden and costs. Multiple 24-h recall can use to estimate usual intake of the individual. As mentioned, this method relies on memory, and a single 24-h recall cannot

measure day-day variations and selective omission of foods eaten may occur (Gibson, 1990; Nelson & Bingham, 1997; Dwyer, 1999).

2.1.3.4.2. Food Frequency Questionnaire (FFQ)

FFQ is commonly used to collect qualitative or semi-quantitative usual food consumption patterns. They are designed to assess the frequency of consumption of certain food items or food groups which are consumed during a specific time period (e.g. weekly, monthly). The list of food items in FFQ vary from very short questionnaire with only nine food items for assessing a single nutrient (Calcium) to very long items (276 items for national study of diet and heart diseases) (Nelson, 2000). The FFQ is one of the principal dietary survey methods in epidemiology studies of chronic diseases. The main advantages of this method include the ease and uniformity of administration, which can overcome problems of interviewer bias, relatively low costs, high response rate, a low respondent burden and repaid analysis. The main limitations of this method are: validation of this method is difficult, lists compiled for the general populations are not useful for subgroups, estimation of total consumption is difficult, error in estimating portion size, respondent burden rises as the number of food item increases (Gibson, 1990; Teufel, 1997; Dwyer, 1999).

2.1.3.4.3. Diet History

In this method the respondent is questioned about typical or usual food intake over the recent past. This method provides a more complete and detailed description of both qualitative and quantitative aspects of food intake and relatively eliminates individual day-to-day and seasonal variations. In contrast this method is very dependent upon the skill of the interviewer, so a highly trained interviewer is often required. It is also difficult to standardize this method because of variability among interviewers in how they carry out the process. Like

other retrospective methods it is reliant on the subjects memory. Because of the high cost and face-to-face interview this method is less commonly used in epidemiology, but it is frequently used in clinics (Nelson & Bingham, 1997).

2.1.3.4.4. Food records

A weighed food record is the most precise method available for estimating usual food and nutrient intake of individuals. In this method all foods and beverages before consumption and any leftovers must be weighed or estimated using household measures of portion sizes. These methods have been used for validation of other methods in many studies, however it has been suggested that this method itself required an external reference for validation (Dwyer, 1999). This method needs highly motivated subjects and is time-consuming. In addition, in this method participants can change their usual diet patterns, and misreading and misreporting, together within high rate of dropouts make this method quite difficult.

2.1.3.4.5. Common errors in dietary assessment

Like the other epidemiology studies, there might be two types of error in dietary data, bias and random error. Random error affects the precision of a method and in theory can be minimized by increasing the number of observations, but it does not have any effect on systemic error. The major sources of error in dietary assessment are respondent biases, coding errors, wrong weighed food, reporting error, variation with time, wrong frequency of consumption and changes in diet (Bingham, 1987). Giving socially desirable answers to interviewers or researcher is another important source of respondent bias, which may cause overestimation of the intake of some foods like fruits and vegetables and underestimation of some other foods like fast food snacks. Physical and psychological characteristics of subjects play an important role in this observed reporting bias (Johansson *et al.*, 1998; Westerterp & Goris, 2002).

Misreporting, specially under-reporting of energy intake is common among participants and the amount of under-reporting among obese subjects is , almost twice as high as in the normal weight subjects (Westerterp & Goris, 2002).

2.1.3.5. Dietary assessment in SHS

In the SHS the information on dietary habits was obtained by a short dietary questionnaire, which is called the 'Dietary Target Monitor' (Lean *et al.*, 2003). This questionnaire was designed specifically to provide information about eating behaviour to evaluate the Scottish Dietary Targets (The Scottish Office, 1996). It was validated against a FFQ (Bolton-Smith & Milne, 1991), which had been used in the Scottish MONICA project. This questionnaire included questions relating to type and frequency of major food items and contained questions relating to the frequency of consumption of fruits and vegetables (including fresh, cooked, frozen), starchy foods (including bread, breakfast cereals, potatoes, pasta and rice), fish intake, chips, meat and meat products, cheese, milk, sweets or chocolate, ice cream, crisps, savoury snacks, soft and fizzy drinks, cakes, scones, sweet pies or pastries and biscuits.

The frequency of consumption of foods were divided into six or more times a day, four or five times a day, two or three times a day, once a day, five or six times a week, two to four times a week, one to three times a month, less often or never. This questionnaire has been validated against the very widely used FFQ (Bolton-Smith & Milne, 1991) for three key food groups: fruit and vegetables, starchy foods and fish. (Lean *et al.*, 2003) and different equations were produced to estimate intake of these three food groups. In the validation study it was assumed that 'times' could be equated to 'portion' and then they calculated the average portion size for fruit and vegetables, starchy foods and fish intake (Lean et al., 2003). For the estimation of

the total fruit and vegetable intake, total frequency of fresh fruit, cooked green vegetables (fresh or frozen), cooked root vegetables (fresh or frozen) and raw vegetables or salad (including tomatoes) was multiplied 1.33 and 80 to generate a g/day figure. In this study total fruit and vegetable consumption was used as a categorical variable divided in three groups, low consumers (< 200g per day), moderate consumers (200 - < 400g per day) and achievers of current targets (≥ 400 g per day) (The Scottish Office, 1996).

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Consumption of starchy foods (portion per day) was estimated by total starchy food from FFQ (sum of frequency of bread, breakfast cereals, potatoes, pasta and rice) multiplied by 8 for men and 6.4 for women before division into tertiles.

Estimation of fish intake (g/week) was made by the multiplication of the sum of frequency of fish intake in FFQ by 0.99 and 120. Total fish intake (g/week) was categorized into three groups: low consumers (0-239 g/week), moderate consumers (240-359 g/week) and high consumers (≥ 360 g/week).

It is difficult, if not impossible, to assess the true food intake of free-living subjects especially in a large sample. Although FFQ is not a perfect method to measure dietary intake and has own limitations and errors, it is a useful and easy tool in nutritional surveys. As the FFQ that was used in SHS was not designed to collect full nutrient intake, therefore, it therefore had limitations to evaluate food intake in this study. However, speed and ease of use were the main advantages of the questionnaire.

2.1.3.6. Body composition

Body composition is one of the common methods to evaluate nutritional status and in humans it can be evaluated at five levels: atomic, molecular, cellular, tissue and whole body level (Wang *et al.*, 1992). At the atomic level, common elements in the body such as oxygen, carbon, hydrogen, nitrogen, calcium and phosphorus can be measured by different techniques. The major components at a molecular level are water, protein, lipid and minerals. At the cellular level three main compartments construct human body, which are different kind of cells, extracellular fluid, and extracellular solids. Variations in body weight at the tissue level arise from variations in adipose tissue, skeletal muscle, bone, viscera, blood and others. These may vary in opposite directions. Many measurement methods use the '2-compartment' method, which assumes that body water is constant in healthy subjects. In the whole body level of body composition different dimensions of whole body such as size, shape and other physical characteristics are considered. All of the mentioned levels can be measured in clinical and research settings with different levels of accuracy and precisions. Different in vitro (such as anatomical dissection and chemical analysis), and in vivo methods (such as densitometry, hydrometry, dual energy x-ray absorptiometry (DEXA), magnetic resonance imagining (MRI), bioimpedance analysis (BIA) and anthropometry) are available (Norgan, 2005). Many of these methods are laboratory methods, requiring technical support, expertise and are expensive to carry out. Densitometry and hydrometry are the most commonly used two components techniques, which measure fat mass (FM) and fat free mass (FFM).

2.1.3.6.1. Anthropometry

In nutritional epidemiology anthropometry is an important element and body size apart from genetic effect typically is a sign of a cumulative exposure to diet (energy balance) and illness (Sjostrom *et al.*, 2004). Anthropometric methods can be validated against criterion methods to

provide estimates of body composition (e.g. fat mass, lean body mass). They are also widely used as indicators of associated risks of morbidity and mortality. The most commonly used measurements are height, weight, body mass, circumferences especially waist and skin fold thickness. It is worth noting that BMI (kg/m^2) is relatively poor indicator of body composition, although it has been used as the basis for defining obesity internationally. Small changes in muscle mass can have large effect on BMI, especially in non-obese individuals. However, for the assessment of obesity-related metabolic risk at the population level, BMI and WC are important indirect indices of obesity (Bosy-Westphal *et al.*, 2006).

For historical reason, SHS reported WHR, not WC or HC separately. WHR has no biological meaning and little association with body composition, but it does relate to some health outcomes.

2.1.3.6.2. Anthropometric measurements

In the SHS interviewers measured height and weight, and nurses measured WC, HC and demi-span. Height was measured to the nearest millimetre with a portable stadiometer in bare feet with a standing position in the Frankfort plane. For subjects who could not stand straight or were unsteady on their feet demi-span, which is an alternative to height as a measure of skeletal size in elderly people was measured. Body weight was measured to the nearest 0.1 kg in bare feet and light clothes with the Soehnle scales. Informants who were pregnant, chair bound, or unsteady on their feet were excluded from the measures. As the scale was inaccurate above 130 kg, the weight over this was estimated by the participants. WC was measured midway between the lateral lower ribs and iliac crests. HC was defined as being the widest circumference over the buttocks and below the iliac crest. WC and HC were measured to the nearest millimetre. WC and HC measurements were made at least twice and the mean value was used. BMI was calculated as weight divided by height squared (kg/m^2).

Overweight and obesity were defined as a BMI of 25 - 29.9 and ≥30 kg/m^2 respectively. WHR was calculated as WC divided by HC.

2.1.3.7. Blood samples

Trained nurses took non-fasting venous blood samples from 84.4% of men and 80.1% of women that they visited. The samples were sent to the Royal Victoria Infirmary (RVI) in Newcastle-upon-Tyne and a small sub-sample of blood samples was sent to the Institute of Food research in Norwich. All analyses were carried out according to Standard Operating Procedures by State Registered Medical Laboratory Scientific Officers (MLSOs). Both blood and saliva analytes were checked with internal quality control and external quality assessment and the results were within expected limits.

Fasting blood sample is more appropriate for measuring many CVD risk factors because different factors including different type of diet and drink and supplements may have considerable influence on the composition of plasma. A high fat diet increases the serum concentration of triglyceride, however the ingestion of different amount of cholesterol may have little effect on the serum cholesterol concentrations. Epidemiological studies like population survey generally need a simple but unbiased measurement of participants. The practical difficulties of obtaining fasting blood sample in population survey have resulted in using non-fasting blood sample. Although this may raise questions about the accuracy of the measurements particularly in clinical setting, some studies showed that total cholesterol, HDL-C and non-HDL-C would be reliable in non-fasting blood sample.

C-reactive protein (CRP) was measured using the N Latex CRP mono Immunoassay on the Behring Nephelometer II Analyzer by the Biochemistry Department at the Royal Victoria Infirmary (RVI). Total cholesterol was measured using the DAX Cholesterol Oxidase assay method calibrated to Center for Disease Control (CDC) guideline at the RVI. HDL-cholesterol was measured using the DAX Cholesterol Oxidase assay method calibrated to CDC after PTA precipitation at RVI. Fibrinogen was measured using the Organon Teknika MDA 180 analyser and a modification of the clauses thrombin clotting method by the Department of Haematology. Non-HDL-C, which contains cholesterol in LDL and VLDL calculated by subtracting HDL-C from total cholesterol (Grundy 2002). Cotinine was measured using a Hewlett Packard hp5890 gas chromatograph machine, with a rapid-liquid chromatography technique by the Nicotine Laboratory at New Cross Hospital, London.

2.1.3.8. Data analysis

Analysis was carried out using the statistical package, SPSS 11.0 (SPSS INC., Chicago, IL, USA). For analyzing data, based on distribution of the data, there are two main methods, either parametric and non-parametric methods. Parametric methods are based on the assumption that the data are a sample from population with a normal distribution. Data that are not compatible with a normal distribution can often be transformed to make them acceptably near to normal. A useful method in such situation is the logarithmic transformation, and in practice, common logarithms, to base 10, are used. In this study, many of the studied variables were skewed and were not normally distributed and log- transformed values of dependent variables were used for improving the normality of distributions and the log transformed mean or geometric mean, was used. Formal tests for normality suggested

non-normal distribution for anthropometric data. (e.g. BMI). The deviation from normality was however very small (a positive skew, as in all similar surveys). In chapter 3 log-transformed anthropometic data were used. However, there were no differences in the conclusion if non-transformed data were used. Therefore, the remaining chapters used non-transformed data, as have all other published studies of this kind, so that the results can more accessible to readers.

A general linear model (GLM) was used to compare the adjusted geometric means of the measured risk factors among different independent factors. This analysis often was stratified by gender and controlled for covariates in the univariate GLM. The GLM incorporating Bonferroni *post hoc* test was used to compare the adjusted geometric means of the dependent variables within different categories of the independent variables. The GLM procedure can describe the relationship between a dependent variable and a set of independent variables.
The logistic regression model was used to compute the odds ratio (OR) and 95% confidence interval (CI) for the probability of having high value for a dependent variable among the subgroups of the independent variables with the reference category. A value of $p<0.05$ was used for statistical significance.

2.2. Smoking cessation study

2.2.1. Subjects and methods

All those who participated in a Health Service funded smoking cessation program based in the community in East Kilbride, North Lanarkshire were invited to participate in the study. The

"Stop Smoking Programme" was delivered at no cost by trained smoking cessation facilitators.

The programme comprised 7 weeks of "closed" group meetings and a further 5 weeks of NRT dispensed by a pharmacist. The weekly sessions lasted approximately 1 hour and participants were asked to attend all sessions. Sessions were conducted in a group setting where participants quit with others who were also attempting to stop smoking in a very informal and friendly atmosphere. Trained smoking cessation advisors assisted the groups and provided participants with information and advice about how to stop smoking and remain a non-smoker. The advisors assessed participants' suitability for NRT and helped them decide which was the most suitable product for them. NRT was prescribed monthly and participants collected their prescriptions on a weekly basis from the pharmacy to ensure access to a pharmacist and to allow any problems participants may have experienced with their product during the week to be addressed. A number of different NRT products were used within the smoking cessation program including gums, inhalers, nasal sprays, mictrotabs and zyban, and the nicotine patch was the frequently used.

The first two sessions (week 1 and 2) of the programme were preparing to quit sessions in which the advisors explained the whole programme, gave the participants information about the different NRT, measured participant's respiratory carbon monoxide (CO) and defined their type of NRT products. The third session was the "quit week" when smoking cessation commenced and the following sessions 4, 5 and 6 were support sessions where coping strategies and future expectations were discussed before the sessions ended at week 7. Additional information about smoking cessation, some general helpful hints about healthy

lifestyles such as healthy diet, physical activity, and weight change were given to the participants during the session's meetings.

For the purpose of this study, additional anthropometric data, measures of dietary intake, physical activity, eating behaviors and some other general demographic information was collected at the end of the class. All measurements were on made four occasions, baseline, week 7, week 12 (completion of NRT) and at week 18, six weeks after the completion of the program.

2.2.2. Anthropometric measurements

Body weight was measured by a portable digital scale in kilograms to the nearest 0.1 kg while the subjects wore light clothes and were bare feet. Height was measured with a portable stadiometer in bare feet with standing position. Waist circumference (WC) was measured midway between the lateral lower ribs and iliac crests with an inelastic measuring tape in centimeters. Hip circumference (HC) was defined as being the widest circumference over the buttocks and below the iliac crest and triceps skin-fold thickness was measured with a skin caliper (Holtain LTD. Crymych U.K.) in millimeters. Height, WC and HC were measured to the nearest millimeter. All of the measurements were made in duplicate and the mean value was used. BMI was calculated as body weight divided by height squared (kg/m^2).

2.2.3.Questionnaires

2.2.3.1. Dietary intake

The dietary questionnaire used in the study was DIETQ food frequency questionnaire (FFQ) (DIETQ V4.1 Tinuviel Software), which was self-administered and, measured food intake

retrospectively. The questionnaire contained a list of different foods that were divided into the following major food groups of: bread; breakfast cereals; meats; fish; vegetables and savoury dishes; biscuits, cakes and puddings; fruit; eggs and milk products; fats; and drinks; and additional questions about height, weight and physical activity. Respondents were asked to state how often they usually consumed each item. Quantitative estimates of the quantity consumed were obtained for some items e.g. the number and the size of slices of bread/day, the amount of milk/day, the number of eggs/week, the number of fresh fruits/week and the amount of butter, margarine, cheese and cream/week. For other items an average portion size was used, e.g. meats, fish and vegetables. From the reported frequencies of foods and drinks an average daily intake of foods, energy and major nutrients were calculated by DIETQ software (DIETQ V4.1 Tinuviel Software).

2.2.3.2. Physical activity

The International Physical Activity Questionnaire (IPAQ) long form instrument was used to estimate the physical activity that participants do as part of their everyday lives. Like the FFQ, participants were asked to report their usual physical activity at baseline, week 7, 12 and 18 using this questionnaire. The questionnaire assessed physical activity undertaken across a comprehensive set of domains including walking, moderate-intensity and vigorous intensity activity within each of the works, transportation, domestic chores and gardening (yard) and leisure-time domains. In this questionnaire, frequency (days) and duration (in minutes) were asked for each domain. Data collected with IPAQ have been expressed as a score in MET minutes by weighting each type of activity by its energy requirements defined in MET minutes. Then energy expended on physical activity every day was calculated from MET-

minute using the following equation: MET min multiplies weight in kilograms divided 60 kilograms.

2.2.3.3. Eating behaviors

Eating behavior was assessed by using a three-factor eating questionnaire revised 18-item (TFEQ-R18) version (Karlsson *et al.*, 2000). The questionnaire referred to current dietary practices and contained 18-items. The TEEQ-R18 measures 3 aspects of eating behavior: cognitive or dietary restraint (6 items), uncontrolled eating (9 items) and emotional eating (3 items). It is a self-assessment questionnaire containing multiple-choice answers and the range of possible scores was from 6 to 24 for restraint, from 9 to 36 for uncontrolled eating and from 3 to 12 for emotional eating, with higher scores representing higher levels of the behaviors. The sum of scores of the each three factors were divided into tertiles, low, medium and high levels of the specific eating behaviours.

Dietary under-reporting was defined by means of the energy intake to basal metabolic rate ratio (EI:BMR) (Goldberg *et al.*, 1991). Basal metabolic rate was calculated using the Schofield equation, and a ratio cut-off point of ≤ 1.35 was chosen for lower-energy reporting and > 2.82 for high-energy reporting.

Other information related to smoking behaviors, including alcohol intake, satisfaction about body weight and shape, dieting at the time of the study, food preferences; attempts to quit in the past and general demographic information were obtained by a general questionnaire.

2.2.4. Data analysis

Analysis was carried out using the statistical package, SPSS 11.0 (SPSS Inc., Chicago, Illinois, USA). As some of the data were not normally distributed both parametric and nonparametric analysis were used. For each outcome a mean and median level was calculated for the different stages of the study (baseline, week 7, 12 and 18). Mean and median levels of changes were also calculated for the measures at different of the stages. Data were analyzed by repeated-measures ANOVA and Friedman test to assess the impact of smoking cessation using NRT on outcome measures during the program. For comparing the changes in each stage of study with baseline or other stages, paired t-test and Wilcoxon signed-rank test were used.

Chapter 3: Relationships between cigarette smoking, body size and body shape.

3.1. Introduction

Tobacco smoking and obesity are the leading causes of preventable death (Mokdad *et al.*, 2004). Tobacco alone contributes 4.9 million deaths world wide annually and is one of the most important causes of morbidity - mainly cardiovascular disease, respiratory disease and cancers (WHO, 2002). Obesity has been estimated to cause 385-500,000 deaths annually in USA (Mokdad *et al.*, 2004). A multiplicative interaction between the risks of smoking and overweight are well established in the aetiology of coronary heart disease (Manson *et al.*, 1990).

Despite awareness of the detrimental health effects of cigarette smoking, many people either prefer to smoke or are unable to cease. Aside from the addictive properties of nicotine, one possible explanation for the continuation of smoking is the belief that it can assist in controlling body weight (Wee *et al.*, 2001; Fulkerson & French, 2003). An increased awareness of the adverse health effects of obesity and overweight together with peer pressure to encourage slimness may encourage the use of smoking as a means of weight control by young adults (Wee *et al.*, 2001). However, the catastrophic increase in health risks when smoking and overweight are combined may not be widely understood (Peeters *et al.*, 2003).

Nicotine addiction is a multifactorial process which influences body weight (Heishman, 1999). Most of studies have shown a negative relationship between smoking and body weight (Albanes *et al.*, 1987; Eisen *et al.*, 1993; Flegal *et al.*, 1995; Hu *et al.*, 2002). However, direction and strength of the association varies considerably among adult populations, according to socio-economic status and smoking duration (Marti *et al.*, 1989; Molarius & Seidell, 1997; Molarius *et al.*, 1997; Oh & Seo, 2001). Studies on-biracial African-American

and white teenagers and younger adults have suggested that in younger people cigarette smoking has minimal effects on body weight control, and may encourage weight gain (Klesges *et al.*, 1998a; Klesges *et al.*, 1998b; Cooper *et al.*, 2003). Furthermore some studies (Barrett-Connor & Khaw, 1989; Shimokata *et al.*, 1989; Lissner *et al.*, 1992; Visser *et al.*, 1999b) have found that smokers have an abnormal body fat distribution, with more central adiposity than non smokers. Early studies measured waist to hip ratio (WHR) to evaluate body shape (Barrett-Connor & Khaw, 1989; Lissner *et al.*, 1992). However, WHR is a poor indication of body fat distribution as measured by CT or MRI scanning (Han *et al.*, 1997). Waist and hip circumferences (WC, HC) measure different aspects of body fat distribution (Snijder *et al.*, 2003) and show independent and opposite effects on cardiovascular risk factors, which are not reflected properly by WHR (Seidell *et al.*, 2001). Thus, closer examinations of the relationships between smoking, BMI and body shape using WC and HC separately in a representative sample of adults with a high prevalence of cigarette smoking are justified and presented in the present study.

3.2. Subjects and methods

3.2.1. Sample

The Scottish Health Survey (SHS) 1998 is a cross sectional nationally representative survey that was designed to provide a comprehensive picture of the health of the Scottish population both to document the prevalence of health risk factors and monitor progress towards health targets. A total of 9047 adults aged 16-74 participated in the 1998 survey, an overall participation rate of 76%. All data were weighted by the inverse of the probability of selection for sector, the address within sector, the household at the address and the individual within the

household. Full details of the survey methods have been published elsewhere (Shaw *et al.*, 2000).

3.2.2. Anthropometric measures and lifestyle factors

Weight, height, WC and HC were measured using standard techniques by trained staff (Shaw *et al.*, 2000). Height was measured with a portable stadiometer in bare feet with a standing position. For subjects who could not stand straight or were unsteady on their feet demi-span, which is an alternative to height as a measure of skeletal size, was measured. Body weight was measured to the nearest 0.1 kg in bare foot and light clothes with Soehnle scales. WC was measured midway between the lateral lower ribs and iliac crests. HC was defined as being the widest circumference over the buttocks and below the iliac crest. Height, WC and HC were measured to the nearest millimetre. WC and HC measurements were made at least twice and the mean was used. BMI was calculated as weight divided by height squared (kg/m^2). Overweight and obesity were defined as a BMI of 25 - 29.9 and ≥ 30 kg/m^2 respectively (WHO, 1998). WHR was calculated as WC divided by HC.

Respondents cigarette smoking status was classified as regular cigarette smokers: those who said they smoked cigarettes at all the time of the interview, ex-smokers: those who smoked cigarettes regularly in the past but not currently, and non-smokers: those who had never smoked cigarettes regularly and were not current smokers. Levels of physical activity were measured by a questionnaire which asked about the frequency, duration and intensity of four major types of activity: activity at home, walks of 15 minutes or more, sports and exercise activities, and activity at work in the four weeks before the interview. These activities were then compiled to calculate overall physical activity and divided in to five categories based on different levels of physical activity recommendations which are: inactive, low activity, at least

30 minutes moderate activity on at least 5 days a week, at least 20 minutes vigorous activity on at least 3 days a week, and 30 minutes moderate activity on 5 days a week plus 20 minutes vigorous activity on 3 days a week. These 5 categories were collapsed into 3 main categories: active, those who reached at least one of the two guideline levels (either 3 occasions of twenty minutes vigorous activity per week or 5 occasions of moderate activity per week or both); less active: those were not active enough to meet either guideline level but were active on at least one day a week; and inactive, those respondents who reported less than one day per week of moderate or vigorous activity of at least 20 minutes duration.

Alcohol consumption over the previous 12 months was assessed using questions on frequency, type, average number of days per week on which alcohol was drunk, the usual quantity consumed on any one day and the finally the weekly units of alcohol consumed calculated. This was then divided into four groups of weekly alcohol intake for both men and women based on recommendations for alcohol drinking. For men, these quantities were under 1 unit (as never drinkers or occasionally drinkers), 1-10 units (as low drinkers), 10-21 units (moderate drinkers) and over 21 units (as heave drinkers). For women these were under 1 unit (as never drinkers or occasionally drinkers), 1-7 units (as low drinkers), 7-14 units (as moderate drinkers) and over 14 units (as heave drinkers).

Social class was based on the Register General's Standard Occupation Classification using the current or last occupation of the chief income earner within the informant's household, in one of six categories: professional, intermediate, skilled (non-manual), skilled (manual), partly skilled or unskilled.

3.2.3. Data analysis

Analysis was carried out using the statistical package, SPSS 11.0 (SPSS INC., Chicago, IL, USA). Analyses were stratified by gender and age group. The majority of response variables showed a skewed distributions and logarithmic transformations were carried out. Mean BMI, WC, HC, WHR, total units of alcohol and physical activity by smoking categories were calculated and the differences were tested by analysis of variance (ANOVA) and Bonferroni post hoc test. General linear model (GLM) was used to compare the adjusted means of the anthropometric indices among different smoking categories in the whole population and age groups. BMI was adjusted for age (except for stratified analysis by age groups), social class, physical activity and alcohol consumption. For WC, HC and WHR, an additional adjustment for BMI was carried out. The data were expressed as the mean and 95% confidence interval. Statistical significance was set at $p<0.05$ for all tests.

3.3. Results

3.3.1. Smoking status and BMI

Of the 9047 respondents, 8125, (50.1% male, 49.9% female), had a valid BMI. Approximately 34% of male and 32% of females were current cigarette smokers, and 22% of male and 18% of female were ex-smokers. The proportion of current smokers fell with increasing the age in contrast to ex-smokers. Figure 1 and 2 reports the prevalence of cigarette smoking, which is higher among obese subjects in the youngest age group but falls with increasing age in both sexes.

Table 1 presents unadjusted mean values for anthropometric data, alcohol consumption and physical activity according to cigarette smoking status and sex. Current smokers had significantly lower, and ex-smokers had significantly higher mean BMI compared to non-

smokers in both sexes ($p<0.001$). Table 2 shows that after adjusting for social class, physical activity, age (for all ages) and alcohol, current smokers are still significantly leaner than non and ex-smokers in total ($p<0.001$), and in men aged over 25 years and in women aged over 55 years ($p<0.05$). Although ex-smokers had the highest mean BMI overall in both sexes ($p<0.05$), there were no significant differences in BMI between ex-smokers and non-smokers of any age group.

3.3.2. Smoking status and body shape

Cigarette smoking in men was associated with a significantly smaller unadjusted mean WC compared to non and ex-smokers ($p<0.001$) (table 1). Unadjusted mean WC in women current smokers was significantly smaller than in women ex-smokers ($p<0.001$), but was not significantly different to non-smokers. WC co-varies with BMI since both are correlated with body fat content (Lean *et al.*, 1996). Mean WC adjusted for social class, physical activity, alcohol consumption, age (for all ages) and BMI in men was significantly different only in those aged 55-64 years amongst whom current smokers had significantly larger WC than non-smokers ($p<0.05$) (table 3). In contrast, women current smokers had a significantly larger WC for entire sample ($p<0.001$) and in those aged between 35- 64 years ($p<0.05$) compared with non-smokers.

Total unadjusted mean HC was significantly higher among ex- smokers and lower among current smokers when compared with non-smokers for both men and women ($p<0.001$) (table 1). Table 4 reports mean HC adjusted for social class, physical activity, alcohol consumption, age (for all ages) and BMI. Examining all smokers as one group showed they had a smaller mean HC than non-smokers ($p<0.001$) for both sexes and than ex-smokers among women

($p<0.001$). Current smokers aged 35-44 years (men) and 16-54 years (women) had a significantly smaller mean HC than non-smokers ($p<0.05$). Ex-smokers aged 16-24 years (men) and 25-54 years (women) had a significantly larger mean HC than that in current smokers ($p<0.05$).

Unadjusted mean WHR for the entire sample was significantly higher among ex-smokers than current or non-smokers in men ($p<0.001$) (table 1). For women, both current and ex-smokers had significantly higher unadjusted mean WHR than non-smokers ($p<0.001$). Women current smokers had a higher adjusted mean WHR than non and ex-smokers ($p<0.001$) (table 5). Women smokers in all age groups, excepting 16-24 years, had significantly higher WHR than non-smokers ($p<0.05$). There were no such differences amongst male smokers.

Alcohol consumption among current smokers was significantly higher than amongst non-smokers and ex-smokers for both sexes ($P<0.001$) and ex-smokers consumed more alcohol than non-smokers only in men ($p<0.001$) (table 1).

Physical activity was lower among male, but not women, current smokers than non-smokers ($p<0.001$) (table 1).

3.4. Discussion

3.4.1. Smoking and BMI

The present study examined the relationships between cigarette smoking status and indices of both weight and shape. The finding that cigarette smokers are generally leaner than never smokers of the same age and sex is in agreement with most previous studies (Albanes *et al.*, 1987; Marti *et al.*, 1989; Eisen *et al.*, 1993; Flegal *et al.*, 1995; Hu *et al.*, 2002). Molarius et al

(Molarius *et al.*, 1997) studied 42 WHO MONICA populations for both men and women and found that regular smokers had significantly lower BMI in 20 (male) and 30 (female) of the 42 populations studied. For some populations there was no association between smoking and body weight. The MONICA populations varied widely in smoking prevalence, and in some of the study populations where smoking was most prevalent, smokers were considerably leaner than never smokers. In contrast, other studies (Albanes *et al.*, 1987; Molarius *et al.*, 1997; Oh & Seo, 2001) have reported that smoking intensity was positively associated with BMI. The reasons for the positive association between heavy smoking and higher BMI remain unclear (Albanes *et al.*, 1987; Molarius *et al.*, 1997; Oh & Seo, 2001) but could be related to negative lifestyle factors including increased frequency of high alcohol consumption (Albanes *et al.*, 1987; Molarius *et al.*, 1997; Oh & Seo, 2001), and physical inactivity, or to the different social classes represented amongst smokers compared with non smokers.

Animal studies have demonstrated that nicotine decreases appetite and energy intake (Blaha *et al.*, 1998; Miyata *et al.*, 2001). Epidemiological studies in adults have shown that the habitual energy intake of smokers is equal or greater than non-smokers and the diet of smokers was more energy dense than that in non-smokers (Dallongeville *et al.*, 1998). Studies of food consumption are usually confounded by mis-reporting (Subar *et al.*, 2003), but cigarette smoking also increases resting metabolic rate. This effect is mediated through nicotine increasing sympathetic nervous system activity and increasing thermogenesis in adipose tissues at least in rodent studies (Yoshida *et al.*, 1999). Perkins (Perkins, 1992b) concluded that cigarette smoking increases whole body metabolism rather than changing energy intake or physical activity levels. However, assessment of energy intake is notoriously confounded by mis-reporting in people with high BMI (Subar *et al.*, 2003). The present study did not attempt to quantify energy intake, but found that male cigarette smokers drank more alcohol and were

less active than non-smokers. The differences in BMI between smokers and non-smokers in this study persisted after adjustment for these lifestyle practices.

3.4.2. Association of Smoking and BMI in younger adults

Age is an important modifying factor for the association between smoking and BMI (Marti *et al.*, 1989). Smokers usually start below the age of 25, and former smokers are necessarily older. BMI tends to increase up to the age of 60 or 70 years. There are also secular trends such that BMI is higher at every age now compared to previous generations, but particularly in younger groups. These inverse relationships between smoking and relative body weight tend to be stronger in older than younger subjects. This may be explained by the influence of a longer smoking duration in older people who may have attained a lower body weight (Molarius *et al.*, 1997). Adolescent and younger adults' smoking initiation and maintenance has been related to weight concern in other studies (Klesges *et al.*, 1998a; Wee *et al.*, 2001; Fulkerson & French, 2003). However, available evidence does not indicate that smoking offers immediate weight control or long term reduction effects. African-American and white teenagers and young adults (Klesges *et al.*, 1998a; Klesges *et al.*, 1998b; Cooper *et al.*, 2003) in both cross sectional and prospective studies have shown no weight control benefit from cigarette smoking. As the majority of smokers start as teenagers, the present results support the view that younger people with weight problems are more likely to start smoking and also that smoking is ineffective as a weight control strategy at least in the short term. In the younger age group the present data cannot exclude an effect from smoking in promoting weight gain in young people, but this would be inconsistent with the data in older age groups. It seems more likely that younger people who recognise a weight problem are lured into smoking in an (unsuccessful) attempt to control the problem. However, this study examined

data from a cross sectional study which only allows associations to be identified, and it does not confirm any causal relationships between smoking status and body weight.

3.4.3. Smoking cessation and BMI

Although ex-smokers in this study had a higher BMI compared with non-smokers before and after adjustment for confounders, there were no significant differences in BMI between former and never smokers in any of the age groupings. In the MONICA project ex-smokers had a significantly higher BMI than never smokers in only 10 out of 42 populations among men, whilst among women there was no consistent pattern (Molarius *et al.*, 1997). Although smoking cessation is known to be accompanied by weight gain acutely (Williamson *et al.*, 1991; O'Hara *et al.*, 1998), increased body weight may not remain in the longer term (Chen *et al.*, 1993; Mizoue *et al.*, 1998). Chen et al (Chen *et al.*, 1993) reported that BMI, especially in women, decreased with increasing duration after cessation. Mizoue et al (Mizoue *et al.*, 1998) used cross-sectional data from work-site health examinations in Japan to show that when "light" and "moderate" smokers (<25 cigarettes per day) stopped smoking they gained almost the same amount of weight as never smokers. Weight gain after smoking cessation can plausibly be attributed to a number of interrelated changes: increased energy intake including fat and sugar rich foods, increased alcohol consumption, decreased metabolic rate and energy expenditure (Talcott *et al.*, 1995). A study of the mechanisms that contribute to weight gain has suggested that smoking cessation was associated with significant changes in adipose cell metabolism in which the adipose tissue lipoprotein lipase (AT-LPL) activity increased. This increase in LPL activity may contribute to the increase in body weight associated with smoking cessation (Ferrara *et al.*, 2001).

The present study showed that ex-smokers as a whole were less active and consumed larger quantities of alcohol than non-smokers. These differences were greatest in men. This suggests that although smoking cessation is accompanied by weight gain, the quantity of weight (0.9 kg/m^2 in men and 0.6 kg/m^2 in women compared with non-smokers, and 2.1kg/m^2 in men and 1.6kg/m^2 in women compared to current smokers) is insufficient to compromise the health benefit of cessation. Opportunities exist for anticipated weight gain to be addressed by the adoption of a healthier lifestyle.

3.4.4. Smoking status and body shape

The WC and HC were used in this study as indices of body shape and fat distribution. In line with other studies that have found smokers have higher WHR (Barrett-Connor & Khaw, 1989; Shimokata *et al.*, 1989; Lissner *et al.*, 1992; Visser *et al.*, 1999b); WHR was significantly higher in smokers than non-smokers for women probably due to both higher waist and lower HC, to which lower leg muscle mass may contribute. In men despite their predisposition central adiposity they had lower HC that may be as a result of reduced muscle mass. Higher central adiposity and smaller HC have been reported amongst smokers in other studies, but the literature is inconsistent (Shimokata *et al.*, 1989; Han *et al.*, 1998). Lissner et al (Lissner *et al.*, 1992) showed that women who continued to smoke had a significantly higher WHR than those who stopped smoking. In contrast, a study carried out in general practice on 601 patients failed to find a specific pattern of body fat distribution, as measured by WHR, in smokers compared to non-smokers (Armellini *et al.*, 1993). Jensen et al (Jensen *et al.*, 1995) reported that total fat and body fat distribution measured by DEXA was similar in smokers and non-smokers, but that HC, not waist, was negatively correlated with 24-hour cotinine excretion. However, the participants of the study were young and the sample sizes were small.

Samaras et al in a study of monozygotic postmenopausal twins showed that for twins discordant for smoking, those who smoked had the lowest total and central fat compared to those who did not (Samaras *et al.*, 1998).

A relatively smaller HC may be related to a higher risk for the development of diabetes because the smaller muscle mass (resulting from inactivity or illness) is associated with reduced capacity for glucose disposal and this poorer insulin action (Seidell *et al.*, 1997). Determinants of HC also may differ between male and female (Snijder *et al.*, 2003), so that gluteal fat mass and pelvic width in female and, muscle mass and pelvic width in male may be the main determinants of HC. WC has been found to be correlated highly with both intra-abdominal and total fat masses (Lean *et al.*, 1996; Han *et al.*, 1997) and increased visceral fat mass lead to increased portal concentration free fatty acids which may lead to hyperinsulinaemia and insulin resistance (Bjorntorp, 1991). In addition central deposition of body fat is associated with dyslipidaemia, hypertension, type 2 diabetes and cardiovascular diseases (Bjorntorp, 1997).

It has been shown that cigarette smoking is an independent and modifiable risk factor for type 2 diabetes (Manson *et al.*, 2000; Wannamethee *et al.*, 2001). Several reasons may explain this association, which include an increased blood glucose level, impaired insulin sensitivity, dyslipidemia, increased abdominal fat, free radical oxidative damage and oxidative stress and toxic effects at nicotine on the pancreas. The present study shows that cigarette smoking does not protect against abdominal fat distribution. Indeed it is negatively associated with body shape and this negative effect was more pronounced in females than males. The altered body shape of smokers, with a relatively broad waist and narrow hip, may partially explain why

smokers are susceptible to cardiovascular disease, diabetes and metabolic syndrome, despite their lower BMI compared with non-smokers.

The mechanism of the positive association between smoking and central fat accumulation reflected by adjusted waist circumference, in the present study could relate either to redistribution of body fat from gluteal to abdominal, or to muscle atrophy. Possible mechanisms could include differences in serum hormone levels between smokers and non-smokers such as sex hormones or some remaining confounding factors such as physical activity, diet, alcohol intake and stress level which could not be fully adjusted in the present study (Seidell *et al.*, 1991).

3.5. Conclusions

Smoking is associated with alterations in body shape, especially with a greater waist circumference particularly in women. This changed body shape may partly explain the higher risk of diabetes and metabolic syndrome in smokers, and have particular implications for the women's health. Smoking fails to offer any benefits in weight control in the young particularly in women, although it is negatively associated with BMI in older adults. A greater BMI associated with smoking cessation (0.9 kg/m^2 in men and 0.6 kg/m^2 in women compared with non-smokers, and 2.1kg/m^2 in men and 1.6kg/m^2 in women compared to current smokers) may have an effect on prevalence of obesity, however the health benefits of smoking cessation would exceed greater the risks associated with this quantity of the excess weight.

Table 3. 1: Unadjusted mean values for anthropometric measures, alcohol consumption and physical activityby sex and cigarette smoking satatus

	Men			Women		
All ages (16-74) Years	Non-smokers	Ex-smokers	Current smokers	Non-smokers	Ex-smokers	Current smokers
n	1791	893	1388	2036	715	1302
BMI (kg/m²)	26.2(26.0-26.4)	27.8(27.5-28.0)**	25.2(25.0-25.4)**++	26.0(25.8-26.2)	27.4(27.1-27.8)**	25.3(25.1-25.6)**++
Hip (cm)	102.2(101.9-102.6)	103.8(103.3-104.3)**	99.6(99.2-100.1)**++	102.1(101.6-102.6)	104.3(103.5-105.2)**	99.6(99.0-100.2)**++
Waist (cm)	91.2(90.6-91.8)	96.6(95.8-97.3)**	89.2(88.6-89.9)**++	80.1(79.6-80.7)	84.4(83.5-85.4)**	80.2(79.5-80.9)++
WHR	0.89(0.89-0.89)	0.93(0.93-0.93)**	0.89(0.89-0.90)++	0.78(0.78-0.79)	0.81(0.80-0.82)**	0.81(0.80-0.81)**
Alcohol/week	15.4(14.6-16.3)	18.2(16.5-19.6)**	23.9(22.4-25.4)**++	5.5(5.2-5.9)	6.1(5.6-6.7)	8.7(8.0-9.4)**++
Activity/days	14.4(13.8-14.9)	10.7(10.0-11.4)**	12.6(12.0-13.2)**++	10.9(10.4-11.3)	9.9(9.2-10.7)	10.6(10.0-11.1)

All data are mean + 95% confidence interval; n, number of subject based on BMI; BMI, Body Mass Index; Hip, Hip circumference; waist, Waist circumference; WHR, Waist-to-Hip ratio; Alcohol/week, total units alcohol consumption per week; Activity/day, total number of days of 30 minutes or more at least moderate physical activity in four weeks; ** Compared with non-smokers P<0.001; ++ Compared with ex-smokers p<0.001

Figure 3. 1: Prevalence of current cigarette smoking by different BMI categories in men

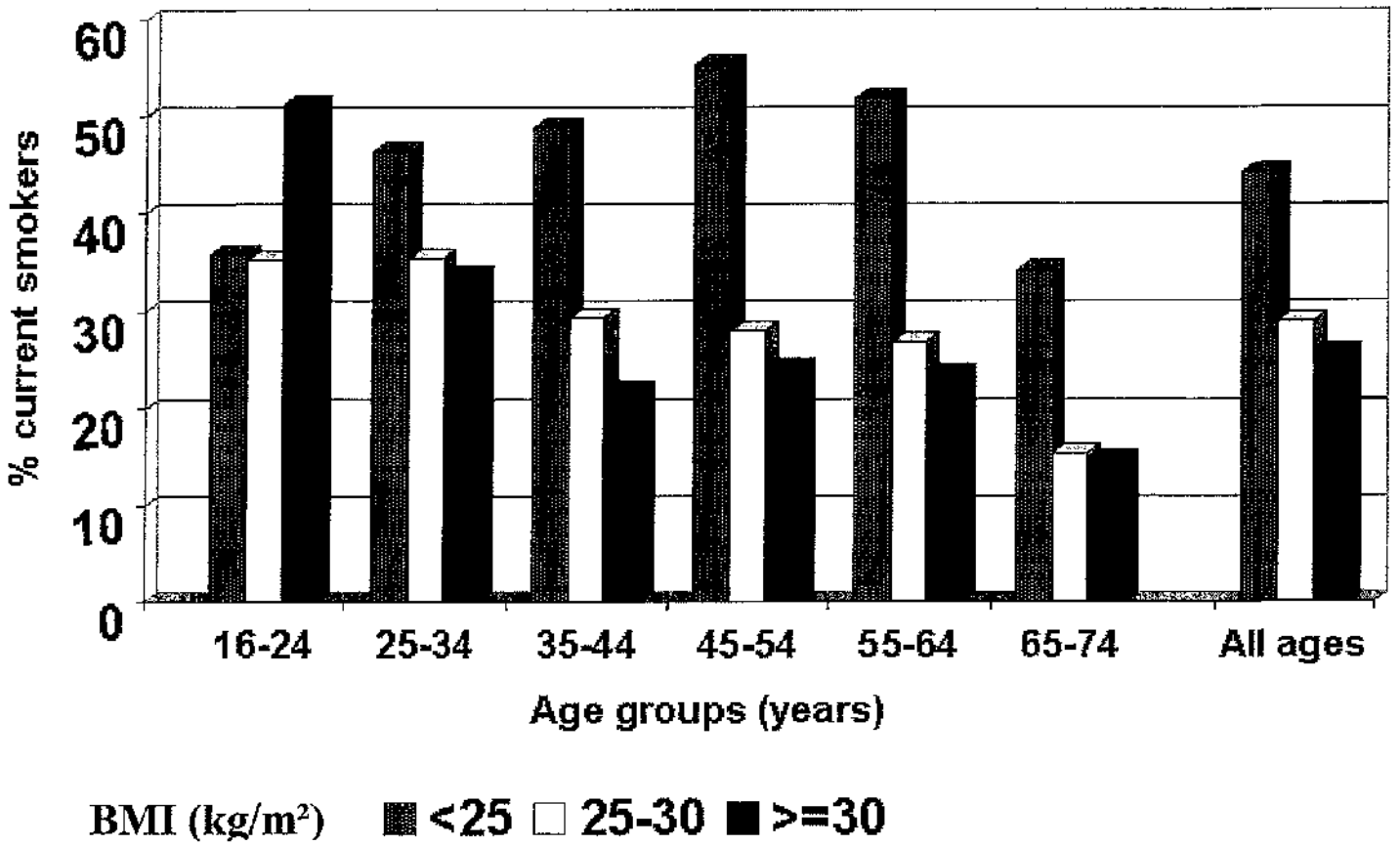


Figure 3. 2: Prevalence of current cigarette smoking by different BMI categories in women.

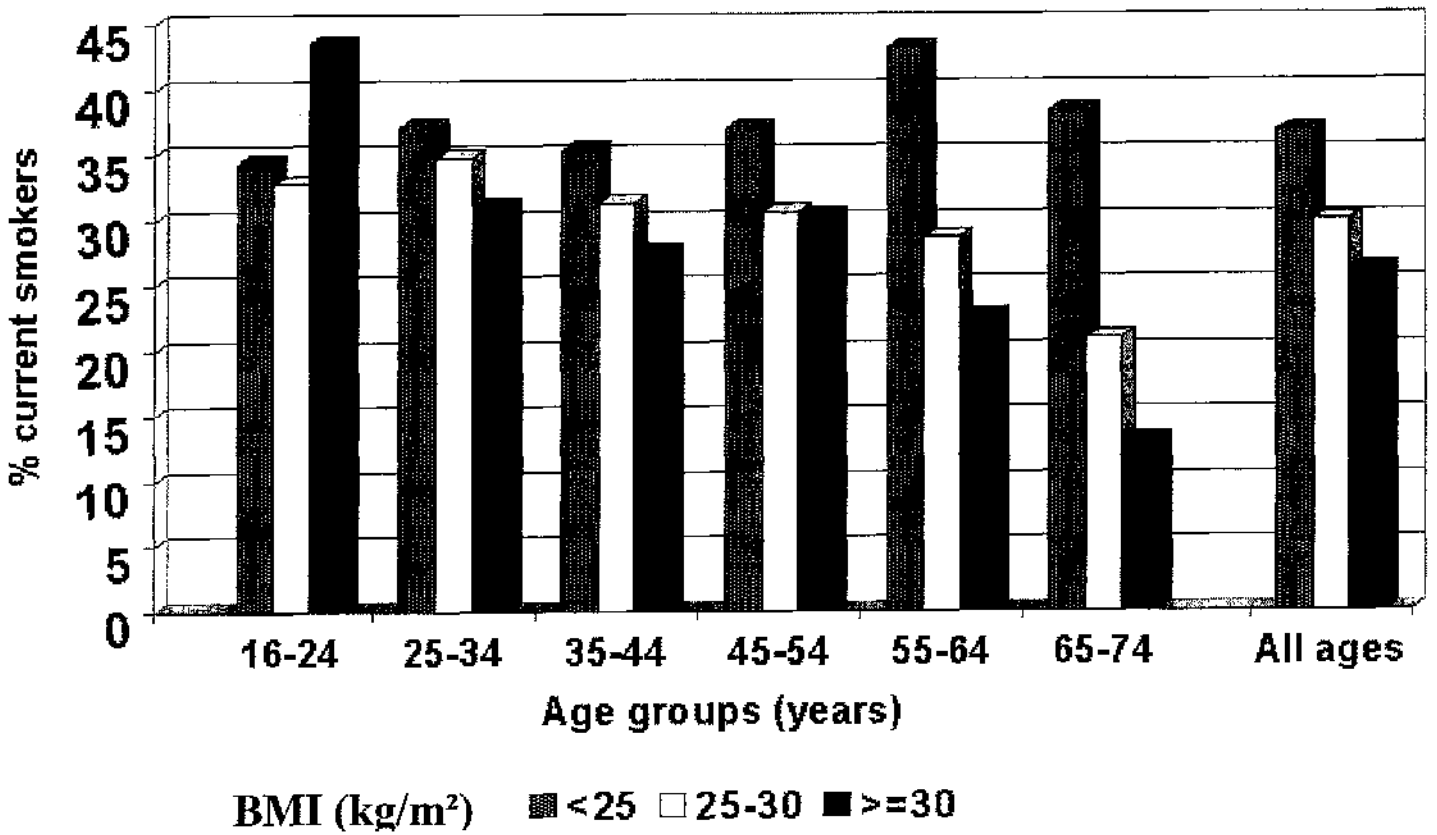


Table 3. 2: Adjusted mean[a] BMI (kg/m²) by smoking status, age and sex

	Men				Women			
Age groups (Year)	N	Non-smokers	Ex-smokers	Current smokers	N	Non-smokers	Ex-smokers	Current smokers
16-24	661	22.5(22.0-23.2)	23.1(21.0-25.3)	23.4(22.7-24.1)	600	23.5(22.7-24.3)	25.0(22.9-27.3)	23.7(22.6-24.7)
25-34	894	26.1(25.6-26.6)	27.0(26.2-27.9)	24.8(24.3-25.3)**++	828	25.2(24.6-25.9)	26.4(25.2-27.5)	24.8(24.0-25.5)+
35-44	833	27.3(26.6-28.0)	27.0(26.0-28.1)	25.5(24.8-26.1)**+	838	26.1(25.2-27.0)	26.3(25.2-27.4)	25.3(24.4-26.2)
45-54	734	28.4(27.6-29.2)	28.8(27.9-29.8)	26.0(25.2-26.8)**++	710	27.7(26.6-28.8)	28.1(26.8-29.3)	26.8(25.7-27.9)
55-64	549	27.7(26.0-29.6)	28.6(26.8-30.5)	26.4(24.7-28.2)*++	590	27.4(26.0-28.9)	28.3(26.8-29.8)	25.6(24.2-27.0)**++
65-74	407	27.0(25.9-28.1)	27.0(26.1-27.9)	24.8(23.8-25.9)**++	500	27.5(25.2-29.9)	27.3(25.1-29.7)	24.0(21.9-26.2)**++
All ages §	4078	26.8(26.4-27.2)	27.7(27.2-28.2)**	25.6(25.2-26.1)**++	4066	26.2(25.8-26.5)	26.8(26.3-27.3)*	25.2(24.8-25.5)**++

All data are mean + 95% confidence interval; [a] Adjusted for social class, physical activity, and alcohol consumption,

*Compared with non-smokers p<0.05, ** Compared with non-smokers p<0.001,

+ Compared with ex-smokers p<0.05, ++ Compared with ex-smokers p<0.001,

§ Addition adjusted by age

Table 3. 3: adjusted mean[a] waist circumference (cm) by smoking status, age and sex

	Men				Women			
Age groups (Year)	N	Non-smokers	Ex-smokers	Current smokers	N	Non-smokers	Ex-smokers	Current smokers
16-24	555	82.0(80.9-83.0)	81.3(78.2-84.5)	80.2(78.5-81.7)	512	72.9(71.8-74.0)	73.3(71.3-75.3)	72.9(71.6-74.3)
25-34	746	89.7(89.1-90.4)	90.4(89.1-91.6)	89.1(88.5-89.9)	713	77.1(76.4-78.0)	77.3(75.9-78.5)	77.8(76.9-78.5)
35-44	745	91.8(91.0-92.7)	91.4(90.2-92.7)	91.4(90.4-92.5)	735	79.4(78.3-80.5)	80.5(79.2-81.8)	80.9(79.8-82.0)*
45-54	670	94.8(93.8-95.9)	95.5(94.2-96.8)	95.1(93.8-96.2)	666	81.8(80.7-83.2)	82.8(81.3-84.1)	83.8(82.4-85.1)*
55-64	526	95.3(94.2-96.6)	96.6(95.5-97.7)	97.3(96.2-98.6)*	530	84.5(82.8-86.3)	84.9(83.0-86.7)	86.7(84.7-88.7)*
65-74	399	96.8(95.3-98.4)	96.6(95.5-97.9)	97.7(96.2-99.5)	460	83.2(80.3-86.1)	84.5(8138-87.5)	84.9(81.8-87.9)
All ages §	3641	91.8(91.4-92.3)	91.8(91.4-92.5)	91.4(91.0-91.8)	3616	79.8(79.4-80.3)	80.3(79.8-80.9)	81.1(80.5-81.7)**

All data are mean + 95% confidence interval; [a] Adjusted for social class, physical activity, BMI, and alcohol consumption,

* Compared with non-smokers $p<0.05$, ** Compared with non-smokers $p<0.001$,

§ Addition adjusted by age

Table 3. 4: Adjusted mean[a] hip circumference (cm) by smoking status, age and sex

	Men				Women			
Age groups (Year)	N	Non-smokers	Ex-smokers	Current smokers	N	Non-smokers	Ex-smokers	Current smokers
16-24	555	98.9(98.2-99.8)	101.6(99.1-104.9)	97.3(95.9-98.6)+	512	98.4(97.3-99.3)	98.2(96.2-100.2)	96.8(95.7-97.9)*
25-34	746	101.9(101.4-102.6)	101.6(100.7-102.6)	101.4(100.9-102.1)	713	100.5(99.5-101.2)	101.4(100.0-102.8)	99.1(98.2-100.0)*+
35-44	746	102.1(101.4-102.8)	101.4(100.2-102.3)	101.2(100.2-101.9)*	735	101.9(101.2-102.8)	101.9(100.7-102.8)	100.5(99.5-101.4)*+
45-54	668	102.1(100.9-103.3)	102.3(101.4-103.5)	101.6(100.5-102.8)	667	103.0(102.1-104.0)	102.6(101.6-103.7)	101.4(100.2-102.3)**+
55-64	526	102.8((101.9-103.5)	102.1(101.4-103.0)	102.1(101.2-103.0)	525	104.0(102.6-105.4)	103.0(101.4-104.7)	103.3(101.6-105.0)
65-74	401	103.8(102.3-105.2)	102.8(101.6-104.0)	102.8(101.4-104.2)	462	103.0(100.5-105.4)	102.3(100.0-104.7)	102.3(99.8-105.0)
All ages §	3642	101.9(101.4-102.3)	101.6(100.9-102.1)	101.2(100.7-101.6)**	3614	101.9(101.4-102.3)	101.6(101.2-102.1)	100.5(100.0-100.9)**++

All data are mean + 95% confidence interval; [a] Adjusted for social class, physical activity, BMI and alcohol consumption,

* Compared with non-smokers $p<0.05$, ** Compared with non-smokers $p<0.001$,

+ Compared with ex-smokers $p<0.05$, ++ Compared with ex-smokers $p<0.001$,

§ Addition adjusted by age

Table 3. 5: Adjusted mean[a] waist-to- hip ratio by smoking status, age and sex

	Men				Women			
Age groups (Year)	N	Non-smokers	Ex-smokers	Current smokers	N	Non-smokers	Ex-smokers	Current smokers
16-24	555	0.83(0.82-0.84)	0.81(0.78-0.84)	0.82(0.80-0.84)	512	0.74(0.73-0.75)	0.75(0.73-0.77)	0.76(0.74-0.77)
25-34	746	0.88(0.87-0.89)	0.89(0.87-0.90)	0.87(0.86-0.89)	712	0.77(0.76-0.78)	0.76(0.75-0.78)	0.79(0.78-0.79)*+
35-44	745	0.90(0.89-0.91)	0.90(0.89-0.91)	0.90(0.89-0.91)	734	0.78(0.77-0.79)	0.79(0.78-0.80)	0.80(0.79-0.81)**
45-54	668	0.92(0.91-0.93)	0.93(0.92-0.94)	0.93(0.92-0.94)	666	0.79(0.78-0.80)	0.81(0.79-0.82)	0.83(0.81-0.84)**+
55-64	526	0.93(0.92-0.94)	0.95(0.93-0.95)	0.95(0.94-0.96)	525	0.81(0.79-0.83)	0.82(0.80-0.84)	0.84(0.82-0.86)**
65-74	398	0.93(0.91-0.94)	0.93(0.92-0.95)	0.94(0.93-0.96)	460	0.81(0.78-0.84)	0.82(0.80-0.85)*	0.83(0.80-0.86)*
All ages §	3638	0.90(0.90-0.90)	0.91(0.90-0.91)	0.91(0.90-0.91)	3609	0.78(0.78-0.79)	0.79(0.79-0.80)*	0.81(0.80-0.81)**++

All data are mean + 95% confidence interval; [a] Adjusted for social class, physical activity, BMI and alcohol consumption,

* Compared with non-smokers $p<0.05$, ** Compared with non-smokers $p<0.001$,

+Compared with ex-smokers $p<0.05$, ++ Compared with ex-smokers $p<0.001$,

§ Addition adjusted by age

Chapter 4: Smoking combined with overweight or obesity markedly elevated cardiovascular risk factors.

4.1. Introduction

Cardiovascular disease (CVD) is a leading cause of morbidity and mortality contributing 16.6 million (29.2%) of all global deaths (WHO, 2003). Multiple risk factors contribute causally to CVD, with either separate or synergistic effects. Cigarette smoking, overweight or obesity, unhealthy diet and inactivity are major lifestyle risk factors (Darnton-Hill *et al.*, 2004), associated with clinical risk factors including raised blood pressure, plasma lipids, coagulation factors and measures of inflammation. Many biochemical risk factors are attributable to overweight or obesity, especially with a central fat distribution.

Smoking promotes CVD, respiratory disease and several cancers (WHO, 2002) leading to premature death and ill health (Scottish Executive, 2004), but obesity is rapidly approaching smoking as the leading preventable cause of CVD and death (Jonsson *et al.*, 2002; Peeters *et al.*, 2003; Mokdad *et al.*, 2004). Overweight or obese smokers thus have at least two independent risk factors for CVD and there may be synergistic effects between them (Jonsson *et al.*, 2002). Obese smokers have about twice the mortality of the obese non-smoker and quadruplethe mortality of non-smokers of healthy BMI (22-24.9 kg/m^2) (Meyer *et al.*, 2002).

Overweight/obesity and smoking both contribute to inflammation, promote atherosclerosis and CVD (Pearson *et al.*, 2003; Engstrom *et al.*, 2004). C-reactive protein (CRP), a non-specific marker of inflammation, and fibrinogen, an inflammatory marker and coagulation protein, both predict CVD (Ridker *et al.*, 2000; Pearson *et al.*, 2003; Danesh *et al.*, 2004). It has been suggested that the inflammation markers may explain some of the link of obesity with CVD since inflammatory cytokines like tumor necrosis factor- α (TNF- α) and interleukine-6 (IL-6)

are produced by adipose tissue (Visser *et al.*, 1999a; Festa *et al.*, 2001; Bazzano *et al.*, 2003). Indeed, up to 30% of the circulating IL-6 may arise from adipose tissue (Mohamed-Ali *et al.*, 1997). Smoking generates free radicals that can activate inflammatory pathways directly, but is also associated with diets that are lower in fruit and vegetables as sources of antioxidants. Thus both direct and indirect mechanisms link smoking with enhanced inflammation. Smoking may further interact with overweight and obesity indirectly by modulating other risk factors such as lipid profiles.

This study aimed to define the impacts of smoking status and overweight or obesity on CVD risk as determined by total cholesterol, HDL-cholesterol (HDL-C, non-HDL-cholesterol (non-HDL-C) a secondary target of the ATPIII guidelines, CRP and fibrinogen in a large representative sample of adults with high prevalences of cigarette smoking, overweight and obesity.

4.2. Methods

4.2.1. Study Subjects

The Scottish Health Survey (SHS) 1998 is a cross-sectional nationally representative survey, designed to provide a comprehensive picture of the health of the Scottish population. Full survey methods have been published elsewhere (Shaw *et al.*, 2000). Of the total 9047 adults (3941 men and 5106 women) who participated, total numbers of valid blood measurements obtained were: total cholesterol 5924, HDL-C 5891, CRP 5988 and fibrinogen 5460.

4.2.2. Anthropometric measures

Weight and height were measured using standard techniques by trained staff (Shaw *et al.*, 2000). Height was measured with a portable stadiometer standing in bare feet and body weight to the nearest 0.1 kg in bare foot and light clothes with the Soehlne scales. BMI was calculated as weight divided by height squared (kg/m^2). Overweight and obesity were defined as a BMI of 25 - 29.9 and ≥ 30 kg/m^2 respectively (WHO, 1998).

Cigarette smoking status was classified as follows: regular cigarette smokers, those who said they smoked cigarettes at all at the time of the interview; ex-smokers, those who smoked cigarettes regularly in the past but did not currently; and non-smokers: those who had never smoked cigarettes regularly and were not current smokers. Reported physical activity was measured by a questionnaire that asked about the frequency, duration and intensity of four major types of activity: activity at home, walks of 15 minutes or more, sports and exercise activities, and activity at work in the four weeks before the interview. These activities were then compiled to calculate an estimated overall physical activity, divided into five categories based on different levels of physical activity recommendations (American College of Sports Medicine, 1990; Blair & Connelly, 1995). These five categories were reduced into three main levels: active, those who reached at least one of the two guideline levels (either 3 occasions of twenty minutes vigorous activity per week or five occasions of thirty minutes moderate activity per week or both); less active: those were not active enough to meet either guideline level but were active on at least one day a week; and inactive, those respondents who reported less than one day per week of moderate or vigorous activity of at least 20 minutes duration.

Habitual alcohol consumption over the previous 12 months was assessed using questions on frequency, type, average number of days per week on which alcohol was drunk, the usual quantity consumed on any one day and the finally the "usual" weekly units of alcohol

consumed calculated. This was then divided into four groups of weekly alcohol intake for both men and women. For men, these quantities were under 1 unit, 1-10 units, 10-21 units and over 21 units. For women these were under 1 unit, 1-7 units, 7-14 units and over 14 units.

Information on dietary habits was obtained by a short dietary questionnaire (Dietary Target Monitor) (Lean *et al.*, 2003). This questionnaire included questions relating to type and frequency of major food items and recently has been validated in three key food groups (Lean *et al.*, 2003). In this study we used total fruit and vegetables consumption as a categorical variable divided in the three groups, low consumers (< 200g per day), moderate consumers (200 - < 400g per day) and achievers of current targets (≥ 400 g per day) (The Scottish Office, 1996).

Social class was based on the Register General's Standard Occupation Classification using the current or last occupation of the chief income earner within informant's household, in one of four categories: professional and intermediate, skilled (non-manual), skilled (manual), partly skilled and unskilled (Shaw *et al.*, 2000).

4.2.3. Blood samples

Non-fasting venous blood samples were obtained, as described by Shaw et al (Shaw *et al.*, 2000). CRP was measured using the N Latex CRP mono Immunoassay on the Behring Nephelometer II Analyzer. Total cholesterol was measured using the DAX Cholesterol Oxidase assay method calibrated to Center for Disease Control (CDC) guideline. HDL-cholesterol was measured using the DAX Cholesterol Oxidase assay method calibrated to CDC after PTA precipitation. Fibrinogen was measured using the Organon Teknika MDA 180 analyser and a modification of the Clauses thrombin clotting method. Non-HDL-C,

which contains cholesterol in LDL and VLDL calculated by subtracting HDL-C from total cholesterol (NCEP, 2001). CVs were range 0.9 – 11% for all parameters measured.

4.2.4. Data analysis

Analysis was carried out using the statistical package, SPSS 11.0 (SPSS INC., Chicago, IL, USA). As data were not normally distributed, log- transformed values of dependent variables were used for improving the normality of distributions.

A general linear model (GLM) was used to compare the adjusted geometric means of the five measured risk factors among different independent lifestyle factors. This analysis was stratified by gender and controlled for age as a covariate in the univariate GLM. To evaluate the combined impacts of smoking status and BMI, cigarette smoking status and BMI were combined into nine categories and non-smokers with BMI below 25 kg/m² were defined as the reference category. GLM incorporating Bonferroni *post hoc* test was used to compare the adjusted geometric means of the risk factors within combined BMI and smoking variable. In this analysis age was used as a covariate and, social class, physical activity, combined smoking and BMI, alcohol consumption and fruit and vegetable consumption as fixed factors. The logistic regression model was used to compute the OR and 95% confidence interval (CI) for the probability of having high value for CRP (≥ 3 mg/l), fibrinogen (≥ 3 g/l), total cholesterol (≥6.2 mmol/l), non-HDL-C (> 4.00 mmol/l) and a low HDL-C (≤ 1 mmol/l) among the subgroups of the combined smoking and BMI with the reference category, non-smokers with BMI below 25 kg/m². A value of $p<0.05$ was used for statistical significance.

4.3. Results

Approximately 34% male and 33% female subjects were current smokers, 64% of men and 54% of women were either overweight or obese and almost 18% men and 16% women were either overweight or obese and were current smokers.

Table 1 and 2 show the age-adjusted means of CVD risk factors based on different lifestyle factors in men and women. Mean CRP and fibrinogen were higher among current smokers in both sexes ($p<0.0001$). Women current smokers had higher total cholesterol concentrations ($p<0.01$), non-HDL-C and lower HDL-C concentrations ($p<0.0001$). BMI was significantly associated with all risk factors in both sexes as obese subjects had the highest concentrations CRP, total and non-HDL-C and fibrinogen and the lowest concentrations of HDL-C.

In both sexes, those who were inactive had the highest concentrations of CRP and fibrinogen and lowest concentrations of HDL-C ($p<0.0001$). Total and non-HDL-C concentrations were lowest in inactive men ($p<0.05$).

Men who consumed 1-21 units of alcohol per week and women who consumed 7-14 had lower CRP concentrations ($p<0.01$). Total cholesterol rose with increasing alcohol consumption, however this relationship was significant only in men ($p<0.001$). Alcohol consumption was inversely associated with fibrinogen in women ($p<0.0001$). HDL-C increased with increasing alcohol consumption in both sexes ($p<0.0001$). Alcohol consumption showed a significant inverse association with non-HDL-C in women ($p<0.001$).

Those in the lowest social class had highest mean CRP in both sexes ($p<0.0001$), fibrinogen in men ($p<0.001$) and in women ($p<0.02$), non-HDL-C in women ($p<0.001$), and lowest HDL-C in women ($p<0.0001$) and non-HDL-C in men ($p<0.02$).

For both men and women who achieved the recommended target for fruit and vegetable consumption, CRP and fibrinogen were lowest ($p<0.001$). Women who consumed larger quantities of fruit and vegetables had higher HDL-C ($p<0.001$) and lower non-HDL-C concentrations ($p<0.01$).

When all independent variables (table 1 and 2) have been entered simultaneously in the GLM model, both BMI and smoking status had significant associations with all CVD risk factors except smoking with cholesterol in males ($p<0.001$).

Figures 1-2 show adjusted geometric mean values of CRP and HDL-C among different categories of smoking and BMI. Cigarette smoking, overweight and obesity were associated with higher CRP concentration compared with non-smokers of BMI less than 25 in both sexes ($p<0.001$). The obese and regular smokers had markedly higher concentrations of CRP in both sexes (figure 1). Smoking alone was significantly associated with lower HDL-C and higher non-HDL-C in females ($p<0.001$) compared with nonsmokers. Overweight and obesity, smokers had significantly reduced HDL-C concentration and increased non-HDL-C in both sexes ($p<0.001$) (figure2) and obese current smokers had the highest levels of fibrinogen ($p<0.001$).

In total, the proportion of subjects who had CRP $\geq$3 mg/l, fibrinogen $\geq$3 g/l, total cholesterol $\geq$6.2 mmol/l, HDL-C $\leq$1 mmol/l and non-HDL-C >4 mmol/l were 30.6%, 34.6%, 27.2%, 15.6% and 47.7% respectively.

Table 3 shows adjusted OR of having the CVD risk factors above the cut off points by smoking status and BMI. Smoking, overweight and obesity were significantly associated with

higher OR of having elevated CRP, fibrinogen, non-HDL-C and higher OR of having lower concentrations of HDL-C than non-smokers with normal BMI ($p<0.001$). Obese current smokers had particularly high OR for HDL-C and CRP ($p<0.001$). The OR of having HDL-C ≤1 mmol/l, CRP ≥3 mg/l, non-HDL-C >4 mmol/l, fibrinogen ≥3 g/l and total cholesterol ≥6.2 mmol/l in obese smokers were 11.6, 9.1, 5.2, 4.7 and 2.7 times that of the reference category respectively ($p<0.001$).

In a separate analysis we excluded subjects with three major existing cardiovascular conditions which might result in secondary behavioral changes (angina, heart attack or stroke) and the results were almost the same as without exclusion of these conditions. In this analysis adjustment was not made for WC because WC and BMI are highly correlated and both are correlated with body fat content. However a further analysis using combination of WC and smoking status in nine groups (a combination of both WC action levels and smoking status in three categories) showed that the combination of WC and smoking predict the risks very similarly to the combination of BMI and smoking. The age stratified analysis in two groups (age < 45 and ≥ 45 years) showed that smoking had a greater effect on total cholesterol in younger age group and on CRP in older age groups. Ex-smokers had lower OR of the risk factors compared with current smokers in different BMI categories.

4.4. Discussion

This study describes links between two well-established CVD risk factors, cigarette smoking and obesity/overweight in a population with high prevalence of all these factors. Jonsson et al (Jonsson *et al.*, 2002) reported that the differences in the incidence of CHD among obese subjects were related to exposure to other risk factors for CVD, of which smoking was the

most important. Meyer et al (Meyer *et al.*, 2002) showed that overall risk associated with obesity increased markedly when combined with smoking. In our study we have determined the strengths of impacts of smoking, and overweight or obesity on other established CVD risk factors. The findings suggested that several other lifestyle factors, physical activity, total fruit and vegetable consumption, alcohol intake and social class are important independent factors for the CVD risk factors. However, after controlling for all these independent lifestyle factors, BMI and smoking remained important CVD risk factors; only these two factors related independently to all five measured risk parameters.

4.4.1. Association between smoking and elevated BMI with inflammatory markers

Epidemiological and clinical studies have indicated strong associations between inflammatory markers, insulin resistance (Hotamisligil, 2003; Yudkin, 2003) and the risk of CVD (Pearson *et al.*, 2003; Engstrom *et al.*, 2004). It has been frequently shown that CRP and fibrinogen are both independent predictors of risk of myocardial infarction, stroke, peripheral arterial disease and sudden cardiac death (Danesh *et al.*, 1998; Kamath & Lip, 2003; Ridker, 2003; Torres & Ridker, 2003). Although the mechanisms responsible for elevated CRP in obese subjects are uncertain, there is a strong relationship between adipocytokines, cytokines secreted by adipose tissue, such as IL6, TNF-α and leptin and inflammatory markers including CRP (Maachi *et al.*, 2004). Thus the secreted cytokines from adipose tissue in the obese may play a role in hepatic production of inflammatory protein like CRP (Maachi *et al.*, 2004; Trayhurn & Wood, 2004).

Bazzano et al (Bazzano *et al.*, 2003) found strong positive associations between cigarette smoking, CRP and fibrinogen in a large representative sample of U.S. population. The third

MONICA Augsburg survey 1994/95 showed strong positive associations between smoking and various markers of systemic inflammation including CRP and fibrinogen in men, but not women and smoking cessation was associated with decreased inflammation markers (Frohlich *et al.*, 2003). In a cross sectional study in women Bermudez et al (Bermudez *et al.*, 2002) found that five markers of systemic vascular inflammation including CRP were associated with smoking. Thus the association of cigarette smoking and atherosclerosis may partly be modulated through inflammation and smoking may initiate or accelerate atherosclorosis through this process.

After adjusting for lifestyle factors, our results are in line with previous findings and demonstrate that both smoking and obesity are independently associated with inflammation marker in both sexes and that smoking cessation is associated with a decreased inflammatory response. Although earlier studies have demonstrated that obesity influences CRP (Visser *et al.*, 1999a; Festa *et al.*, 2001; Maachi *et al.*, 2004) and others that smoking also does (Bermudez *et al.*, 2002; Bazzano *et al.*, 2003; Frohlich *et al.*, 2003) ours is one the first to document the cumulative effects of smoking together with obesity on such markers.

4.4.2. Association between smoking and elevated BMI with plasma lipids

Elevated total plasma cholesterol is a vitally important risk factor for coronary heart disease (Grundy, 1997; Kromhout, 2001). Both obesity and smoking are associated with various lipid abnormalities including elevated total cholesterol, triglycerides and lower HDL-C (Muscat *et al.*, 1991; Hu *et al.*, 2000). After controlling for lifestyle factors, overweight and obesity were strongly and independently associated with lipid abnormalities. Although cigarette smoking had no independent association with lipid abnormalities in men and only a weak association in

women in BMI less than 25 kg/m^2, a combination of overweight and obesity with smoking increased the risk of lipid abnormalities.

Native American Indians participating in the Strong Heart Study showed that HDL-C is decreased in obese men and women (Hu *et al.*, 2000). Data from 27 populations in WHO MONICA project also showed a statistically significant positive association between hypercholesterolaemia (defined as cholesterol $\geq$ 6.5 mmol/l) and BMI (Gostynski *et al.*, 2004). Norwegians surveyed over an 8 year period showed BMI was associated with elevated total cholesterol and triglycerides, and decreased HDL-C (Wilsgaard & Arnesen, 2004).

A meta-analysis defining the associations between smoking and plasma lipoproteins revealed that smokers had significantly higher plasma concentrations of cholesterol, triglyceride, VLDL-C, LDL-C and lower serum HDL-C compared with non-smokers (Craig *et al.*, 1989). Smoking acutely impairs glucose tolerance and insulin sensitivity, and enhances plasma cholesterol and triglyceride concentrations (Frati *et al.*, 1996). A study on British women revealed that current smokers had higher plasma concentrations of LDL-C, total/HDL-C, triglyceride and lower HDL-C compared with non-smokers (Razay & Heaton, 1995). Eliasson et al (Eliasson *et al.*, 1997) demonstrated that smokers were insulin resistant and had some disorders related with insulin resistance syndromes such as lower HDL-C, raised fasting triglyceride with lipid intolerance and impaired elimination of triglyceride from a mixed meal, potentially encouraging atherosclerosis (Mero *et al.*, 1997). The association between smoking and blood pressure is not clear and controversial views had been reported on the chronic effects of cigarette smoking on blood pressure in which some studies showed no associations, but some others reported that smokers had either lower or higher blood pressure compared

with non-smokers. Because of lack of association between smoking and blood pressure in the present study, this risk factor has not been included in this study.

The present results are thus in line with the literature and confirm that overweight, obesity and smoking are important and independent risk factors for atherogenic lipids profiles. The stronger relationships between obesity, lipids and smoking in women may be explained by greater central fat accumulation (Akbartabartoori *et al.*, 2005) in women smokers compared to men.

How exactly smoking affects plasma lipids concentrations remains unclear. Several mechanisms have been suggested. Cigarette smoking has multiple effects including the elevation of cathecolamines, growth hormone, cortisol, and insulin concentrations, which in turn might induce changes in lipolytic enzymes and in lipoprotein metabolism in the liver (Mero *et al.*, 1997). We have reported that in spite of lower BMI in current smokers, they had higher central adiposity than non-smokers especially in women (chapter 3) (Akbartabartoori *et al.*, 2005) and central deposition of body fat is associated with dyslipidaemia, hypertension, type 2 diabetes and cardiovascular diseases (Bjorntorp, 1997).

Lifestyle behaviors including physical activity, diet, smoking, and drinking habits are major modifiable factors that affect CVD and metabolic syndrome. Zhu et al (Zhu *et al.*, 2004) using data from the Third National Health and Nutrition Examination Survey (NHANES III) reported that the risk of having the metabolic syndrome is substantially lower in individuals who are physically active, non-smoking, consume a relatively low carbohydrate intake, moderate alcohol consumption, and who maintain a BMI in the non-obese range. This study also revealed that subjects who had all of these low risk behaviors combined with a BMI of <

30 kg/m^2 had a much lower risk of having metabolic syndrome. Overweight, obesity, physical inactivity and an atherogenic diet are major underlying risk factors for coronary heart disease (CHD) and metabolic syndrome (NCEP, 2001). Lifestyle changes have been shown to be the most cost-effective means to reduce CHD and metabolic syndrome (NCEP, 2001). In England and Wales between 1981 and 2000 modest reduction in major risk factors principally smoking, cholesterol, and blood pressure levels led to 4 times greater gains in life-years than did cardiological treatments (Unal *et al.*, 2005). The authors concluded that effective policies to promote healthy diets and control tobacco use might yield substantial additional years of life. Almost the same result has been reported in Scotland between 1975 and 1994 (Critchley *et al.*, 2003).

Longitudinal studies are ideally needed to confirm the causal relationships of the interaction between the lifestyle factors with overweight or obesity on CVD risk factors. The present results extend the rather consistent evidence-base and the recognition of combined impacts of smoking and overweight / obesity on some of the components of metabolic syndrome and justifies early intervention for overweight /obese smokers. Smoking cessation might be valuably incorporated into algorithms for initiating treatment for those at high CVD risk, such as in the metabolic syndrome.

4.5. Conclusion

Cigarette smoking, overweight and obesity are independently associated with CVD risk factors. They increase serum concentrations of CRP, fibrinogen, and plasma total cholesterol, non-HDL-C and decrease HDL-C. In addition to these independent effects, a combination of smoking and obesity elevates CVD risk factors, especially towards a higher CRP and lower

HDL-C. This is an additive not synergistic effect. Early aggressive interventions to tackle weight management and smoking cessation simultaneously are justified in obese and overweight smokers given the present evidence that smoking together with overweight or obesity aggravate CVD risk factors so markedly.

Table 4. 1: Age adjusted geometric means of CVD risk factors by some lifestyle factors in men

	n	CRP mg/l	Cholesterol mmol/l	Fibrinogen g/l	HDL-c mmol/l	Non-HDL-c mmol/l
Smoking						
Non-smokers	1034	1.08	5.33	2.46	1.27	3.95
Ex-smokers	591	1.32	5.42	2.49	1.24	4.06
Current smokers	849	1.89	5.37	2.76	1.24	4.01
p value for trend		<0.0001	<0.3	<0.0001	<0.05	<0.2
BMI (kg/m²)						
< 25	842	1.02	5.08	2.54	1.39	3.57
25-30	1033	1.34	5.51	2.54	1.23	4.18
≥ 30	442	2.21	5.57	2.65	1.09	4.39
p value for trend		<0.0001	<0.001	<0.01	<0.0001	<0.0001
Physical activity						
Active	950	1.17	5.38	2.49	1.31	3.97
Less active	969	1.30	5.41	5.56	1.23	4.06
Inactive	549	1.87	5.26	2.69	1.21	3.92
p value for trend		<0.0001	<0.03	<0.0001	<0.0001	<0.04
Alcohol (unit per week)						
0-1	323	1.55	5.20	2.64	1.14	3.96
1-10	753	1.28	5.32	2.56	1.19	4.03
10-21	614	1.29	5.39	2.53	1.28	4.02
> 21	763	1.49	5.47	2.58	1.34	4.00
p value for trend		<0.01	<0.001	<0.08	<0.0001	<0.9
Social class						
I&II	834	1.19	5.44	2.51	1.26	4.10
IIINM	277	1.44	5.36	2.59	1.23	4.01
IIIM	804	1.46	5.36	2.59	1.24	4.01
IV&V	482	1.53	5.30	2.64	1.26	3.89
P value for trend		<0.0001	<0.07	<0.001	<0.6	<0.02
Fruit & vegetables						
Achievers of target	411	1.08	5.37	2.49	1.27	4.00
Moderate consumer	879	1.31	5.38	2.54	1.25	4.02
Low consumer	1190	1.54	5.34	2.61	1.25	3.98
p value for trend		<0.0001	<0.7	<0.001	<0.6	<0.8

The General linear model univariate was used. When all independent variables entered in the GLM model both BMI and smoking had association with all the risk factors except smoking with cholesterol $p<0.001$

Table 4. 2: Age adjusted geometric means of CVD risk factors by some lifestyle factors in women

	n	CRP mg/l	Cholesterol mmol/l	Fibrinogen g/l	HDL-c mmol/l	Non HDL-c mmol/l
Smoking						
Non-smokers	1442	1.41	5.39	2.71	1.55	3.73
Ex-smokers	543	1.72	5.35	2.69	1.53	3.70
Current smokers	986	1.92	5.50	2.90	1.40	3.97
p value for trend		<0.0001	<0.01	<0.0001	<0.0001	<0.0001
BMI (kg/m²)						
< 25	1261	1.01	5.25	2.62	1.62	3.51
25-30	910	1.70	5.51	2.77	1.49	3.93
≥ 30	592	3.48	5.61	3.05	1.31	4.21
p value for trend		<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
Physical activity						
Active	849	1.29	5.42	2.69	1.55	3.76
Less active	1494	1.58	5.43	2.75	1.50	3.81
Inactive	624	2.25	5.40	2.92	1.44	3.83
p value for trend		<0.0001	<0.7	<0.0001	<0.0001	<0.4
Alcohol (unit per week)						
0-1	818	1.86	5.39	2.88	1.37	3.92
1-7	1199	1.56	5.42	2.76	1.51	3.79
7-14	532	1.42	5.44	2.69	1.58	3.75
> 14	410	1.64	5.46	2.67	1.66	3.68
p value for trend		<0.001	<0.7	<0.0001	<0.0001	<0.001
Social class						
I&II	988	1.40	5.43	2.72	1.57	3.72
IIINM	602	1.66	5.42	2.80	1.50	3.80
IIIM	648	1.75	5.37	2.79	1.47	3.79
IV&V	658	1.87	5.50	2.81	1.43	3.94
p value for trend		<0.0001	<0.2	<0.02	<0.0001	<0.001
Fruit & vegetables						
Achievers of target	856	1.36	5.38	2.71	1.56	3.71
Moderate consumer	1175	1.60	5.43	2.72	1.50	3.81
Low consumer	949	1.91	5.44	2.87	1.44	3.87
p value for trend		<0.0001	<0.4	<0.0001	<0.0001	<0.01

The General linear model univariate was used. When all independent variables entered in the GLM model both BMI and smoking had association with all the risk factors p<0.001.

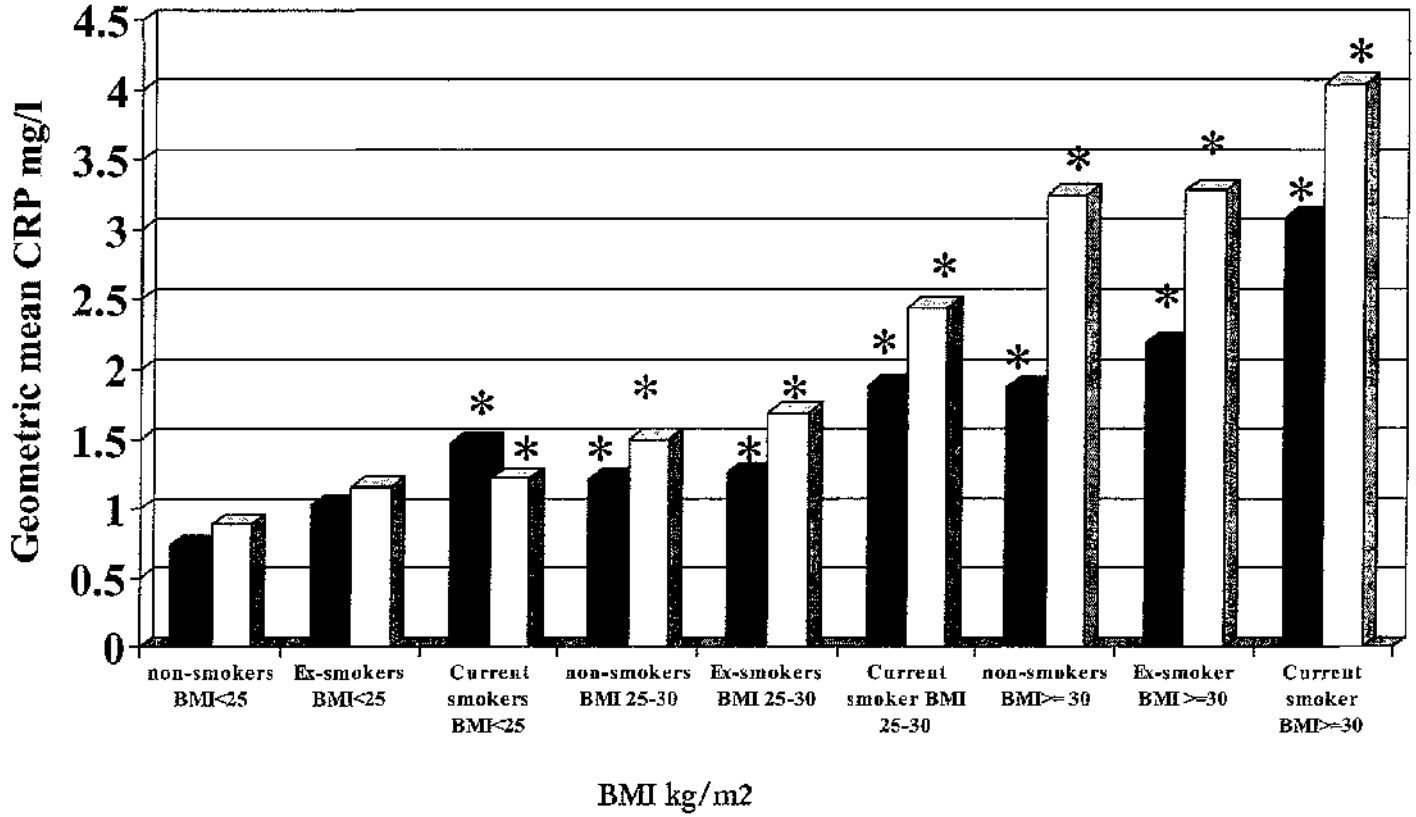
Table 4. 3: Adjusted odds ratio of cardiovascular risk factors by smoking and BMI

	n	Cholesterol ≥ 6.2 mmol/l (*n* 5260)		HDL-cholesterol ≤ 1 mmol/l (*n* 5233)		Fibrinogen ≥ 3 g/l(*n 4869*)		C-reactive protein ≥3 mg/l (*n5314*)		Non-HDL-cholesterol > 4 mmol/l (*n* 5231)	
		OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
BMI<25 kg/m²											
Non-smokers	960	1		1		1		1		1	
Ex-smokers	287	0.85	(0.60-1.20)	1.52	(0.92-2.50)	1.44 †	(1.05-1.98)	1.60 †	(1.13-2.26)	0.84	(0.62-1.13)
Current smokers	847	1.21	(0.93-1.60)	2.52 *	(1.77-3.58)	2.29 *	(1.81-2.90)	2.20 *	(1.70-2.84)	1.53 *	(1.23-1.91)
BMI 25-30 kg/m²											
Non-smokers	879	1.76 *	(1.39-2.24)	2.13 *	(1.51-3.01)	1.57 *	(1.24-1.99)	1.92 *	(1.49-2.48)	2.22 *	(1.81-2.74)
Ex-smokers	489	1.70 *	(1.30-2.20)	2.62 *	(1.79-3.82)	1.39 †	(1.06-1.82)	2.28 *	(1.72-3.03)	2.27 *	(1.78-2.91)
Current smokers	572	2.31 *	(1.77-3.00)	4.76 *	(3.35-6.78)	3.30 *	(2.54-4.25)	4.08 *	(3.13-5.32)	3.23 *	(2.54-4.10)
BMI >=30 kg/m²											
Non-smokers	477	2.31 *	(1.77-3.01)	4.73 *	(3.31-6.78)	2.61 *	(2.00-3.40)	5.69 *	(4.35-7.44)	3.89 *	(3.04-4.98)
Ex-smokers	276	1.89 *	(1.39-2.56)	6.90 *	(4.36-9.48)	2.52 *	(1.85-3.44)	6.45 *	(4.76-8.73)	3.15 *	(3.35-4.23)
Current smokers	281	2.70 *	(1.98-3.68)	11.57 *	(7.91-16.92)	4.66 *	(3.42-6.35)	9.11 *	(6.67-12.5)	5.20 *	(3.84-7.02)

Results adjusted for social class, physical activity, age, gender, alcohol consumption and fruit and vegetables. OR = odds ratio,

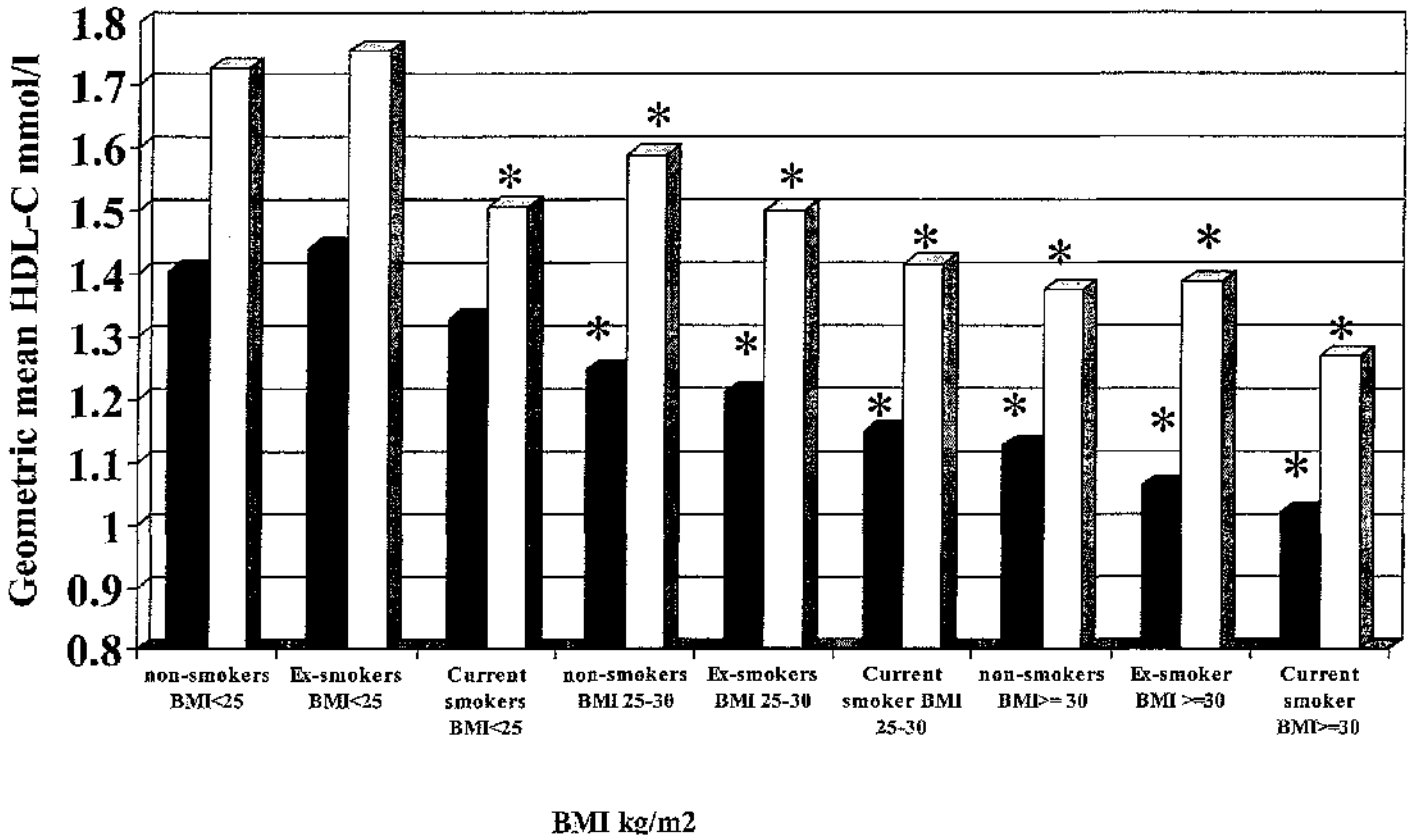
CI = confidence interval. Significantly different from reference category: * $p<0.001$, † $p<0.03$.

Figure 4. 1 Geometric mean of C-reactive protein by smoking and BMI.



■ Male, □ female, * Compared with non-smokers BMI < 25 kg/m²: p < 0.001. The mean has been adjusted for age, physical activity, alcohol consumption, total fruit and vegetable consumption and social class.

Figure 4. 2: Geometric mean of HDL-cholesterol by smoking and BMI.



■ Male, □ female, * Compared with non-smokers BMI < 25 kg/m²: $p < 0.001$. The mean has been adjusted for age, physical activity, alcohol consumption, total fruit and vegetable consumption and social class.

Chapter 5: Physical activity, BMI, body shape and cardiovascular risk factors

5.1. Introduction

Physical activity is an important component of lifestyle that has many benefits for physical, mental and social health (WHO, 2003). It is a major determinant of energy expenditure and an essential component to maintain energy balance and encourage weight control. The increasing of a prevalence of obesity continuing to increase worldwide makes the prevention of weight gain and the maintenance of a healthy body weight increasingly important. Lifestyle factors, including physical activity and habitual diet, have major roles in maintaining energy balance and weight management. Prentice et al reported that modern inactive lifestyles possibly represent the dominant factor in the aetiology of obesity in Britain (Prentice & Jebb, 1995). American analyses suggest that the falling activity levels may have triggered the first phase of the resent obesity epidemic 1970-1990, following which it has been fuelled by hyperphagia coupled with increased food availability and a fall in the real cost of high fat high energy foods (Putnam *et al.*, 2002)

To improve public health, different physical activity levels have been recommended. Based on the current recommendations, which were advised in the United States, every adult should accumulate 30 minutes or more of moderate-intensity physical activity on most days of the week (Blair & Connelly, 1995). Previous recommendations for cardiovascular health advised more vigorous –intensity activity: 20 minutes vigorous activity at least three times a week (American College of Sports Medicine, 1990). Recommendations for maintaining a healthy body weight and preventing unhealthy weight gain are one hour of moderate intensity activity per day on most days of the week. However, there is still a need to evaluate the achievement of these recommendations at population level, and to determine their relationship with overweight and obesity.

Overweight, obesity and inactivity are major risk factors for cardiovascular disease and all cause mortality (WHO, 2003). Apart from the effect of physical activity on controlling body weight, evidence shows it is also associated with reduced risk for cardiovascular disease, diabetes and metabolic syndrome (Hu *et al.*, 2004b; Katzmarzyk *et al.*, 2004; Katzmarzyk *et al.*, 2005).

Physical activity and physical fitness can modify obesity related chronic diseases and mortality, and evidence suggests that overweight or obese people who are active and fit have less cardiovascular disease and lower all cause mortality than normal weight unfit people (Blair & Brodney, 1999; Church *et al.*, 2004; Wessel *et al.*, 2004). Katzmarzyk et al (Katzmarzyk *et al.*, 2005) have reported that cardiorespiratory fitness, assessed by a maximal treadmill exercise test, modifies the relationships between obesity, metabolic status and mortality in men and can protect against premature mortality regardless of body weight status of the presence of Metabolic Syndrome.

In contrast, in a prospective study on Russian and US men aged 40-59 years; Stevens et al (Stevens *et al.*, 2004) concluded that the effects of fitness might be more robust across populations than are the effects of fatness. Stevens et al (Stevens *et al.*, 2002) in previous study reported that both 'fitness and fatness' are opposing risk factors for mortality, but that being fit does not completely reverse the increased risk associated with excess adiposity.

Similarly, Meyer et al (Meyer *et al.*, 2002) found that even among men who reported a high level of physical activity during leisure time, estimated by questionnaire, obesity was associated with an increased total mortality. In women participating in the nurses's health study both BMI and the level of physical activity significantly and independently predicted

mortality, but a high physical activity level did not eliminate the excess of deaths associated with obesity (Hu *et al.*, 2004a). A study of healthy men showed that fatness was more strongly and consistently associated with cardiovascular disease risk than aerobic fitness, assessed by maximal oxygen consumption (Christou *et al.*, 2005). Weinstein et al (Weinstein *et al.*, 2004) found that both BMI and physical activity were important for the development of type 2 diabetes in women. However, BMI was a better predictor than recreational physical activity, which was measured by a validated questionnaire, in predicting the incidence of type 2 diabetes.

There is thus still debate as to the magnitude of influence these two factors have in combination on health outcomes, perhaps because of the differences among study populations, methods and outcomes (Blair & Church, 2004) and the recommendations for physical activity vary.

Therefore, there are two main research questions for this study:

1) What is the association between currently recommended physical activity levels with BMI and body shape WC, HC and WHR?

2) What is the association between the combination of current recommended physical activity levels and BMI with CVD risk factors?

5.2. Subjects and methods

5.2.1. Sample

The Scottish Health Survey is a cross sectional nationally representative survey programme that was designed to provide a comprehensive picture of the health of the Scottish population and to document the prevalence of health risk factors as well as

monitor progress towards health targets. Full details of the survey methods have been published elsewhere (Shaw *et al.*, 2000) and mentioned in chapter 2.

Of the total 9047 adults aged 16-74 years (3941 men and 5106 women) who participated in the 1998 survey, 8100 subjects had a valid BMI and reported their physical activity. The total numbers of valid blood samples that have been used in this analysis were: total plasma cholesterol 5924, high-density lipoprotein cholesterol (HDL-C) 5891, c-reactive protein (CRP) 5988, fibrinogen 5460, Systolic blood pressure 6221 and general health questionnaire (GHQ12) 8045.

5.2.2. Anthropometric measures

Weight and height were measured using standard techniques by trained staff (Shaw *et al.*, 2000). Height was measured in a standing position with a portable stadiometer. Body weight was measured to the nearest 0.1 kg in bare feet and light clothes with the Soehlne scales. BMI was calculated as weight divided by height squared (kg/m^2). Healthy body weight, overweight and obesity were defined as a BMI of 18.5-24.9, 25 - 29.9 and ≥ 30 kg/m^2 respectively (WHO, 1998).

Cigarette smoking status was classified as follows: regular cigarette smokers, those who said they smoked cigarette at all at the time of the interview; ex-smokers, those who smoked cigarettes regularly in the past but not currently; and non-smokers: those who had never smoked cigarettes regularly and were not current smokers. Reported levels of physical activity were measured by a questionnaire that asked about the frequency, duration and intensity of four major types of activity: activity at home, walks of 15 minutes or more, sports and exercise activities, and activity at work in the four weeks before the interview (Shaw *et al.*, 2000). These activities were then summed up to calculate

estimated total physical activity, which was divided into five categories based on different levels of physical activity recommendations (American College of Sports Medicine, 1990; Blair & Connelly, 1995). The categories were: inactive, low activity, at least 30 minutes moderate activity on at least 5 days a week, at least 20 minutes vigorous activity on at least 3 days a week, and 30 minutes moderate activity on 5 days a week plus 20 minutes vigorous activity on 3 days a week. Due to the small sample size of some of the subcategories, these 5 categories were collapsed into 3: active, those who reached at least one of the two guideline levels (either 3 occasions of twenty minutes vigorous activity per week or 5 occasions of moderate activity per week or both); less active: those were not active enough to meet either guideline level but were active on at least one day a week; and inactive, those respondents who reported less than one day per week of moderate or vigorous activity of at least 20 minutes duration.

Habitual alcohol consumption over the previous 12 months was assessed with questions on frequency, type, average number of days per week on which alcohol was drunk, the usual quantity consumed on any one day and the finally the "usual" weekly units of alcohol consumed calculated. This was then divided into 4 groups of weekly alcohol intake for both men and women. For men, these quantities were under 1 unit, 1-10 units, 10-21 units and over 21 units. For women these were under 1 unit, 1-7 units, 7-14 units and over 14 units.

Information on dietary habits was obtained by a short dietary questionnaire, which included questions relating to type and frequency of major food items (Lean *et al.*, 2003). In this study total fruit and vegetable consumption was accessed using a categorical variable divided in three groups; low consumers (< 200g per day), moderate consumers

(200 - < 400g per day) and achievers of current targets (≥ 400 gram per day) (The Scottish Office, 1996).

Social class was based on the Registrar General's Standard Occupation Classification using the current or last occupation of the chief income earner within informant's household, in one of 4 categories: professional and intermediate, skilled (non-manual), skilled (manual), partly skilled and unskilled (Shaw *et al.*, 2000).

Blood pressure was measured by using an automated device, the Dinamap 8100 monitor. Three blood pressure readings were taken on the right arm in a seated position after five minutes rest. The mean of the second and third readings were used as the blood pressure (Shaw *et al.*, 2000).

The GHQ12 has been used to assess the psychosocial health of participants (Goldberg & Williams, 1988; Shaw *et al.*, 2000). Participants were asked to complete a self-completion booklet which comprised 12 questions about general levels of happiness, anxiety, depression, stress and sleep disturbance over the past few weeks prior to the interview. An overall GHQ12 score equal or greater than four has been used to identify subjects with a poor psychological health.

5.2.3. Metabolic Syndrome and predicted coronary heart disease risk

The current criteria for Metabolic Syndrome (ATP III)(NCEP, 2001) were not all available. Metabolic Syndrome was defined for this study using the data available in the Scottish health Survey database for any three of the following waist circumference>102 cm

in men and > 88 cm in women, blood pressure ≥ 130/85 mmHg, HDL-C ≤ 1mmol/l in men and <1.3 mmol/l in women, non-high-density lipoprotein cholesterol (non-HDL-C) > 4mmol/l and a medical diagnosis of diabetes (NCEP, 2001). Ten year total coronary heart disease risk was calculated in 1877 men and 2323 women aged 30-74 years by using the 1998 Framingham sex-specific risk equations based on total cholesterol (Wilson *et al.*, 1998). Subjects with three major existing cardiovascular conditions (angina, heart attack or stroke) were excluded before calculating the risk.

5.2.4. Blood samples analyses

Non-fasting venous blood samples were obtained and analysis for CRP, total cholesterol, HDL-C and fibrinogen carried out using standardized methods (Shaw *et al.*, 2000). Non-HDL-C, which contains cholesterol in low-density lipoprotein and very low-density lipoprotein, was calculated by subtracting HDL-C from total cholesterol (Grundy, 2002).

5.2.5. Data analyses

Analysis was carried out using the statistical package, SPSS 11.0 (SPSS INC., Chicago, IL, USA). As data were not normally distributed, log transformed values of some of the dependent variables were used for improving the normality of distributions.

For the association of physical activity and anthropometric data, analyses were stratified by gender and age group. Mean anthropometric data by physical activity were calculated among different age and sex categories.

To evaluate the combined impact of physical activity and BMI, physical activity status and BMI were combined into nine categories and inactive subjects with BMI below 25 kg/m² were defined as the reference category. A general linear model incorporating the

Bonferroni *post hoc* test was used to compare the adjusted means of anthropometric data within physical activity, age and sex categories and the adjusted geometric means risk factors within a combined BMI and physical activity variable in a stratified analysis by sex. In this analysis age was used as a covariate and social class, cigarette smoking, combined activity and BMI, alcohol consumption and fruit and vegetable consumption as fixed factors. The logistic regression model was used to compute the OR for the probability of obesity by different levels of physical activity and having high value for CRP (≥ 3 mg/l), fibrinogen (≥ 3 g/l), total cholesterol (≥6.2 mmol/l), low HDL-C (≤ 1 mmol/l), systolic blood pressure (≥ 130 mmHg) and GHQ12 (≥ 4) and Metabolic Syndrome among the subgroups of the combined physical activity and BMI with the reference category (inactive subjects with a BMI below 25 kg/m²). A value of $p<0.05$ was used for statistical significance.

5.3. Results

5.3.1. Physical activity levels with BMI and body shape

Prevalence of obesity was highest in inactive men and women with 29% and 35% respectively (table 1). Normal weight defined by BMI 18.5-25 kg/m² predominated among people who performed level '3×20 vigorous' plus '5×30 moderate' of activity with 50% in men and 58% in women.

Table 2 shows the adjusted odds ratio of obesity (BMI ≥ 30 kg/m²) versus healthy body weight (BMI = 18.5-24.9 kg/m²). Apart from low activity level (in men) and '3×20 vigorous' activity level (in both men and women), all kinds of activity levels were associated with lower risk of prevalence of obesity, but not prevalence of overweight in both sexes.

Participants who were performed only '5×30 mod' level or '3×20 vigorous' plus '5×30 moderate' level of activity had significantly lower BMI, WC, and WHR compared with inactive persons in both sexes (figures 1, 2, 3 and 4). All physical activity levels were associated with a lower mean BMI and WC (after further adjustment for BMI), but '3×20 vigorous' group in men and women and low activity level in men were not significantly associated with BMI and WC (figures 1 and 2). Figures 3 and 4 show the associations between mean WC and WHR and physical activity without BMI adjustment. In this figure we can see that without BMI adjustment, all activity levels except '3×20 vigorous' group in women were significantly associated with lower WC and WHR. Mean HC did not differ across different activity levels when it was further adjusted for BMI. However, mean HC were significantly higher in men participants who performed '5×30 mod' level or '3×20 vigorous' plus '5×30 moderate' compared with inactive subjects when extra adjustment was mad for WC. This association was not significant in women (figure 5).

Table 3 presents adjusted mean BMI according to physical activity levels, age groups and sex. Inactive subjects had significantly higher mean BMI compared with active subjects as a whole in both sexes ($p<0.001$). There were also significant differences between the lower but active group with the active group in both sexes ($p < 0.05$). Participants who were in the lower but active group had a lower BMI compared with the inactive group. However it was significant only in females ($p<0.05$). Inactive participants in males aged older 34 years and in women older than 24 years had higher BMI compared with active counterparts, but the differences were significant only in men aged 35-54, and in females aged 25-34 and 55-74 years ($p < 0.05$). There were no significant differences among youngest group (16-24 years) in terms of activity levels. In this group, in both sexes, inactive subjects were lighter than the other levels.

Active subjects had lower adjusted mean WC when compared with inactive subjects in all age groups and in both sexes (table 4) even after adjustment for BMI. The differences were significant in all subjects as one group in both sexes ($p < 0.001$), and in men in the age group 45-64 years and in women in the age group 25-34 years ($p<0.05$). Subjects who were in the lower but active group had a higher WC compared with active subjects, but lower compared with inactive subjects. There were same associations between WC and physical activity without BMI adjustment, however further adjustment for BMI reduced the mean WC differences between active and inactive subjects, particularly in women.

Although active younger men had slightly higher adjusted mean HC than inactive ones when adjusted for BMI, there were no significant differences among them (table 5). However, without further adjustment for BMI, inactive subjects had a significantly higher HC than the active group in both sexes ($p< 0.05$). Whereas, mean HC adjusted for WC in men (table 6) showed that physical activity was significantly associated with higher HC in men particularly in younger age groups. There were no such associations in women.

Mean WHR was significantly lower among those who were active compared with inactive subjects in both men and women in all ages, and as a group ($p<0.001$) (table 7). In men in all age groups within the exception of the oldest age group (65-74 years) and in women only in the age group 25-34 years, the mean WHR were significantly higher in inactive compared with active groups. Further analysis without BMI adjustment did not changed these associations very much.

5.3.2. Physical activity, BMI and cardiovascular disease risk factors

General characteristics of the study population who had valid blood sample are presented in table 8. Approximately 34% male and 33% female subjects were current smokers, 63% of men and 54% of women were either overweight or obese and around 20% of total sample were obese. The proportion of the total subjects who achieved the recommended physical activity levels (either 3×20 vigorous or 5×30 moderate or both of them) were 33%, 38% men and 29% women. Around 31 % of men consumed more than 21 units of alcohol per week and among women almost 14 % more than 14 units per week. Only 16.6% of men and 28.7% of women reached the current targets of five portions daily for total fruit and vegetables consumption. Almost 22 % of the population was classified as partly skilled and unskilled by the social class of chief income earners.

Figures 6-8 show the adjusted geometric mean values of the risk factors among different categories of combined physical activity and BMI in men and women. Initial analyses used three separate categories for people who achieved the recommended activity levels (either 3×20 vigorous or 5×30 moderate or both). These have not been presented in detail due to the small numbers of subjects in these groups, e.g. 7 men and 13 women with BMI ≥ 30 kg/m^2 had reported 3×20 vigorous activity level. The patterns of cardiovascular risk for these 3 categories were similar, so they were collapsed into a single "active" category. Overweight and obese subjects had a significantly lower mean HDL-C concentrations, regardless of physical activity levels, when compared with inactive subjects with BMI < 25 kg/m^2 in both sexes ($p<0.001$), (figure 6). Mean cholesterol and non-HDL-C were significantly higher in overweight and obese subjects ($p<0.01$) and activity levels did not change the results significantly. Mean CRP concentration was highest in obese inactive subjects. Although mean CRP concentrations were lower in active subjects in all BMI categories compared with inactive people, particularly in active subjects with BMI < 30 kg/m^2, the mean CRP was still significantly higher in obese active subjects in both sexes

compared with reference categories ($p<0.05$), (figure 7). Mean systolic blood pressure rose with increasing BMI in different activity levels, however it was significantly higher in female obese subjects in all activity levels, compared with those inactive with a BMI < 25 kg/m^2, ($p<0.001$). Female obese subjects had significantly higher mean fibrinogen levels across the physical activity levels than the reference group ($p<0.001$). Active males and less active females with BMI < 25 kg/m^2 had significantly lower mean fibrinogen concentration when compared with inactive subjects with BMI < 25 kg/m^2 ($p<0.05$), (figure 8).

Table 9 shows adjusted OR of having the cardiovascular disease risk factors and GHQ scores above the cut-off points by physical activity status and BMI. After controlling for age, gender, social class, smoking, alcohol intake and fruit and vegetable consumption; inactivity, overweight and obesity were associated significantly with higher OR for elevated cholesterol, CRP, systolic blood pressure, non-HDL-C and lower HDL-C than inactive with BMI <25 kg/m^2 ($p<0.05$). Physical activity improved GHQ scores in all BMI categories ($p<0.001$). Physical activity reduced the likelihood of lower HDL-C and higher CRP in obese subjects, but it did not eliminate the higher risk of the measured cardiovascular disease risk factors in this group and OR of these two risk factors were still high. These were 4.39 and 2.67 respectively compared with the reference group ($p<0.001$). Increasing physical activity did not change the OR of having higher systolic blood pressure values, but overweight and obesity significantly increased the OR across different physical activity levels. Overweight and obese subjects had significantly higher OR for higher non-HDL-C in the different activity categories ($p<0.001$). The OR of having higher fibrinogen decreased in active subjects with BMI < 30 kg/m^2 ($p<0.001$), however it did not change in obese participants.

Table 10 shows OR for subjects who felt within criteria for metabolic syndrome (NCEP, 2001). Fasting glucose and triglyceride values were not available; therefore medically diagnosed diabetes and non-HDL-C values were used to estimate metabolic syndrome. With this definition, almost 20% of men and women were categorized with Metabolic Syndrome and the OR of having Metabolic Syndrome was significantly higher in overweight and obese subjects within each category. Being physically active had a protective effect with Metabolic Syndrome lower in the obese active compared with obese inactive subjects particularly in men, however prevalence of Metabolic Syndrome was still high among obese active subjects.

About 30% of men and 9% of women had a predicted 10-year coronary heart disease risk ≥15%. Amongst these, obese inactive subjects had the highest proportion of the risk, 57.6% in men and 26.5% in women. The median coronary heart disease risk was highest among obese inactive (16.6% in men and 10.3% in women) and the lowest among active subjects with BMI < 25 kg/m² (5.4% in men and 1.8% in women). The geometric mean of coronary heart disease risk was significantly lower in obese active and active subjects with BMI < 25 kg/m² compared with their counterparts ($p<0.001$). The mean coronary heart disease risk was not significantly different between obese active and inactive subjects with BMI < 25 kg/m². However, it should be noted that inactive groups were older than active groups and because age was part of the risk equations, it was not controlled for in these analyses. The distribution of predicted coronary heart disease risk among BMI/activity categories across ages 30-74 has been shown in figures 9 and 10. Figure 9 shows, in men average coronary heart disease risk was highest in the obese who were inactive and lowest in the active subjects with BMI < 25 kg/m². Obese active men had lower average coronary heart disease risk than obese inactive, but higher than the inactive group with BMI < 25 kg/m². In women, figure 10, obese groups had higher average coronary heart disease risk

than group with BMI < 25 kg/m², however physical activity reduced the risk slightly only in older obese subjects.

5.4. Discussion

5.4.1. Physical activity levels with BMI and body shape

Regular physical activity has a major role in preventing weight gain and managing overweight and obesity. The present study has examined the associations between current recommendations of physical activity levels with BMI, WC, HC, and WHR in a large sample. This showed that the proportion of people that who are doing 3×20 vigorous activity per week were few and that these people were mostly in the younger age groups. In the Allied Dunbar National Fitness Survey (Allied Dunbar, 1992) also only 14% of men and 4 % of women were '3×20 vigorously' active. There was a negative association between physical activity and BMI and the active subjects had the lower mean BMI. Although subjects who reported achieved the recommended level of physical activity had healthier BMI than inactive subjects, almost 50% of active people still had unhealthy BMI (BMI $\geq$ 25 kg/m²). It has also been revealed that current physical activity recommendations have a negative association with WC and WHR as an index of central obesity independent of BMI.

A review of several prospective studies showed that there is a moderately strong relationship between low levels of physical activity and the risk of developing obesity (Grundy *et al.*, 1999). Many cross-sectional studies have revealed an inverse association between physical activity and body weight or BMI (DiPietro, 1999), however longitudinal studies showed that habitual physical activity are more effective to reduce weight gain rather that encourage weight loss.(DiPietro, 1999). In spite of a negative association between physical activity and BMI, evidence has shown that there is still a high prevalence

of overweight and obesity in groups with high physical activity levels (Erlichman *et al.*, 2002).

In the European Investigation into Cancer and Nutrition study (EPIC) (Trichopoulou *et al.*, 2001), a higher energy expenditure, as estimated by physical activity questionnaire, was significantly associated with lower WHR after controlling for BMI in men, but not in women. Our findings are in line with some of these studies indicating a negative association between physical activity and BMI or WC. However, this level of physical activity is not enough to prevent development of an unhealthy BMI. The cross-sectional design of the SHS means causal relationships between physical activity and body weight and shape cannot be proven. We do not know that the physical activity levels are the cause of the anthropometric changes, or the consequences of the anthropometric changes. However, the association between HC and physical activity in larger subjects is interesting. A high HC probably reflects increased body fat in older and more overweight subjects, but in younger people, and those within or near to normal body fat content, HC it may reflect increased muscle mass. Changes in HC may also show falls in muscle mass (e.g. in people developing type 2 diabetes). BMI also reflects differences in muscle mass, most obviously in sports men. Adjusting HC for BMI is therefore unhelpful, but adjusting HC for WC may help to remove the effect of body fat.

5.4.2. Physical activity, BMI and cardiovascular disease risk factors

Many studies of different types have demonstrated that physical activity has protective effects for chronic diseases, including CHD, hypertension, diabetes, osteoporosis, colon cancer, and anxiety and depression (Pate *et al.*, 1995). Apart from the effects of physical activity on obesity, it is reported that physical activity or physical fitness has additional health benefits, independent of BMI.

Physical activity is defined as "any bodily movement produced by skeletal muscles that results in energy expenditure" and physical fitness is a "set of attributes that people have or achieve that relates to the ability to perform physical activity" (Caspersen *et al.*, 1985; Pate *et al.*, 1995). The amount of physical activity necessary for preventing cardiovascular disease risk is not clear; however different amounts and types of physical activity have been recommended (American College of Sports Medicine, 1990; Blair & Connelly, 1995).

Cardiorespiratory fitness, assessed with maximal treadmill exercise to calculate the maximal oxygen uptake, is stronger and more accurate than self-reported physical activity as a predictor of health outcome (Blair *et al.*, 2001). However the most accurate methods of measuring fitness, such as VO2 max are often unavailable and are not feasible for large population studies. Although more accurate methods are needed to measure total physical activity, a physical activity questionnaire is the most practical and widely used instrument for measuring physical activity in population studies. An evaluation of the effects of recommendations for physical activity levels for public health on cardiovascular disease risk factors would be beneficial to clarify the health effects of specific amount of physical activity along with BMI. In the present study we have assumed that reported physical activity reflects fitness, and evaluated the associations between recommended levels of physical activity, in combination with overweight and obesity, and cardiovascular risk factors.

Being active in this study was defined as: at least 30 minutes moderate activity on at least 5 days a week or at least 20 minutes vigorous activity on at least 3 days a week or both. About 38% of all men, and 29% of all women fell into this category. Overweight and

obesity were strongly linked with raised risk factors and predicted coronary heart disease risk. Subjects who were overweight or obese had greater mean and OR for most of the cardiovacular risk factors, Metabolic Syndrome and predicted coronary heart disease risk than subjects with BMI <25 kg/m². Recommended physical activity levels reduced the risk associated overweight and obesity for CRP, HDL-C, predicted coronary heart disease risk and Metabolic Syndrome, particularly in men when compared with the reference group of inactive subjective with BMI <25 kg/m². However, this level of activity could not eliminate the health risks associated with obesity and those who achieved this level still were at elevated risk compared to the non obese reference group. The results showed that physical activity improved self-assessed health scores across all BMI categories.

Our finding that BMI was a more important factor than physical activity in association with cardiovascular disease risk factors and predicted coronary heart disease risk is in agreement with some other studies (Meyer *et al.*, 2002; Hu *et al.*, 2004a; Weinstein *et al.*, 2004; Christou *et al.*, 2005). Meyer et al (Meyer *et al.*, 2002) found that in all categories (sedentary, moderate, intermediate and intensive) of self-reported physical activity during leisure time, obese men had a similar increased relative risk of death compared with normal weight individuals in the same category of physical activity. Weinstein et al (Weinstein *et al.*, 2004) examined the combined relationship of BMI and physical activity (self-reported recreational activity during the past years) in women and found that increasing physical activity had a modest reduction in the risk of diabetes compared to a large increase in the risk with increasing BMI. In a cross-sectional study of 135 healthy men, fatness was a better and stronger predictor of 18 established cardiovascular disease risk factors including total cholesterol, HDL-C, systolic blood pressure and fibrinogen than aerobic fitness assessed by Vo^2 max (Christou *et al.*, 2005). Stevens at al studied a cohort of the Lipid Research Clinics Study of American men and women and reported that both

high levels of fatness and low levels of fitness (assessed using a treadmill test) increased mortality from all cause and from cardiovascular disease (Stevens *et al.*, 2002). This suggested that to reduce the mortality risk a combination of both a moderate level of fitness and low fatness were required.

In contrast, a number of other published studies support the theory that physical fitness is more important than fatness. Katzmarzyk et al in a follow-up study revealed that cardiorespiratory fitness had a protective effect against all-cause and cardiovascular mortality in healthy men and men with the Metabolic Syndrome (Katzmarzyk *et al.*, 2004. In this study body weight status was not an important modifier of mortality risk when cardiorespiratory fitness was taken into account. In Russian men fitness assessed by a treadmill test, but not fatness, was associated with all cause and cardiovascular disease mortality. In US men fatness and fitness were both associated with all-cause and cardiovascular disease mortality {Stevens, 2004 #7; Stevens *et al.*, 2004). Katja et al (Katja *et al.*, 2006) in a cross sectional study of Finnish adults, adjusted for confounding factors including WHR as a measure of obesity showed that self assessed fitness and aerobic fitness measured by questionnaire were inversely associated with CRP concentrations. Another study in adults that examined cardiorespiratory fitness and its association with Metabolic Syndrome followed a prospective design and showed that cardiorespiratory fitness was inversely associated with the incidence of Metabolic Syndrome (LaMonte *et al.*, 2005). This data also showed that second and third cardiorespiratory fitness tertiles were significantly associated with lower risk of developing Metabolic Syndrome even in those men who are overweight or obese (BMI $\geq$ 25 kg/m^2). This association was not significant in women, possibly due to thire small numbers in the study. Katzmarzyk et al (Katzmarzyk *et al.*, 2005) reported that obesity and Metabolic

Syndrome were associated with an increased risk of all-cause and cardiovascular disease mortality, but these risks were largely related to low cardiorespiratory fitness.

Around 450 million people worldwide suffer from mental or behavioral disorders (WHO, 2001) such as depression and anxiety. Good mental health is essential to the overall well being of individuals and societies. Physical activity has been shown to have a positive impact on mental health and psychological well being (Stephens, 1988). The results of a study using a large data set from the US population showed that obesity was negatively associated with health-related quality of life, including mental health. Individuals who used exercise alone or together with diet to lose weight reported better health-related quality of life scores (Hassan *et al.*, 2003). Schmitz et al in the German National Health Interview and Examination Survey found that self-reported physical activity was associated with a better quality of life and higher levels of physical activity were associated with higher health related quality of life among persons with mental disorders (Schmitz *et al.*, 2004). Dunn et al (Dunn *et al.*, 2005) in a randomized placebo control study found that aerobic exercise at a dose compatible with public health recommendations (17.5 kcal/kg/week) was effective in the treatment of mild to moderate major depressive disorder when compared to a lower dose of exercise (7.0 kcal/kg/week) or to control. Our results support these findings and have indicated that active subjects may have suffered less current psychological problems than inactive in all BMI categories. The mechanisms that explain the beneficial effects of physical activity on mental health are unclear. However, various psychological hypotheses such as improvements in distraction, self-efficacy and social interaction, and physiological hypotheses like increased monoamines and endorphins have been proposed (Peluso & Andrade, 2005).

The main limitations of the present study are the cross-sectional design and the self-reported rather than measured physical activity and lack of fasting blood samples necessary to conclusively define Metabolic Syndrome. A measure of fasting blood glucose and HDL-C would have assisted greatly with a conclusive definition of Metabolic Syndrome. Both shortcomings represent practical limitations with large-scale representative survey. A lack of information on total energy intake may also be another limitation of this study.

Our results show that for those reporting having achieved the recommended physical activity levels, some cardiovascular disease risk factors were reduced and improve psychosocial health improved. These benefits cannot eliminate the extra health risks imposed by overweight/obesity. Our data cannot be used to suggest that a higher recommendation for physical activity in obese people might be necessary to reverse their increased cardiovascular disease risk, but more active populations would appear to be the healthier ones from these data.

5.5. Conclusion

Current recommendations of physical activity were associated with lower BMI and risk of obesity (BMI $\geq$ 30 kg/m^2), but did not alter the prevalence of overweight (BMI 25-30 kg/m^2). Moderate activity of 30 minutes 5 days per week (in combination with vigorous activity 20×3 days per week) was associated with lower central obesity independent of BMI and may help prevent weight gain and abdominal fat accumulation. Vigorous activity alone has more limited value, but may help to reduce obesity in men. These associations do not necessarily imply causality, but do support the recommendation of 5×30 moderate rather than the 3×20 alone.

Overweight and obesity were associated with significantly greater mean and/or OR for elevated total cholesterol, CRP, systolic blood pressure, lower HDL-C, higher prevalence of Metabolic Syndrome and predicted 10-year coronary heart disease risk than BMI <25 kg/m^2. Currently recommended physical activity levels for cardiovascular health modestly reduced the risk of lower HDL-C, higher CRP concentrations and predicted coronary heart disease risk. The higher cardiovascular disease risk factors in active obese subjects were not eliminated when compared to inactive subjects with BMI<25 kg/m^2. Physical activity improved general health scores across all BMI categories, therefore obese active subjects reported feeling better according to their GHQ score. These data support messages, which stress the importance of both physical activity and reducing body weight in obese subjects to challenge cardiovascular disease risk.

Table 5. 1: Prevalence of normal weight (BMI 18.5-25 kg/m²), overweight (BMI 25-30 kg/m²) and obesity (BMI 30 kg/m²) according to sex and physical activity levels.

Activity levels	Male				Female			
	n	BMI 18.5-25 kg/m²	BMI 25-30 kg/m²	BMI ≥ 30 kg/m²	n	BMI 18.5-25 kg/m²	BMI 25-30 kg/m²	BMI ≥ 30 kg/m²
Inactive	835	28.9	42.6	28.5	971	32.7	32.5	34.7
Low activity	1365	33.1	45.1	21.8	2218	42.5	34.8	22.7
5×30 moderate #	951	38.6	45.2	16.2	1012	51.6	31.7	16.7
3×20 vigorous #	71	36.6	53.5	9.9	53	50.9	24.5	24.5
3×20 plus 5×30#	315	49.8	40	10.2	173	57.8	27.2	15.0

5×30 moderate: 30 minutes moderate activity on 5 days a week, 3×20 vigorous: 20 minutes vigorous activity on 3 days a week, 3×20 vigorous plus 5×30 moderate: 20 minutes vigorous activity on 3 days a week plus 30 minutes moderate activity on 5 days a week.

Table 5. 2: Odds ratio[a] of obesity (BMI ≥ 30 kg/m²) versus healthy BMI (18.5- 25 kg/m²) by sex and physical activity levels.

Activity levels	Male		Female	
	BMI ≥ 30 Odds (95% CI) †	P Value	BMI ≥ 30 Odds (95% CI)	P Value
Inactive	1		1	
Low activity	0.83(0.64-1.10)	0.2	0.71(0.58-0.88)	<0.01
5×30 moderate	0.61(0.45-0.82)	<0.01	0.48(0.38-0.63)	<0.001
3×20 vigorous	0.41(0.16-1.10)	<0.07	0.79(0.38-1.70)	0.54
3×20 plus 5×30	0.34(0.21-0.54)	<0.001	0.44(0.27-0.72)	<0.01

[a,] adjusted for age, social class, smoking and alcohol consumption † CI: confidence interval,

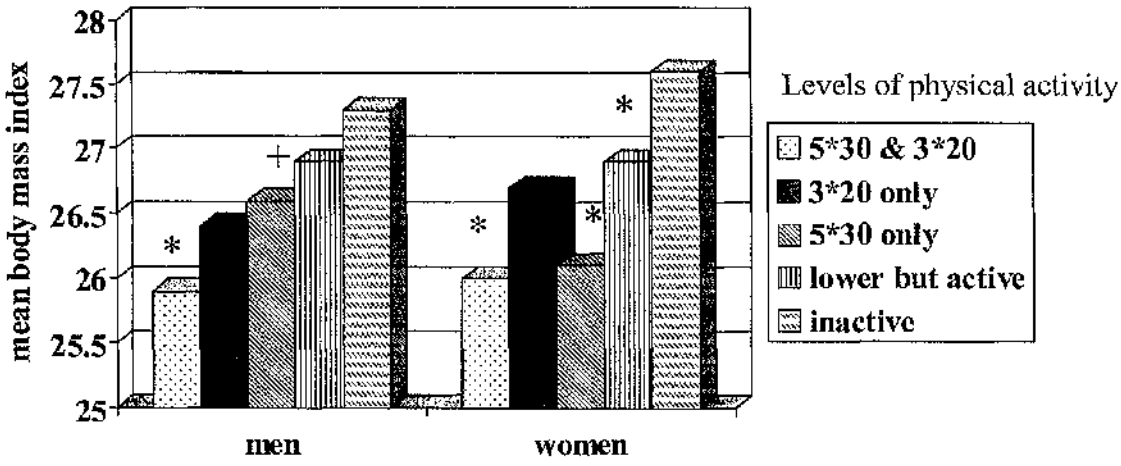


Figure 5. 1: Mean BMI (adjusted for age, social class, cigarette smoking and alcohol consumption) by physical activity levels

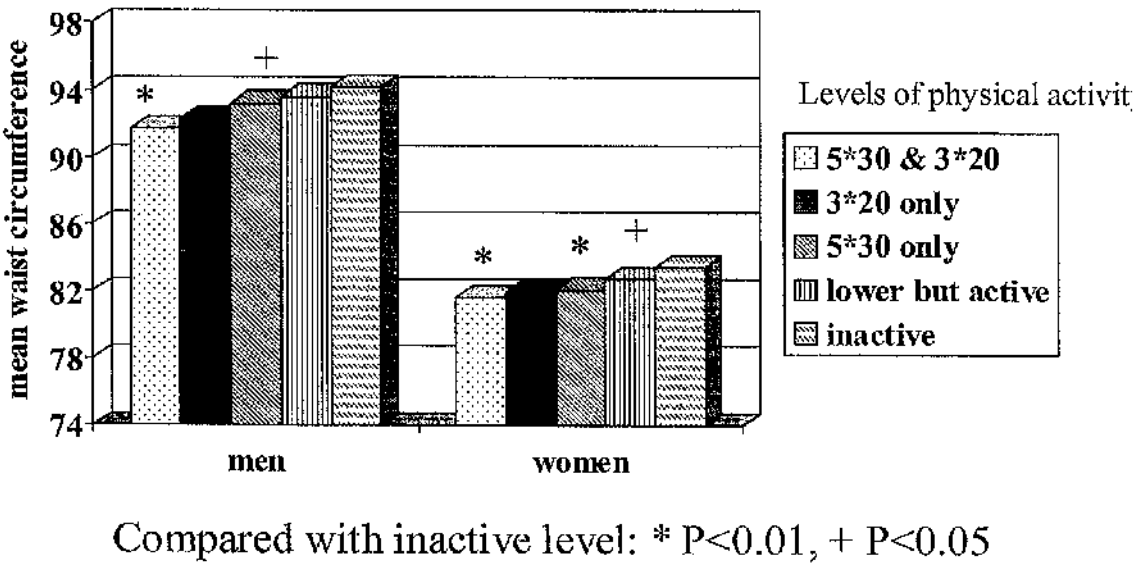


Figure 5. 2: Mean waist circumference (adjusted for age, social class, cigarette smoking, BMI and alcohol consumption) by physical activity levels

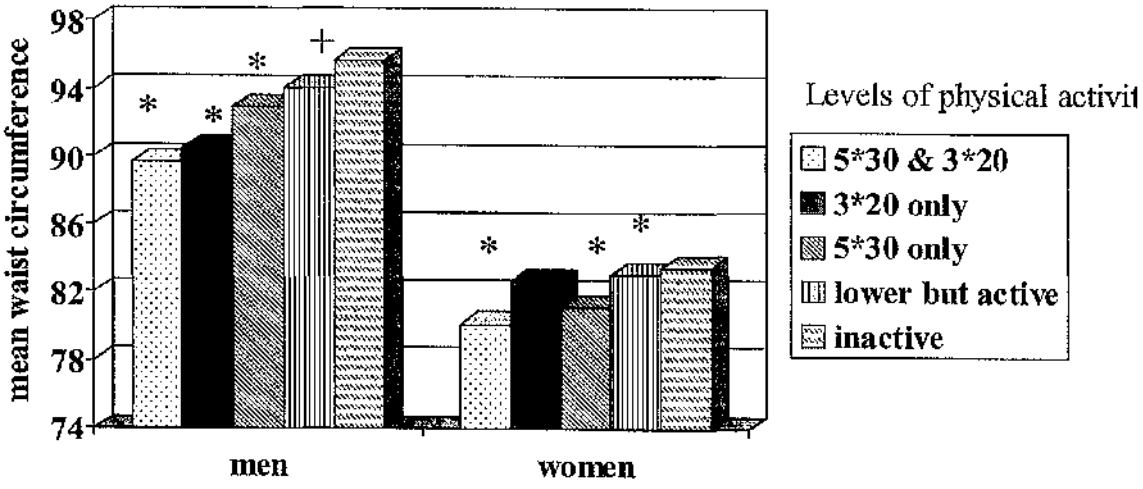


Figure 5. 3: Mean waist circumference (adjusted for age, social class, cigarette smoking and alcohol consumption) by physical activity levels

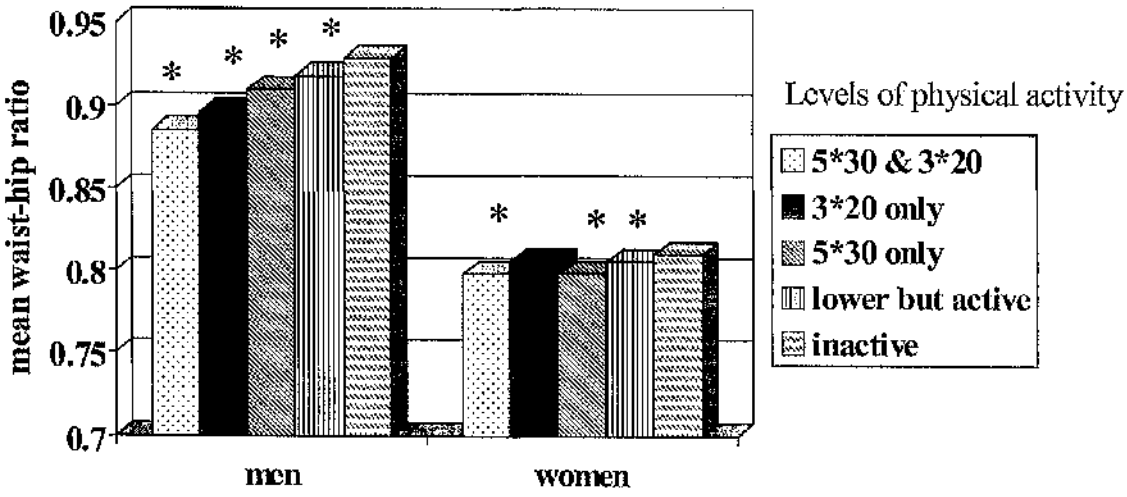


Figure 5. 4: Mean waist-hip ratio (adjusted for age, social class, cigarette smoking and alcohol consumption) by physical activity levels

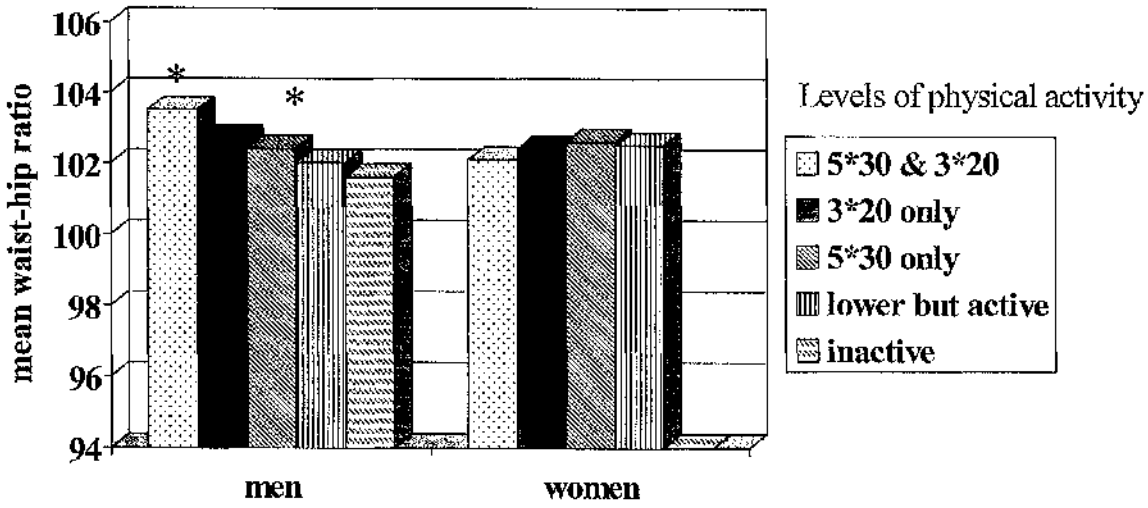


Figure 5. 5: Mean hip circumference (adjusted for age, social class, cigarette smoking, alcohol consumption and waist circumference) by physical activity levels

Table 5. 3: Adjusted mean[a] BMI (kg/m^2) by physical activity, age and sex

		Men				Women		
Age groups (Year)	n	Active	Lower but active	Inactive	n	Active	Lower but active	Inactive
16-24	371	23.0(22.1-23.9)	23.5(22.5-24.6)	22.6(20.6-24.6)	470	23.9(23.1-24.8)	24.7(23.9-25.5)	23.1(21.6-24.5)
25-34	703	26.0(25.4-26.6)	26.3(25.7-27.0)	26.0(25.0-27.1)	864	25.4(24.7-26.0)	26.0(25.4-26.6)	28.0(26.8-29.2)+**
35-44	761	26.4(25.8-26.9)	26.6(26.1-27.1)	27.7(26.8-28.5)*	915	25.9(25.3-26.5)	26.7(26.2-27.3)	26.6(25.6-27.6)
45-54	644	27.3(26.7-28.0)	28.3(27.7-28.9)	28.9(28.1-29.6)*	803	26.6(25.8-27.3)	27.6(27.0-28.2)	27.8(26.9-28.7)
55-64	617	27.1(26.4-27.8)	27.7(27.1-28.3)	28.1(27.5-28.7)	716	27.0(26.0-27.9)	28.0(27.3-28.7)	29.3(28.5-30.1)+**
65-74	495	26.6(25.6-27.6)	27.0(26.4-27.6)	27.6(27.0-28.2)	755	26.0(24.8-27.2)	27.1(26.4-27.8)	28.3(27.6-29.0)**
All ages §	3591	26.5(26.2-26.7)	26.9(26.7-27.2)*	27.3(27.0-27.6)**	4523	26.1(25.8-26.4)	26.9(26.6-27.1)**	27.6(27.2-27.9)+**

All data are mean + 95% confidence interval; [a] Adjusted for social class, smoking, total fruit and vegetable consumption and alcohol consumption, *Compared with active $p<0.05$, ** Compared with active $p<0.001$, + Compared with lower but active $p<0.05$,

§ Additionally adjusted by age.

Table 5. 4: Adjusted mean[a] waist circumference (cm) by physical activity, age and sex.

	Men				Women			
Age groups (Year)	n	Active	Lower but active	Inactive	n	Active	Lower but active	Active
16-24	306	81.1(80.0-82.3)	82.6(81.25-84.0)	82.8(80.2-85.4)	389	74.5(73.4-75.5)	74.4(73.5-75.4)	74.7(73.0-76.4)
25-34	595	89.6(88.8-90.4)	90.1(89.3-91.0)	91.0(89.6-92.4)	745	78.1(77.2-78.9)	79.6(78.8-80.3)*	80.5(79.0-81.9)*
35-44	679	91.7(90.9-92.5)	92.2(91.4-93.0)	92.8(91.6-94.1)	801	80.6(79.9-81.3)	81.3(80.7-82.0)	80.6(79.4-81.8)
45-54	585	95.4(94.6-96.3)	96.8(96.0-97.6)*	97.1(96.1-98.1)*	747	83.2(82.4-84.0)	84.1(83.4-84.7)	84.3(83.3-85.4)
55-64	584	96.7(95.7-97.7)	97.4(96.5-98.2)	98.2(97.4-99.1)*	651	86.9(85.7-88.1)	86.8(86.1-87.6)	88.0(87.0-88.9)
65-74	485	97.2(95.6-98.8)	96.9(96.0-97.9)	97.3(96.4-98.2)	691	86.2(84.5-87.8)	86.5(85.5-87.5)	88.3(87.3-89.3)+
All ages §	3234	92.8(92.5-93.2)	93.5(93.2-93.9)*	94.1(93.6-94.5)**	4024	82.0(81.6-82.4)	82.7(82.4-83.0)*	83.5(83.0-83.9+**

All data are mean ± 95% confidence interval; [a] Adjusted for social class, smoking, BMI, total fruit and vegetable consumption and alcohol consumption, * Compared with active $p<0.05$, ** Compared with active $p<0.001$, + Compared with lower but active $p<0.05$
§ Additionally adjusted by age

Table 5. 5: Adjusted mean[a] hip circumference (cm) by physical activity, age and sex

	Men				Women			
Age groups (Year)	n	Active	Lower but active	Inactive	n	Active	Lower but active	Inactive
16-24	306	98.8(97.7-99.8)	98.3(97.1-99.6)	96.6(64.3-98.9)	390	97.8(96.8-98.7)	97.4(96.6-98.3)	97.6(96.99.3)
25-34	595	102.0(101.3-102.7)	101.5(100.7-102.3)	101.2(99.9-102.5)	745	101.0(100.2-101.7)	101.8(101.2-102.5)	101.0(99.7-102.3)
35-44	680	102.5(101.9-103.1)	102.3(101.7-103.0)	101.9(101.0-102.9)	800	101.4(100.8-102.0)	101.9(101.3-102.4)	101.3(100.4-102.4)
45-54	584	103.0(102.3-103.7)	103.4(102.8-104.1)	102.5(101.6-103.3)	748	103.0(102.3-103.6)	103.1(102.6-103.6)	102.9(102.1-103.7)
55-64	585	102.9(102.1-103.7)	103.1(102.4-103.8)	102.6(102.0-103.3)	649	104.6(103.5-105.7)	105.3(104.6-106.1)	105.1(104.3-106.0)
65-74	486	103.1(101.7-104.6)	102.5(101.6-103.3)	103.7(102.8-104.6)	694	103.8(102.5-105.1)	103.6(102.5-104.4)	104.2(103.4-105.0)
All ages §	3236	102.4(102.0-102.7)	102.2(101.9-102.5)	102.1(101.7-102.5)	4026	102.1(101.8-102.5)	102.5(102.2-102.7)	102.4(102.1-102.8

All data are mean + 95% confidence interval; [a] Adjusted for social class, smoking, BMI, total fruit and vegetable consumption and alcohol consumption, § Additionally adjusted by age.

Table 5. 6: Adjusted mean[a] hip circumference (cm) by physical activity, age and sex

	Men				Women			
Age groups (Year)	N	Active	Lower but active	Inactive	n	Active	Lower but active	Inactive
16-24	306	99.2(98.1,100.3)	97.9(96.7,99.1)	96.1(93.9,98.4)*	390	97.9(96.9,99.0)	97.6(96.6,98.6)	97.8(96.1,99.5)
25-34	595	102.2(101.6,102.8)	101.4(100.7,102.1)	100.7(99.6,101.8)*	745	102.1(101.3,102.9)	101.8(101.1,102.4)	101.0(99.7,102.2)
35-44	680	102.9(102.3,103.5)	102.4(101.9,103.0)	101.6(100.7,102.5)*	800	101.3(100.7,102.0)	101.4(100.8,102.0)	101.5(100.4,102.6)
45-54	584	103.5(102.9,104.1)	103.2(102.7,103.8)	102.2(101.5,102.9)*	748	103.0(102.3,103.8)	103.0(102.4,103.5)	102.4(101.5,103.3)
55-64	585	103.3(102.6,104.1)	103.1(102.4,103.7)	102.4(101.8,103.0)	649	104.4(103.2,105.6)	105.5(104.7,106.3)	105.0(104.1,106.0)
65-74	486	103.4(102.0,104.7)	102.6(101.8,103.4)	103.7(103.0,104.5)	694	103.8(102.2,105.4)	104.1(103.1,105.0)	104.0(103.1,105.0)
All ages §	3236	102.7(102.4,103.0)	102.2(101.9,102.4)*	101.8(101.4,102.1)**	4026	102.5(102.1,102.9)	102.5(102.3,102.8)	102.2(101.8,102.6)

All data are mean + 95% confidence interval; [a] Adjusted for social class, smoking, **waist circumference**, total fruit and vegetable consumption and alcohol consumption, * Compared with active $p<0.05$, ** Compared with active $p<0.001$, § Additionally adjusted by age.

Table 5. 7: Adjusted mean[a] waist-to-hip ratio by physical activity, age and sex

	Men				Women			
Age groups (Year)	n	Active	Lower but active	Inactive	n	Active	Lower but active	Inactive
16-24	306	0.82(0.81-0.83)	0.84(0.82-0.85)*	0.86(083-0.88)*	389	0.76(0.75-0.77)	0.76(0.75-0.77)	0.76(0.75-0.78)
25-34	595	0.88(0.87-0.88)	0.89(0.88-0.89)	0.90(0.88-0.91)*	745	0.77(0.76-0.78)	0.78(0.77-0.79)	0.79(0.78-0.81)*
35-44	679	0.89(0.89-0.90)	0.90(0.89-0.91)	0.91(0.90-0.92)*	800	0.79(0.79-0.80)	0.80(0.79-0.80)	0.79(0.78-0.80)
45-54	584	0.92(0.91-0.93)	0.93(0.93-0.94)	0.95(0.64-0.95)**	747	0.81(0.80-0.81)	0.81(0.81-0.82)	0.82(0.81-0.83)
55-64	584	0.94(0.93-0.95)	0.94(0.93-0.95)	0.96(0.95-0.96)+*	648	0.83(0.82-0.84)	0.82(0.82-0.83)	0.84(0.83-0.85)
65-74	484	0.94(0.93-0.96)	0.94(0.94-0.95)	0.94(0.93-0.95)	691	0.83(0.81-0.85)	0.83(0.82-0.84)	0.85(0.84-0.86)
All ages §	3232	0.90(0.90-0.91)	0.91(0.91-0.92)**	0.92(0.91-0.92)+**	4020	0.80(0.80-0.81)	0.80(0.80-0.81)	0.81(0.81-0.82)+**

All data are mean + 95% confidence interval; [a] Adjusted for social class, smoking, BMI, total fruit and vegetable consumption and alcohol consumption, * Compared with active $p<0.05$, ** Compared with active $p<0.001$, +Compared with lower but active $p<0.05$, ++ Compared with lower but active $p<0.001$, § Additionally adjusted by age

Table 5. 8: General characteristics of the total study population, for whom blood sample were taken by sex.

	Male %	Female %	Total n	%
Smoking				
Non-smokers	41.8	48.5	2476	45.5
Ex-smokers	23.9	18.3	1134	20.8
Current smokers	34.3	33.2	1835	33.7
BMI (kg/m²)				
< 25	36.7	45.8	2129	41.7
25-30	44.3	32.8	1945	38.1
≥ 30	18.9	21.3	1034	20.2
Physical activity				
Active	38.5	28.6	1799	33.1
Less active	39.3	50.4	2463	45.3
Inactive	22.2	21.0	1173	21.6
Alcohol (u/w)				
Male 0-1	13.2		323	
1-10	30.7		753	
10-21	25.0		614	
> 21	31.1		763	
Female 0-1		27.6	818	
1-7		40.5	1199	
7-14		18.0	532	
>14		13.9	410	
Social class				
I&II	34.9	34.2	1838	34.5
IIINM	11.5	20.7	879	16.5
IIIM	33.5	22.4	1458	27.4
IV&V	20.1	22.7	1145	21.5
Fruit & vegetables				
Achievers of target	16.6	28.7	1272	23.2
Moderate consumer	35.4	39.4	2062	37.6
Low consumer	48.0	31.9	2155	39.3

n: sample size based on total fibrinogen ,the lowest valid sample among the variables. u/w: units per week

Table 5. 9: Adjusted odds ratio of cardiovascular risk factors and GHQ by activity and BMI

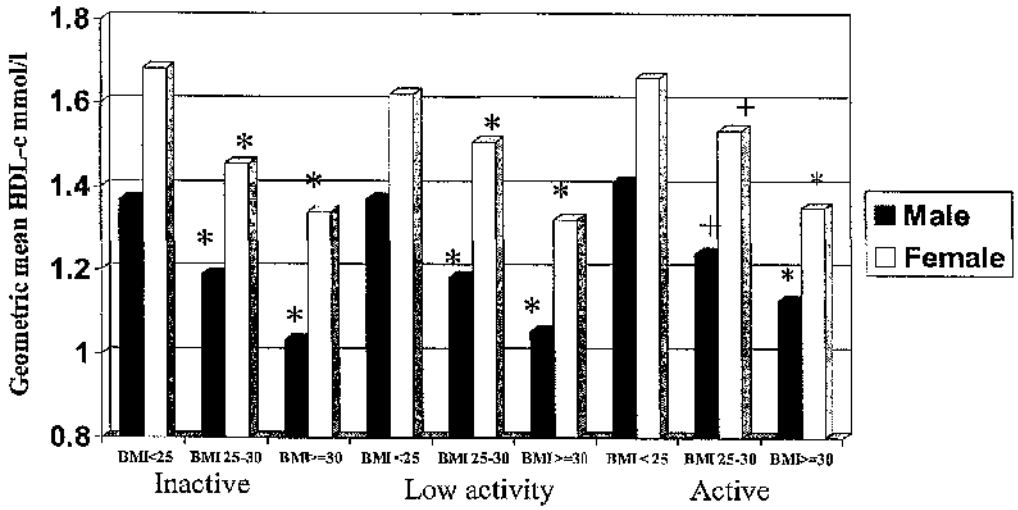
BMI kg/m² /activity categories	n	HDL-c ≤1mmol/l (n 5434)	Cholesterol ≥ 6.2 mmol (n 5463)	CRP ≥ 3 mg/l (n 5525)	Fibrinogen ≥ 3 g/l (n 5057)	Systolic BP ≥130mmHg (n 6221)	GHQ ≥ 4 score (n 8045)
Inactive		OR(95% CI)	OR(95% CI)	OR(95% CI)	OR(95% CI)	OR(95% CI)	OR(95% CI)
BMI<25	357	1	1	1	1	1	1
25-30	369	3.1 (2.0-4.9)*	1.6(1.2-2.2)†	1.8(1.3-2.4)*	1.0(0.7-1.4)	1.5(1.1-2.0)†	0.8(0.6-1.1)
≥30	315	6.4 (4.1-10.1)*	2.3(1.6-3.2)*	4.9(3.5-6.7)*	1.2(0.8-1.6)	2.8(2.0-3.8)*	1.0(0.7-1.3)
Less active							
BMI <25	954	1.4(0.9-2.2)	1.1(0.8-1.5)	0.6(0.5-0.8)†	0.5(0.4-0.6)*	1.0(0.7-1.3)	0.5(0.4-0.7)*
25-30	906	2.5(1.6-3.8)*	2.2(1.7-3.0)*	1.2(0.9-1.6)	0.8(0.6-1.1)	1.5(1.2-2.0)†	0.4(0.3-0.5)*
≥30	459	6.0(3.9-9.2)*	2.5(1.8-3.4)*	3.1(2.3-4.1)*	1.3(1.0-1.8)	2.3(1.7-3.1)*	0.5(0.4-0.6)*
Active							
BMI <25	785	1.0(0.6-1.7)	1.1(0.8-1.5)	0.6(0.5-0.8)†	0.4(0.3-0.6)*	1.0(0.7-1.3)	0.4(0.3-0.5)*
25-30	656	1.7(1.1-2.7)†	2.1(1.5-2.9)*	0.9(0.9-1.2)	0.6(0.4-0.8)*	1.9(1.4-2.4)*	0.3(0.2-0.4)*
≥ 30	256	4.4(2.7-7.1)*	2.4(1.7-3.6)*	2.7(1.9-3.8)*	1.3(0.9-1.8)	2.5(1.8-3.5)*	0.4(0.3-0.6)*

Results adjusted for age, gender, social class, cigarette smoking, alcohol consumption and fruit and vegetables. HDL-c: HDL–cholesterol, CRP = C-reactive protein, BP = Blood pressure, GHQ = General health questionnaire, Active= people who performed at least 3 days 20 minutes vigorous activity or 5 days 30 minutes moderate activity or both of them, Low active = people who were still active but less than current recommendations. Significantly different from reference category: * $p<0.001$, †$p<0.05$.

Table 5. 10 : Odds ratio for Metabolic Syndrome according to physical activity and BMI

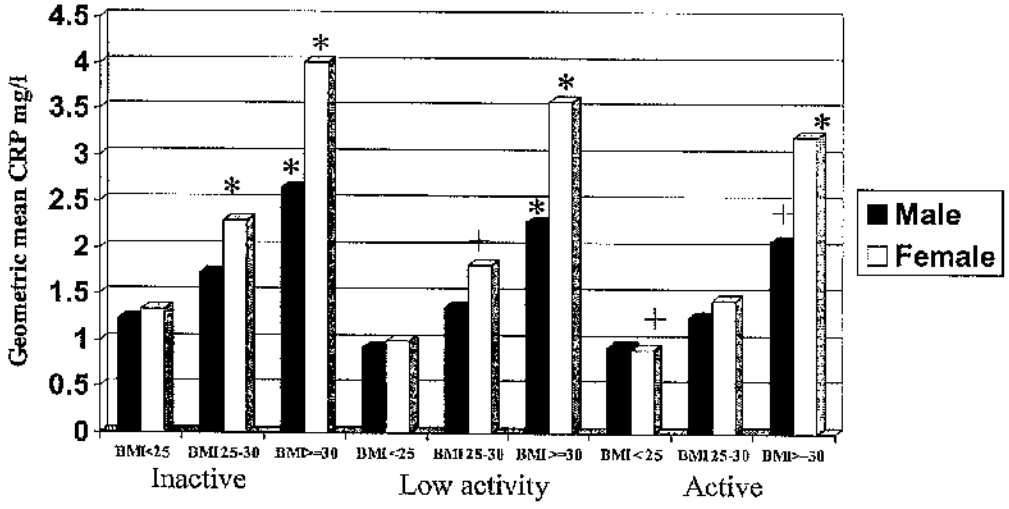
BMI kg/m² /activity	Men			Women		
categories	n	MS (%)	OR (95% CI)	n	MS (%)	OR (95% CI)
Inactive						
BMI<25	154	3 (1.9)	1	202	11 (5.4)	1
25-30	206	48 (23.3)	14.7 (4.4-48.6)*	174	51 (29.3)	6.3 (3.4-12.9)*
≥30	128	90 (70.3)	132.0 (39.1-445.4)*	188	126 (67.0)	35.7 (17.4-73.1)*
Less active						
BMI <25	322	20 (6.2)	4.7 (1.3-15.8)+	604	26 (4.3)	1.3 (0.6-2.7)
25-30	413	69 (16.7)	12.5 (3.8-40.9)*	488	82 (16.8)	5.3 (2.7-10.4)*
≥30	200	109 (54.5)	79.9(24.2-263.9)*	275	144 (52.4)	37.2 (18.5-74.7)*
Active						
BMI <25	345	13 (3.8)	3.3 (0.9-12.1)	400	8 (2.0)	0.8 (0.3-2.0)
25-30	386	47 (12.2)	11.2 (3.4-37.2)*	230	31 (13.5)	4.9 (2.3-10.5)*
≥ 30	124	52 (41.9)	56.4 (16.7-190.6)*	119	50 (42.0)	33.1 (15.4-71.2)*

MS; Metabolic Syndrome: defined using available data in SHS database any three of WC>102 cm (M) and > 88 cm (F), BP ≥ 130/85 mmHg, HDL-C ≤ 1mmol/l (M) and <1.3 mmol/l (F), Non-HDL-C >4mmol/l and diagnosed diabetes. Results adjusted for age, social class, cigarette smoking, alcohol consumption and fruit and vegetable intake. Significantly different from the reference category: + p<0.05, * p<0.001



reference group inactive BMI < 25, + P < 0.05,* P < 0.001

Figure 5. 6: Adjusted geometric mean HDL-c by physical activity and BMI categories & sex



reference group inactive BMI < 25, + P < 0.05,* P < 0.001

Figure 5. 7: Adjusted geometric mean C-reactive protein by physical activity and BMI categories & sex

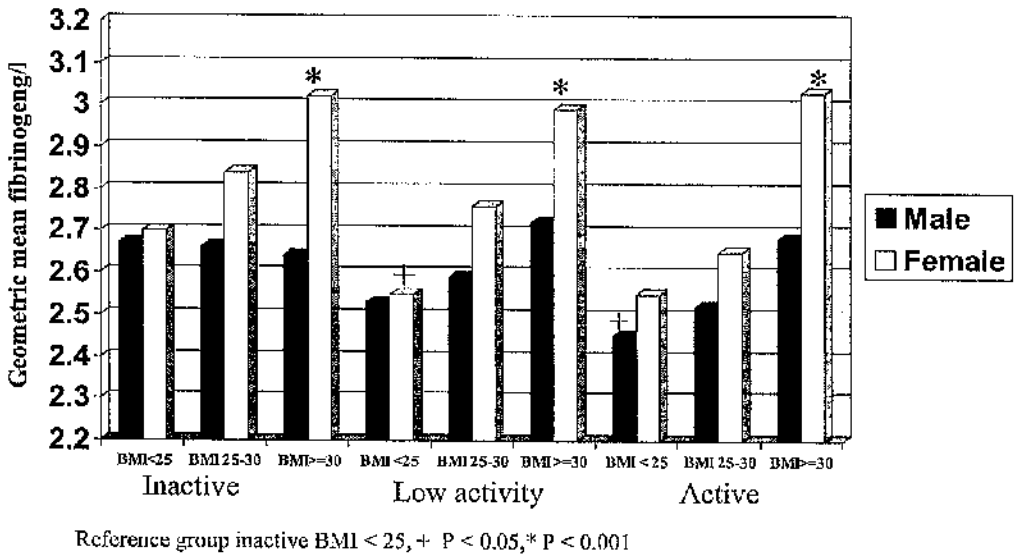


Figure 5. 8: Adjusted geometric mean fibrinogen by physical activity and BMI categories & sex

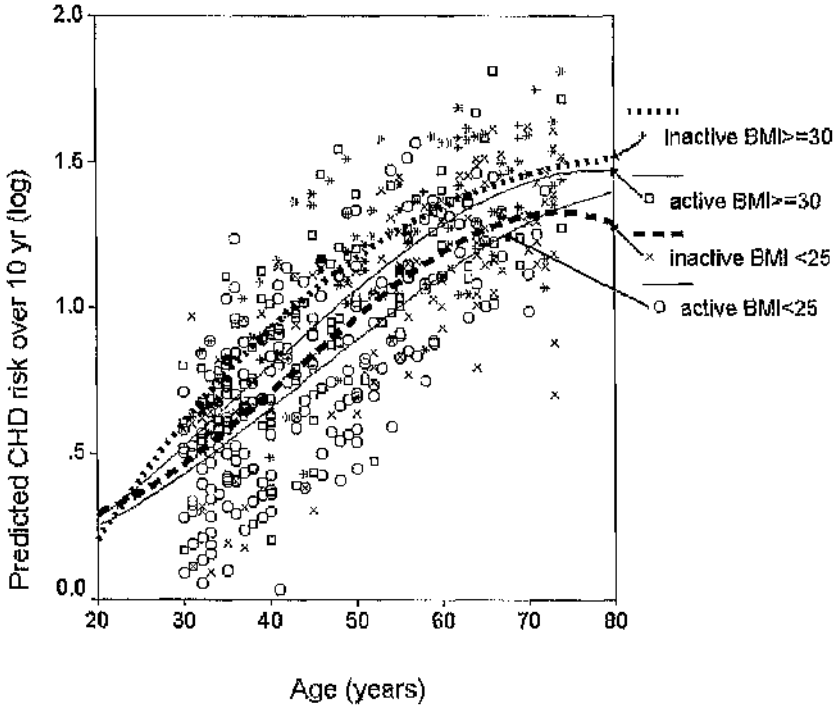


Figure 5. 9: Predicted 10 year Coronary Heart Disease risk distribution by age according to physical activity and BMI categories in men (the fit lines are based on cubic function)

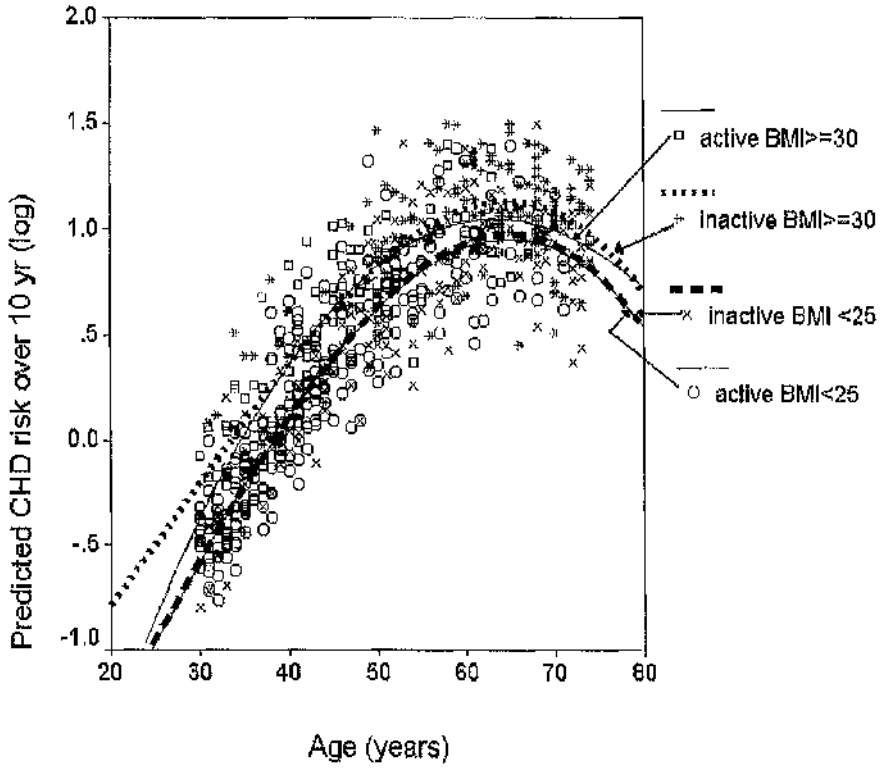


Figure 5. 10: Predicted 10 year Coronary Heart Disease risk distribution by age according to physical activity and BMI categories in women (the fit lines are based on cubic function)

Chapter 6: Relationships between lifestyle factors and dietary habits.

6.1. Introduction

The burden of chronic diseases including obesity, diabetes mellitus, cardiovascular diseases, hypertension, stroke and some types of cancer, is rapidly increasing worldwide and by 2020 it has been estimated that these diseases will account for almost three quarters of all deaths in the world (WHO, 2003). Dietary habits are major modifiable factor for good health throughout life, and have a strong effect on the incidence and management of chronic diseases.

Many high risk behaviors such as smoking and physical activity and dietary habits modify health status and all these take place in a social, cultural, political and economic environment. Food consumption patterns are shaped by many of these factors and the complex interaction between them. The association of tobacco with chronic diseases may be in part due to its association with an unhealthy diet (Preston, 1991).

Cigarette smoking is a major contributor to morbidity and mortality from many diseases including several cancers, heart disease, stroke, chronic respiratory disease and a range of other problems (WHO, 2002). Many studies have shown that smokers have unhealthy dietary patterns. A meta-analysis of fifty-one published nutritional surveys from 15 different countries has shown that smokers have unhealthy patterns of nutrient intake compared with non-smokers. On average smokers reported significantly higher intakes of energy, total fat, saturated fat, cholesterol and alcohol, and lower intake of polyunsaturated fat, fibre, vitamin C, vitamin E, beta-carotene, calcium and iron than non-smokers (Dallongeville *et al.*, 1998). Dyer et al (Dyer *et al.*, 2003) in the INTERMAP study confirmed the finding that smokers had less healthy diets than non-smokers. In addition this study also revealed that current smokers, when compared with never smokers, consumed more energy from alcohol and saturated fats,

and less energy from vegetables protein and carbohydrates, higher dietary and urinary Na/K, but not Na intake. The dietary intake of ex-smokers was almost similar to non-smokers. The dietary and nutritional survey of British adults (Margetts & Jackson, 1993) showed that smokers ate more white bread, sugar, cooked meat dishes, butter, and whole milk and less whole meal bread, high fibre breakfast cereals, fruit, and carrots than non-smokers. Palaniappan reported that smokers consumed a significantly lower quantity of fruit and vegetables and higher carbonated beverages, coffee and tea than non-smokers (Palaniappan *et al.*, 2001). In this study the average number of portions of fruit and vegetables in smokers was below the minimum recommended portion size, 5 portions per day (4.0 in men and 3.7 in women) and only 30% of smokers compared 48% of non-smokers met this target. Tobacco smoke contains many oxidants and generates free radicals that can cause oxidative damage in the body, primarily in the lungs. In smokers the oxidative tissue damage can be as a result of both the direct effect of oxidants in cigarette smoke and the consequences of lower antioxidant status as a result of poor dietary habits associated with smoking (Kim *et al.*, 2003). The reasons that make smokers eat less healthy diets are not clear. Besides the factors that shape dietary habits, it might be possible that the effect of smoking on taste independently influences food choices.

Smoking is more prevalent in the lower socioeconomic groups and these socioeconomic differences in smoking habits are more prominent among younger than among older age groups. These are the same groups, which are likely to have the poorest dietary habits, have the highest levels of both under nutrition and obesity. This inequality may increase the morbidity and mortality related to smoking in the future (Cavelaars *et al.*, 2000).

In Scotland, smoking, an unhealthy diet, inactivity and excess alcohol consumption have also been identified as major risk factors for poor health. Therefore, a study of the inter-relationship among these risk factors is important. The aim of this study was to examine the relationships between some lifestyle factors such as physical activity, alcohol consumption, smoking, BMI and social class and dietary habits in a representative sample of the Scottish population. A population, which has one of the highest CVD prevalence in Europe, and almost the poorest dietary habits.

6.2. Subjects and methods

Information about the SHS survey and most of the variables that have been used in this study have been explained in chapter 2.

6.2.1. Dietary habits

In the SHS, information on dietary habits was obtained by a short dietary questionnaire the Dietary Target Monitor(Shaw *et al.*, 2000; Lean *et al.*, 2003). This includes questions relating to type and frequency of major food items, but is not quantitative. This questionnaire was designed specifically to provide information about eating behaviors in terms of evaluation of the Scottish dietary targets (The Scottish Office, 1996).

The eating habits monitor included questions relating to type and frequency of consumption of major food items. It includes questions relating to frequency of consumption of fruits and vegetables (including fresh, cooked, frozen), starchy foods consumption (including bread, breakfast cereals, potatoes, pasta and rice), fish intake, chips, meat and meat products, cheese,

milk, sweets or chocolate, ice cream, crisps, savoury snacks, soft and fizzy drinks, cakes, scones, sweet pies or pastries and biscuits.

The frequency with which foods were consumed was divided into the following categories: six or more times a day, four or five times a day, two or three times a day, once a day, five or six times a week, two to four times a week, one to three times a month, less often or never. This questionnaire has been validated against the very widely used Scottish food frequency questionnaire (FFQ) (Bolton-Smith & Milne, 1991) in three key food groups: fruit and vegetables, starchy foods and fish (Lean *et al.*, 2003). Different equations were produced to estimate the intakes of these three food groups. In the validation study it was assumed that 'times' could be equated to 'portion' (Lean *et al.*, 2003). For the estimation of the total fruit and vegetable intake, total frequency of fresh fruit, cooked green vegetables (fresh or frozen), cooked root vegetables (fresh or frozen) and raw vegetables or salad (including tomatoes) was multiplied 1.33 and 80 to generate a g/day figure. Then total fruit and vegetable consumption was divided into three groups, low consumers (< 200 g/day), moderate consumers (200 - < 400 g/ day) and achievers of current targets (≥ 400 g/day) (The Scottish Office, 1996).

Consumption of starchy foods (portion per day) was estimated by total starchy food from FFQ (sum of frequency of bread, breakfast cereals, potatoes, pasta and rice) multiplied by 8 for men and 6.4 for women before division into tertiles.

Estimation of fish intake (g/week) was made by the multiplication of the sum of frequency of fish intake in FFQ by 0.99 and 120. Total fish intake (g/week) was categorized into three groups: low consumers (0-239 g/week), moderate consumers (240-359 g/week) and high consumers (≥ 360 g/week).

The sum of the frequency of the variables ' cakes, scones sweet pies, pastries or pudding', 'soft drinks, not including diet or low-calorie types', ' chocolates, crisps or biscuits, including savoury biscuits such as cream crackers' and 'sweets or ice-cream' defined as 'snacks'. These data were then collapsed into tertiles.

To evaluate the consumption of fat rich foods, the consumption of the variables ' number of pats of spread' such as butter or margarine, 'fried food, including fish, chips, cooked breakfast, samosas', ' meat, such as beef, lamb, pork and meat product not poultry' and ' cheese, not including cottage cheese or other reduced fat cheeses' have been added together and defined as 'fat foods'. This variable was divided into tertiles for comparison of the different levels of independent variables.

The amount of milk consumed per day was converted to portions per day as follows: 'less than a quarter of a pint' equal to a quarter of a portion, 'about a quarter of a pint' equal to half of a portion, 'about half a pint' equal to a portion, 'three quarter of a pint' equal to one and half portion, and ' one pint or more' equal or more to two portions.

6.2.2. Data Analysis

The frequency of all the dietary habits variables were not normally distributed; so as a result, data have been shown as the median and inter-quartile ranges. Multinomial logistic regressions were used to evaluate the relationships between consumption of fruit and vegetables, starchy foods, milk, fat food, snack food and adding salt to food with lifestyle

variables stratified by gender. The reference category for the dependent variable for total fruit and vegetable, starchy food and milk intake was high intake, and for the fat foods, snack foods and adding salt was low or rarely. Binary logistic regressions were used for fish intake and adding sugar to tea or coffee in which high fish intake and no sugar were the dependent reference category. In the logistic regression analyses, all variables were entered simultaneously to evaluate the independent association of the individual variables studied.

6.3. Results

The median and interquartiles of food groups (total fruit and vegetable consumption, total fish intake, starchy foods, fat food and snacks) are shown in men and women in tables 1 and 2. The associations between age groups and lifestyle factors with the food groups are shown in tables 6.3-6.18.

6.3.1. Fruits and vegetables

The median and the percentage of subjects who achieved the recommended levels of consumption of total fruits and vegetables per day in younger age groups, current smokers, people with BMI < 25 kg/m^2, inactive subjects, people who did not drink or drank high amount of alcohol and people from the lower social class in both men and women were low. After adjustment for all the variables, men aged over 45 years and women aged over 35 years were associated with higher fruit and vegetable consumption than the age group 16-24 years (reference group) ($p<0.001$). Current smokers, both less active and inactive groups, and low social class subjects were significantly associated with low fruit and vegetable consumption in both men and women compared with their reference groups ($p<0.01$) (table 6.3 & 6.4).

Current smokers, inactive people and lower social class participants also were more likely to be moderate fruit and vegetable consumers compared with their counterparts. Overweight and obesity did not show any significant associations with total fruit and vegetable consumption. Female moderate alcohol consumers were less likely to be low consumers of fruit and vegetables compared with the reference group ($p<0.005$).

6.3.2. Total fish consumption

Subjects who had lower median consumption of total fish were younger, smokers and from the lowest social classes for both sexes. After adjustment for the all the variables, older subjects for both genders consumed more fish than the reference group aged 16-24 years ($p< 0.01$). Physically inactive men ($p<0.01$), both less active and inactive women ($p<0.005$) and female current smokers ($p<0.05$) were more likely to have low and moderate consumption of fish compared with their counterparts. Women of lower social class had significantly low and moderate fish intake ($p<0.005$), whereas in men only social class IIIM showed a significant association with low and moderate fish intake compared with the highest social class as the reference group ($p<0.05$).

6.3.3. Starchy foods

Among men, people in the lower social class consumed significantly more starchy food, whereas increasing age; being physically less active or inactive and obesity were associated with consumption of less starchy food than their counterparts ($p<0.05$). Among women, the lowest social class group was associated with more starchy food intake ($p< 0.05$). A higher number of female current smokers were more in lower starchy food consumer group ($p<0.05$) compared with non-smokers.

6.3.4. Fat foods

The highest median of fat food intake in men was seen among the youngest age group, current smokers, those with a BMI less than 25 kg/m², physically active subjects, those in the highest alcohol intake category and the lowest social class. All of these variables showed significant associations with fat food in intake in men. Whereas in women the lowest social class group and the current smokers consumed significantly more fat food compared with their reference categories.

6.3.5. Snacks

Age, alcohol intake and BMI were negatively associated with snack food consumption in both men and women in which older adults, heavier drinkers and overweight and obese subjects (among men) had a lower snack food consumption compared to their counterparts. Low social class and inactivity were both associated with higher snack foods, particularly amongst women.

6.3.6. Milk

The median consumption of milk for all different categories and in both sexes was one portion per day. Age was positively associated with low consumption of milk. Older adults (aged >45 years) consumed significantly less milk than the youngest age group (age 16-24 years). Current smokers in both sexes consumed more milk than non-smokers ($p<0.001$). Overweight and obese subjects reported consuming less milk than BMI < 25 kg/m², but this was significant only in men ($p<0.05$). Inactive subjects were lower milk consumers than active subjects. People who drank less alcohol were more likely to be higher milk consumers,

especially women. Those in the lowest social class consumed more milk than higher social class in men.

6.3.7. Adds sugar to tea or coffee

Younger subjects, current smokers, people with BMI <25 kg/m² and people in low social class were more likely to add sugar to their tea or coffee than their counterparts in both sexes. In women, physical activity and alcohol consumption showed a significant association with adding sugar to tea or coffee in which inactive subjects and those who consumed between zero and one unit of alcohol per week reported more adding sugar to their hot drinks.

6.3.8. Adds salt to food

Informants were asked whether they added salt to their food at the table. Women aged 25-64 years were more likely to add salt to food than those aged 16-24 years. Current smokers for both men and women were more likely to add salt to food compared with non-smokers. Both high alcohol intake and low social class groups were positively associated with adding salt to food. People who were heavy drinkers and those in the lower social classes were more likely to add salt to food at the table.

6.4. Discussion

Nutrition is a major determinant of general health and many diseases particularly chronic diseases and evidences show that modification of diet has strong effects on health all over life (WHO, 2003). Therefore it has been recognized that a healthy diet has the potential to make significant improvements to our health and well being.

Due to the complexity of dietary habits, in nutritional epidemiology and in the relations between dietary factors with disease, the intercorrelations among dietary habits and the correlations of those habits with other behaviors should be considered (Freudenheim, 1999). Many factors including smoking, physical activity, social-economic status, alcohol consumption, overweight and obesity are associated with health and diseases and may also have associations with dietary habits. Although the associations between many individual behavioral risk factors such as smoking, alcohol consumption, physical activity and BMI, and dietary habits have been studied, we need to evaluate whether a composite of healthy lifestyle factors is associated with healthy dietary habits. Therefore the aim of this study was to evaluate the independent relationship between those factors with dietary habits. Others have used cluster analysis or principal components analysis to address questions in this field. These approaches are often used to define patterns of diet and lifestyle, which tend to occur together. Such approaches are interesting in a descriptive, and qualitative way, but do not lend themselves towards informing advice or interventions. For public health promotion, targeted messages are favoured, hence the decision to use some of the pre-defined dietary targets for health promotion.

The SHS FFQ asked about the frequency of consumption of a limited of major food groups, which relates directly to quantitative dietary targets. It did not provide quantitative information about other dietary components. In this study we used six food or food groups as indicators of healthy and unhealthy dietary habits. Three of them (fruit and vegetable, starchy foods and fish) have been validated (Lean *et al.*, 2003). The consumption of salt and sugar as a sign of unhealthy dietary habits also has been included. People who add salt or sugar are

likely to have higher overall consumption of them and also other unhealthy foods, but no evidence exists to support this common-sense view.

The results of the present study have shown that smoking, being in a low social class and of a younger age are the major determinants of unhealthy diet. Individuals in these groups consumed the lowest amount of fruit and vegetables, highest amount of fat rich foods and added more sugar and salt to their food and drinks. Alcohol consumption has shown an inconsistent association with dietary habits in this study. Moderate alcohol consumptions were associated with higher fruit and vegetable consumption, whereas heavy alcohol intakes were associated with lower consumption of snack foods, but had positive associations with salt intake.

Billson et al analyzed the dietary and nutritional survey of British adults aged 16-64 years in 1986-1987 and reported that manual social class, current smoking and younger age were negativity associated with fruit and vegetable consumption, which is in line with the present results (Billson *et al.*, 1999). In a cross-sectional study based on three Dutch National Food Consumption Surveys (Hulshof *et al.*, 2003) reported that subjects from higher social economic class consumed a more healthy diet than the lower social economic classes. Ricciuto et al (Ricciuto *et al.*, 2006) reported that household socio-demographic characteristics are major determinants of food purchasing and that a lower income is a major restriction on the purchase of healthy diet, as measured by the following components of a healthy diet, vegetables, fruit and milk. However, in a questionnaire survey mailed to homes owned by a large UK housing association, Dibsdall et al (Dibsdall *et al.*, 2003) showed that access or affordability to fruit and vegetables was not the major barrier of eating enough fruit and vegetables, but that other factors such as motivation, psychosocial or lifestyle factors were

stronger barriers. Our results confirm that apart from social class other factors such as smoking, physical activity, alcohol consumption and age were all influential in determining dietary habits.

In this study, contrary to expectation, overweight and obese subjects reported consuming less energy dense foods and sugar compared with those of a BMI < 25 kg/m^2 in men and in women there was no significant relationships between energy dense foods and BMI, but they reported less sugar. A systematic literature review of 30 published studies examined the association between food intake patterns (assessed by diet index, factor analysis or cluster analysis) and BMI or obesity showed that no consistent associations were observed (Togo *et al.*, 2001). In this review, ten studies found a positive association between the intake pattern, categorized as ether fatty, sweet or energy dense foods, and BMI or obesity. The results of four studies were in line with the present results, showing there was a negative association between food intake patterns and BMI. A further 11 studies found no significant associations. The present results were also in agreement with the findings in Sanchez-Villegas et al (Sanchez-Villegas *et al.*, 2003) in a study that found history of obesity inversely associated with a "western" diet factors which contained fat-food, French fries, high-fat dairy products, processed meals and red meats. However the authors believed that the diagnosis of several diseases in this group might encourage them to choose a healthier diet. Togo et al (Togo *et al.*, 2004)in a longitudinal observation study in adult Danes showed no consistent association between food intake patterns and changes in BMI or obesity. A cross-sectional analysis of this data showed that there was a negative association between the "sweet" factor, which included more unhealthy diet and BMI.

One of the main reasons for the inverse associations between unhealthy diet, particularly high-energy dense foods, with BMI or obesity might be misreporting, especially under-reporting. Johansson et al (Johansson *et al.*, 2001) in a cross-sectional study using repeated 24-hour recalls dietary assessment methods found that BMI was one of the most consistent factors to be linked with under-reporting. It has also been shown that obese men selectively under-report fat intake (Goris *et al.*, 2000). In the current study it was impossible to address under-reporting, as total energy intake and energy expenditure were not available to estimate it. Weight loss diet and selecting healthier diet among overweight/obese subjects because of the diseases related with extra body fat might be the cause of these inverse associations. In simultaneous measurements of BMI and dietary habits in a cross-sectional study, obese subjects may intentionally be on a weight loss diet, whereas they might have followed or consumed an unhealthy diet during fat accumulation (Togo *et al.*, 2001).

The present findings regarding physical activity and dietary habits confirmed the other findings in this field (Slattery *et al.*, 1998; Williams *et al.*, 2000; Kromhout, 2001; Sanchez-Villegas *et al.*, 2003) that overall, active subjects had a healthier diet than inactive subjects in both sexes. Sanchez-Villegas et al (Sanchez-Villegas *et al.*, 2003) showed that physical activity during leisure time, assessed by self-reported questionnaires was positively associated with healthier dietary patterns and negatively associated with unhealthy dietary patterns. However, in spite of overall healthier food intake in active people in the current study, active men reported consuming higher high-energy dense foods compared with inactive subjects, which is inconsistent with the healthy foods pattern. Although we can not clarify the reasons for this inconsistency, it might possible those men might think that higher activity needs more

energy or vice versa, people who consumed higher energy dense believe that they need more physical activity to burn the extra energy.

A cross-sectional design and the use of a short food frequency questionnaire are the major limitations of the current study. Some major food categories are not included in the SHS FFQ and are therefore absent from this study. However, it is recognized that there is no perfect way to assess food and nutrient intake in large population study. Misclassifications of food groups in this study were possible, which again might cause misinterpretation of the results. The comparison of the findings of this study with the other studies, in which statistical approaches such as factor analysis have been used to define dietary patterns, may not be fully relevant. This is because in this study some of the pre-defined dietary targets were used plus some other indicators of healthy and unhealthy diet. However, the methods used in SHS seem is capable to detect known effects of lifestyle on dietary habits. Since the sample sizes of the other dietary surveys in Scotland such as the National Diet and Nutritional Survey in UK are insufficient to assess the effects of lifestyle on dietary habits components. The large representative sample size in SHS is an important strength of the present analysis.

6.5. Conclusion

The study of dietary patterns and their determinants in population is quite important. In this study we used some of the pre-defined dietary targets for health promotion. This study has shown that those in the youngest age group, current smokers, inactive people and those from lower social classes were more likely to have unhealthy dietary habits and in contrast, older adults, non-smokers, active subjects and people from higher social class were more likely to reach the recommended dietary targets. However, the findings of this study also revealed that

there were some inconsistency between healthy lifestyle and healthy dietary patterns. In order to effectively influence and to encourage healthy eating, public health policy should focus more on changing all major unhealthy behaviors together in these and younger age groups.

Table 6. 1: Median and interquartiles ranges for food groups in men

	n	Fruit and & vegetables (g/day)	Starchy foods (portion /day)	Fish (g/week)	Fat foods (Portion/day)	Snacks (Portion/day)	Milk (Portion/day)
Age groups							
16-24	399	152(84,293)	6.4(4.5,8.0)	119(59.356)	4.5(3.2,6.0)	3.4(2.0,5.5)	1.5(1.0,2.0)
25-34	763	175(106,258)	6.3(4.2,7.5)	119(59,239)	4.0(2.9,5.6)	2.1(1.1,3.8)	1.0(1.0,2.0)
35-44	826	205(114,312)	6.1(4.0,7.4)	178(119,356)	3.9(2.9,5.6)	1.7(1.1,3.0)	1.0(1.0,2.0)
45-54	694	243(144,365)	5.6(4.0,7.3)	178(119,356)	4.0(2.9,5.6)	1.5(0.9,2.6)	1.0(1.0,2.0)
55-64	683	243(145,365)	5.6(4.0,7.3)	178(119,416)	3.9(2.7,5.3)	1.5(0.6,2.6)	1.0(1.0,2.0)
65-74	572	239(144,365)	5.1(4.0,6.9)	238(119,416)	3.9(2.9,4.9)	1.6(0.9,2.6)	1.0(1.0,2.0)
Smoking							
Non-smokers	1600	243(144,372)	6.1(4.2,7.4)	178(119,356)	3.7(2.6,5.2)	1.9(1.1,3.2)	1.0(0.5,2.0)
Ex-smokers	955	243(152,364)	5.1(4.0,7.3)	178(119,416)	3.6(2.6,5.3)	1.6(1.0,2.9)	1.0(0.5,2.0)
Current smokers	1373	152(91,243)	6.0(4.0,7.4)	119(59,358)	4.4(3.1,6.1)	1.9(1.0,3.4)	1.0(0.5,1.5)
BMI (kg/m^2)							
<25	1296	190(106,304)	6.2(4.1,7.4)	178(59,356)	4.3(3.0,5.9)	2.1(1.1,3.7)	1.0(0.5,2.0)
25-30	1575	213(137,342)	5.8(4.2,7.4)	178(11,356)	3.9(2.9,5.3)	1.7(1.0,2.9)	1.0(0.5,2.0)
≥30	733	220(137,365)	5.6(4.0,7.2)	178(119,356)	3.6(2.8,5.3)	1.6(0.9,2.8)	1.0(0.5,1.75)
Physical activity							
Active	1448	213(129,365)	6.3(4.5,7.8)	178(119,356)	4.3(3.0,5.9)	2.0(1.1,3.5)	1.0(0.5,2.0)
Less active	1480	213(122,334)	5.6(4.0,7.3)	178(119,356)	3.9(2.8,5.4)	1.7(1.0,3.1)	1.0(0.5,2.0)
Inactive	990	198(106,304)	5.1(4.0,6.8)	178(119,356)	3.8(2.7,5.2)	1.6(0.8,2.9)	1.0(0.5,2.0)
Alcohol (units per week)							
0–1	559	198(114,350)	5.3(4.0,7.4)	178(119,356)	3.7(2.7,5.2)	2.0(1.1,3.4)	1.0(0.5,2.0)
1-10	1160	236(144,365)	6.1(4.5,7.4)	178(119,356)	3.9(2.7,5.4)	2.0(1.1,3.4)	1.0(0.5,2.0)
10-21	939	213(137,334)	6.0(4.2,7.4)	178(119,356)	3.9(2.9,5.6)	1.7(1.2,2.9)	1.0(0.5,2.0)
>21	1238	182(106,304)	5.7(4.0,7.4)	178(119,356)	4.3(3.0,5.6)	1.6(0.9,3.1)	1.0(0.5,2.0)
Social class							
I, II	1281	243(167,372)	5.1(4.0,7.3)	178(119,416)	3.6(2.6,5.0)	1.7(1.0,3.0)	1.0(0.5,1.5)
IIINM	420	205(122,334)	5.6(4.0,7.2)	178(119,356)	3.8(2.7,5.3)	1.9(1.0,3.1)	1.0(0.5,2.0)
IIIM	1290	190(114,312)	6.1(4.0,7.4)	178(119,356)	4.1(2.9,5.6)	2.0(1.1,3.4)	1.0(0.5,2.0)
IV, V	798	167(91,251)	6.3(4.2,7.4)	119(59,356)	4.6(3.3,6.1)	2.0(1.0,3.2)	1.0(0.5,2.0)

Data are median (quartile 1, quartile 3)

Table 6. 2: Median and interquartiles ranges for food groups in women.

	n	Fruit and & vegetables (g/day)	Starchy foods (portion /day)	Fish (g/week)	Fat food (Portion / day)	Snacks (Portion / day)	Milk (Portion/day)
Age groups							
16-24	528	220(122,342)	3.7(3.0,5.0)	119(0.0,238)	3.0(2.1,4.1)	2.4(1.4,3.9)	1.0(0.5,2.0)
25-34	972	236(144,364)	3.6(2.7,4.6)	119(59,238)	2.9(2.0,4.1)	1.7(1.0,3.1)	1.0(0.5,1.5)
35-44	1008	251(175,403)	3.6(2.7,4.5)	178(119,356)	2.9(1.9,3.9)	1.5(0.7,2.6)	1.0(0.5,1.0)
45-54	896	274(182,403)	3.6(2.7,4.9)	178(119,356)	3.0(2.0,4.3)	1.4(0.8,2.4)	1.0(0.5,1.0)
55-64	808	304(190,441)	3.6(3.0,4.8)	238(119,416)	2.9(2.0,4.1)	1.3(0.6,2.2)	1.0(0.5,1.0)
65-74	890	281(182,426)	3.9(3.2,5.0)	238(119,416)	3.0(2.0,4.3)	1.5(0.9,2.7)	1.0(0.5,1.0)
Smoking							
Non-smokers	2380	304(198,433)	3.6(3.1,4.8)	178(119,416)	2.9(2.0,3.9)	1.6(1.0,2.7)	1.0(0.5,1.0)
Ex-smokers	940	319(205,441)	3.6(3.0,4.9)	178(119,416)	2.9(2.0,4.0)	1.4(0.8,2.6)	1.0(0.5,1.0)
Current smokers	1763	205(114,312)	3.6(2.7,4.9)	119(59,356)	3.1(2.2,4.4)	1.5(0.8,3.0)	1.0(0.5,2.0)
BMI (kg/m^2)							
<25	2003	251(152,403)	3.6(2.7,5.0)	178(119,356)	3.0(2.0,4.2)	1.6(1.0,2.9)	1.0(0.5,1.5)
25–30	1479	274(182,403)	3.6(3.0,4.8)	178(119,356)	2.9(2.0,4.0)	1.4(0.8,2.6)	1.0(0.5,1.5)
≥30	1052	266(152,410)	3.6(3.0,4.6)	178(119,356)	2.9(2.0,4.0)	1.5(0.8,2.7)	1.0(0.5,1.0)
Physical activity							
Active	1382	296(190,426)	3.6(2.8,5.0)	178(119,416)	2.9(2.0,4.1)	1.5(0.9,2.6)	1.0(0.5,1.5)
Less active	2506	258(160,403)	3.6(3.0,4.8)	178(119,356)	3.0(2.0,4.0)	1.6(0.9,2.7)	1.0(0.5,1.5)
Inactive	1192	243(137,365)	3.6(2.8,4.8)	178(119,356)	2.9(2.0,4.1)	1.6(0.6,3.0)	1.0(0.5,1.0)
Alcohol (units per week)							
0–1	1530	243(152,403)	3.6(2.8,4.0)	178(119,356)	2.9(2.0,4.1)	1.6(1.0,3.0)	1.0(0.5,1.5)
1 7	2003	274(182,410)	3.6(3.0,4.9)	178(119,356)	2.9(2.0,4.0)	1.6(0.9,2.8)	1.0(0.5,1.0)
7–14	869	274(182,403)	3.6(3.0,4.6)	178(119,416)	2.9(2.0,4.2)	1.4(0.8,2.4)	1.0(0.5,1.0)
>14	658	251(152,403)	3.4(2.7,5.0)	178(59,356)	3.0(2.0,4.3)	1.4(0.6,2.6)	1.0(0.5,1.0)
Social class							
I, II	1581	334(213,448)	3.6(2.7,4.5)	178(119,416)	2.9(1.9,3.9)	1.4(0.9,2.4)	1.0(0.5,1.5)
IIINM	1036	274(175,403)	3.6(3.0,4.8)	178(119,416)	2.9(2.0,3.9)	1.6(0.9,2.9)	1.0(0.5,1.0)
IIIM	1094	243(152,380)	3.6(2.7,4.6)	178(59,356)	2.9(2.0,4.0)	1.6(1.0,2.9)	1.0(0.5,1.5)
IV, V	1185	205(122,334)	3.6(3.0,5.0)	119(59,356)	3.2(2.3,4.5)	1.6(0.9,2.9)	1.0(0.5,1.5)

Data are median (quartile 1, quartile 3)

Table 6. 3: Relationship between **fruit and vegetable** intake by some lifestyle factors in men.

	Fruit & vegetable intake			Low relative to high		Moderate relative to high	
	Low	Moderate	High	OR (95% CI)	p Value	OR (95% CI)	p Value
Age groups(years)	%	%	%				
16-24	62	25	13	1	Ref.	1	Ref.
25-34	59	30	11	1.2(0.8-1.9)	0.4	1.6(.0-2.5)	0.07
35-44	49	35	16	0.7(0.5-1.1)	0.2	1.3(0.8-2.1)	0.3
45-54	40	37	23	0.4(0.2-0.6)	0.001	0.9(0.6-1.5)	0.9
55-64	39	40	21	0.3(0.2-0.4)	0.001	1.0(0.6-1.5)	0.9
65-74	41	40	19	0.4(0.2-0.6)	0.001	1.0(0.6-1.6)	1
Smoking							
Non-smokers	40	37	23	1	Ref.	1	Ref.
Ex-smokers	37	43	20	1.4(1.1-1.8)	0.02	1.5(1.1-1.9)	0.005
Current smokers	64	28	8	3.4(2.7-4.5)	0.001	1.8(1.4-2.3)	0.001
BMI (kg/m^2)							
<25	52	33	15	1	Ref.	1	Ref.
25-30	46	36	18	1.0(0.8-1.3)	1	0.9(0.7-1.2)	0.5
≥30	43	37	20	0.8(0.6-1.1)	0.2	0.8(0.6-1.0)	0.07
Physical activity							
Active	46	34	20	1	Ref.	1	Ref.
Less active	47	36	17	1.8(1.4-2.2)	0.001	1.5(1.2-1.8)	0.005
Inactive	52	35	13	2.4(1.8-3.2)	0.001	1.7(1.3-2.3)	0.001
Alcohol (units per week)							
0–1	50	32	18	1	Ref.	1	Ref.
1-10	42	38	20	0.8(0.6-1.1)	0.3	1.2(0.9-1.7)	0.3
10-21	44	38	18	0.9(0.6-1.2)	0.5	1.3(0.9-1.8)	0.2
>21	54	32	14	1.2(0.91.7)	0.3	1.4(1.0-1.9)	0.09
Social class							
I, II	34	42	24	1	Ref.	1	Ref.
IIINM	50	33	18	2.0(1.5-2.8)	0.001	1.1(0.8-1.5)	0.7
IIIM	53	32	15	2.4(1.9-3.1)	0.001	1.2(1.0-1.5)	0.1
IV, V	62	29	9	4.4(3.2-6.1)	0.001	1.9(1.4-2.6)	0.001

Ref. = reference category

Table 6. 4: Relationship between **fruit and vegetable** intake by some lifestyle factors in women.

	Fruit & vegetable intake			Low relative to high		Moderate relative to high	
	Low	Moderate	High	OR (95% CI)	p Value	OR (95% CI)	p Value
Age groups(years)	%	%	%				
16-24	45	34	20	1	Ref.	1	Ref.
25-34	41	37	21	0.8(0.6-1.1)	0.2	1.1(0.7-1.5)	0.9
35-44	34	38	28	0.6(0.4-0.8)	0.001	0.9(0.6-1.2)	0.4
45-54	29	42	29	0.4(0.3-0.5)	0.001	0.8(0.6-1.2)	0.3
55-64	27	38	35	0.2(0.2-0.3)	0.001	0.6(0.4-.08)	0.005
65-74	28	41	31	0.2(0.2-0.3)	0.001	0.6(0.5-0.9)	0.02
Smoking							
Non-smokers	26	41	33	1	Ref.	1	Ref.
Ex-smokers	24	38	37	0.9(0.7-1.2)	0.6	0.8(0.7-1.0)	0.06
Current smokers	49	36	15	3.6(2.9-4.4)	0.001	1.8(1.5-2.2)	0.001
BMI (kg/m^2)							
<25	35	39	26	1	Ref.	1	Ref.
25-30	30	40	30	0.9(0.8-1.1)	0.5	1.0(0.08-1.2)	0.9
≥30	35	36	29	1.0(0.8-1.3)	1.0	0.9(0.7-1.0)	0.2
Physical activity							
Active	27	39	34	1	Ref.	1	Ref.
Less active	34	40	26	1.9(1.5-2.3)	0.001	1.4(1.2-1.6)	0.001
Inactive	41	36	23	2.8(2.2-3.6)	0.001	1.4(1.1-1.8)	0.005
Alcohol (units per week)							
0–1	37	38	25	1	Ref.	1	Ref.
1-7	31	39	30	0.7(0.6-0.9)	0.005	0.9(0.7-1.0)	0.2
7-14	30	40	29	0.7(0.5-0.9)	0.005	0.9(0.7-1.1)	0.3
>14	37	38	25	0.8(0.6-1.1)	0.2	0.9(0.7-1.2)	0.6
Social class							
I, II	21	41	38	1	Ref.	1	Ref.
IIINM	31	42	27	1.8(1.4-2.3)	0.001	1.4(1.1-1.7)	0.005
IIIM	36	40	24	2.1(1.7-2.7)	0.001	1.5(1.2-1.8)	0.001
IV, V	50	32	18	3.8(3.0-4.8)	0.001	1.5(1.2-1.9)	0.001

Ref. = reference category

Table 6. 5: Relationship between **starchy food** intake by some lifestyle factors in men.

	Starchy food intake			Low relative to high		Moderate relative to high	
	Low	Moderate	High	OR (95% CI)	p Value	OR (95% CI)	p Value
Age groups(years)	%	%	%				
16-24	27	29	44	1	Ref.	1	Ref.
25-34	34	29	37	1.5(1.1-2.2)	0.02	1.1(0.8-1.6)	0.5
35-44	35	31	35	1.5(1.1-2.1)	0.03	1.2(0.9-1.7)	0.3
45-54	35	35	30	1.6(1.1-2.3)	0.02	1.5(1.1-2.1)	0.03
55-64	37	33	30	1.7(1.1-2.4)	0.01	1.4(1.0-2.1)	0.06
65-74	39	36	25	1.9(1.3-2.8)	0.005	1.7(1.2-2.5)	0.01
Smoking							
Non-smokers	32	33	35	1	Ref.	1	Ref.
Ex-smokers	37	33	30	1.1(0.9-1.3)	0.6	0.9(0.7-1.2)	0.5
Current smokers	36	31	33	1.2(1.0-1.5)	0.09	1.1(0.9-1.3)	0.6
BMI (kg/m^2)							
<25	33	29	38	1	Ref.	1	Ref.
25-30	35	34	32	1.2(1.0-1.4)	0.08	1.3(1.1-1.6)	0.01
≥30	36	36	28	1.3(1.0-1.7)	0.05	1.5(1.1-1.9)	0.005
Physical activity							
Active	30	29	41	1	Ref.	1	Ref.
Less active	36	33	31	1.5(1.2-1.8)	0.001	1.4(1.1-1.6)	0.005
Inactive	40	35	25	2.0(1.6-2.6)	0.001	1.7(1.3-2.2)	0.001
Alcohol (units per week)							
0–1	36	34	30	1	Ref.	1	Ref.
1-10	31	33	36	0.8(0.6-1.1)	0.3	0.9(0.7-1.2)	0.7
10-21	35	33	33	1.1(0.8-1.4)	0.7	1.1(0.8-1.4)	0.8
>21	38	31	32	1.2(0.9-1.6)	0.3	1.1(0.8-1.4)	0.6
Social class							
I, II	37	34	29	1	Ref.	1	Ref.
IIINM	35	38	27	1.0(0.8-1.4)	0.9	1.2(0.9-1.6)	0.3
IIIM	35	31	35	0.7(0.6-0.9)	0.01	0.8(0.6-.0.9)	0.02
IV, V	31	30	39	0.6(0.5-0.8)	0.001	0.7(0.5-0.8)	0.005

Ref. = reference category

Table 6. 6: Relationship between **starchy food** intake by some lifestyle factors in women.

	Starchy food intake			Low relative to high		Moderate relative to high	
	Low	Moderate	High	OR (95% CI)	p Value	OR (95% CI)	p Value
Age groups(years)	%	%	%				
16-24	29	35	37	1	Ref.	1	Ref.
25-34	33	39	28	1.3(0.9-1.7)	0.2	1.3(0.9-1.7)	0.2
35-44	36	38	26	1.5(1.1-2.0)	0.01	1.3(1.0-1.7)	0.09
45-54	33	39	29	1.3(0.9-1.7)	0.2	1.2(0.9-1.6)	0.3
55-64	30	42	28	1.1(0.8-1.6)	0.5	1.2(0.9-1.7)	0.2
65-74	23	46	31	0.7(0.5-1.0)	0.09	1.3(0.9-1.8)	0.2
Smoking							
Non-smokers	28	44	28	1	Ref.	1	1
Ex-smokers	29	44	27	1.3(1.0-1.6)	0.06	1.1(0.9-1.3)	0.4
Current smokers	35	33	32	1.2(1.0-1.5)	0.04	0.7(0.6-0.8)	0.001
BMI (kg/m^2)							
<25	32	38	31	1	Ref.	1	Ref.
25-30	29	42	28	1.1(0.9-1.3)	0.6	1.2(1.0-1.4)	0.08
≥30	31	41	28	1.1(0.9-1.4)	0.4	1.1(0.9-1.4)	0.3
Physical activity							
Active	31	39	30	1	Ref.	1	Ref.
Less active	30	41	29	1.1(0.9-1.3)	0.4	1.1(1.0-1.4)	0.2
Inactive	31	39	30	1.2(0.9-1.5)	0.2	1.0(0.8-1.3)	1.0
Alcohol (units per week)							
0–1	31	40	29	1	Ref.	1	Ref.
1-7	29	41	30	0.9(0.7-1.0)	0.2	1.0(0.8-1.2)	0.8
7-14	31	42	27	0.9(0.7-1.1)	0.4	1.1(0.9-1.4)	0.4
>14	36	32	32	0.9(0.7-1.2)	0.6	0.8(0.6-1.1)	0.1
Social class							
I, II	31	43	26	1	Ref.	1	Ref.
IIINM	29	42	29	1.0(0.8-1.2)	0.9	0.9(0.8-1.2)	0.6
IIIM	33	39	28	1.0(0.8-1.2)	1.0	0.9(0.7-1.1)	0.2
IV, V	31	37	33	0.8(0.6-0.99)	0.05	0.7(0.6-0.9)	0.01

Ref. = reference category

Table 6. 7: Odds ratio of low **fish** intake by some lifestyle factors in men.

	Fish intake		OR (95% CI)	p value
	Low& moderate	High		
Age groups(years)	%	%		
16-24	80	20	1	Ref.
25-34	82	18	1.2(0.8-1.6)	0.5
35-44	78	22	0.9(0.6-1.3)	0.6
45-54	78	22	0.8(0.6-1.2)	0.3
55-64	74	26	0.6(0.4-0.9)	0.01
65-74	70	30	0.5(0.3-0.7)	0.001
Smoking				
Non-smokers	76	24	1	Ref.
Ex-smokers	74	26	1.0(0.8-1.3)	0.9
Current smokers	80	20	1.2(0.9-1.4)	0.2
BMI (kg/m^2)				
<25	78	22	1	Ref.
25-30	76	24	1.0(0.8-1.2)	0.9
≥30	78	22	1.1(0.8-1.4)	0.6
Physical activity				
Active	76	24	1	Ref.
Less active	77	23	1.1(0.9-1.3)	0.3
Inactive	78	22	1.4(1.1-1.8)	0.01
Alcohol (units per week)				
0–1	79	21	1	Ref.
1-10	76	24	0.8(0.6-1.1)	0.2
10-21	76	24	0.8(0.6-1.1)	0.2
>21	77	23	0.8(0.6-1.1)	0.3
Social class				
I, II	75	25	1	Ref.
IIINM	77	23	1.2(0.9-1.6)	0.2
IIIM	78	22	1.2(1.01-1.5)	0.05
IV, V	79	21	1.3(1.0-1.6)	0.06

Ref. = reference category

Table 6. 8: Odds ratio of low **fish** intake by some lifestyle factors in women.

	Fish intake		OR (95% CI)	p value
	Low& moderate	High		
Age groups(years)	%	%		
16-24	82	18	1	Ref.
25-34	81	19	0.9(0.7-1.3)	0.6
35-44	76	24	0.7(0.5-0.9)	0.05
45-54	75	25	0.6(0.5-0.9)	0.005
55-64	70	30	0.4(0.3-0.6)	0.001
65-74	72	28	0.5(0.3-0.6)	0.001
Smoking				
Non-smokers	74	26	1	Ref.
Ex-smokers	72	28	1.0(0.8-1.2)	0.8
Current smokers	79	21	1.2(1.01-1.4)	0.05
BMI (kg/m^2)				
<25	76	24	1	Ref.
25-30	76	24	1.1(0.9-1.3)	0.4
≥30	75	25	1.0(0.8-1.2)	1.0
Physical activity				
Active	72	28	1	Ref.
Less active	77	23	1.3(1.1-1.5)	0.005
Inactive	78	22	1.5(1.2-1.9)	0.001
Alcohol (units per week)				
0–1	77	23	1	Ref.
1-7	76	24	1.2(1.0-1.6)	0.2
7-14	72	28	1.1(0.9-1.4)	0.5
>14	76	24	0.9(0.7-1.1)	0.3
Social class				
I, II	72	28	1	Ref.
IIINM	71	29	0.9(0.7-1.1)	0.2
IIIM	79	21	1.4(1.1-1.7)	0.005
IV, V	80	20	1.5(1.2-1.8)	0.001

Ref. = reference category

Table 6. 9: Relationship between **fat food** intake by some lifestyle factors in men.

	Fat food intake			High relative to low		Moderate relative to low	
	Low	Moderate	High	OR (95% CI)	p Value	OR (95% CI)	p Value
Age groups(years)	%	%	%				
16-24	26	32	43	1	Ref.	1	Ref.
25-34	33	34	34	0.7(0.5-0.99)	0.05	0.8(0.6-1.2)	0.4
35-44	34	33	34	0.7(0.5-1.0)	0.09	0.8(0.5-1.2)	0.2
45-54	32	33	35	0.8(0.6-1.2)	0.4	0.9(0.6-1.3)	0.6
55-64	37	32	31	0.6(0.4-0.9)	0.02	0.8(0.5-1.1)	0.2
65-74	36	40	24	0.6(0.4-0.9)	0.03	1.0(0.7-1.6)	0.9
Smoking							
Non-smokers	37	35	27	1	Ref.	1	Ref.
Ex-smokers	37	34	29	1.3(1.01-1.7)	0.05	0.9(0.7-1.1)	0.5
Current smokers	26	32	42	2.1(1.7-2.5)	0.001	1.2(1.0-1.5)	0.2
BMI (kg/m^2)							
<25	28	33	39	1	Ref.	1	Ref.
25-30	35	35	30	0.7(0.6-0.9)	0.005	0.9(0.7-1.1)	0.2
≥30	35	35	30	0.8(0.6-1.0)	0.06	0.9(0.7-1.2)	0.7
Physical activity							
Active	30	32	38	1	Ref.	1	Ref.
Less active	35	34	31	0.8(0.6-0.99)	0.05	1.0(0.8-1.2)	0.7
Inactive	36	36	28	0.6(0.5-0.8)	0.001	0.9(0.7-1.2)	0.5
Alcohol (units per week)							
0–1	38	35	27	1	Ref.	1	Ref.
1-10	34	34	32	1.3(1.0-1.7)	0.2	1.2(0.9-1.5)	0.4
10-21	35	31	34	1.3(1.1-1.8)	0.06	1.1(0.8-1.4)	0.8
>21	29	35	36	1.5(1.1-2.0)	0.01	1.4(1.1-1.9)	0.02
Social class							
I, II	39	36	26	1	Ref.	1	Ref.
IIINM	39	31	30	1.1(0.8-1.5)	0.5	0.9(0.7-1.2)	0.5
IIIM	31	34	36	1.8(1.4-2.2)	0.001	1.2(1.01-1.5)	0.05
IV, V	25	34	42	2.3(1.8-3.0)	0.001	1.5(1.2-1.9)	0.005

Ref. = reference category

Table 6. 10: Relationship between **fat food** intake by some lifestyle factors in women.

	Fat food intake			High relative to low		Moderate relative to low	
	Low	Moderate	High	OR (95% CI)	p Value	OR (95% CI)	p Value
Age groups(years)	%	%	%				
16-24	31	33	35	1	Ref	1	Ref.
25-34	34	33	33	0.9(0.6-1.2)	0.4	1.0(0.6-1.2)	0.9
35-44	37	32	31	0.8(0.6-1.1)	0.3	0.8(0.6-1.2)	0.3
45-54	34	31	35	1.0(0.7-1.4)	1.0	0.9(0.7-1.3)	0.7
55-64	35	32	33	0.9(0.6-1.2)	0.5	0.8(0.6-1.1)	0.2
65-74	31	33	36	1.1(0.8-1.6)	0.4	1.0(0.7-1.3)	0.8
Smoking							
Non-smokers	37	33	30	1	Ref.	1	Ref.
Ex-smokers	35	34	32	1.0(0.8-1.2)	0.9	1.0(0.8-1.3)	0.7
Current smokers	30	31	40	1.4(1.2-1.7)	0.001	1.1(0.9-1.3)	0.4
BMI (kg/m^2)							
<25	34	31	35	1	Ref.	1	Ref.
25-30	33	34	33	1.0(0.8-1.2)	1.0	1.1(0.9-1.4)	0.2
≥30	35	33	32	0.9(0.8-1.1)	0.6	1.1(0.8-1.3)	0.8
Physical activity							
Active	36	31	34	1	Ref.	1	Ref.
Less active	33	34	34	1.0(0.9-1.3)	0.7	1.2(1.0-1.4)	0.1
Inactive	33	32	35	1.0(0.8-1.2)	0.9	1.1(0.8-1.4)	0.6
Alcohol (units per week)							
0–1	34	33	33	1	Ref.	1	Ref.
1-7	34	34	32	1.1(0.9-1.3)	0.5	1.1(0.9-1.3)	0.6
7-14	35	31	34	1.1(0.6-1.4)	0.6	0.9(0.7-1.2)	0.6
>14	33	28	35	1.2(0.9-1.5)	0.3	0.8(0.6-1.1)	0.2
Social class							
I, II	38	32	30	1	Ref.	1	Ref.
IIINM	34	36	31	1.0(0.8-1.3)	1.0	1.1(0.9-1.4)	0.2
IIIM	35	31	36	1.2(0.9-1.4)	0.3	1.0(0.8-1.2)	1.0
IV, V	29	31	40	1.5(1.2-1.8)	0.001	1.2(0.9-1.4)	0.3

Ref. = reference category

Table 6. 11: Relationship between **snack food** intake by some lifestyle factors in men.

	Snack food intake			High relative to low		Moderate relative to low	
	Low	Moderate	High	OR (95% CI)	p Value	OR (95% CI)	p Value
Age groups(years)	%	%	%				
16-24	11	25	64	1	Ref.	1	Ref.
25-34	27	29	44	0.3(0.2-0.5)	0.001	0.5(0.3-0.8)	0.005
35-44	32	36	32	0.2(01-0.3)	0.001	0.6(0.4-0.9)	0.02
45-54	43	34	23	0.1(0.1-0.2)	0.001	0.4(0.3-0.6)	0.001
55-64	41	35	24	0.1(0.1-0.2)	0.001	0.4(0.3-0.7)	0.001
65-74	37	38	25	0.1(0.1-0.2)	0.001	0.5(0.3-0.8)	0.01
Smoking							
Non-smokers	30	35	35	1	Ref.	1	Ref.
Ex-smokers	37	36	27	1.0(0.8-1.2)	1.0	0.9(0.8-1.2)	0.6
Current smokers	34	30	36	1.0(0.8-1.2)	0.7	0.8(0.7.1.0)	0.08
BMI (kg/m^2)							
<25	26	32	42	1	Ref.	1	Ref.
25-30	36	35	30	0.7(0.6-0.9)	0.005	0.9(0.7-1.0)	0.2
≥30	38	35	28	0.7(0.5-0.9)	0.005	0.8(0.7-1.0)	0.2
Physical activity							
Active	29	33	38	1	Ref.	1	Ref.
Less active	34	34	32	0.9(0.7-1.1)	0.3	0.9(0.7-1.1)	0.2
Inactive	38	32	30	1.0(0.8-1.3)	1.0	0.9(0.7-1.1)	0.2
Alcohol (units per week)							
0–1	29	34	37	1	Ref.	1	Ref.
1-10	31	33	36	0.8(0.6-1.1)	0.2	0.8(0.6-1.1)	0.2
10-21	34	36	30	0.5(0.4-0.7)	0.001	0.8(0.6-1.0)	0.06
>21	37	32	31	0.5(0.4-0.7)	0.001	0.7(0.5-0.9)	0.005
Social class							
I, II	35	35	30	1	Ref.	1	Ref.
IIINM	33	35	32	1.0(0.7-13)	0.9	1.0(0.6-1.3)	1.0
IIIM	31	33	36	1.5(1.2-1.8)	0.001	1.1(0.9-1.4)	0.3
IV, V	33	32	35	1.2(0.9-1.5)	0.3	1.0(0.8-1.2)	0.8

Ref. = reference category

Table 6. 12: Relationship between **snack food** intake by some lifestyle factors in women.

	Snack food intake			High relative to low		Moderate relative to low	
	Low	Moderate	High	OR (95% CI)	p Value	OR (95% CI)	p Value
Age groups(ycars)	%	%	%				
16-24	18	29	53	1	Ref.	1	Ref.
25-34	28	33	39	0.4(0.3-0.6)	0.001	0.7(0.5-0.98)	0.05
35-44	38	30	32	0.3(0.2-0.4)	0.001	0.5(0.3-0.7)	0.001
45-54	38	34	28	0.2(0.2-0.3)	0.001	0.6(0.4-0.8)	0.005
55-64	41	35	24	0.2(0.1-0.2)	0.001	0.5(0.4-0.8)	0.001
65-74	34	35	31	0.2(0.1-0.3)	0.001	0.6(0.4-0.9)	0.01
Smoking							
Non-smokers	31	36	33	1	Ref.	1	Ref.
Ex-smokers	37	33	30	0.9(0.7-1.1)	0.3	0.8(0.7-1.0)	0.08
Current smokers	37	28	35	0.8(0.7-0.99)	0.05	0.6(0.5-0.7)	0.001
BMI (kg/m^2)							
<25	31	34	36	1	Ref.	1	Ref.
25-30	36	34	30	0.9(0.7-1.1)	0.2	0.8(0.7-0.99)	0.05
≥30	36	32	32	0.9(0.6-1.1)	0.5	0.8(0.7-0.99)	0.05
Physical activity							
Active	35	32	33	1	Ref.	1	Ref.
Less active	33	34	33	1.2(1.0-1.4)	0.2	1.2(1.0-1.4)	0.09
Inactive	34	31	35	1.3(1.01-1.6)	0.05	1.10.81.3)	0.8
Alcohol (units per week)							
0–1	31	33	37	1	Ref.	1	Ref.
1-7	33	33	34	0.7(0.6-0.9)	0.005	0.9(0.7-1.1)	0.3
7-14	38	34	28	0.6(0.4-0.7)	0.001	0.8(0.6-1.1)	0.1
>14	40	28	32	0.5(0.4-0.7)	0.001	0.7(0.5-0.8)	0.005
Social class							
I, II	38	33	29	1	Ref.	1	Ref.
IIINM	33	33	34	1.1(0.9-1.4)	0.2	1.1(0.9-1.3)	0.5
IIIM	31	33	35	1.3(1.1-1.7)	0.01	1.3(1.0-1.6)	0.02
IV, V	33	32	35	1.2(1.0-1.5)	0.05	1.2(0.9-1.4)	0.2

Ref. = reference category

Table 6. 13: Relationship between **milk** intake by some lifestyle factors in men.

	Milk intake			Low relative to high		Moderate relative to high	
	Low	Moderate	High	OR (95% CI)	p Value	OR (95% CI)	p Value
Age groups(years)	%	%	%				
16-24	23	30	47	1	Ref.	1	Ref.
25-34	26	34	42	1.1(0.7-1.5)	0.9	1.3(0.9-1.8)	0.2
35-44	27	42	31	1.4(1.0-1.9)	0.09	2.0(1.4-2.8)	0.001
45-54	36	42	22	2.6(1.8-3.8)	0.001	3.1(2.1-4.4)	0.001
55-64	39	40	22	2.6(1.8-3.8)	0.001	2.5(1.7-3.6)	0.001
65-74	37	46	19	2.7(1.8-4.1)	0.001	3.2(2.1-4.7)	0.001
Smoking							
Non-smokers	30	41	28	1	Ref.	1	Ref.
Ex-smokers	35	44	21	1.0(0.8-1.3)	1.0	1.1(0.9-1.5)	0.3
Current smokers	29	33	38	0.7(0.5-0.8)	0.001	0.6(0.5-0.8)	0.001
BMI (kg/m^2)							
<25	25	38	37	1	Ref.	1	Ref.
25-30	32	42	26	1.4(1.1-1.7)	0.005	1.2(1.01-1.5)	0.05
≥30	36	39	25	1.4(1.1-1.9)	0.01	1.0(0.8-1.3)	0.9
Physical activity							
Active	28	36	36	1	Ref.	1	Ref.
Less active	31	41	28	1.2(0.9-1.4)	0.2	1.1(0.9-1.4)	0.3
Inactive	36	42	22	1.5(1.2-1.9)	0.005	1.5(1.2-1.9)	0.005
Alcohol (units per week)							
0–1	30	40	30	1	Ref.	1	Ref.
1-10	29	43	28	1.4(1.0-1.8)	0.05	1.4(1.1-1.8)	0.03
10-21	33	40	28	1.5(1.1-2.1)	0.01	1.4(1.02-1.8)	0.05
>21	32	36	32	1.3(1.0-1.8)	0.06	1.1(0.8-1.5)	0.5
Social class							
I, II	32	43	25	1	Ref.	1	Ref.
IIINM	35	39	26	1.1(0.8-1.5)	0.6	0.9(0.7-1.3)	0.6
IIIM	30	37	32	0.7(0.6-0.9)	0.005	0.7(0.6-0.8)	0.001
IV, V	28	37	35	0.7(-0.5-0.9)	0.01	0.7(0.5-0.9)	0.01

Ref. = reference category

Table 6. 14: Relationship between **milk** intake by some lifestyle factors in women.

Age groups(years)	%	%	%				
16-24	40	34	25	1	Ref.	1	Ref.
25-34	37	42	22	1.0(0.7-1.4)	1.0	1.4(1.0-1.9)	0.06
35-44	39	44	17	1.3(0.9-1.8)	0.2	1.7(1.2-2.4)	0.005
45-54	44	40	16	1.6(1.1-2.3)	0.01	1.8(1.3-2.6)	0.005
55-64	44	38	18	1.5(1.0-2.1)	0.05	1.6(1.1-2.3)	0.02
65-74	40	45	15	1.5(1.04-2.2)	0.05	2.3(1.6-3.4)	0.001
Smoking							
Non-smokers	42	44	14	1	Ref.	1	Ref.
Ex-smokers	42	42	16	0.8(0.6-1.1)	0.2	0.8(0.6-1.0)	0.2
Current smokers	38	37	25	0.5(0.4-0.6)	0.001	0.5(0.4-0.6)	0.001
BMI (kg/m^2)							
<25	39	41	19	1	Ref.	1	Ref.
25-30	41	42	17	1.1(0.9-1.3)	0.6	1.0(0.8-1.3)	0.8
≥30	44	41	15	1.3(1.0-1.6)	0.06	1.1(0.9-1.4)	0.3
Physical activity							
Active	40	40	20	1	Ref.	1	Ref.
Less active	40	42	18	1.1(0.9-1.4)	0.3	1.1(0.9-1.4)	0.3
Inactive	41	42	17	1.4(1.01-1.8)	0.05	1.4(1.1-1.8)	0.05
Alcohol (units per week)							
0–1	40	38	22	1	Ref.	1	Ref.
1-7	40	43	17	1.5(1.2-1.9)	0.001	1.7(1.4-2.1)	0.001
7-14	39	46	15	1.8(1.3-2.3)	0.001	2.1(1.6-2.8)	0.001
>14	44	38	18	2.1(1.5-2.8)	0.001	1.7(1.2-2.3)	0.005
Social class							
I, II	41	42	17	1	Ref.	1	Ref.
IIINM	39	45	16	1.1(0.8-1.4)	0.6	1.2(0.9-1.6)	0.2
IIIM	43	38	19	1.0(0.8-1.3)	0.9	0.9(0.7-1.1)	0.3
IV, V	40	38	22	0.9(0.7-1.1)	0.4	0.8(0.7-1.1)	0.2

Ref. = reference category

Table 6. 15: Odds ratio of **adding sugar** to tea or coffee by some lifestyle factors in men.

	Add sugar		OR (95% CI)	p value
	Yes	No		
Age groups(years)	%	%		
16-24	79	21	1	Ref.
25-34	68	32	0.7(0.5,0.98)	0.037
35-44	56	44	0.4(0.3,0.6)	0.000
45-54	59	41	0.5(0.4,0.7)	0.000
55-64	61	39	0.5(0.3,0.7)	0.000
65-74	59	41	0.5(0.3,0.8)	0.001
Smoking				
Non-smokers	54	46	1	Ref.
Ex-smokers	55	45	1.2(1.0,1.5)	0.40
Current smokers	77	23	2.4(2.0,2.9)	0.000
BMI (kg/m^2)				
<25	73	27	1	Ref.
25-30	59	41	0.7(0.5,0.8)	0.000
≥30	50	50	0.4(0.4,0.6)	0.000
Physical activity				
Active	65	35	1	Ref.
Less active	59	41	0.9(0.8,1.1)	0.6
Inactive	64	36	1.0(0.8,1.2)	0.9
Alcohol (units per week)				
0–1	65	35	1	Ref.
1-10	59	41	0.9(0.7,1.2)	0.6
10-21	59	41	0.9(0.7,1.2)	0.4
>21	66	34	1.0(0.8,1.4)	0.7
Social class				
I, II	50	50	1	Ref.
IIINM	60	40	1.4(1.1,1.9)	0.006
IIIM	69	31	2.1(1.7,2.5)	0.000
IV, V	73	27	2.2(1.6,2.7)	0.000

Ref. = reference category

Table 6. 16: Odds ratio of **adding sugar** to tea or coffee by some lifestyle factors in women.

Age groups(years)	%	%		
16-24	62	38	1	Ref.
25-34	43	57	0.4(0.3,0.5)	0.000
35-44	35	65	0.3(0.2,0.4)	0.000
45-54	31	69	0.3(0.2,0.3)	0.000
55-64	38	62	0.3(0.2,0.4)	0.000
65-74	41	59	0.3(0.2,0.4)	0.000
Smoking				
Non-smokers	33	67	1	Ref.
Ex-smokers	33	67	1.1(0.9,1.4)	0.2
Current smokers	52	48	2.1(1.8,2.4)	0.000
BMI (kg/m^2)				
<25	47	53	1	Ref.
25-30	35	65	0.7(0.6,0.8)	0.000
≥30	34	66	0.6(0.5,0.7)	0.000
Physical activity				
Active	36	64	1	Ref.
Less active	39	61	1.2(1.0,1.4)	0.027
Inactive	47	53	1.6(1.3,2.0)	0.000
Alcohol (units per week)				
0–1	49	51	1	Ref.
1-7	37	63	0.8(0.5,0.7)	0.000
7-14	33	67	0.5(0.4(0.6)	0.000
>14	37	63	0.4(0.3,0.6)	0.000
Social class				
I, II	31	69	1	Ref.
IIINM	36	64	0.9(0.8,1.2)	0.61
IIIM	43	57	1.4(1.1,1.7)	0.001
IV, V	50	50	1.6(1.3,1.9)	0.000

Ref. = reference category

Table 6. 17: Relationship between **adding salt** to food by some lifestyle factors in men.

	Adding salt to food			Yes relative to rarely		Occasionally relative to rarely	
	Rarely	Occasionally	Yes	OR (95% CI)	p Value	OR (95% CI)	p Value
Age groups(ycars)	%	%	%				
16-24	36	20	44	1	Ref	1	Ref.
25-34	32	18	49	1.2(0.9-1.7)	0.3	1.0(0.7-1.5)	1.0
35-44	31	19	50	1.3(0.9-1.7)	0.2	1.1(0.7-1.6)	0.8
45-54	31	19	50	1.3(1.0-1.9)	0.1	1.3(0.8-1.9)	0.3
55-64	27	19	54	1.5(1.1-2.2)	0.02	1.4(0.9-2.1)	0.2
65-74	30	23	47	1.3(0.9-1.8)	0.3	1.4(0.9-2.2)	0.2
Smoking							
Non-smokers	37	21	42	1	Ref.	1	Ref.
Ex-smokers	29	22	49	1.4(1.1-1.7)	0.005	1.1(0.8-1.3)	0.8
Current smokers	25	16	59	1.8(1.5-2.2)	0.001	1.0(0.8-1.3)	1.0
BMI (kg/m^2)							
<25	32	19	49	1	Ref.	1	Ref.
25-30	31	20	49	1.1(0.9-1.3)	0.5	1.1(0.8-1.3)	0.6
≥30	30	20	50	1.1(0.9-1.4)	0.4	1.0(0.8-1.4)	1.0
Physical activity							
Active	32	20	48	1	Ref.	1	Ref.
Less active	32	19	49	1.0(0.9-1.2)	0.8	0.8(0.7-1.0)	0.2
Inactive	28	20	53	1.0(0.8-1.3)	0.8	1.0(0.8-1.3)	1.0
Alcohol (units per week)							
0–1	39	20	42	1	Ref.	1	Ref.
1-10	33	20	47	1.5(1.2-2.0)	0.005	1.3(0.9-1.8)	0.2
10-21	29	21	50	1.9(1.4-2.5)	0.001	1.7(1.2-2.3)	0.005
>21	26	19	55	2.1(1.6-2.7)	0.001	1.7(1.2-2.3)	0.005
Social class							
I, II	37	23	40	1	Ref.	1	Ref.
IIINM	31	20	49	1.5(1.2-2.0)	0.005	1.1(0.8-1.6)	0.5
IIIM	27	17	56	1.8(1.5-2.2)	0.001	1.1(0.8-1.3)	0.6
IV, V	25	17	58	2.0(1.6-2.5)	0.001	1.1(0.8-1.5)	0.4

Ref. = reference category

Table 6. 18: Relationship between **adding salt** to food by some lifestyle factors in women.

Age groups(years)	%	%	%				
16-24	45	19	36	1	Ref.	1	Ref.
25-34	34	21	45	1.7(1.3-2.2)	0.001	1.3(0.9-1.8)	0.2
35-44	39	20	41	1.4(1.1-1.9)	0.02	1.1(0.8-1.5)	0.7
45-54	36	24	41	1.5(1.1-2.0)	0.01	1.5(1.1-2.0)	0.03
55-64	37	24	40	1.4(1.1-1.9)	0.02	1.3(0.9-1.9)	0.2
65-74	43	22	35	1.1(0.8-1.5)	0.6	1.1(0.8-1.5)	0.7
Smoking							
Non-smokers	43	23	34	1	Ref.	1	Ref.
Ex-smokers	40	23	37	1.2(1.0-1.4)	0.2	1.1(0.9-1.4)	0.4
Current smokers	31	20	49	1.8(1.5-2.1)	0.001	1.2(1.0-1.4)	0.08
BMI (kg/m^2)							
<25	40	20	40	1	Ref.	1	Ref.
25-30	39	21	39	1.0(0.8-1.2)	1.0	1.1(0.9-1.3)	0.6
≥30	36	24	40	1.2(1.0-1.4)	0.1	1.3(1.04-1.6)	0.02
Physical activity							
Active	40	22	38	1	Ref.	1	Ref.
Less active	39	21	41	1.2(1.01-1.4)	0.05	1.0(0.8-1.2)	0.9
Inactive	36	23	41	1.3(1.1-1.6)	0.02	1.2(1.0-1.6)	0.09
Alcohol (units per week)							
0–1	41	21	38	1	Ref.	1	Ref.
1-7	39	22	39	1.3(1.1-1.5)	0.02	1.2(1.0-1.5)	0.07
7-14	35	24	41	1.4(1.1-1.7)	0.005	1.5(1.2-1.9)	0.005
>14	35	19	46	1.5(1.2-2.0)	0.001	1.1(0.8-1.4)	0.7
Social class							
I, II	42	25	33	1	Ref.	1	Ref.
IIINM	43	20	37	1.0(0.8-1.3)	0.7	0.8(0.6-0.96)	0.03
IIIM	37	21	43	1.4(1.1-1.7)	0.005	0.9(0.7-1.2)	0.6
IV, V	32	20	48	1.6(1.3-1.9)	0.001	1.0(0.8-1.2)	0.9

Ref. = reference category

Chapter 7: The effect of smoking cessation on energy balance dietary patterns and physical activity

7.1. Introduction

Smoking is one of the most important causes of morbidity worldwide increasing CVD, respiratory disease, some cancers and cause of premature death and ill health (WHO, 2002). Smoking cessation has substantial health benefits and is associated with a considerable reduction in risk of all cause mortality among patients with CHD and it reduces by 36% the risk of mortality in all patients with CHD (Critchley & Capewell, 2003).

Weight gain is one of the most common consequences of smoking cessation as a result of changing energy balance during this period (Perkins *et al.*, 1990). Concern about weight gain after smoking cessation is one of the primary reasons not to give up smoking or as a reason to terminate the efforts to quit smoking very early. It has been shown that women with strong concerns about their weight are less likely to even engage in a smoking cessation attempt (Pomerleau *et al.*, 2000). Although females are more likely to be weight concerned (Meyers *et al.*, 1997) it has been shown that motivation to quit in male smokers with weight concern was significantly lower than non-smokers (Clark *et al.*, 2004).

The amount of post cessation weight gain varies widely according to type of study, duration of smoking cessation, type of population, age, sex and social-economic status. Different amounts of weight gain have been reported from different studies: from 2.8 to 7.6 kg for men and 3.8 to 8.7 kg for women (Williamson *et al.*, 1991; Kawachi *et al.*, 1996; O'Hara *et al.*, 1998). It has been reported that the rate of weight gain during smoking cessation is high in the first weeks or months and plateaus after 6 months before body weight stabilises at the higher level ((Hall *et al.*, 1986). The number of studies that have evaluated the effects of smoking cessation on body

weight prospectively are few, and many of them have methodological problems (Filozof *et al.*, 2004).

The association between smoking cessation and changes in body composition and shape are inconsistent and data in this field are limited. It has been shown that past and present smoking habits are positively associated with abdominal fat as measured by WHR and waist circumference in older men, but not in older women (Visser *et al.*, 1999b). However, Swedish women who stopped smoking gained weight, but their WHR did not increase as much as would be expected from associated weight gain (Lissner *et al.*, 1992). In this study continuing smokers gained 0.5 and quitters gained 1.4 units of BMI kg/m^2 after 6 years. After controlling for changes in BMI, smokers increased by 0.05 and quitters increased 0.03 units of WHR. In another study the amount of increase in WHR among those who quit smoking was significantly less than that expected had smoking continued (Shimokata *et al.*, 1989).

Nicotine in tobacco smoke may be the active factor. Nicotine imposes multiple influences, including specific effects on the central and peripheral nervous systems and control of food intake, as well as increases in metabolic rate (Li *et al.*, 2003). Thus, replacement of nicotine during abstinence by nicotine gum, transdermal patch or intranasal spray might reduce, prevent or delay weight gain (Emont & Cummings, 1987; Gross *et al.*, 1989; Dale *et al.*, 1998).

Smokers are known to consume poorer diets in terms of the type of fat, cholesterol, alcohol, fibre and certain vitamins than those who do not smoke (Dallongeville *et al.*, 1998) which contributes to their poorer health. Evidence shows that food intake may increase over a short time following smoking cessation and may decrease transiently after relapse (Hall *et al.*, 1989; Perkins, 1993).

Information about the dietary patterns during smoking cessation and during maintenance of cessation is lacking.

Few studies exist to describe the effects of a programme of smoking cessation on the dietary patterns, body weight and shape and physical activity of its participants in continuous abstainers.

The present study aimed to examine the effects of smoking cessation delivered programme within a 12 week and following an approach widely accepted in Scotland. Nicotine replacement therapy (NRT) was offered routinely to all participants. Dietary intake, body weight and shape, and physical activity were measured at baseline and after cessation in free living subjects.

7.2. Subjects and methods

Study design and setting have been described in method section in chapter 2.

7.3. Results

7.3.1. Study numbers

Fifty-five (44% male and 56% female) current smokers were recruited through a smoking cessation program at baseline as they prepared to cease smoking. For repeat measurements at subsequent follow-up sessions, 32 subjects (34% male and 66% female) attended week 7, 21 subjects (29% male and 71%) at week 12 and 18 subjects (33% male and 67% female) at week 18. The total number of subjects who completed all questionnaires (general questions, food frequency questionnaire and physical activity questionnaire) were 33, 24, 19, 17 at baseline, weeks 7, 12 and 18 respectively. Therefore the attrition rates at week 7, 12 and 18 of smoking

cessation program were 42%, 62% and 67% respectively for anthropometric data. These attrition rates for the questionnaires only were 27.3%, 42% and 49% respectively. It should be noted that apart from this study, the amount of attrition rate from smoking cessation on its own was almost 40-60% in different programmes.

It was originally proposed to follow two programmes of smoking cessation, however, because of the high attrition rates five programmes of smoking cessation were followed. All were based on the same part of west of Scotland (Easkilbride) and all delivered using the same approach.

7.3.2. Baseline characteristics

Baseline participant characteristics are shown in table 1 for all subjects and for those who completed the 18-week study. Participants mean ages were 47.2, range 16 – 67 years. Fifty five percent of the subjects were overweight or obese (BMI $\geq$ 25 kg/m^2). Most of the subjects were moderate and heavy smokers with a long duration of smoking (28.8 years, range 2-53 years). All of them planned to quit in the next 30 days and almost 90 % had tried to give up smoking in the past with an average of 3 attempts. However, almost 52% of them had quit for at least 24 hours in the past year and therefore when categorized terms of stages of change, at least 52% were in the preparation stage (Mcilvain HE 1998).

Almost three quarter (73%) of the participants reported that they were not satisfied with their body weight and body shape and 21% of the subjects said that they are on a diet to lose body weight, but mostly without external support. Apart from smoking cessation, 73% of the participants said that they had planned to change their physical activity, 61% to change their food intake and 22% to change their alcohol intake during smoking cessation. More than half (55%)

of the subjects at baseline reported that they would like to eat specific types of foods and of those half of them preferred to eat savoury foods.

The mean scores for restrained eating were 10.8 ± 3.9, for uncontrolled eating was 15.0 ± 5.0 and for emotional eating was 5.3 ± 2.5. Twenty five percent of the subjects had high score (3rd tertiles) of restraint and emotional eating, and 31% had high score (3rd tertiles) of uncontrolled eating.

There were no big differences between baseline characteristics of all subjects and of completers, although the completers were older by at least 4 years than all subjects. Comparison between those who completed the study and those who dropped out (table 7.1) shows that non-completers were younger but slightly heavier (BMI 27.2 kg/m²) than completers (BMI =26.6 kg/m²). Compliers also were more likely to have medical condition (50%) than non-compliers (33%).

7.3.3. Anthropometric changes during and after smoking cessation

Anthropometric changes during smoking cessation are shown in tables 2 and 3 and figures 1-7.

7.3.3.1. Body Weight and body mass index

Table 2 shows the mean and standard deviation of the anthropometric data and table 3 shows the median and interquartiles differences between participants of the study during smoking cessation program. The weight changes were between -6.20 to +8.60 kg over 18 weeks. Fifteen (83%) subjects had gained weight and 2 subjects lost weight and one subject remained weight stable. Mean weight gain among subjects who completed the 18 weeks of the study was 2.9 kg (median = 3.6 kg) in which males gained 1.1 kg (median = 2.7 kg) whereas females gained 3.8 kg (median = 4.3 kg). The weight change over 18 weeks was significantly different from baseline for all participants ($p<0.01$) and in females ($p< 0.01$), but not in males. The result was similar

with a Wilcoxon signed-rank test (non-parametric test). The trend of weight changes over the weeks of smoking abstinence (at week 7, 12 and 18) was significant based on both parametric and non-parametric tests in total ($P<0.05$ and $p<0.001$) and in females ($p<0.05$ and $p<0.001$) (figure 1). The trend for males was not significant ($p = 0.67$ and $p = 0.25$). Comparing weight changes at week 7, 12 and 18 with baseline shows that in females and in total the differences are significant ($p< 0.05$). Weight at week 18 significantly increased compared with week 12 ($p< 0.05$) in females not in males and the group as a whole, however the non-parametric Wilcoxon signed rank test showed that this was significant in the group as a whole ($p<0.05$). Individual weight changes have been shown in figures 4 and 5.

Mean BMI at week 18 increased 1.1 kg/m^2 in total (0.37 kg/m^2 in males and 1.49 kg/m^2 in females) from baseline and these changes were significant in total ($p < 0.01$) and in females ($p < 0.01$). The trend for changes in BMI from baseline to week 18 was significant in total subjects ($p<0.05$) and in females ($p<0.01$) (figure 3). Similar to body weight, BMI changes at week 7, 12, and 18 were significant from baseline in total and in female subjects ($p<0.05$), but there were no significant differences among males. Individual BMI changes are shown in figures 6 and 7.

7.3.3.2. Waist circumference and percent of body fat

Mean WC of females at the end of week 18 increased significantly, by almost 4 cm, compared with baseline ($p< 0.01$) whereas mean WC in males decreased insignificantly, almost 0.9 cm, during this period. The mean WC in females at week 7 decreased slightly, 0.13 cm, which was insignificant compared with baseline, but the trend of WC increase in females over the weeks was significant ($p < 0.01$) (figure 2).

The percentage of total body fat was calculated by using triceps skin fold and waist circumference. Total mean body fat percent significantly increased among females from baseline to week 18, almost 3 percent ($p < 0.01$). Males had body fat percent at week 18 slightly increased in total almost 0.5 percent, but these changes were not statistically significant. There was no significant changes in percent of body fat at week 7, however the trend at week 7, 12and 18 was significant ($p < 0.05$).

The mean WHR increase at week 18 was 0.02 in females, which was significant increased from baseline ($p < 0.01$), but there were no significant change among males during this period. There was also no significant changes at week 18 compared week 12 in females.

7.3.4. Energy Intake and physical activity

The reported energy intake and energy expended on physical activity per day are shown in table 4. The mean reported energy intake decreased -165 kcal/day at week 7 (males -49 kcal/day and female -224 kcal/day). This decrease was significant among females ($p < 0.05$) and all subjects as a group ($p < 0.05$), but not in males. At the end of week 18 mean energy intake increased 12 kcal per day in males and decreased 129 kcal per day in females. Although the reported energy intake decreased in females during the smoking cessation program, the trend was not significant over this period for all subjects and both males and females. On an individual basis, among the subjects who completed the study 53% at week 7, 71% at week 12 and 77% at week 18 showed a decrease in energy intake as measured by the diet Q questionnaire when compared to baseline, for both sexes. However, in contrast 12 subjects (71%) in response to this question "how much have changed your food intake?" at week 7, 12 and 18 said that their food have increased since their last session which was 5-6 weeks.

Table 5 shows the intake and macronutrients changes of nutrients during and after smoking cessation. In general there were no important changes in terms of nutrient intake, and diet composition during this study. The percentage of energy from total fat and saturated fat decreased slightly at the end of the study compared with baseline. The consumption of fruit increased during the study by on average 2 times per week by the end of the study ($p< 0.05$), but in contrast the amount of vegetables appeared to decrease, although these differences did not reach significace.

Energy expenditure by physical activity was estimated by calculating the reported physical activity to MET equivalent which was then converted to kcal energy per day. The amount of energy expended on physical activity, decreased from week 1 to week 18. The mean decrease was 44 kcal/day for total subjects and 112 kcal/day in females, but in males the mean energy expended on physical activity increased almost 84 kcal/day at week 18 compared with baseline. However, these changes were not statistically significant. The percentage of subjects who increased their physical activity by the end of the study compared with baseline was 47% in all subjects (83% males and 27% females).

7.3.5. Misreporting of energy intake

Mean EI/BMR for those who completed the study (n=17) was 1.34, 1.29, 1.26, and 1.25 at baseline, week 7, 12 and 18 respectively among all subjects. The mean in males was unchanged from baseline (1.22) to week 18 (1.21), however it decreased at week 12 (1.13). In females the mean decreased from 1.41 at baseline to 1.27 at week 18. Overall, the prevalence of under-

reporting, EI/BMR ≤ 1.35, was 71% (64% males and 83% females) at the end of the study. There was no over reporting, EI:BMR > 2.82, in total and both sexes.

7.4. Discussion

The use of NRT is effective when stopping smoking and can double the chances of success, especially when accompanied by intensive behavioral support (Silagy *et al.*, 2002; Molyneux, 2004). However, surprisingly evidence shows that only 17 % of smokers who had used NRT successfully quit at follow-up compared with only 10% of smokers in the control group (Silagy *et al.*, 2002). In a randomized controlled trial using minimal structured counseling and nicotine patches for three months the rate of continuous abstinence at 12 months was higher than the control group (20.2% vs. 8.7%)(Rodriguez-Artalejo *et al.*, 2003). In the present study, the rate of success in quitting after 18 weeks was at least 33%, which seems average, however direct comparison of the rate between present study and the mentioned figures could be misleading. It should be mentioned that the rate of continuous abstinence was higher among subjects who completed the questionnaires at the baseline (51%) that might be related with higher motivation among them at the baseline.

7.4.1. Anthropomertric changes

Studies showed that weight gain is common after smoking cessation. It is widely accepted that nicotine, which is the main addictive component of tobacco, is primarily responsible for the effects of smoking on body weight. Thus, replacement of nicotine during abstinence by gum, transdermal patch or nasal spray might in theory reduce or prevent weight gain.

A study by Moffatt et al (Moffatt & Owens, 1991) on women showed that smoking cessation for 60 days was accompanied by a 3.6 kg increase in body weight of which 72% percent was attributable to body fat. This amount of weight gain was partially due to a decreased RMR and increased caloric intake, however the authors also mentioned the other possible contributors such as decreased energy expenditure during daily activities, a lessened thermic response to food and a more efficient absorption of nutrients. Gross et al (Gross *et al.*, 1989) reported that after 10 weeks smoking abstinence using nicotine gum weight gain was 3.8 lbs compared with 7.8 lbs for placebo gum users and this decrease was greater in higher nicotine dose. In a study by using placebo, 2mg or 4mg of nicotine gum after 90 days post cessation the gum users gained 3.7, 2.1 and 1.7 kg respectively (Doherty *et al.*, 1996). It has been reported that there was a negative correlation between 8 week weight change and percentage of cotinine replacement (Dale *et al.*, 1998) and their 8 week weight change from baseline was 3.0 ± 2 kg. In this study men had higher weight gain at 8 weeks (4.0 ±1.8 kg) than women (2.1 ±1.7 kg). Allen et al (Allen *et al.*, 2005) in a randomized controlled trial of 94 postmenopausal female smokers showed that after 2 weeks of abstinence with nicotine patch, abstainers gained less weight than the placebo group (0.47 kg vs. 1.0 kg).

The present study result is in line with many other studies showed that weight gain after a short time cessation is common with NRT particularly in females. Although it is impossible to compare this result with the other studies directly, due to differences in study type, sample size, duration and attrition rate, the amount of weight gain seems high in compared with others, particularly for females. Furthermore, in this study we did not exclude subjects who had a medical condition or were on a diet, as we aimed to assess this program as a whole. It may partially explain the reason for a high weight gain in such a group; particularly in this study

completers were older and had more medical conditions than those who dropped out from the study. In addition, we were not able to collect the anthropometric data from non-compliers in order to compare the amount of weight gain between the two groups. Our study also confirmed the findings of other ((Moffatt & Owens, 1991) that not all individuals gained weight following cessation, despite the mean increase across the group. Small sample size in men precluded us from reaching any conclusions in terms of statistics.

Research on the association between smoking cessation and changes in body composition are limited. Visser et all (Visser *et al.*, 1999b)has shown that past and current smoking habits are positively associated with increased abdominal fat in older men, but the other studies showed that WHR had not increased as much as would be expected from associated weight gain in ex-smokers (Shimokata *et al.*, 1989; Lissner *et al.*, 1992). In the present study we have found increasing WC to be another important issue in smoking cessation in females with and without NRT, but weight gain was not linked to waist increase in men who became non-smoker. These findings require to be confirmed by other studies.

7.4.2. Dietary intake and physical activity

There are several factors possibly related to post smoking cessation weight gain. These include increasing the intake of foods that are high in fat and sugar, increasing alcohol consumption, changing physical activity, decreasing metabolic rate and energy expenditure (Talcott *et al.*, 1995). Allen et al (Allen *et al.*, 2005) reported that women who used nicotine patches had a higher energy intake than placebo group (173.6 vs. –100.4 kcal/day) after 2 weeks abstinence. This study also revealed that nicotine patch users consumed more fat and sweet carbohydrate than the control group. In contrast, Gross et al (Gross *et al.*, 1989) showed that nicotine gum

users had decreased hunger and reduced eating compared to those assigned to placebo. In a study by Hughes et al (Hughes & Hatsukami, 1997) subjects who stopped smoking for 6 weeks, caloric, carbohydrate and fat intake, hunger, and weight increased in those without NRT, but the use of a nicotine patch decreased caloric intake and intake of carbohydrate and fats in a dose related manner. Furthermore higher doses of nicotine completely reversed the increase observed in the placebo group. In a study (Gilbert & Pope, 1982) showed that most of the increased energy intake during smoking cessation took the form of extra snacks.

In the present study total energy intakes among participants were lower than their actual requirements and also with regard to gaining weight during smoking cessation. There may be numerous reasons that the subjects reported less energy intake than they needed. Misreporting and intention to under report may be one important explanation. This may also cause changes in reported dietary intake based particularly on the advice given to participants in group sessions. Apart from slightly decreased total and saturated fat, and vegetables intake and increased in fruit intake, the composition of the diet did not change significantly. Therefore, an intervention to improve the quality of diet along with smoking cessation is needed and this might be helpful for smoking cessation.

Allen et al (Allen *et al.*, 2005) showed that nicotine patches did not affect physical activity in the treatment compared with the placebo group of ceased smokers. However in this study the patch users decreased their physical activity after a further 2 weeks abstinence (-1.4 hours/week).

7.4.3. Limitations of the present study

This study has confirmed that it is possible to carry out research in the context of current smoking cessation programs. Limitations include the use of self-reported dietary intake and physical activity rather than interview, small sample size, and high attrition rates. Given that this was an observational study there were no control subjects or placebo to compare the amount of changes in weight, dietary intake and physical activity in NRT users with control or placebo or even non-compliers. We could not identify any possible factors to predict these changes and felt it was impossible to distinguish between the effects of different NRT products on outcomes. Additionally, information has been provided for further study so that sample size can be estimated.

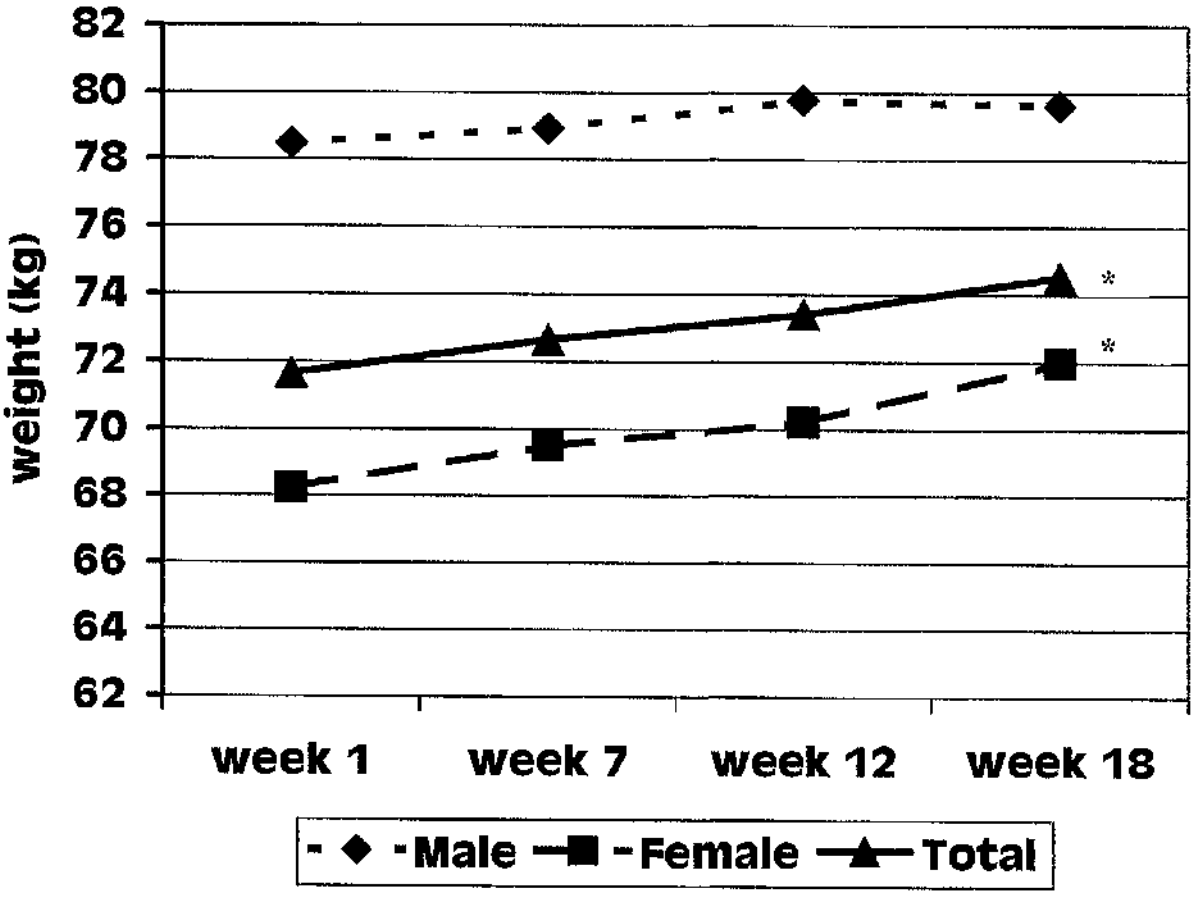
7.5. Conclusion

Weight gain and increased WC are common among people who are participating in current smoking cessation program using NRT particularly in women. Attrition rates from smoking cessation programmes are high and effective weight maintenance strategies have the potential to improve this. This small study was unable to determine the factors that might affect these anthropometric changes. However results from this study may be useful as a pilot for a larger study using similar methods with clarifying the limitations of current study would be required to confirm the findings of this study.

Table 7. 1: Baseline participant characteristics for all those who participated in the study and those who completed the 18-week follow up

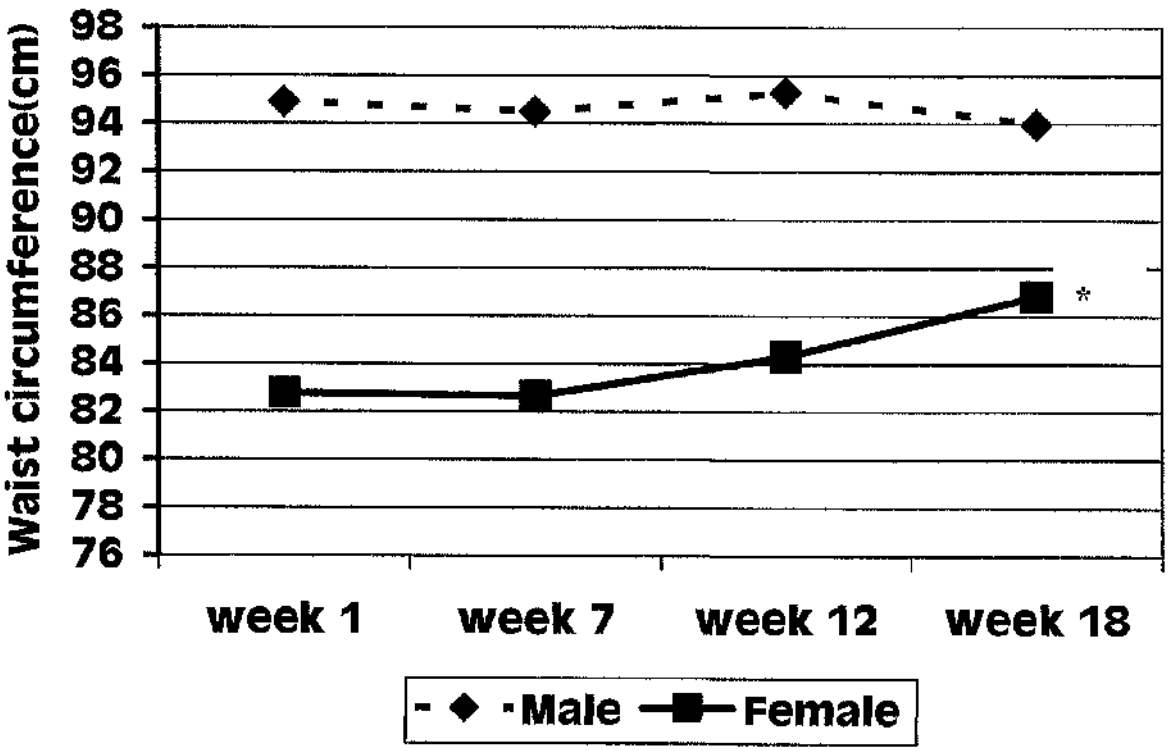
	All subjects (n = 33)	Completers (n = 18)	Non completers (n=15)
Age (year)	47.2 ± 12.5	51.4 ± 11.2	42.2 ±12.5
Sex	12 (36)	6 (33)	6 (40)
Male	21 (64)	12 (67)	9(60)
Female			
BMI (kg/m²)	26.9 ± 5.3	26.6 ± 4.9	27.2 ±5.8
Marital status			
Single	9 (27)	5 (28)	4 (27)
Married	22 (67)	13 (72)	9 (60)
Divorced	2 (6)	---	2 (13)
Medical conditions			
Yes	14 (42)	9 (50)	5 (33)
No	19 (58)	9 (50)	10(67)
Reported number of cigarettes smoked daily	19.2 ± 9.1	19.3 ±10.0	19.1 ±8.4
Duration of smoking (year)	28.8 ± 12.8	29.9 ± 12.7	27.4 ±13.2
Stages of change			
Contemplation	16 (48)	8 (44)	13 (78)
Preparation	17 (52)	10 (56)	2 (13)
Reported alcohol consumption			
Daily	5 (16)	3 (17)	2 (13)
Weekly	18 (56)	9 (50)	9 (60)
Less often or never	9 (28)	6 (33)	4 (27)
Body weight satisfaction			
Satisfied	9 (27)	5 (28)	4 (27)
Dissatisfied	24 (73)	13 (72)	11 (73)
Cognitively restrained *			
Low	9 (28)	6 (33)	3 (21)
Medium	15 (47)	7 (39)	8 (57)
High	8 (25)	3 (28)	3 (22)
Uncontrolled eating *			
Low	11 (34)	6 (33)	5 (36)
Medium	11 (34)	6 (33)	5 (36)
High	10 (31)	6 (33)	4 (29)
Emotional eating *			
Low	15 (47)	8 (44)	7 (50)
Medium	9 (28)	4 (22)	5 (36)
High	5 (25)	6 (33)	2 (14)

Data are mean ± S.D or n (%), * assessed by three-factor eating questionnaire revised 18-itm (TFEQ-R18)



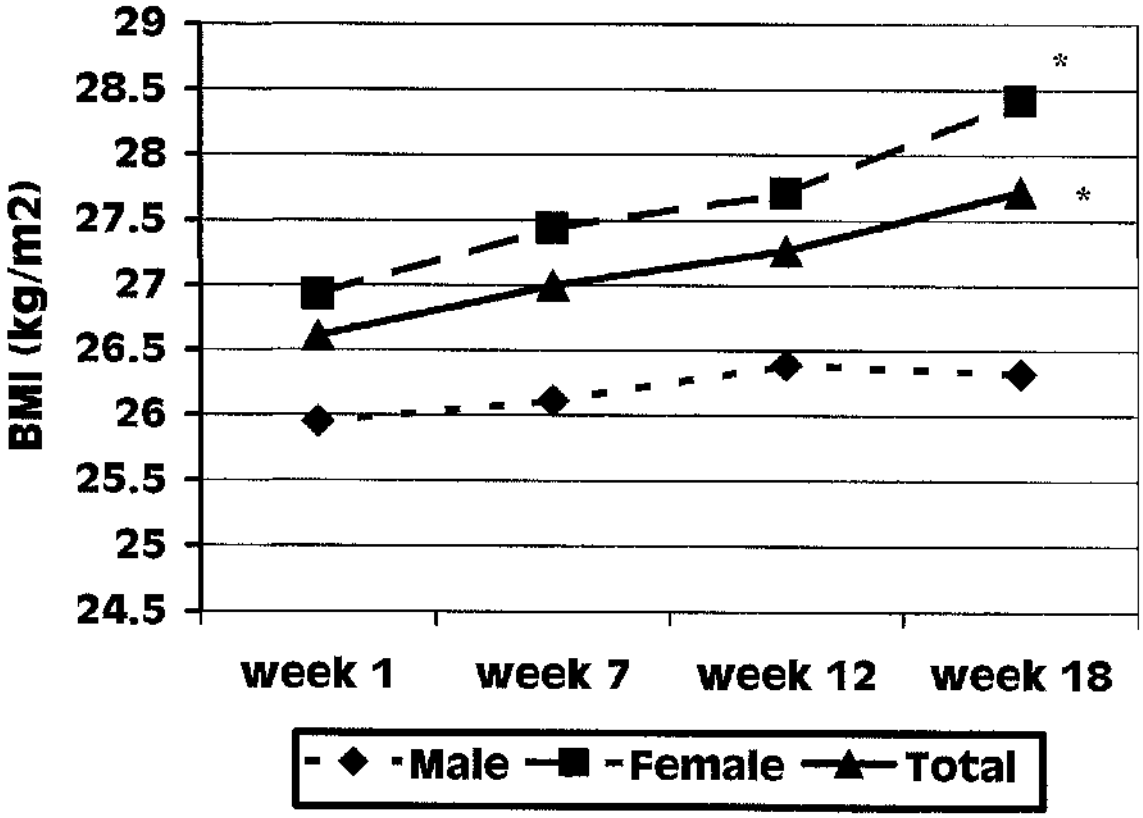
N = 18 (6 males, 12 females), * p for trend < 0.05

Figure 7. 1: Mean changes in body weight during smoking cessation program



N = 18 (6 males, 12 females), * p for trend < 0.01

Figure 7. 2: Mean changes in waist circumference during smoking cessation



N = 18 (6 males, 12 females), * p for trend < 0.05

Figure 7. 3: Mean changes in BMI during smoking cessation

Table 7. 2: Anthropometric changes during and after the smoking cessation program[A].

	Baseline (M= 11, F=21)	Week 7	Baseline (M=6, F=15)	Week12	Baseline (M=6, F=12)	Week18	Week12 (M=6, F=12)	Week18
Weight (kg)								
Male	85.4(22.7)	86.4(22.7)	78.5(22.5)	79.8(23.1)	78.5(22.5)	79.6(23.6)	79.8(23.1)	79.6(23.6)
Female	66.1(12.0)	66.9(12.5)+	66.5(12.7)	68.7(13.3)*	68.2(13.8)	72.0(14.9)*	70.2(14.5)	72.0(14.9)+
Total	72.7(18.7)	73.6(19.1)*	69.9(16.5)	71.9(16.8) *	71.6(17.2)	74.5(17.9)*	73.4(17.7)	74.5(17.9)
BMI (kg/m^2)								
Male	27.5(5.9)	27.8(6.1)	25.9(5.9)	26.4(6.1)	25.9(5.9)	26.3(6.2)	26.4(6.1)	26.3(6.2)
Female	26.1(4.3)	26.4(4.4)+	26.0(4.6)	26.8(4.7) *	26.9(4.6)	28.4(5.1)*	27.7(4.8)	28.4(5.1)+
Total	26.6(4.8)	26.9(5.0) *	26.0(4.7)	26.7(5.0)*	26.6(4.9)	27.7(5.4)+	27.3(5.1)	27.7(5.4)
Waist circumference (cm)								
Male	99.1(17.1)	99.0(16.4)	94.9(17.9)	95.3(15.9)	94.9(17.9)	94.0(17.4)	95.3(15.9)	94.0(17.4)
Female	80.5(10.9)	80.3(11.3)	80.5(11.9)	82.6(11.0)+	82.8(12.3)	86.8(12.7)*	84.3(11.7)	86.8(12.7) *
Percent of body fat								
Male	33.7(12.3)	33.9(12.8)	30.7(12.9)	31.6(13.2)	30.7(12.9)	31.2(12.9)	31.6(13.2)	31.2(12.9)
Female	37.9(7.9)	38.1(8.1)	38.5(7.6)	40.5(8.0)*	40.0(7.5)	42.9(8.2)*	41.8(8.2)	42.9(8.2)+
Waist-hip-ratio								
Male	0.97(0.08)	0.98(0.08)	0.97(0.09)	0.98(0.09)	0.97(0.06)	0.97(0.08)	0.98(0.09)	0.97(0.08)
Female	0.82(0.07)	0.82(0.07)	0.82(0.07)	0.84(0.07)*	0.83(0.08)	0.85(0.08)*	0.84(0.08)	0.85(.08)

A: data are mean (S.D.), Paired t test: + $p< 0.05$, * $p< 0.01$

Table 7. 3: Anthropometric changes during and after smoking cessation program [A].

	Week7- Baseline M= 11 F=21	Week12-Baseline M=6 F=15	Week18-Baseline M=6 F=12	Week 12-Week7 M=6 F=15	Week18-Week12 M=6 F=12
Weight (kg)					
Male	1.6(-0.7,2.6)	1.9(-0.6,3.8)	2.7(-1.6,3.8)	1.4(-0.5,2.0)	0.6(-2.1,1.7)
Female	0.80(-0.49,2.00)+	2.50(1.20,3.60)*	4.25(1.25,5.62)*	1.20(0.20,1.90)+	1.25(0.18,2.28)+
Total	1.05(-0.54,2.28)*	2.30(1.05,3.55)*	3.55(0.90,4.60)*	1.20(0.15,1.90)*	1.15(-0.18,2.23)+
BMI (kg/m²)					
Male	0.49(-0.24,0.87)	0.62(-0.19,0.62)	0.92(-0.52,1.21)	0.47(-0.15,0.62)	0.18(-0.72,0.56)
Female	0.34(-0.19,0.79)+	0.99(0.56,1.37)*	1.68(0.55,2.17)*	0.48(0.09,0.73)+	0.46(0.06,0.88)+
Total	0.42(-0.20,0.83)*	0.87(0.40,1.28)*	1.19(0.41,1.94)*	0.48(0.07,0.66)+	0.43(-0.07,0.85)+
Waist circumference (cm)					
Male	-0.75(-1.5,1.2)	0.20(-2.25,4.37)	-0.13(-3.56,2.73)	1.0(-1.1,3.31)	-1.5(-4.62,2.37)
Female	0.45(-1.50,1.75)	2.00(0.50,4.5)+	4.35(1.31,6.37)*	2.00(0.50,4.50)+	1.87(0.33,3.43)*
Percent of body fat					
Male	0.20(-0.58,1.43)	1.08(-0.53,2.28)	0.67(-2.39,2.86)	0.92(-47,1.45)	-0.86(-3.67,2.78)
Female	0.05(-0.53,1.41)	1.89(0.58,3.42)*	2.32(0.56,5.20)*	1.53(0.44,2.35)*	0.89(0.29,1.94)+
Waist-hip-ratio					
Male	0.007(-0.006,0.032)	0.017(0.002,0.030)	0.003(-0.026,0.032)	0.019(-0.025,0.030)	-0.008(-0.053,0.023)
Female	0.007(-0.009,0.012)	0.017(0.005,0.031)+	0.023(0.007,0.045)*	0.015(0.000,0.024)+	0.002(0.000,0.022)

A: data are median changes (interquartiles), wilcoxon signed rank test: + $p<0.05$, * $p<0.01$

Table 7. 4: Energy intake and energy expenditure by physical activity during smoking cessation program [A].

	Baseline (M= 8, F=16)	Week 7	Baseline (M=6, F=13)	Week12	Baseline (M=6, F=11)	Week18	Week12 (M=6, F=11)	Week18
Total energy Intake (kcal/day)								
Male	2213(766)	2163(581)	2099(867)	1958(647)	2099(667)	2111(588)	1958(647)	2111(588)
Female	2052(678)	1828(564)*	1921(585)	1774(433)	1840(581)	1711(565)	1772(468)	1711(565)
Total	2105(696)	1939(580)*	1977(667)	1832(499)	1931(680)	1852(588)	1838(526)	1852(588)
Energy expenditure on physical activity (kcal/day)								
Male	565(459)	494(443)	337(216)	316(199)	337(216)	421(402)	316(199)	421(402)
Female	596(418)	585(392)	524(390)	369(232)	512(416)	400(321)	352(245)	400(321)
Total	586(423)	555(402)	465(350)	352(218)	451(361)	407(339)	339(224)	407(339)

A: data are based on mean (S.D.). Paired t test: * $p < 0.05$.

Table 7. 5: diet composition and some nutrient intake during and after smoking cessation program [A].

	Baseline n=17	Week 7 n=17	Week12 n=17	Week 18 n =17
Total protein (g)	84.5 (22)	82.6 (19)	832 (19)	84 (17)
Total fat (g)	68.8 (31)	64.9 (30)	61.9 (28)	62.2 (29)
Total carbohydrate (g)	227.0 (91)	227 (63)	224.8 (61)	224.2 (72)
Total fibre (g)	12.3 (4)	13.3 (5)	12.9 (4)	13.1 (4)
Total sugar (g)	117.3 (71)	112.7 (39)	113 (37)	109.2 (45)
Percent of energy from protein	19.1 (6)	18.2 (3)	18.5 (2)	19.1 (4)
Percent of energy from carbohydrate	43.9 (8)	45.9 (6)	46.3 (6)	45.4 (6)
Percent of energy from fat	31.8 (7)	30.5 (8)	29.7 (7)	29.8 (8)
Percent of energy from saturated fat	14.1 (5)	13.3 (4)	12.9 (4)	12.7 (4)*
Total vegetable intake per week (portion)	10.3 (4)	9.2 (6)	8.8 (5)	8.6 (5)
Total fruit per week (portion)	7.8 (5)	9.8 (6)	9.4 (5)	9.8 (6)+

A: data are mean (S.D.), Paired t test compared with baseline: * $p< 0.05$, + $p = 0.05$

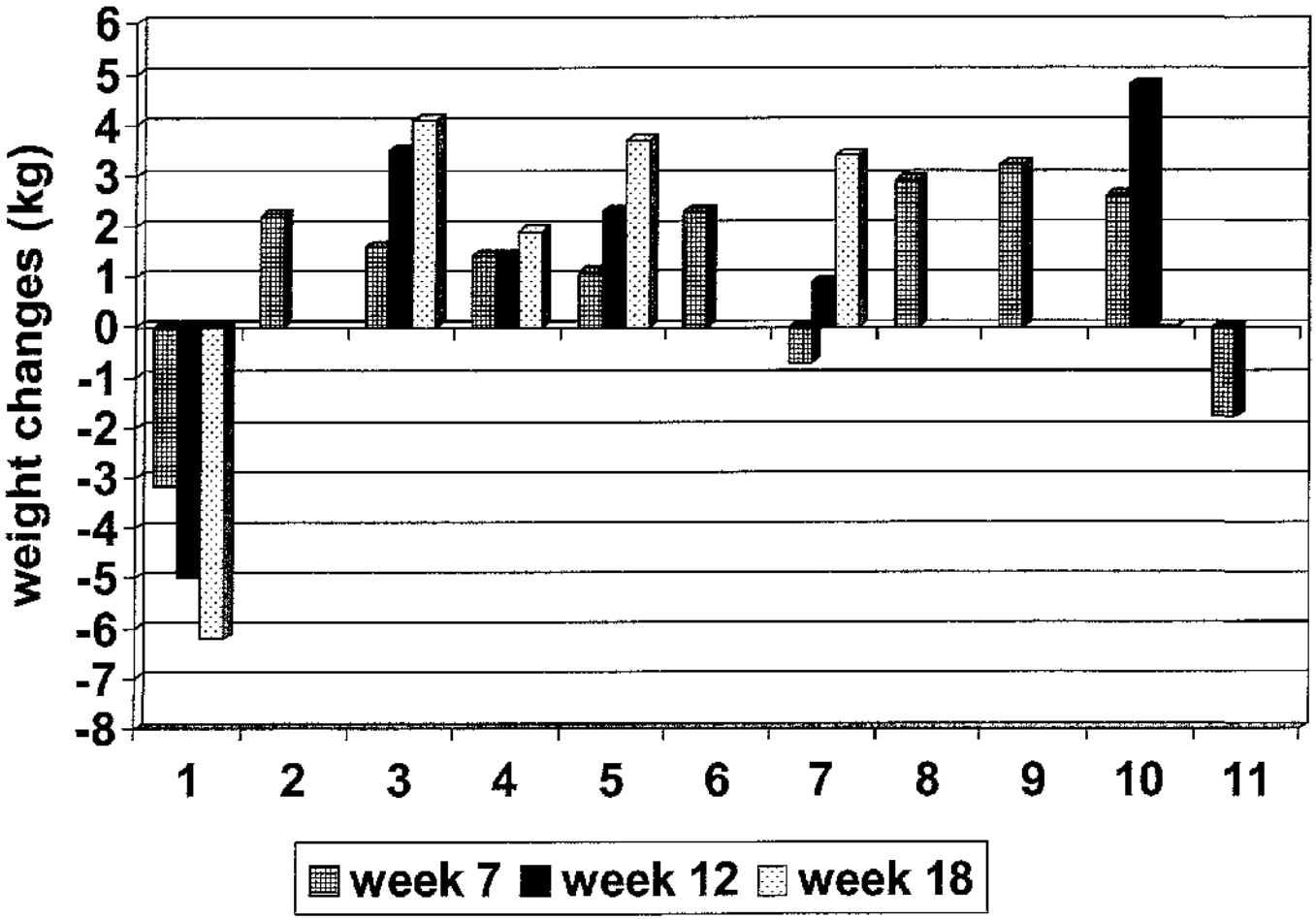


Figure 7. 4: Individual body weight changes during smoking cessation in males.

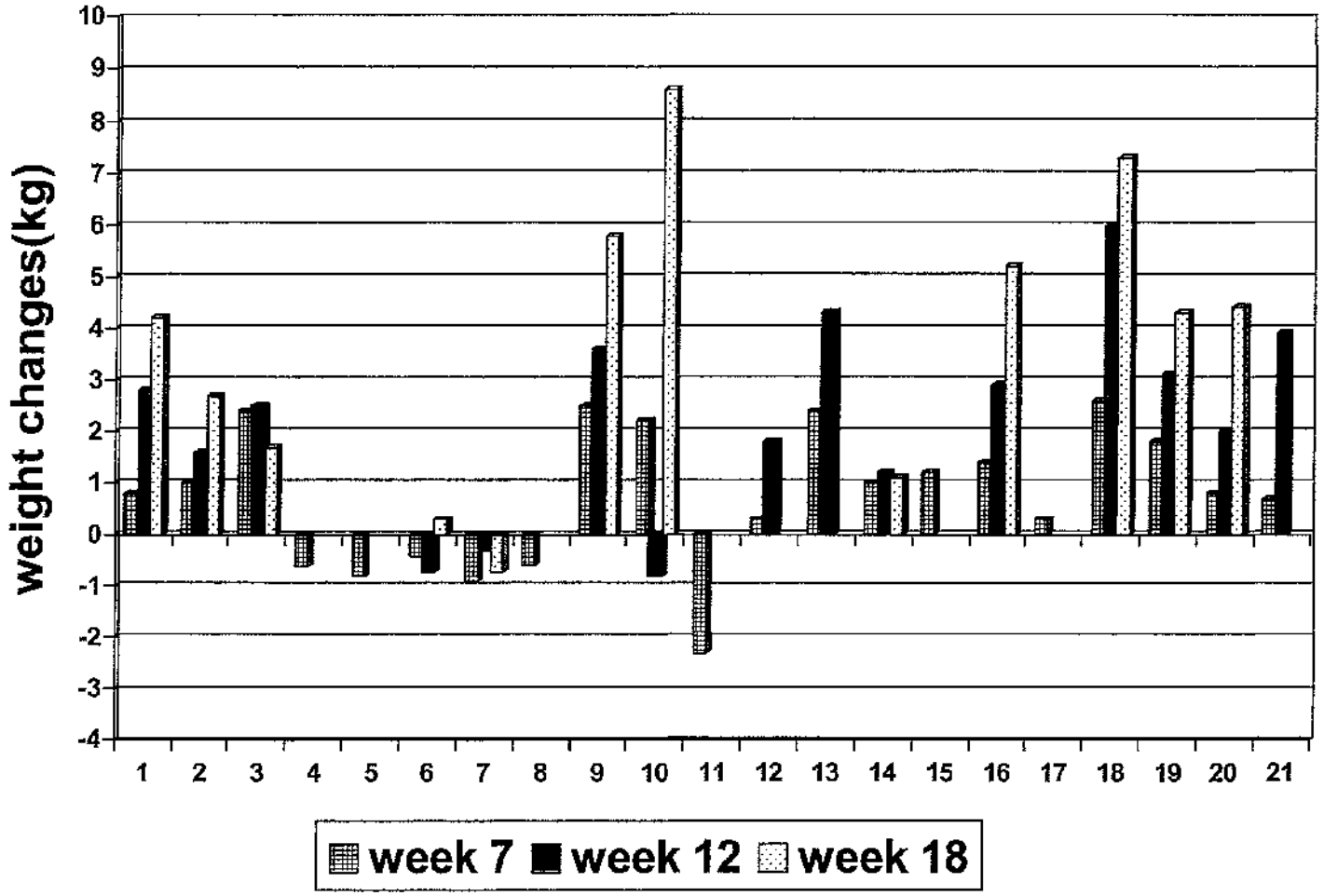


Figure 7. 5: Individual body weight changes during smoking cessation in females.

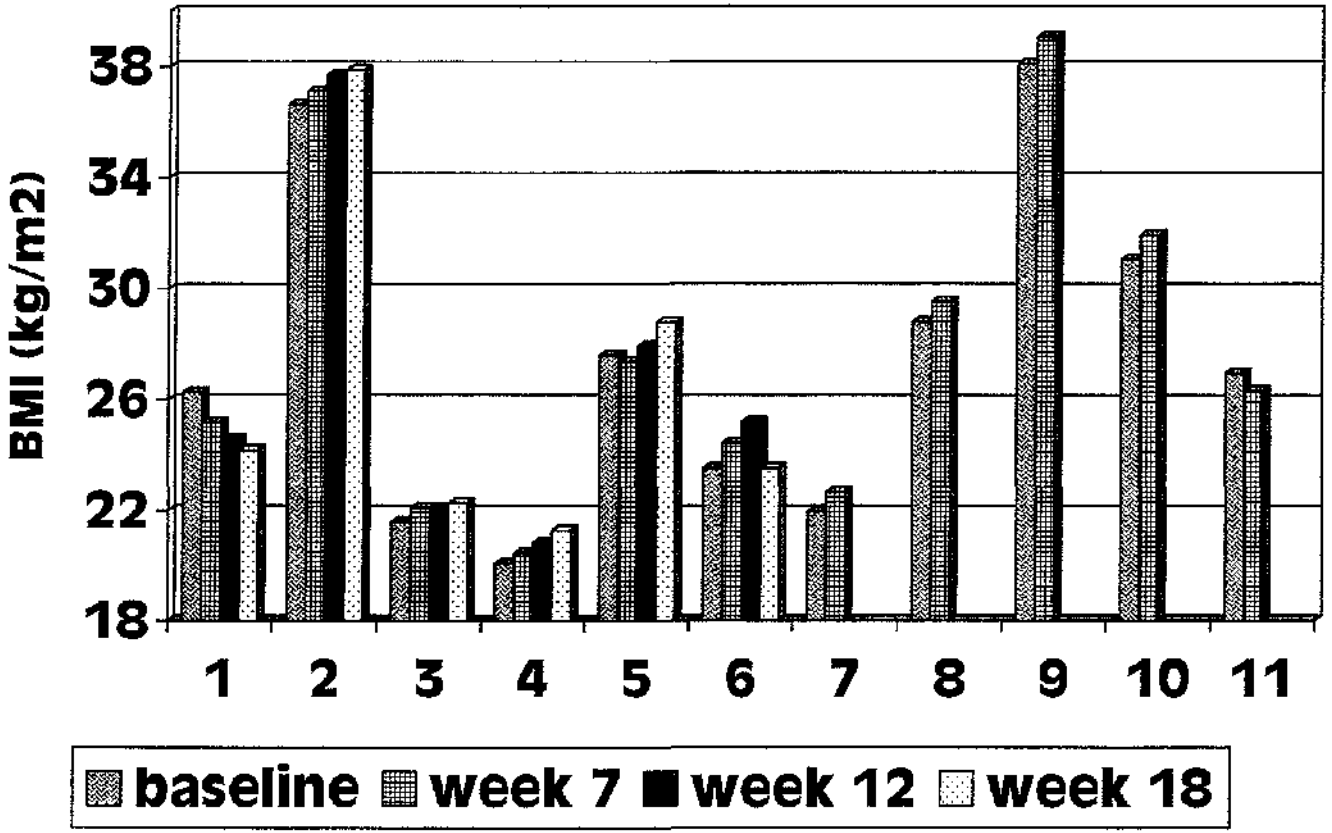


Figure 7. 6: Individual BMI during smoking cessation in males

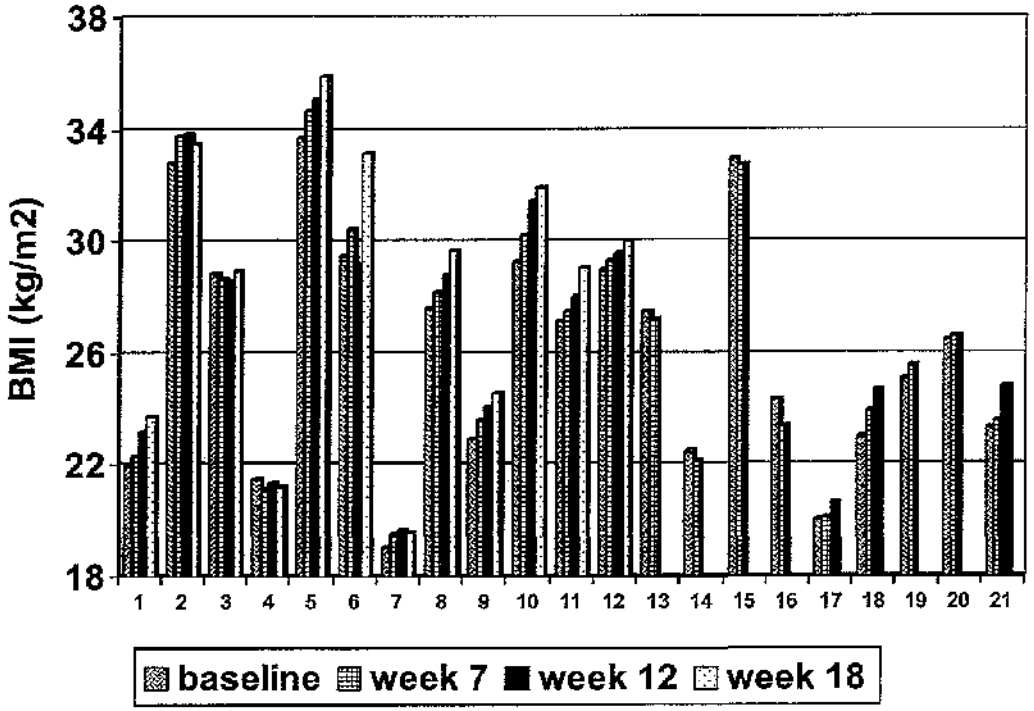


Figure 7. 7: Individual BMI during smoking cessation in females

Chapter 8: General discussion

8.1. Introduction

One of the major health problems worldwide is the rapid increase in the incidence of chronic non-communicable diseases. Obesity, whose prevalence is growing worldwide, is a major public health problem, linked closely with chronic disease occurrence and having trebled internationally since 1980. In Scotland, the Scottish Health Survey 2003 showed that 26% of women and 22.4% of men were obese. Any health promotion initiations should be based on a comprehensive understanding of the health problems and existing health behaviours of the population.

It is generally accepted that the rapid changes in diet and reduced or at least unchanged physical activity levels are associated with the epidemic of obesity worldwide. Global changes in urbanization; economic growth; changes in technology in work, leisure, food processing and mass media growth, have resulted in shifts in diet towards a higher energy dense, sweeter, and more processed foods with higher animal fats and a lower intake of fibre on one hand, and falling physical activity towards reduced energy expenditure on the other hand (Popkin, 2006).

Epidemiological evidence shows that there are direct and indirect associations between lifestyle factors such as physical activity, smoking, dietary habits, morbidity and mortality. Physical activity and dietary factors can affect directly overweight and obesity, hypertension, dyslipidemia, metabolic abnormality and incidence of chronic diseases, and indirectly via obesity or metabolic abnormalities can affect on chronic disease and mortality. Smoking is slightly different from the other lifestyle factors. It is usually associated with a lower BMI on one hand and higher metabolic abnormalities and chronic disease on the other. Smoking

cessation is associated with weight gain and results in slightly increased prevalence of obesity, however it improves metabolic profiles and may reduce the incidence of chronic disease.

In the present thesis, the association of the most modifiable lifestyle factors, smoking physical activity and dietary habits, with obesity and available cardiovascular risk factors, and the association of the combination of smoking and physical activity with obesity, with CVD risk factors have been examined. In addition, in an observational study "The effect of smoking cessation program using NRT on energy balance, dietary habits and physical activity" has been considered in a context of an ongoing smoking cessation programme.

8.2. Research questions and answers

This thesis has provided the answer for the research questions (chapter 1). This reports the main findings with regard to the questions, along with design limitations and further research topics.

RQ 1) what is the association between smoking status, BMI and body shape?

In chapter 3 the association between smoking and anthropometric indices were presented. The main findings of this chapter showed that those who smoked had a lower BMI, but higher central obesity compared with non-smokers especially in women. The positive associations between smoking and abdominal obesity have subsequently been confirmed by another large cross-sectional study from Norfolk, UK study (Canoy *et al.*, 2005). Another important finding of this chapter was the lack of any association between body weight and smoking in younger adults. Against a general belief that smoking can control body weight especially in younger and women, this study showed that smoking was not associated with a lower body weight in

younger adults. In addition, it was negatively linked to body composition, which is itself associated with adverse metabolic outcomes. This may be an important public health message to reassure young people that smoking may not be helpful to control body weight in short time period. Although smoking in older adults of a long duration may have negative associations with body weight, it should be remembered that a long duration of smoking causes a substantial damage to health. Therefore using smoking as a long-term weight management strategy probably offers no net benefits, causing considerable damage to health. Smoking cessation is essential to avoid these health negative consequences of smoking.

In the present study ex-smokers had a higher mean BMI compared with non- and current smokers and the prevalence of obesity was higher in this group compared to others. The mean and the prevalence of differences between ex- and current smoker was much higher than the differences between ex- and non-smokers. The inverse association between smoking and body weight, and weight gain after smoking cessation raised the question as to whether smoking cessation may partly be responsible for the increased prevalence of obesity in U.S. Smoking cessation might be associated with small increase in the prevalence of overweight in U.S (Flegal *et al.*, 1995). Another study, demonstrated that a higher cigarette price was associated with reduced smoking prevalence and increased rate of obesity in U.S (Chou *et al.*, 2004). However, these findings were not confirmed by other work that suggested a falling prevalence of smoking had little effect on the rising prevalence of obesity in U.S (Gruber & Frakes, 2006). In Scotland, overall smoking prevalence reduced 4% from 1995 to 2003, but the prevalence of overweight and obesity increased in this period by 8% in men and 10% in women. Therefore, the association of smoking cessation with increased prevalence of overweight and obesity should be studied in future at a population level. Apart from the associations between weight gain after smoking cessation, and the prevalence of overweight

and obesity at a population level, it may also affect those who are weight concerned and discourage them either to stop smoking or to withdraw from smoking cessation. Therefore, public health authorities should consider weight management along with smoking cessation programmes.

RQ 2) what is the independent and combined association between smoking and BMI with CVD risk factors?

Obesity and smoking are the two important major modifiable risk factors that contribute towards overall morbidity and mortality, especially from CVD. In the present study (chapter 4) the independent and joint associations of smoking and overweight/obesity with some established CVD risk factors have been studied. The main findings of this study were that both smoking and obesity were independently associated with inflammation markers and lipid abnormalities. Among all the lifestyle factors that have been studied, BMI was the most and smoking the second most influential factor in multivariable analysis. The combination of overweight and obesity with smoking markedly increased the risk of the CVD risk factors especially HDL-C and CRP.

Among the Scottish adult population in 1998, 54% had at least one of the two risk factors of obesity or smoking, and 5.4% were obese and smoked. These figures compared with the U.S national health interview survey in 2002, in which 4.7% of adults over 18 years old were obese and smoked, were higher (Healton *et al.*, 2006). The prevalence of the co-occurrence of these two risk factors was higher in women (5.8%) than men (5.0%) in Scottish population, whereas in U.S it was higher in men (5.3%) than women (4.2%). Expanding the joint prevalence of these two factors in the total population results in a considerable number of people suffering from the adverse health effects of this condition. From the public health

point of view this group of people should be in a higher priority for treatment and also prevention. Lifestyle changes may be one of the most effective means to reduce some of these risks, and more research is required to determine more effective ways to tackle both risk factors simultaneously.

RQ 3) what is the association between the currently recommended physical activity levels with BMI and body shape?

In the present study the associations between current recommendations for physical activity and anthropometric indices and CVD risk factors were examined (chapter 5). Physical activity, at least 30 minutes moderate activity on at least 5 days a week or at least 20 minutes vigorous activity on at least 3 days a week or both, were negatively associated with BMI and WC and WHR and positively with HC. However, almost half of the active subjects had an unhealthy BMI (BMI $\geq$ 25 kg/m^2) indicating that this level of physical activity was probably insufficient to prevent it.

RQ 4) what is the independent and combined association between currently recommended physical activity levels and BMI with CVD risk factors?

Plenty of evidence exists to show that high levels of physical activity or physical fitness, reduce the risk of chronic diseases including diabetes and CVD. In contrast, obesity increases the risk of these diseases, and in turn mortality from them. The present study showed that overweight and obesity were strongly linked with raised CVD risk factors, predicted 10 years coronary heart disease risk and Metabolic Syndrome. The recommended physical activity level was associated with a lower CRP, HDL-C, predicted CHD risk and Metabolic Syndrome, particularly in men, and it was also associated with better psychosocial health. However,

overweight and obese subjects who were active still were at elevated risk compared to the inactive non-overweight/obese group.

The cross sectional data prevented distinguishing between the effects of extra body fat, low physical activity, or both on CVD risk factors. However, the associations between obesity with the risk factors were stronger than the associations of physical activity with them. These findings showed that at least this level of self-reported physical activity might not be enough to eliminate the extra risk imposed by obesity. Both physical inactivity and obesity are major CVD risk factors. It is also possible that habitual physical activity indirectly affects CVD risk factors by reducing body weight and body fat, as has been seen in this study.

There is no doubt that physical activity or physical fitness has many physical, social and mental benefits. Evidence from randomised trials showed that moderate to high intensity exercise at least for eight weeks durations could reduce abdominal fats assessed by imaging methods in middle to older aged subjects (Kay & Fiatarone Singh, 2006). Some reviews of evidence showed that people who are fit or physically active enough have lower risk of morbidity or morbidity than normal weight unfit counterparts (Blair & Brodney, 1999; Grundy *et al.*, 1999). This raised the argument as to whether being fat but active or fit would be better than being inactive or unfit and thin. A small number of studies that looked at the joint relationship of physical activity and high BMI on mortality have found conflicting results and most were discussed earlier (chapter 5). The possible reasons for these different findings may be due to study design, methodologies or inaccurate measurements of exposure. A recent review of literature by Weinstein (Weinstein & Sesso, 2006) considered the joint effects of physical activity and body weight on diabetes and CVD and showed that only few studies had looked at the combined effects of physical activity and body weight. Based on this review, the

joint effects of obesity and activity are different based on these outcomes, and it seems on balance that physical activity is more important in CVD, and body weight or BMI has a greater effect on the development of diabetes. It has been suggested that it is essential to evaluate the independent and combined exposures of physical activity and BMI on health outcomes, and accurate measurements of the exposures are necessary.(Lamonte & Blair, 2006)

RQ 5) what are the associations between dietary habits and other lifestyle factors?

In chapter 6 the associations between lifestyle factors and dietary habits were presented. Younger people and those from lower social classes reported more unhealthy dietary habits than their older and higher class counterparts. Among modifiable lifestyle behaviours, smoking was one of the most important factors that showed consistent positive association with most unhealthy dietary habits. Cigarette smokers consumed fewer fruit and vegetables, more high fat rich foods, added more salt to their foods, and consumed more sugar with tea or coffee than non-smokers. Physical activity was another modifiable behaviour that showed positive associations to healthy dietary habits, but active men consumed more fat rich foods. Overall, moderate alcohol drinkers were shown to have more healthy dietary habits, however, there was inconsistency in these associations. Overweight/obese subjects reported healthier dietary patterns and less energy dense foods than non-overweight/obese subjects, which may be associated with under-reporting in these groups. Having specific dietary patterns may be as markers for an overall lifestyle, which may have a direct relationship to the health status of individuals. This study showed inconsistent associations between healthy lifestyle behaviors and healthy dietary patterns, and some healthy behaviours were associated with unhealthy dietary habits.

RQ 6) how do smoking cessation programmes using NRT affect body weight and shape, dietary habits and physical activity?

In the observational study (chapter 7) smoking cessation over a short time period, even using NRT, increased body weight particularly in women. It was also associated with increased WC in women. Although this study was small with high attrition rates, the amount of weight gain and increased central adiposity was considerable in free-living subjects. Confirmation by further research is required. In this study, the reported dietary intake was lower than the individual requirements and reduced slightly during the study, bot this change was not significant. Reported physical activity did not show any associations with the weight change, however the overall physical activity did not increase during the study.

In conclusion, lifestyle factors were associated with and clearly play an important role in both developing and preventing obesity and CVD risk factors. It is important to understand the associations of the important lifestyle factors on health outcomes. People with multiple unhealthy behaviours exist, and in most cases all healthy lifestyles would not happen simultaneously in one person, or even in a community or group. The benefits of one or more healthy lifestyle behaviours may diminish or be attenuated by other unhealthy lifestyles. Therefore in health promotion, special attention should be paid identifies people who have multiple unhealthy lifestyles focus on components of a healthy lifestyle, and to try to increase these healthy lifestyle components as much as possible at individual or community level.

8.3. Limitations of these studies

The main limitation of the present studies was the cross sectional study design of SHS. The SHS has been designed originally for measuring and monitoring health risk factors in Scottish population and using this data for other purposes may have some limitations especially searching the associations among the subgroups of the population. However the large sample size and relatively appropriate response rate overcomes this limitation to some extent. In a cross sectional study both exposures and outcomes are determined simultaneously for each subject, and therefore only associations can be examined, but not cause and effect. Longitudinal studies are ideally needed to confirm the causal relationships of the interaction between the lifestyle factors with overweight or obesity on CVD risk factors.

Dietary habits in SHS were assessed by a short FFQ indicating a limited number of food items. Therefore it was not possible to evaluate the detailed role of diet on the CVD risk factors.

Self reported physical activity with a questionnaire and misclassifications of subjects in terms of activity are other limitations of this study. Both obese and sedentary individuals are more likely to overestimate their habitual physical activity than those who are regularly active.

8.4. Possible future research topics

This work in this thesis has generated several further research questions for future. The association of long term smoking with a lower body weight in older adults is well recognized, but there are still many areas of this field that require further research. The mechanism underlying the process where smoking can reduce body weight is unclear as yet. Many

researchers have suggested different conclusions, however the role of the two main lifestyle factors, which are linked with energy balance, dietary habits and physical activity, are not clear. The possible research questions in this field are:

- How do specific components of energy balance contribute to body weight changes during smoking and smoking cessation?
- What is the role of body weight (overweight and obesity) and body shape in smoking initiation particularly in younger adults and teenagers?
- How long does it take that smoking can affect on body weight especially in the young people?
- What is the effect of smoking and smoking cessation on body fat distribution in a prospective study with detailed body composition analysis to assess body fat and fat free mass compartments?
- Can NRT prevent long-term weight gain after smoking cessation?
- Does smoking cessation increase the prevalence of overweight/obesity at a population levels?
- Does incorporating weight management into ongoing smoking cessation programmes, improve smoking cessation and reduce weight gain?
- Can habitual physical activity eliminate the health risk associated with obesity in obese subjects in a prospective study?

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Appendices

General questions

Name:_____ ID number: _____ Week: ____

Address: _____

Postcode: _____

Date of interview (dd/mm/yy): _____ Sex: Male ☐ Female ☐

Date of Birth (dd/mm/yy): _____ Place of birth: _____

Education: _____

Your current occupation: _____

Marital Status: Single ☐ Married ☐ Divorced ☐ Widowed ☐

-Have you been diagnosed with any medical conditions? Yes ☐ No ☐

-If yes, can you provide the details of the conditions? _____

Are you taking any drugs to treat your medical problems: Yes ☐ No☐

-If yes, can you provide the name of the drugs? _____

The following questions are about smoking status.

-What do you smoke nowadays? Cigar ☐ Cigarette ☐ Pipe ☐ Do not smoke ☐

-If you smoke nowadays, are you planning to quit in the next six months? Yes ☐ No ☐

-If yes, are you planning to quit in the next 30 days? Yes ☐ No ☐

- How many cigarettes a day do you usually smoke on weekdays: _____ at weekends: ____

-What is the tar level of the cigarettes you usually smoke: 1 high tar (over 18mg)
2 middle tar (15<18mg)
3 low to middle tar (10<15)
4 low tar (1<10mg)
5 varies

- How many cigars (if any) a day do you usually smoke on weekdays: ____ at weekends: ____

-How much tobacco (if any) do you usually smoke on weekdays (grams): _____
at weekends: _____

- How long have you been smoking regularly: In years: _____ In months: _____

- What was/ were the reason(s) to start smoking?
Stress ☐ Influence of peers ☐ To control your Weight ☐ Don't know ☐
Other: _____

- Why would you like to give up smoking (now)?
Because of current health problem ☐ To prevent a health problem in the future ☐
To live longer ☐ Family or peer pressure ☐ Economic reasons ☐
Other: _____

-Have you ever tried to give up smoking in the past? Yes ☐ No ☐

If yes, how many times: _____

- Have you made a quit attempt of 24 hours or more in the past year? Yes ☐ No ☐

- If yes, why did you fail to give up smoking in the past?

_____

_____

The following questions are about alcohol consumption.

- How often do you ever drink alcohol nowadays, including drinks you may brew or make at home?

Daily☐ Weekly ☐ Monthly ☐ Less often ☐ No, never ☐

- If you are an ex-drinker, when did you stop drinking? In years: _____ In months: _____

- If you are a regular or occasional drinker, how long have you been drinking:

In years: _____ In months: _____

- Could you provide an average weekly estimation of the type, frequency and amount of alcohol you consumed during last 4 weeks?

Type of alcohol	How often during last 4 weeks (Number per week)	How much on any day				
		Bottle	Pint	Small glass	Large glass	Shot
Normal strength (less than 6% alcohol) beer, lager, stout, cider, or shandy						
Strong (6% or more alcohol) beer or cider like Tennants, Extra, special Brew, Diamond White						
Spirits or liqueurs, such as gin, whisky, brandy, rum, vodka, advocaat or cocktails						
Sherry or martini including port, vermouth, Cinzano and Dubonnet						
Wine including Babycham and champagne						
Alcoholic soft drink (alcopop) such as Two Dogs or Acola						
Cooler/mixer/blender type drinks (eg. Bacardi Breezer, Castaway						
Any other types of alcohol:						

- Thinking now about all kinds of drink, how often have you had an alcoholic drink of any kind during the last 12 months? _____

The following questions are about weight, diet and physical activity

-How satisfied /dissatisfied are you about your current body weight?

Very satisfied ☐ Satisfied ☐ Dissatisfied ☐ very dissatisfied ☐

-If not satisfied, how much weight would you like to lose (kg)? _____

or to gain (kg)? _____

-How satisfied /dissatisfied are you about your current body shape?

Very satisfied ☐ Satisfied ☐ Dissatisfied ☐ very dissatisfied ☐

-Are you currently on a diet to lose weight? Yes ☐ No ☐

-If yes, what are you doing to lose weight?

Attending a slimming club ☐ a health center ☐ dieting on your own ☐

-Apart from stopping smoking do you plan to change any other aspect of your lifestyles?

a) how active you are? Yes ☐ No ☐ Do not know ☐

b) the foods that you eat? Yes ☐ No ☐ Do not know ☐

c) how much alcohol you drink? Yes ☐ No ☐ Do not know ☐

-Is there a type of foods would you like to eat more nowadays?

Yes ☐ No ☐

- If yes, please specify the type of food:

Sweet foods ☐ Salty foods ☐ savoury foods ☐ fatty foods ☐

The Three-Factor Eating questionnaire

Please answer the following questions by circling the number that is appropriate to you

1. I deliberately take small helpings as a means of controlling my weight.

1-definitely true 2- mostly true 3-mostly false 4- definitely false

2. I consciously hold back at meals in order not to gain weight.
1-definitely true 2- mostly true 3- mostly false 4- definitely false

3. I do not eat some foods because they make me fat.
1-definitely true 2- mostly true 3- mostly false 4- definitely false

4. How frequently do you avoid 'stocking up' on tempting foods?
1-almost never 2- seldom 3- usually 4- almost always

5. How likely are you to consciously eat less than you want?
1-Unlikely 2-slightly likely 3-moderately likely 4-very likely

6. On a scale of 1 to 8, where 1 means no restraint in eating (eating whatever you want, whenever you want it) and 8 means total restraint (constantly limiting food intake and never 'giving in'), what number would you give yourself?

7. When I smell a sizzling steak or a juicy piece of meat, I find it very difficult to keep from eating, even if I have just finished a meal.
1- definitely true 2- mostly true 3- mostly false 4- definitely false

8. Sometimes when I start eating, I just can't seem to stop.
1-definitely true 2- mostly true 3- mostly false 4- definitely false

9. Being with someone who is eating often makes me hungry enough to eat also.
1-definitely true 2- mostly true 3- mostly false 4- definitely false

10. When I see a real delicacy, I often get so hungry that I have to eat right away.
1-definitely true 2- mostly true 3- mostly false 4- definitely false

11. I get so hungry that my stomach often seems like a bottomless pit.
1-definitely true 2- mostly true 3- mostly false 4-definitely false

12. I am always hungry so it is hard for me to stop eating before I finish the food on my plate.
1-definitely true 2- mostly true 3- mostly false 4- definitely false

13. I am always hungry enough to eat at any time.
1-definitely true 2- mostly true 3- mostly false 4- definitely false

14. How often do you feel hungry?
1-only at mealtimes 2-sometimes between meals 3-often between meals 4- almost always

15. Do you go on eating binges though you are not hungry?
1-never 2- rarely 3- sometimes 4-at least once a week

16. When I feel anxious, I end myself eating.
1-definitely true 2- mostly true 3- mostly false 4- definitely false

17. When I feel blue, I often overeat.
1-definitely true 2- mostly true 3- mostly false 4- definitely false

18. When I feel lonely, I console myself by eating.
1- definitely true 2- mostly true 3- mostly false 4- definitely false

PHYSICAL ACTIVITY QUESTIONNAIRE

We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the **last 7 days**. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Think about all the **vigorous** and **moderate** activities that you did in the **last 7 days**. **Vigorous** physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. **Moderate** activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal.

PART 1: JOB-RELATED PHYSICAL ACTIVITY

The first section is about your work. This includes paid jobs, farming, volunteer work, course work, and any other unpaid work that you did outside your home. Do not include unpaid work you might do around your home, like housework, yard work, general maintenance, and caring for your family. These are asked in Part 3.

1. Do you currently have a job or do any unpaid work outside your home?

   ☐ Yes

   ☐ No → ***Skip to PART 2: TRANSPORTATION***

The next questions are about all the physical activity you did in the **last 7 days** as part of your paid or unpaid work. This does not include traveling to and from work.

2. During the **last 7 days**, on how many days did you do **vigorous** physical activities like heavy lifting, digging, heavy construction, or climbing up stairs **as part of your work**? Think about only those physical activities that you did for at least 10 minutes at a time.

   _____ **days per week**

   ☐ No vigorous job-related physical activity → ***Skip to question 4***

3. How much time did you usually spend on one of those days doing **vigorous** physical activities as part of your work?

   _____ **hours per day**
   _____ **minutes per day**

4. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the **last 7 days**, on how many days did you do **moderate** physical activities like carrying light loads **as part of your work**? Please do not include walking.

   _____ **days per week**

   ☐ No moderate job-related physical activity → ***Skip to question 6***

5. How much time did you usually spend on one of those days doing **moderate** physical activities as part of your work?

_____ **hours per day**
_____ **minutes per day**

6. During the **last 7 days**, on how many days did you **walk** for at least 10 minutes at a time **as part of your work**? Please do not count any walking you did to travel to or from work.

_____ **days per week**

☐ No job-related walking → ***Skip to PART 2: TRANSPORTATION***

7. How much time did you usually spend on one of those days **walking** as part of your work?

_____ **hours per day**
_____ **minutes per day**

PART 2: TRANSPORTATION PHYSICAL ACTIVITY

These questions are about how you traveled from place to place, including to places like work, stores, movies, and so on.

8. During the **last 7 days**, on how many days did you **travel in a motor vehicle** like a train, bus, car, or tram?

_____ **days per week**

☐ No traveling in a motor vehicle → ***Skip to question 10***

9. How much time did you usually spend on one of those days **traveling** in a train, bus, car, tram, or other kind of motor vehicle?

_____ **hours per day**
_____ **minutes per day**

Now think only about the **bicycling** and **walking** you might have done to travel to and from work, to do errands, or to go from place to place.

10. During the **last 7 days**, on how many days did you **bicycle** for at least 10 minutes at a time to go **from place to place**?

_____ **days per week**

☐ No bicycling from place to place → ***Skip to question 12***

11. How much time did you usually spend on one of those days to **bicycle** from place to place?

_____ **hours per day**
_____ **minutes per day**

12. During the **last 7 days**, on how many days did you **walk** for at least 10 minutes at a time to go **from place to place**?

_____ **days per week**

☐ No walking from place to place → ***Skip to PART 3: HOUSEWORK, HOUSE MAINTENANCE, AND CARING FOR FAMILY***

13. How much time did you usually spend on one of those days **walking** from place to place?

_____ **hours per day**
_____ **minutes per day**

PART 3: HOUSEWORK, HOUSE MAINTENANCE, AND CARING FOR FAMILY

This section is about some of the physical activities you might have done in the **last 7 days** in and around your home, like housework, gardening, yard work, general maintenance work, and caring for your family.

14. Think about only those physical activities that you did for at least 10 minutes at a time. During the **last 7 days**, on how many days did you do **vigorous** physical activities like heavy lifting, chopping wood, shoveling snow, or digging **in the garden or yard**?

_____ **days per week**

☐ No vigorous activity in garden or yard → ***Skip to question 16***

15. How much time did you usually spend on one of those days doing **vigorous** physical activities in the garden or yard?

_____ **hours per day**
_____ **minutes per day**

16. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the **last 7 days**, on how many days did you do **moderate** activities like carrying light loads, sweeping, washing windows, and raking **in the garden or yard**?

_____ **days per week**

☐ No moderate activity in garden or yard → ***Skip to question 18***

17. How much time did you usually spend on one of those days doing **moderate** physical activities in the garden or yard?

_____ **hours per day**
_____ **minutes per day**

18. Once again, think about only those physical activities that you did for at least 10 minutes at a time. During the **last 7 days**, on how many days did you do **moderate** activities like carrying light loads, washing windows, scrubbing floors and sweeping **inside your home**?

_____ **days per week**

☐ No moderate activity inside home → ***Skip to PART 4: RECREATION, SPORT AND LEISURE-TIME PHYSICAL ACTIVITY***

19. How much time did you usually spend on one of those days doing **moderate** physical activities inside your home?

_____ **hours per day**
_____ **minutes per day**

PART 4: RECREATION, SPORT, AND LEISURE-TIME PHYSICAL ACTIVITY

This section is about all the physical activities that you did in the **last 7 days** solely for recreation, sport, exercise or leisure. Please do not include any activities you have already mentioned.

20. Not counting any walking you have already mentioned, during the **last 7 days**, on how many days did you **walk** for at least 10 minutes at a time **in your leisure time**?

_____ **days per week**

☐ No walking in leisure time → ***Skip to question 22***

21. How much time did you usually spend on one of those days **walking** in your leisure time?

_____ **hours per day**
_____ **minutes per day**

22. Think about only those physical activities that you did for at least 10 minutes at a time. During the **last 7 days**, on how many days did you do **vigorous** physical activities like aerobics, running, fast bicycling, or fast swimming **in your leisure time**?

_____ **days per week**

☐ No vigorous activity in leisure time → ***Skip to question 24***

23. How much time did you usually spend on one of those days doing **vigorous** physical activities in your leisure time?

_____ **hours per day**
_____ **minutes per day**

24. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the **last 7 days**, on how many days did you do **moderate** physical activities like bicycling at a regular pace, swimming at a regular pace, and doubles tennis **in your leisure time?**

_____ **days per week**

☐ No moderate activity in leisure time → ***Skip to PART 5: TIME SPENT SITTING***

25. How much time did you usually spend on one of those days doing **moderate** physical activities in your leisure time?
_____ **hours per day**
_____ **minutes per day**

PART 5: TIME SPENT SITTING

The last questions are about the time you spend sitting while at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading or sitting or lying down to watch television. Do not include any time spent sitting in a motor vehicle that you have already told me about.

26. During the **last 7 days**, how much time did you usually spend **sitting** on a **weekday?**

_____ **hours per day**
_____ **minutes per day**

27. During the **last 7 days**, how much time did you usually spend **sitting** on a **weekend day?**

_____ **hours per day**
_____ **minutes per day**

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FOOD INTAKE QUESTIONNAIRE

Surname..... .... Subject ID

First Name(s) ....

Address .... Questionnaire No

.... Group Code

Phone No. .. .. Survey No

Male / Female

Date of Birth .... Date of Survey ....

The following questions are about the foods you USUALLY eat.
Please indicate the number of days per week that you eat each item on average. Ring the answer as in these examples:

If you eat the food every day, ring 7	(7) 6 5 4 3 2 1 F R
If you eat the food three days/week, ring 3	7 6 5 4 (3) 2 1 F R
If you eat the food once a fortnight, ring F	7 6 5 4 3 2 1 (F) R
If you rarely or NEVER eat the food, ring R	7 6 5 4 3 2 1 F (R)

PLEASE ANSWER EVERY QUESTION

BREAD

How often do you eat the following breads and how many slices do you have per day?

	No. days/week	*No. slices or rolls per day*	*Size of slices or rolls*
White or high fibre white	7 6 5 4 3 2 1 F R	.....	Thick/medium/thin Large/small
Brown or wheatgerm	7 6 5 4 3 2 1 F R	.....	Thick/medium/thin Large/small
Wholemeal/chapatis	7 6 5 4 3 2 1 F R	.....	Thick/medium/thin Large/small Chapatis
Bread rolls/crumpets	7 6 5 4 3 2 1 F R	.....	White or crumpets /brown/wholemeal
Crispbread, Ryvita or cream crackers	7 6 5 4 3 2 1 F R	.....	

How often do you eat jam, marmalade or honey on bread? 7 6 5 4 3 2 1 F R

1-4 5-8 9 10 11-12 13 14-15 16-17 18-19/162-163 20-21 22-23 24-25/164-165 26-28 29-31 32-34 35-37 38-39 40

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BREAKFAST CEREALS

How often do you eat the following cereals?

1. Cornflakes	7 6 5 4 3 2 1 F R	
2. Sugar Puffs, Special K, Ricicles, Rice Krispies, Coco Pops, Frosties or Crunchy Nut Cornflakes	7 6 5 4 3 2 1 F R	41-42
3. Muesli, Fruit n' Fibre or Cheerios	7 6 5 4 3 2 1 F R	43-44
4. Weetabix, Wheat Flakes or Shredded Wheat	7 6 5 4 3 2 1 F R	
5. Bran Flakes or Sultana Bran	7 6 5 4 3 2 1 F R	
6. Porridge or Ready Brek	7 6 5 4 3 2 1 F R	
7. All Bran	7 6 5 4 3 2 1 F R	
Other Cereal	7 6 5 4 3 2 1 F R	
Please specify brand/type	.....	
How many teaspoons of sugar/honey do you add?	.....	45
How often do you have wheat bran?	7 6 5 4 3 2 1 F R	46

MEATS

How often do you have the following meats?
Include all forms of each meat, eg use in stews, casseroles, lasagne, curry etc.

Beef (including beefburgers)	7 6 5 4 3 2 1 F R	47
Lamb	7 6 5 4 3 2 1 F R	48
Pork	7 6 5 4 3 2 1 F R	49
Bacon	7 6 5 4 3 2 1 F R	50
Ham	7 6 5 4 3 2 1 F R	51
Chicken or other poultry	7 6 5 4 3 2 1 F R	52
Canned meat (e.g., corned beef), paté or meat spread	7 6 5 4 3 2 1 F R	53
Sausages	7 6 5 4 3 2 1 F R	54
What type of sausages do you have?	1 Pork 2 Beef 3 Pork and Beef 4 Turkey 5 Low Fat	55
Meat pie/pastie/sausage roll/samosa - shop bought	7 6 5 4 3 2 1 F R	56
Meat pie/pastie/sausage roll/samosa - home made	7 6 5 4 3 2 1 F R	57
Liver/kidney/heart	7 6 5 4 3 2 1 F R	58
Do you usually eat the fat on meat?	Yes / No	59

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FISH

How often do you eat the following fish?

White fish (cod/haddock/plaice/fish fingers/fish cakes)	7 6 5 4 3 2 1 F R	60
Kipper/herring/mackerel/trout (including canned)	7 6 5 4 3 2 1 F R	61
Pilchards/sardines/salmon (including canned)	7 6 5 4 3 2 1 F R	62
Tuna (including canned)	7 6 5 4 3 2 1 F R	63

VEGETABLES & SAVOURY DISHES

How often do you have the following vegetables or dishes?

Potatoes - boiled or mashed	7 6 5 4 3 2 1 F R	64
Potatoes - jacket	7 6 5 4 3 2 1 F R	65
Chips - shop bought, 'oven/microwave chips' or hash browns	7 6 5 4 3 2 1 F R	66
Chips - homecooked	7 6 5 4 3 2 1 F R	67
Potatoes - roast	7 6 5 4 3 2 1 F R	68
Peas	7 6 5 4 3 2 1 F R	69
Other green vegetables, salads or tomatoes	7 6 5 4 3 2 1 F R	70
Carrots	7 6 5 4 3 2 1 F R	71
Parsnips, swedes, turnips or sweetcorn	7 6 5 4 3 2 1 F R	72
Baked beans	7 6 5 4 3 2 1 F R	73
Butter beans, broad beans or red kidney beans	7 6 5 4 3 2 1 F R	74
Lentils, chick peas or dahl	7 6 5 4 3 2 1 F R	75
Onions (cooked/raw/pickled)	7 6 5 4 3 2 1 F R	76
Spaghetti, other pasta or noodles	7 6 5 4 3 2 1 F R	77
Rice (NOT pudding rice)	7 6 5 4 3 2 1 F R	78
Quiche	7 6 5 4 3 2 1 F R	79
Pizza	7 6 5 4 3 2 1 F R	80
Vegetable pie/pasty/samosa	7 6 5 4 3 2 1 F R	81

BISCUITS, CAKES & PUDDINGS

How often do you eat the following items?

Digestive biscuits/plain biscuits	7 6 5 4 3 2 1 F R	82
Other sweet biscuits	7 6 5 4 3 2 1 F R	83
Chocolate, e.g., Galaxy, Mars Bar, Twix, KitKat	7 6 5 4 3 2 1 F R	84

Sweets, e.g., fruit gums, pastilles, mints	7 6 5 4 3 2 1 F R	85
Crisps/savoury snacks, e.g., Quavers, tortilla chips	7 6 5 4 3 2 1 F R	86
Nuts	7 6 5 4 3 2 1 F R	87
Ice cream, iced dessert, fool, mousse or trifle	7 6 5 4 3 2 1 F R	88
Low fat yogurt	7 6 5 4 3 2 1 F R	89
Low calorie yogurt e.g., Shape	7 6 5 4 3 2 1 F R	90
Other yogurt/fromage frais, e.g., thick & creamy	7 6 5 4 3 2 1 F R	91
Fruitcake/sponge cake/sponge pudding - shop bought	7 6 5 4 3 2 1 F R	92
Fruitcake/sponge cake/sponge pudding - homemade	7 6 5 4 3 2 1 F R	93
Fruit tart/jam tart/doughnut/Danish pastry - shopbought	7 6 5 4 3 2 1 F R	94
Fruit tart/jam tart - home made	7 6 5 4 3 2 1 F R	95
Milk pudding e.g., rice/tapioca/macaroni	7 6 5 4 3 2 1 F R	96

What type of milk do you use for milk pudding? 97

1 Ordinary/whole
2 Semi-skimmed
3 Skimmed
4 Canned milk pudding - ordinary
5 Canned milk pudding - low fat

FRUIT

How often do you have fruit canned in syrup?	7 6 5 4 3 2 1 F R	98
How often do you have fruit canned in juice?	7 6 5 4 3 2 1 F R	99
How many apples do you have per week?	.....	100
How many pears do you have per week?	.....	101
How many oranges/tangerines/satsumas/clementines/ grapefruit do you have per week?	.....	102
How many bananas do you have per week?	.....	103

EGGS & MILK PRODUCTS

How many eggs do you usually eat per week? 104-105

Roughly how much milk do you drink in a day in tea/coffee/milky drinks/cereals?

1 None
2 Half a pint or less
3 Between half and one pint
4 One pint or more

106

What type of milk do you have?

1 Whole
2 Semi-skimmed
3 Skimmed
4 More than one type

107

How much cream do you use per week?

(1 tablespoon=20g; small carton=150g; large carton=300g) .....g

108-110

How much cheese (excluding cottage cheese) do you usually eat per week? .....g

111-113

(Suggestion: divide amount bought for household by number of people in house)

How often do you eat cottage cheese? 7 6 5 4 3 2 1 F R

114

FATS

What do you usually spread on bread?

1 Butter
2 Polyunsaturated margarine/spread
3 Other soft marg/spread (tub) (not olive spread)
4 Hard margarine (block)
5 Low fat spread - polyunsaturated
6 Low fat spread - other
7 Lard, dripping, solid vegetable oil
8 Very low fat spread (25% fat)
9 Olive oil spread
0 Bread eaten dry

115-116

Brand name & description on packet/tub .....

How much butter/margarine/spread do you usually eat per week?.....g

117-119

(One block or small tub = 250g. Spread on one slice of bread: Thinly=5g; Medium=8g; Thickly=13g.)

How often do you have food that is fried?
(fish/onions/mushrooms/tomatoes/eggs) 7 6 5 4 3 2 1 F R

120

What types and BRANDS of fat do you use in cooking?

Frying	.....	solid/liquid
Chips	.....	solid/liquid
Roast Potatoes	.....	solid/liquid/eaten out
Home made cake	.....	
Home made pastry	.....	

121
122
123
124
125

DRINKS

How many cups of tea do you have per day? .....

126-127

How many teaspoons of sugar/honey per cup? .....

128

How many cups of coffee do you have per day? .....

129-130

How many teaspoons of sugar/honey per cup? .....

131

How often do you have fruit juice/squash/fizzy drinks (NOT low calorie)? 7 6 5 4 3 2 1 F R

132

Which of these do you usually have?

1 Natural Juice
2 Squash
3 Fizzy Drink
4 More than one

133

How often do you have drinks containing alcohol? 7 6 5 4 3 2 1 F R

134

When you drink, how many do you have? .....

Please specify how many drinks of each type per occasion:

Beer/lager/stout/cider	Number of pints	.....
Wine	Number of glasses	.....
Sherry/port/vermouth	Number of glasses	.....
Spirits/liqueurs	No. of single measures	.....

135-136
137
138
139

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HEIGHT, WEIGHT & ACTIVITY

What is your height? ..... ft ..... ins **OR** .....cm

What is your weight? ..... st ..... lbs **OR** .....kg

How physically active is your occupation?

1 Not very active
2 Moderately active
3 Very active
4 Not working

How physically active is your leisure time?

1 Not very Active
2 Moderately active
3 Very active

Questions for women only..

Are you pregnant? Yes / No

Are you breast feeding? Yes / No

ADDITIONAL QUESTIONS

How often do you have..

Dishes made with TVP (soya mince) or Quorn? 7 6 5 4 3 2 1 F R

Vegetarian sausages / Vegetarian burgers? 7 6 5 4 3 2 1 F R

Are there any other foods that you eat regularly, but which are not recorded in the questionnaire? Yes / No

If Yes, please state each food and how often you usually eat it

Food	Frequency

Diet Code

140-143 . 144-147

148

149

150

151

152

153

154

155-157

158-160

161

27th July 2004

NHS
Lanarkshire

Mr Mehdi Akbartabartoori
Postgraduate Student
University of Glasgow
Human Nutrition, Queen Elizabeth Building,
Glasgow Royal Infirmary, Glasgow
G312ER

Dear Mr. Akbartabartoori

Full title of study: The effect of smoking cessation on energy balance dietary patterns and physical activity
REC reference number: 04/S1001/32
Protocol number: 1

Thank you for your letter of 22nd July 2004 responding to the Committee's request for further information on the above research and submitting revised documentation.

The further information has been considered on behalf of the Committee by the Chairman.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised.

Conditions of approval

The favourable opinion is given provided that you comply with the conditions set out in the attached document. You are advised to study the conditions carefully.

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document Type: Application
Version: 3
Dated: 28/06/2004
Date Received: 02/07/2004

Document Type: Investigator CV
Version: 1
Dated: 02/07/2004
Date Received: 02/07/2004

Document Type: Protocol
Version: 1
Dated: 24/06/2004
Date Received: 02/07/2004

Document Type: Covering Letter
Version: 1
Dated: 01/07/2004
Date Received: 02/07/2004

Document Type: Copy of Questionnaire
Version: 1
Dated: 01/07/2004
Date Received: 02/07/2004

Document Type: Sample Diary/Patient Card
Version: 1
Dated: 02/07/2004
Date Received: 02/07/2004

Document Type: Participant Information Sheet
Version: 2
Dated: 21/07/2004
Date Received: 22/07/2004

Document Type: Participant Consent Form
Version: 1
Dated: 02/07/2004
Date Received: 02/07/2004

Document Type: Other
Version: 1
Dated: 02/07/2004
Date Received: 02/07/2004

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

REC reference number:04/S1001/32 Please quote this number on all correspondence

Yours sincerely,

DR. D. GORDON
CHAIRMAN

Enclosures *Standard approval conditions [SL-AC1 or SL-AC2]*