

Associations between self-reported dietary intake in randomly selected men and women in relation to the metabolic syndrome and the individual components of the syndrome.

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## Abstract

Master Thesis, Programme in Medicine

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Introduction: The metabolic syndrome (MetS) is a collection of five medical conditions: central obesity, increased triglycerides, reduced HDL-cholesterol, hypertension and increased fasting plasma glucose. The components of the MetS cover a subset of known cardiovascular risk factors. Several additional adverse health effects are associated with the MetS for instance increased risk to develop type 2 diabetes, Alzheimer disease and cancer. A vast body of scientific research have earlier investigated the relationship between diet and the MetS; but the conclusions are still inconsistent.

Aim: To investigate the impact of self-reported dietary intake, in a randomly selected population of men and women, on the MetS and individual components of the syndrome.

Methods: A cross-sectional population-based study, of randomly selected adult men and women, was used to analyse the associations between self-reported dietary intake and the MetS and individual components of the syndrome.

Results: In men sugar (mono- and disaccharides) intake was significantly positively associated with the MetS. The strongest associations included sugar intake being negatively associated with HDL-cholesterol in men. In women total carbohydrate intake was negatively associated with HDL-cholesterol and fasting blood glucose. In men polyunsaturated fatty acids was negatively associated with systolic and diastolic blood pressure.

Conclusion: In men, simple carbohydrates, was positively associated with the MetS. Simple and total carbohydrates were negatively associated with HDL-cholesterol in men and women, respectively. Polyunsaturated fatty acids were negatively associated with both systolic and diastolic blood pressure. The results from this cross-sectional study are in concordance with present dietary recommendations to prevent common disease risk factors: to limit sugar intake and to increase intake of polyunsaturated fatty acids. The difference, in influence of diet on the MetS, between sexes is intriguing and demand further research on whether men and women are affected differently in various diet composition.

Key words: metabolic syndrome, dietary intake, randomly selected sample, cross-sectional study.

## **Introduction**

### *The metabolic syndrome*

The metabolic syndrome (MetS) is a collection of five medical conditions: central obesity, increased triglycerides, reduced HDL-cholesterol (HDL-C), hypertension and increased fasting plasma glucose (Table 1). An individual with three or more of these components is considered having the MetS. In a consensus statement, from the International Diabetes Federation (IDF) (1), on the MetS, published in 2006 (updated as a joint scientific statement in 2009 (2)), the IDF state that obesity plays a crucial role in the increasing prevalence of the MetS. The components of the MetS cover a subset of known cardiovascular risk factors. Presence of the MetS has been associated with increased risk for coronary heart disease (CHD)(3, 4). Several observational studies have suggested an increased risk of cardiovascular disease (CVD) and cardiovascular as well as all-cause mortality in persons with the MetS (5-8). In addition to predict risk, CVD-studies have suggested that individuals with the MetS has as much as a five-fold risk of developing type 2 diabetes (T2DM) (4, 9) and it has been suggested that the greatest benefit of the MetS is to use it as a risk-estimate of T2DM (10). All studies are not solely positive and criticism has been raised against the clinical relevance of the MetS. Existing competing algorithms to estimate risk of CVD and T2DM i.e. Framingham Risk Score (11) and Diabetes Risk Score (12) are suggested to be superior.

Yet, the list of adverse effects associated with the MetS is still growing and spans from chronic kidney disease, non-alcoholic fatty liver disease (NAFLD), Alzheimer disease, cancer and worsening of health-related quality of life (13-17).

The IDF acknowledge that there are many unanswered questions related to the MetS, one of the greater questions being the aetiology of the MetS.

### *Diet and the metabolic syndrome*

Historically overweight and obesity have been argued to be the most important risk factors for

the MetS. The prevalence and incidence of T2DM, dyslipidaemia and CVD increase with increasing BMI, but there is heterogeneity in presence of illness, between individuals with similar BMI values. It has been suggested that more than 20% of normal-weight individuals are metabolically unhealthy, while more than 30% of obese adults are metabolically healthy (18). This has led researchers to the conclusion that obesity rather being a marker of metabolic dysfunction in contrast of being its cause (19). An obvious question that comes with all diseases closely related to an unhealthy metabolism is what impact various dietary nutrients have on the disease? Exploring dietary nutrients in relation to disease is an appealing thought, but does at the same time present great challenges. Rigorous scientific research providing causal evidence (i.e. randomised controlled trials, RCTs) is expensive and requires extensive periods of time which makes these studies hard to fund and implement in great scale. Observational studies are easier to accomplish with sufficient material and time but do not provide causal inference.

The MetS is closely related to insulin resistance; in fact *The Insulin resistance syndrome* is one of several earlier names the MetS has been referred to as (1). In a study by Samaha et al. a carbohydrate-restricted (<30 grams/day) diet was shown to increase insulin sensitivity and glycaemic control in 132 severely obese subjects with high prevalence of T2DM and the MetS (20). This effect remained even after adjustment was made for the weight loss achieved. Other potential effects of a carbohydrate-restricted diet are illustrated in a study by Volek et al. where 12 healthy normal-weight men demonstrated a significant decrease of 34% in serum insulin concentration after six weeks on a carbohydrate-restricted (8 percent of energy intake, E%) diet (21).

It is an intriguing thought that a carbohydrate-restricted diet would reduce the postprandial glucose level and insulin response and thereby be advantageous for improving components of the MetS, with the greatest effects seen in triglyceride and HDL-C levels suggestively (22).

This has also been suggested through observational studies where a high carbohydrate intake was associated with adverse effects on the components of the MetS (23-25).

Several RCTs, ranging from six weeks to two years follow-up, have tested the hypothesis that restricting total carbohydrate intake compared to restricting total fat intake will have a beneficial outcome regarding the MetS variables (20, 26-32), especially in lowering triglyceride concentration and increasing HDL-C concentration. Other studies have not shown any differences regarding the impact of altering the macronutrient composition on the MetS (33, 34), some rather recognizing the weight loss achieved by any diet as the most important positive influence on the MetS. Further aspects have been added to the discussion whether the definition of a carbohydrate-restricted diet is congruently defined between studies (35). In a Cochrane report published in 2007 the authors suggest that a diet with a low glycaemic index (LGI) or load has favourable effects concerning the lipid profile compared to a diet with a higher glycaemic index (HGI) or load. This fact might propose the quality of the carbohydrates consumed is more important than the total carbohydrate intake. With this in mind a LGI diet might be paralleled by a decrease in total carbohydrate intake (36), but not always.

In respect of the discussion regarding weight loss and the MetS, one aspect of diet and weight loss in relation to metabolic risk factors is shown by Krauss et al. (37). In an interpretation of the study by Krauss et al., Feinman and Volek suggest that there was a significant improvement in metabolic profile on a carbohydrate-restricted diet when compared to a low-fat (LF) diet, even in the absence of weight loss. On a LF diet, improvement in metabolic profile was only seen when accompanied with weight reduction (38).

The Swedish Council on Health Technology Assessment (SBU) (22) note in their systematic comparison on dietary advice for obese individuals that a low carbohydrate diet compared to a low fat diet results in increased HDL cholesterol without negative effects on LDL cholesterol,

at least during the first six months of intervention. The effect was seen in both a moderate low carbohydrate diet (carbohydrate intake <40E% ) as well as a very low carbohydrate diet (<20 E%). The very low carbohydrate diet also indicated improved glucose values in the short term along with reduced triglycerides (22).

These findings raise intriguing questions regarding the associations between habitual carbohydrate intake and metabolic risk factors in randomly selected adult men and women.

## **Aim**

The aim of this study is to examine the impact of energy and macronutrient intake on components of the MetS and the MetS as such, in randomly selected men and women.

## **Subjects and Methods**

### *Study population*

The study population was the reference group to the Swedish Obese Subjects (SOS) study (39). The Swedish Obese Subjects reference study (40) included a total of 1135 subjects, 524 men and 611 women, 37-61 years of age. The participants were randomly selected, from a population registry, living in the cities of Mölndal and Örebro, Sweden. The examinations were performed between August 1994 and December 1999. The participation rates were 53.7% for men and 57.6% for women (40).

### *Habitual food intake*

A, 51 questions, semi-quantitative dietary questionnaire on habitual food and beverage intake during the last 3 months was completed by the subjects.

The questionnaire was developed within the SOS study. To validate the dietary questionnaire Lindroos et al. (41) carried out a study including 45 obese and 19 non-obese subjects, aged 21-61 years. The validation process of the questionnaire included a 4-day food registration where the subjects kept food records for four consecutive days, after filling out the dietary

questionnaire. An open-circuit indirect calorimetry system was used to determine the subjects basic metabolic rate (BMR), the calculation included oxygen consumption, carbon dioxide production and urinary nitrogen excretion. The 24h energy expenditure (24EE) was estimated based on each individuals BMR along with self-reported physical activity. The 24EE was later compared with the calculated energy intake from the diet records and the questionnaire. Mean energy intake from the dietary questionnaire in comparison with the 24EE proposed a 5% (non-significant) discrepancy in normal weight individuals and a 4% non-significant difference in the obese (41). The authors concluded that the use of the dietary questionnaire for recording energy intake was as valid in the obese as in normal weight individuals. Looking at selected nutrients (e.g. protein, total carbohydrate and total fat) the nutrient estimates from the questionnaire were all significantly correlated compared to the food records, when adjustment was made for total energy intake (41).

### **Statistical methods**

Initially, to visualise the basic characteristics of the study population, Student's t-test was used to compare the population according to gender. Prevalence of the MetS was also calculated. Values are presented as means (s.d.) if not stated otherwise. Dietary variables (e.g. carbohydrate, fat and protein) were analysed as continuous absolute variables (grams) as well as relative measures, E%. Due to statistical reasons and because of the simpler representation of data continuous absolute variables are presented throughout the study. Men and women were analysed separately throughout the study. To explore correlation between continuous dietary variables and components of the MetS, Pearson's correlation was used. Self-reported dietary data is accompanied with a non-negligible amount of uncertainty due to recall bias. One way to deal with this fact is to categorize continuous variables into ordinals (i.e. comparing groups with increasing intake of a specific dietary nutrient). Hence dividing the study subjects into quartiles in respect of dietary nutrients was used in this study. When



expecting great measurement error of the dietary variables one way to increase the contrast between the groups of “lowest” and “highest” intake of a specific macronutrient, is to pool quartiles into “tertiles” (i.e. low intake equals 1<sup>st</sup> quartile, medium intake equals 2<sup>nd</sup> plus 3<sup>rd</sup> quartile, high intake equals 4<sup>th</sup> quartile). Consequently three groups were formed for each individual macronutrient. Throughout the report these groups are referred to as quartiles. To examine correlation between quartiles and the MetS variables rank correlation (Spearman’s correlation) was used. This procedure is customary when analysing correlation for ordinal data. To illustrate the correlation between continuous macronutrients as well as quartiles and variables of the MetS scatterplots as well as boxplots were drawn. To utilize the statistically reinforced contrast between 1<sup>st</sup> quartile (low intake) and 4<sup>th</sup> quartile (high intake) Student’s t-test was performed to compare means of the variables of the MetS according to various dietary data. Further analyses included bivariate and multivariate linear regression to examine the impact of dietary factors on the individual MetS variables. In order to visualise the correlation between macronutrient quartiles (absolute measure, g/day) and prevalence of the MetS, crosstabs and Chi<sup>2</sup>-test were performed. To further investigate any potential correlation between macronutrients and prevalence of the MetS, logistic multivariate regression was performed. During both linear and logistic regression adjustment was made for age, BMI, physical activity (during leisure time) and total energy intake (kcal/day).

Analyses were performed with SPSS Statistics Version 22 for Windows.

### **Ethics**

As expressed by Larsson et al. (40), “the ethical committee of Göteborg University approved the protocol and informed consent was obtained from all subjects before the examinations.”

### **Results**

The clinical criteria specifying the MetS are described in Table 1.

Table 2 displays descriptive characteristics of the study population. Data are presented for the total group as well as for men and women, respectively. The population was comprised of 46.2% men and 53.8% women. Men and women were of similar age, while men had a statistically significant higher BMI, blood pressure and self-reported total energy intake compared to women. In relative terms men reported lower E% carbohydrate intake but higher E% fat intake than women (Table 2). A statistically significant higher prevalence of the MetS was found in men compared to women (Table 2).

Table 4 (a and b) displays rank correlation coefficients between macronutrient quartiles and the variables of the MetS. Table 5 (a and b) displays the comparison of means of the variables of the MetS between the 1<sup>st</sup> quartile (low intake) and 4<sup>th</sup> quartile (high intake) according to various macronutrients. To increase the readability and simplifying presentation of the results of this study the results (Table 6a, 6b, 7a and 7b) from analysing macronutrients (continuous) and their correlation with the components of the MetS, are presented in the appendix. These results are depicted and described in the report but results from analysing quartiles (Table 4a, 4b, 5a and 5b) are stressed. Table 6 (a and b, appendix) displays Pearson's correlation coefficients between macronutrients and each of the variables of the MetS while Table 7 (a and b, appendix) demonstrate linear multivariate regression of macronutrients and variables of the MetS.

Figures 1 through 3 visualise the strongest rank correlation coefficients ( $r < -0.120$ ) as presented in Table 4b. Figures 4 through 7 (appendix) visualise the strongest Pearson's correlation coefficients ( $r < -0.120$ ,  $r > 0.120$ ) as presented in Table 6a and 6b (appendix).

### *Carbohydrate intake*

In men, those with the highest sugar (mono- and disaccharides are referred to as sugar throughout the entire report) intake (4<sup>th</sup> quartile) also had significantly higher prevalence of

the MetS compared with lower intakes ( $p=0.032$ ). No such association was found among women (Table 3). In men, prevalence of the MetS in the 1<sup>st</sup> quartile (low intake) was 19.2%, in 2<sup>nd</sup> plus 3<sup>rd</sup> quartiles (medium intake) 18.2% and in the 4<sup>th</sup> quartile (high intake) 29.6%. No other self-reported macronutrient intake in either sex was significantly associated with the prevalence of the MetS. In women, carbohydrate intake indicated a trend toward significant association with prevalence of the MetS ( $p=0.067$ ), not shown.

In women, carbohydrate intake was negatively associated with HDL-C and fasting glucose, using rank correlation (Table 4a) and comparing means of 1<sup>st</sup> and 4<sup>th</sup> quartile (Table 5a). In men, sugar intake was negatively associated with HDL-C (Table 4b). Comparing means of 1<sup>st</sup> and 4<sup>th</sup> quartile in men, sugar intake was positively associated with triglyceride level and negatively associated with HDL-C (Table 5b). Pearson correlation (Table 6a, appendix), showed that total carbohydrate intake was positively associated with triglyceride level and negatively associated with HDL-C, in men. In women, total carbohydrate intake was negatively associated with fasting blood glucose using Pearson correlation (Table 6a, appendix). Analysing sugar intake showed a positive association with BMI and triglyceride level along with a negative association with HDL-C, in men (Table 6b, appendix). Total fibre intake was positively associated with triglycerides, in men (Table 6 b, appendix).

In men, employing linear regression on the data at hand, the results were concordant in the negative correlation between carbohydrate intake and HDL-C. Carbohydrate ( $p=0.002$ , Table 7a, appendix) and sugar ( $p=0.018$ , Table 7b, appendix) intake was negatively associated with waist circumference, in men. Sugar intake also showed a negative association with HDL-C, in men (Table 7b, appendix). Dietary fibre was positively associated with triglycerides in men (Table 7b, appendix). However, residual plots rejected the results because the triglyceride variable did not meet the criteria of normal distribution. This fact was true for all results that

included triglycerides in the linear multivariate regression analysis (Table 7a and 7b, appendix).

### *Fat intake*

When quartiles of total fat intake were examined with rank correlation, a negative association with systolic blood pressure (SBP) was found, in men. In women, total fat intake was negatively associated with diastolic blood pressure (DBP) (Table 4a). When quartiles of subgroups of fat intake were analysed poly-unsaturated fatty acids (PUFA) were negatively associated with SBP and DBP, in men. In women, PUFA were negatively associated with HDL-cholesterol and DBP (Table 4b). In women, mono-unsaturated fatty acids (MUFA) were negatively associated with DBP in women using rank correlation (Table 4b). Total fat intake was negatively associated with DBP, in women, when means of 1<sup>st</sup> and 4<sup>th</sup> quartiles were compared (Table 5a). PUFA were negatively associated with SBP and DBP in men (Table 5b). In women, PUFA were negatively associated with HDL-C and DBP (Table 5b). MUFA intake presented a negative correlation with DBP when means of 1<sup>st</sup> and 4<sup>th</sup> quartiles were compared (Table 5b). PUFA also displayed a negative correlation with HDL-C (Table 5b). Total fat intake (continuous) was negatively associated with both SBP and DBP in men (Table 6a, appendix). When subgroups of fat were analysed the Pearson's correlations remained and were strengthened for PUFA, in men. PUFA were also negatively associated with HDL-cholesterol in women. MUFA exhibited a negative association with SBP along with a positive association with triglycerides, in men. In women, MUFA were negatively associated with DBP (Table 6b, appendix).

Dividing fat intake into subgroups, linear multivariate regression did strengthen the results of PUFA being negatively associated with both DBP and SBP, in men. PUFA also showed a weak positive association with waist circumference, in men (Table 7b, appendix).

### *Protein intake*

Protein intake was negatively associated with HDL-C in women (Table 5a).

In women, linear multivariate regression showed a negative association between protein intake and triglycerides (Table 7a, appendix).

### *Total energy intake*

In women, total energy intake was negatively associated with HDL-C using rank correlation (Table 4a). This was also true when comparing means of 1<sup>st</sup> and 4<sup>th</sup> quartiles, in women (Table 5b).

Total energy intake was positively associated with triglycerides, in men, using Pearson correlation ( $p < 0.05$ ) (Table 6a, appendix). This correlation did not remain with rank correlation ( $p = 0.967$ ) (Table 4a).

Total energy intake was positively associated with triglycerides, in men, but did not fulfil the criteria of normally distributed residuals as stated earlier (Table 7b, appendix).

## **Discussion**

In this study on a randomly selected population of adult middle-aged men and women, self-reported energy and macronutrient intake was analysed in relation to the prevalence of the metabolic syndrome as well as the separate components included in the criteria of the syndrome. Utilizing the contrast of analysing lowest and highest quartiles of various dietary variables and focusing on the strongest correlations the main results of this study are, in men, self-reported sugar intake was positively associated with triglyceride level and negatively associated with HDL-C. In women total carbohydrate intake was negatively associated with HDL-C and fasting B-glucose. In men, PUFA was negatively associated with DBP and SBP. In women, total fat, MUFA and PUFA were negatively associated with DBP. PUFA also

showed a negative association with HDL-C. In women, protein intake and total energy intake displayed a negative association with HDL-C.

Carbohydrate intake in general was negatively associated with HDL-C in women; in men this association was true for sugar intake. In men, sugar intake was also positively associated with triglyceride level. These associations are in resemblance with earlier studies performed with other research designs (27, 32, 42).

In women, carbohydrate intake was negatively associated with fasting B-glucose; these results are somewhat surprising, as one would expect a positive association between carbohydrate intake and glucose level. This might reflect the well-known fact that some foods are notoriously underreported. More specifically, often underreported foods are those that are commonly known that one should not eat, for example, sweets, chocolate, cookies, buns and ice-cream, all with high carbohydrate content. Underreporting of the mentioned kinds of foods might lead to the paradoxical association seen in this study because the underreporting may be greater in those consuming the highest quantity of these foods, compared with groups with less frequent consumption of these foods. In this study no associations between sugar intake and components of the MetS were seen in women (as opposed to men), this along with the known fact that women more frequently underreport dietary intake compared to men (43) might explain these differences.

The negative association between total fat intake and blood pressure might reflect the impact MUFA and PUFA have on blood pressure. Indeed the negative associations in this study strengthened when PUFA alone was analysed in relation to blood pressure. PUFA or more specifically fish oil (predominantly eicosapentaenoic (EPA) and docosahexaenoic acid (DHA)) has been found to lower blood pressure levels as well as decreasing the risk of coronary death and total mortality (44, 45). The blood pressure lowering effect is evidently

more pronounced in older and hypertensive subjects (44). Hypertension is a major risk factor for cardiovascular events so the results of this study could be considered in line with previous scientific research. The negative association between MUFA and DBP in women is to some extent consistent with the scientific literature. A Mediterranean diet rich in olive oil (MUFA) has been argued to have a potential role in lowering blood pressure (46). Saturated fatty acids (SFA) presented no association with adverse metabolic outcomes in the current study; this is also in accordance with earlier studies. Several meta-analyses have previously shown SFA neutral to all-cause mortality, CVD, total CHD, fatal CHD, ischaemic stroke and T2DM (47-50). To identify deleterious effects from dietary nutrients on health, a long time period of observation is needed. This is provided by observational studies, which in turn only provide associations. Randomised controlled clinical trials are needed to provide causal evidence of the obtained associations. With RCTs much of the discussion concerning the results are whether differences of the study population is of importance (e.g. primary or secondary prevention). This is indeed an area of scientific research of much controversy. It has been proposed changing SFA for PUFA is beneficial from a cardiovascular health perspective but this is still debated (42, 51).

In women, protein intake was negatively associated with HDL-C, at least when analysing quartiles of protein intake (Table 5a). This finding contradicts earlier studies where subjects consuming a high-protein diet either maintains higher circulating levels of, or shows an improvement in, HDL-C (31, 52). The fact that this association was not seen with rank correlation (Table 4a) weakens this association.

Total energy intake was negatively associated with HDL-C in women (Table 4a).

Epidemiological studies have previously shown that an increase in total carbohydrate intake was paralleled with an increase in total energy intake; this covariance might account for the present association (53). The rank correlation was significant though in the lower range, in

this study correlations approximate to  $\pm 0.1$ , and greater, was considered being of interest; which in this case weaken these results.

It has been proposed that total carbohydrate intake *per se* should not be considered in association with the MetS but rather subgroups i.e. glucose and fructose (19, 54, 55). In the current study information about sugar (mono- and disaccharides i.e. glucose, fructose, galactose, sucrose and lactose were analysed together in this study) intake was available but individual subgroups were not. Access to the data about specific subgroups of macronutrients would potentially strengthen the associations of this study and make it possible to further investigate the relationship between habitual food intake and the MetS.

### *Methodological considerations*

In this study, men and women were analysed separately with respect to the MetS and energy and macronutrients. This approach can be discussed. Analysing men and women collectively would result in a greater number of measure points in one group and would broaden the range of data points for the various dietary variables. With a larger set of data one could expect correlations to strengthen which would be preferable. With respect to this the approach chosen has its advantages. First off the clinical criteria of the MetS do distinguish between men and women on two measures (Table 1); waist circumference and HDL-C, which have sex-dependant cut-off limits. This makes analyses of correlation between each individual component of the MetS and dietary variables harder to interpret. Men and women do differentiate to a greater or lesser extent in respect of habitual dietary intake on all macronutrients analysed (Table 2). This fact makes the analysis of quartiles of dietary variables and variables of the MetS less straight forward if men and women were to be analysed as a single group.



Analysing various dietary factors impact on health related variables is indeed a difficult task to take on. One could argue about which is the better way to present and analyse intake of different macronutrients. In this study presenting macronutrients as absolute continuous variables, in contrast to percent of total energy intake, was chosen. The fact that absolute measures are easier to visualise and calculations are easier to perform affected this decision. When performing multivariate regression the question of co-variation between a specific macronutrient and total energy intake has to be considered. Analysing a macronutrient and adjusting for total energy intake, which are not independent variables, can be discussed. Willet et al. have in earlier studies argued these adjustments can still be made (56).

Due to the great uncertainty of self-reported dietary data correlations often exhibit low Pearson and Spearman correlation coefficients. A Pearson or Spearman correlation coefficient approximate to  $\pm 0.1$ , or greater, was in this study considered being of interest.

### *Limitations*

The data set used in this study does not contain any information about medication that might impact variables of the MetS. The most commonly used drugs targeting dyslipidaemias are statins, their main actions are considered lowering LDL-cholesterol and total cholesterol, which are not part of the MetS (57). Therefore omitting information on the use of statins in this study might not impact the results to a great extent. Alternative medications that could alternate variables of the MetS are fibrates and nicotinic acid. These are the most commonly used drugs that reduce triglycerides and raise HDL-C. The use of high dose Omega-3 fatty acids does impact triglycerides hence this information would contribute to the study (2).

Furthermore there is no available information on antihypertensive drug treatment, in patients with previous hypertension, which is an alternative to fulfilling the criteria of hypertension of the MetS. Since antihypertensive drug treatment is rather frequent in the population this data would most certainly influence the prevalence of the MetS. An alternative indicator of the

elevated fasting glucose-criteria is drug treatment of elevated glucose; the same reasoning goes for these drugs.

Simultaneous disease could indeed affect the MetS. Patients with T2DM will commonly fulfil the criteria of the MetS, naturally omitting all drug treatments used for these diseases will underestimate the prevalence.

Smoking is a known cardio metabolic risk factor and available smoking status should preferably have been controlled for when performing various types of regression. The fact that there was no available information on the individual level regarding smoking is a great weakness of this study. Intake and use of alcohol is also a potential metabolic risk factor, thus in an optimal estimation of the impact of self-reported dietary intake on the MetS, this should have been taken into account. Information about alcohol intake was registered in the SOS reference study (40) but was not used in the analyses of this study due to statistical reasons. This is another limitation of this study.

In the SOS reference study (40) participants and non-participants were compared in respect of smoking habits. Only non-participants from the Mölndal part of the study 1994-1996 were examined, through telephone interview. Non-participants were significantly more often smokers and less often ex-smokers compared with participants. This fact may underestimate the impact smoking has on the cardio metabolic risk and the prevalence of the MetS in the general population.

Participants and non-participants did not significantly differ in respect of medication nor the prevalence of disease, which consolidate the possibility of extrapolating the results to the general population.

In this study there was no information about the ethnicity of the participants. In the current definition of the MetS different cut-points of waist circumference are based on ethnicity. This may or may not influence the outcome of this study.

Furthermore, the validity of the dietary questionnaire might be questioned when used in the present study. In the validation process normal weight and obese individuals were compared. In the present study men and women are compared and overweight individuals are also included in the randomly selected study group, which were not included in the validation process. It has been proposed that women generally have a greater level of underreporting dietary intake compared to men (43). In the validating study selected nutrients were overestimated 5-35% using the questionnaire compared to the food records. The between method correlation was at its lowest for mono + disaccharides ( $r=0.33$ ) and  $r=0.43$  for total carbohydrate intake (41). Recall bias is important to consider when using retrospective questionnaires on dietary patterns. All methods measuring habitual food intake are prone to mis- or underreporting. The uncertainty of this study is in comparison with similar studies (43).

There are a variety of food-frequency questionnaires (FFQs) being used when analysing dietary variables in relation to disease. Since validation of the FFQ is a complicated process, and the fact that there is no gold standard in validating the questionnaire, this does add a great deal of uncertainty to the self-reported habitual food intake. In the present case the FFQ was designed specifically for the SOS Study that gives the advantage of the authors being able to detail the requirements of the FFQ, trying to answer the questions asked. The FFQ was originally designed to answer questions about habitual food intake in relation to obesity. In this study the information collected was used to explore the association between habitual food intake and the MetS; a question which the FFQ was not initially designed to answer. This

might be a weakness of the present study, but the close covariation between obesity and the MetS might imply this is not a great issue.

The use of epidemiological studies when analysing habitual food intake in relation to disease does indeed come with weaknesses. At first, from a cross-sectional study only associations can be determined and causal inference cannot be made. RCTs focusing on individual nutrients, outlined by observational evidence, often fail to demonstrate the hypothesised effect (58). The use of dietary patterns can still be proven useful in the search of any influence of habitual food intake in relation to disease, and is still important in the process of establishing new hypotheses.

Marks et al. suggest that gender is one of the personal characteristics that are most strongly associated with estimate errors when reporting food intake, using FFQs (59). Agreement between reported and actual intake also varies to a great extent between food groups, vegetables being one of the groups with the poorest consistency. This implies that separation according to gender might be preferred before analysing the data, as practised in this study. On the other hand different FFQs used depending on gender might be a better way to make use of this proposition.

The FFQ used in this study requested the study subjects to answer questions about a variety of food groups. The purpose of the study was to examine the effect of a variety of macronutrients on the MetS. A preferable approach when analysing single macronutrients is to present study subjects with a comprehensive food list (43). This is a weakness of the usage of the current FFQ. One way of increasing the certainty of the analyses would be to analyse individual food groups instead of macronutrients and their impact on the MetS. Individual food groups have previously been associated with the MetS e.g. sugar-sweetened beverages which Malik et al. address in a meta-analysis (60). From the current study one could obtain

associations that later must be tested in more rigorous ways i.e. RCTs to eventually express ideas of causality.

The participation rate of the study was 53.7% for men and 57.6% for women. The great non-participation rate is a considerable weakness of the study. Making statements of the general population from the results of the study might not be advisable in this case.

### *Definitions of the metabolic syndrome*

Today the IDF consensus statement is considered the most revised definition of MetS.

Previous to the current report, valid definitions include the World Health Organization (WHO)(61), which has been considered the general definition. Parallel with the WHO definition further definitions include The European Group for the Study of Insulin Resistance (EGIR)(62) which is a modified version of the WHO definitions, both published in 1999. In 2001 a definition more suited for clinical practice was published in the Journal of the American Medical Association, the National Cholesterol Education Program – Third Adult Treatment Panel (NCEP ATP III)(63). The 2002 American Association of Clinical Endocrinology Position Statement (AACE)(64) has also been used as a clinical guideline. All definitions and statements share basic parts i.e. obesity, insulin resistance, dyslipidaemia and hypertension, but differ regarding some criteria and cut-off values. The need for a compact, clinically accessible and scientifically valid definition was identified by the IDF and their statement was published in 2006 and updated in 2009.

The prevalence of the MetS increases with age and the fact that different studies build on different definitions of the MetS a coherent figure is sometimes hard to define. In Sweden, the prevalence of the MetS has been appreciated to range from 18.8 - 21.9% depending on the definition (65). Using the IDF definition results in a higher prevalence (21.9%) in comparison with previous definitions. Despite this fact the IDF definition did not have a preferable

prediction rate, compared to earlier definitions, regarding cardiovascular events. A coincident study on 60-year-old men and women in Stockholm reported an incidence of the MetS being 24% and 19% for men and women respectively (66). In the H70 study carried out in Gothenburg the 70-year-old cohort found an overall prevalence of the MetS of 22.6% (26.3% in men and 19.2% in women) (67). Due to the fact that different MetS-definitions are used, it is hard to compare studies regarding prevalence of the MetS. Several components have impact on the prevalence of the MetS, a collection of which includes lifestyle, genetic factors and age that further complicates the comparison between studies (68). The fact that the MetS varies between ethnic groups has led to the introduction of region-specific cut-points regarding waist circumference in the IDF-definition. Using NHANES data Aguilar et al. (69) report that the prevalence of the MetS has remained stable around 35% since 2007, at least in an North American population.

Hence the use of different definitions of the MetS does influence prevalence and is important to keep in mind when comparing different studies.

### **Conclusions and implications**

The results of this study are largely consistent with earlier scientific research. The lack of consensus in the interpretation of the scientific literature requires more rigorous scientific research to be undertaken. An important concern of this study is the great uncertainty of self-reported dietary intake. Diagnose and an efficient way of treating the MetS would have great health benefits for the general population since the prevalence of the MetS is high. According to this study and the current scientific literature in general, a diet restricted, preferentially in simple carbohydrates and favourable fat quality, have a role in the treatment of the MetS and may reduce the risk of the components of the syndrome.

## Populärvetenskaplig sammanfattning

Titel: Association mellan självrapporterat kostintag och det metabola syndromet och de ingående variablerna i syndromet, hos slumpmässigt utvalda män och kvinnor.

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Sammanfattning: Det metabola syndromet är en samling av fem medicinska tillstånd: bukfetma, påverkade blodfetter (förhöjda triglycerider, lågt HDL-kolesterol (det ”goda” kolesterolet)), högt blodtryck samt förhöjt fasteblodssocker. En individ som uppfyller kriterierna för det metabola syndromet har bland annat ökad risk för att utveckla hjärtkärlsjukdom, typ 2 diabetes, Alzheimers sjukdom och cancer. Kostens koppling till det metabola syndromet har upprepade gånger undersökts i vetenskapliga studier. Detta till trots råder det ingen enighet angående kostrekommendationer för någon som uppfyller kriterierna för det metabola syndromet. I denna studie har kopplingen mellan kostintag som studiedeltagare själva rapporterat och det metabola syndromet undersökts, hos slumpmässigt utvalda män och kvinnor. Mellan 1994 och 1999 samlades information om kostintag in genom att slumpmässigt utvalda män och kvinnor i Mölndal och Örebro fick svara på enkäter angående sina kostvanor. Blodprover och övriga prover mättes vid ett tillfälle. Koppling mellan det rapporterade kostintaget och kriterierna för det metabola syndromet har sedan statistiskt analyserats.

Resultat i den här studien inkluderar: intag av enkla sockerarter (mono- och disackarider, t.ex. bordssocker, fruktsocker och mjölksocker) var kopplat till ökad förekomst av metabola syndromet hos män. De starkaste kopplingarna mellan kostintag och enskilda mätvärden i det metabola syndromet i den här studien var en koppling mellan sockerintag och lägre blodfett (HDL-kolesterol) hos män. Hos kvinnor var totalt kolhydratintag kopplat till lägre blodfett (HDL-kolesterol) och lägre fasteblodssocker. Hos män var intag av fleromättade fetter kopplat till lägre blodtryck.

Hos män var enkla sockerarter kopplat till ökad risk att ha metabola syndromet. Intag av enkla sockerarter och totalt kolhydratintag var kopplat till en lägre nivå av blodfett (HDL-kolesterol) hos både män och kvinnor. Fleromättade fetter var kopplat till lägre blodtryck (systoliskt och diastoliskt). Resultaten från den här tvärsnittsstudien stämmer till stor del överens med de nuvarande kostrekommendationerna för att motverka vanliga riskfaktorer för ämnesomsättningsjukdom: att minska intag av socker och ett ökat intag av fleromättade fetter. Skillnaden mellan könen är intressant och det krävs ytterligare forskning för att utröna om män och kvinnor påverkas olika av olika kostsammansättning.

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## Figures and tables

**Table 1.** Clinical criteria of the metabolic syndrome.

Measure	Cut-off points
Waist circumference (cm)	Males $\geq 94$ , Females $\geq 80$
S-Triglycerides (mmol/l) <sup>1</sup>	$\geq 1.7$
S-HDL-cholesterol(mmol/l) <sup>1</sup>	Males $<1.0$ , Females $< 1.3$
Blood pressure (mmHg) <sup>2</sup>	SBP $\geq 130$ and/or DBP $\geq 85$
Fasting P-glucose (mmol/l) <sup>3</sup>	$\geq 5.6$

HDL: high-density lipoprotein, SBP: systolic blood pressure, DBP: diastolic blood pressure, P: plasma.

<sup>1</sup>drug treatment used for elevated triglycerides or reduced HDL-C is also used as an indicator.

<sup>2</sup>drug treatment for previously diagnosed hypertension is also used as an indicator.

<sup>3</sup>drug treatment for elevated plasma glucose is also used as an indicator.

**Table 2.** Descriptive characteristics of the study population.

	Total n=1135	Males n=524	Females n=611	P-value*
Age (years)	49.5(7.0)	49.8(7.0)	49.3(7.0)	0.226
BMI (kg/m <sup>2</sup> )	25.2(3.8)	25.9(3.4)	24.7(4.1)	<0.001
MetS variables				
Waist circumference (cm)	87.9(11.2)	92.6(9.8)	83.8(10.8)	<0.001
Systolic blood pressure (mmHg)	125.2(19.1)	128.5(19.0)	122.3(18.6)	<0.001
Diastolic blood pressure (mmHg)	76.6(11.3)	79.2(11.2)	74.5(11.0)	<0.001
S-Triglycerides (mmol/l)	1.32(0.79)	1.52(0.91)	1.13(0.62)	<0.001
S-HDL-cholesterol (mmol/l)	1.45(0.40)	1.28(0.33)	1.60(0.41)	<0.001
Fasting B-glucose (mmol/l)	4.4(0.9)	4.5(1.0)	4.3(1.0)	<0.001
Dietary variables				
Total energy (kcal/day)	2473.9(834.0)	2777.0(865.9)	2219.7(713.7)	<0.001
Carbohydrate (E%)	45.6(5.4)	44.8(5.3)	46.3(5.4)	<0.001
Fat (E%)	35.5(4.8)	35.9(4.7)	35.2(4.7)	0.012
Protein (E%)	15.5(2.1)	15.1(2.0)	15.7(2.1)	<0.001
Carbohydrate (g/day)	281(99)	310(102)	257(89)	<0.001
Fat (g/day)	99(40)	112(42)	87(33)	<0.001
Protein (g/day)	95(32)	104(33)	87(28)	<0.001
Sugar <sup>1</sup> (g/day)	118(58)	127(62)	110(54)	<0.001
Fibre (g/day)	22(8)	23(8)	22(7)	0.007
MUFA (g/day)	32(13)	37(14)	29(11)	<0.001
PUFA (g/day)	14(6)	16(6)	12(5)	<0.001
SFA (g/day)	42(19)	48(20)	37(16)	<0.001
MetS prevalence (n,%)	197(17.4)	111(21.2)	86(14.1)	0.002

HDL: high-density lipoprotein, E%: percent of total energy intake, kcal: kilocalories, MetS: metabolic syndrome, MUFA: mono-unsaturated fatty acids, PUFA: poly-unsaturated fatty acids, SFA: saturated fatty acids, B: blood.

Values expressed as mean (s.d.), except MetS prevalence n (%).

\* Independent samples t-test comparing sex, p<0.05 is considered significant.

<sup>1</sup>mono- and disaccharides analysed.

**Table 3.** Crosstabs sugar<sup>1</sup> quartiles and MetS prevalence.

Males	Quartile 1	Quartile 2-3	Quartile 4	p -value*
MetS prevalence	19.20%	18.20%	29.60%	<b>0.032</b>

Females	Quartile 1	Quartile 2-3	Quartile 4	p -value*
MetS prevalence	16.20%	11.70%	16.70%	0.252

\*Pearson Chi<sup>2</sup>

Quartile 1: low intake (<84g), Quartile 2-3: medium intake (84-153g), Quartile 4: high intake (>153g), MetS: metabolic syndrome.

Calculations performed on sugar quartiles (g/day).

<sup>1</sup>mono- and disaccharides analysed.



**Table 4a.** Correlation coefficients for the association between quartiles of macronutrient intake and MetS variables.

Males	CHO	Fat	Protein	Kcal
BMI (kg/m <sup>2</sup> )	-0.018	-0.055	0.02	-0.037
Waist circumference (cm)	-0.027	-0.007	0.042	-0.005
S-Triglycerides (mmol/l)	-0.016	-0.029	-0.054	-0.002
S-HDL-cholesterol (mmol/l)	-0.066	-0.042	-0.015	-0.021
Systolic blood pressure (mmHg)	-0.045	<b>-0.090*</b>	-0.079	-0.064
Diastolic blood pressure (mmHg)	-0.062	-0.08	-0.055	-0.061
Fasting B-glucose (mmol/l)	-0.025	-0.017	-0.038	-0.026
Females	CHO	Fat	Protein	Kcal
BMI (kg/m <sup>2</sup> )	-0.045	-0.078	-0.019	-0.027
Waist circumference (cm)	0.016	0.009	0.056	0.058
S-Triglycerides (mmol/l)	0.042	-0.034	0	0.017
S-HDL-cholesterol (mmol/l)	<b>-0.096*</b>	-0.055	-0.08	<b>-0.081*</b>
Systolic blood pressure (mmHg)	-0.003	-0.035	0.005	0.029
Diastolic blood pressure (mmHg)	-0.067	<b>-0.090*</b>	-0.046	-0.044
Fasting B-glucose (mmol/l)	<b>-0.099*</b>	-0.063	-0.056	-0.08

HDL: high-density lipoprotein, MetS: metabolic syndrome, CHO: carbohydrates, Kcal: kilocalories, B: blood.

Calculations performed on macronutrient quartiles (g/day) and total energy intake (kcal/day).

All correlations expressed as rank correlation (Spearman's rho,  $r_s$ ).

\* Correlation is significant at the 0.05 level.

\*\* Correlation is significant at the 0.01 level.

**Table 4b.** Correlation coefficients for the association between quartiles of sugar, dietary fibre, mono-unsaturated fatty acid, poly-unsaturated fatty acid, saturated fatty acid intake and MetS variables.

Males	Sugar <sup>1</sup>	Fibre	MUFA	PUFA	SFA
BMI (kg/m <sup>2</sup> )	0.082	-0.02	-0.041	-0.071	-0.061
Waist circumference (cm)	0.071	-0.023	-0.01	-0.029	-0.044
S-Triglycerides (mmol/l)	0.06	0.01	0.005	-0.01	-0.031
S-HDL-cholesterol (mmol/l)	<b>-0.145**</b>	-0.023	-0.025	-0.009	-0.029
Systolic blood pressure (mmHg)	0.009	-0.03	-0.083	<b>-0.127**</b>	-0.081
Diastolic blood pressure (mmHg)	-0.032	-0.044	-0.075	<b>-0.131**</b>	-0.056
Fasting B-glucose (mmol/l)	-0.015	-0.023	-0.013	0	0.004
Females	Sugar <sup>1</sup>	Fibre	MUFA	PUFA	SFA
BMI (kg/m <sup>2</sup> )	-0.031	0.01	-0.068	-0.052	-0.072
Waist circumference (cm)	0.024	0.053	0.016	0.016	-0.002
S-Triglycerides (mmol/l)	0.044	0.063	-0.048	-0.016	-0.011
S-HDL-cholesterol (mmol/l)	-0.062	-0.028	-0.066	<b>-0.085*</b>	-0.064
Systolic blood pressure (mmHg)	0.071	0	-0.051	-0.063	0.025
Diastolic blood pressure (mmHg)	-0.011	-0.048	<b>-0.096*</b>	<b>-0.090*</b>	-0.03
Fasting B-glucose (mmol/l)	-0.048	-0.042	-0.06	-0.073	-0.063

HDL: high-density lipoprotein, MetS: metabolic syndrome, MUFA: mono-unsaturated fatty acids, PUFA: poly-unsaturated fatty acids, SFA: saturated fatty acids, B: blood.

Calculations performed on macronutrient quartiles (g/day).

All correlations expressed as rank correlation (Spearman's rho,  $r_s$ ).

\* Correlation is significant at the 0.05 level.

\*\* Correlation is significant at the 0.01 level.

<sup>1</sup>mono- and disaccharides analysed.

**Table 5a.** Student's t-test quartiles of macronutrient intake and MetS variables.

Males	CHO			Fat			Protein		
	Q1	Q4	p	Q1	Q4	p	Q1	Q4	p
BMI (kg/m <sup>2</sup> )	26.2(3.5)	26.0(3.7)	0.695	26.3(3.5)	25.8(3.4)	0.185	26.1(3.6)	26.2(3.4)	0.942
Waist circ. (cm)	93.8(10.0)	92.7(10.4)	0.39	93.6(9.5)	93.0(9.7)	0.64	93.1(10.1)	93.6(9.8)	0.694
S-TG (mmol/l)	1.51(0.79)	1.56(1.01)	0.654	1.60(0.92)	1.61(1.05)	0.904	1.58(0.85)	1.57(1.05)	0.91
S-HDL-C (mmol/l)	1.29(0.29)	1.23(0.30)	0.092	1.27(0.28)	1.24(0.30)	0.375	1.27(0.30)	1.25(0.31)	0.626
SBP (mmHg)	130(20)	128(19)	0.289	130(19)	126(19)	0.08	130(19)	127(19)	0.155
DBP (mmHg)	80(11)	78(12)	0.186	80(11)	78(13)	0.168	80(11)	78(11)	0.15
FG (mmol/l)	4.54(0.87)	4.48(0.76)	0.606	4.54(0.78)	4.51(0.75)	0.745	4.52(0.77)	4.50(0.86)	0.822
Females	CHO			Fat			Protein		
	Q1	Q4	p	Q1	Q4	p	Q1	Q4	p
BMI (kg/m <sup>2</sup> )	24.8(4.0)	24.6(4.7)	0.594	25.2(3.8)	24.9(5.1)	0.598	24.7(3.8)	25.0(5.1)	0.566
Waist circ. (cm)	83.5(10.4)	84.0(11.9)	0.702	84.0(9.8)	84.9(12.6)	0.473	82.6(10.4)	84.7(12.1)	0.128
S-TG (mmol/l)	1.12(0.58)	1.21(0.70)	0.217	1.16(0.58)	1.16(0.68)	0.965	1.11(0.52)	1.14(0.59)	0.581
S-HDL-C (mmol/l)	<b>1.68 (0.43)</b>	<b>1.56 (0.41)</b>	<b>0.02</b>	1.63(0.46)	1.56(0.40)	0.162	<b>1.66 (0.46)</b>	<b>1.56 (0.39)</b>	<b>0.043</b>
SBP (mmHg)	124(20)	123(17)	0.64	124(19)	122(18)	0.464	122(18)	123(17)	0.98
DBP (mmHg)	76(12)	74(11)	0.124	<b>76 (11)</b>	<b>73 (11)</b>	<b>0.04</b>	75(11)	74(9.7)	0.179
FG (mmol/l)	<b>4.39 (0.77)</b>	<b>4.18 (0.56)</b>	<b>0.011</b>	4.39(1.11)	4.30(1.19)	0.496	4.31(0.66)	4.28(1.15)	0.785

TG: triglycerides, HDL: high-density lipoprotein, MetS: metabolic syndrome, CHO: carbohydrates, SBP: systolic blood pressure, DBP: diastolic blood pressure, FG: fasting blood glucose, Q: quartile.

Calculations performed on macronutrient quartiles (g/day).

Values expressed as mean (s.d.).

p-values <0.05 is considered significant.

**Table 5b.** Student's t-test quartiles of sugar, dietary fibre, mono-unsaturated fatty acid, poly-unsaturated fatty acid, saturated fatty acid intake and MetS variables.

Males	Kcal			Sugar <sup>1</sup>			Fibre		
	Q1	Q4	p	Q1	Q4	p	Q1	Q4	p
BMI (kg/m <sup>2</sup> )	26.3(3.4)	26.0(3.7)	0.493	25.7(3.5)	26.5(3.7)	0.093	26.0(3.6)	25.8(3.3)	0.537
Waist circ. (cm)	93.6(9.8)	93.1(10.4)	0.719	92.6(9.7)	94.1(10.1)	0.239	93.6(10.1)	92.7(9.3)	0.444
S-TG (mmol/l)	1.52(0.79)	1.63(1.07)	0.346	<b>1.49(0.78)</b>	<b>1.75(1.15)</b>	<b>0.039</b>	1.52(0.85)	1.65(1.11)	0.274
S-HDL-C (mmol/l)	1.28(0.29)	1.26(0.30)	0.559	<b>1.31(0.30)</b>	<b>1.18(0.29)</b>	<b>&lt;0.001</b>	1.27(0.31)	1.25(0.33)	0.616
SBP (mmHg)	130(19)	127(19)	0.2	128(20)	128(17)	0.786	130(20)	128(19)	0.418
DBP (mmHg)	80(11)	78(12)	0.22	79(11)	78(11)	0.542	80(12)	78(11)	0.173
FG (mmol/l)	4.52(0.76)	4.49(0.76)	0.815	4.55(0.88)	4.48(0.67)	0.493	4.50(0.77)	4.47(0.74)	0.733
Females	Kcal			Sugar <sup>1</sup>			Fibre		
	Q1	Q4	p	Q1	Q4	p	Q1	Q4	p
BMI (kg/m <sup>2</sup> )	24.9(3.9)	25.0(5.0)	0.815	25.0(4.1)	24.8(4.6)	0.628	24.7(4.0)	25.0(4.5)	0.613
Waist circ. (cm)	83.1(9.9)	85.2(12.5)	0.112	83.9(10.7)	84.5(11.8)	0.658	83.8(10.7)	85.3(10.9)	0.238
S-TG (mmol/l)	1.12(0.56)	1.19(0.68)	0.363	1.14(0.61)	1.22(0.68)	0.284	1.13(0.58)	1.22(0.62)	0.173
S-HDL-C (mmol/l)	<b>1.65(0.44)</b>	<b>1.54(0.39)</b>	<b>0.037</b>	1.64(0.44)	1.57(0.42)	0.136	1.63(0.41)	1.59(0.37)	0.42
SBP (mmHg)	122(18)	123(18)	0.509	122(20)	125(18)	0.177	123(20)	122(18)	0.76
DBP (mmHg)	75(11)	74(10)	0.265	75(12)	74(11)	0.675	76(12)	74(10)	0.212
FG (mmol/l)	4.40(0.80)	4.32(1.17)	0.469	4.37(0.78)	4.29(1.04)	0.442	4.41(1.13)	4.31(1.16)	0.482
Males	MUFA			PUFA			SFA		
	Q1	Q4	p	Q1	Q4	p	Q1	Q4	p
BMI (kg/m <sup>2</sup> )	26.4(3.5)	25.9(3.4)	0.272	26.3(3.3)	25.7(3.4)	0.12	26.3(3.5)	25.7(3.3)	0.124
Waist circ. (cm)	93.8(9.6)	93.2(9.7)	0.612	93.6(9.0)	92.7(9.4)	0.462	93.9(9.4)	92.4(9.4)	0.202
S-TG (mmol/l)	1.55(0.91)	1.63(1.04)	0.552	1.45(0.66)	1.59(1.05)	0.204	1.59(0.93)	1.55(0.94)	0.742
S-HDL-C (mmol/l)	1.27(0.28)	1.25(0.31)	0.598	1.26(0.28)	1.25(0.31)	0.756	1.28(0.29)	1.25(0.31)	0.533
SBP (mmHg)	131(20)	127(19)	0.104	<b>130(19)</b>	<b>124(16)</b>	<b>0.003</b>	130(18)	127(19)	0.156
DBP (mmHg)	80(11)	78(13)	0.224	<b>80(11)</b>	<b>77(12)</b>	<b>0.013</b>	80(10)	78(13)	0.332
FG (mmol/l)	4.55(0.77)	4.54(0.84)	0.925	4.49(0.75)	4.51(0.86)	0.797	4.47(0.73)	4.46(0.57)	0.836
Females	MUFA			PUFA			SFA		
	Q1	Q4	p	Q1	Q4	p	Q1	Q4	p
BMI (kg/m <sup>2</sup> )	25.1(3.8)	24.9(5.1)	0.732	24.9(3.7)	24.9(5.2)	0.996	25.0(3.7)	24.8(5.0)	0.628
Waist circ. (cm)	83.6(9.9)	84.8(12.7)	0.381	83.5(9.9)	84.8(13.1)	0.372	83.7(9.9)	84.3(12.3)	0.67
S-TG (mmol/l)	1.16(0.57)	1.12(0.62)	0.62	1.14(0.51)	1.20(0.72)	0.462	1.13(0.58)	1.15(0.66)	0.816
S-HDL-C (mmol/l)	1.64(0.45)	1.56(0.41)	0.12	<b>1.67(0.44)</b>	<b>1.56(0.40)</b>	<b>0.036</b>	1.63(0.43)	1.56(0.39)	0.147
SBP (mmHg)	124(19)	121(17)	0.183	124(18)	121(18)	0.173	121(19)	123(18)	0.575
DBP (mmHg)	<b>75(11)</b>	<b>73(10)</b>	<b>0.021</b>	<b>76(11)</b>	<b>73(10)</b>	<b>0.034</b>	74(11)	73(11)	0.474
FG (mmol/l)	4.37(1.09)	4.18(0.70)	0.092	4.29(0.64)	4.20(0.84)	0.357	4.40(1.16)	4.25(1.1)	0.253

Kcal: kilocalories, TG: triglycerides, HDL: high-density lipoprotein, MetS: metabolic syndrome, SBP: systolic blood pressure, DBP: diastolic blood pressure, FG: fasting blood glucose, MUFA: mono-unsaturated fatty acids, PUFA: poly-unsaturated fatty acids, SFA: saturated fatty acids, Q: quartile.

Calculations performed on total energy intake quartiles (kcal/day) and macronutrient quartiles (g/day).

Values expressed as mean (s.d.).

p-values <0.05 is considered significant.

<sup>1</sup>mono- and disaccharides analysed.

Figure 1

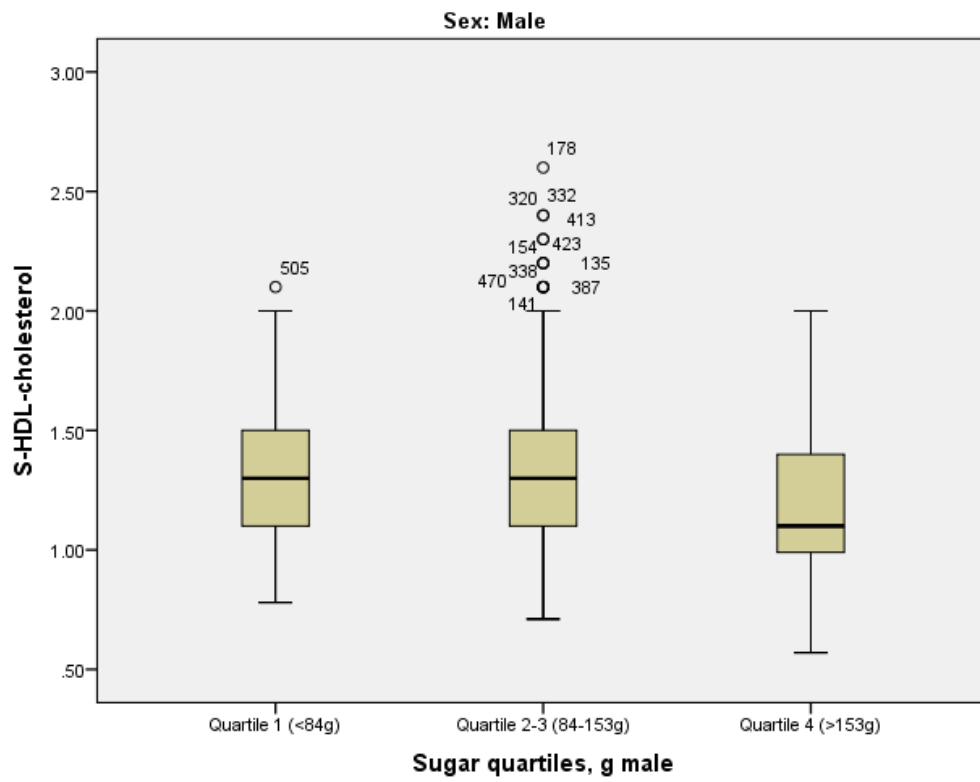
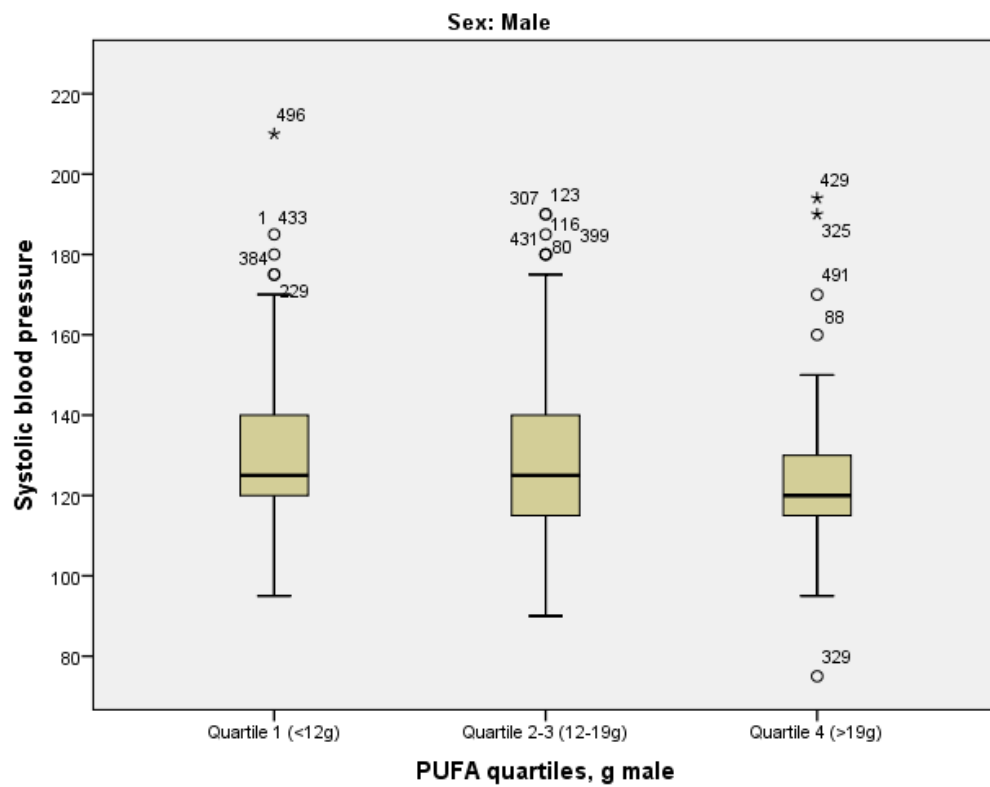
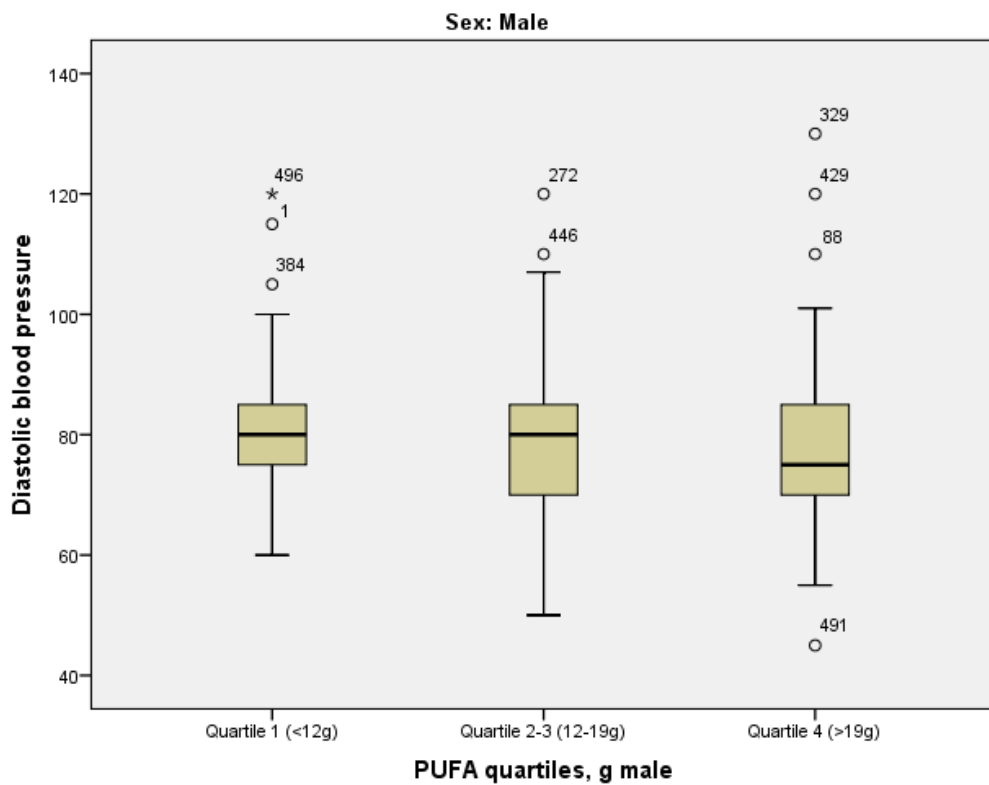


Figure 2



**Figure 3**



*Legend*

**Figure 1**

Boxplot illustrating correlation between HDL-cholesterol and quartiles of mono- and disaccharides in men. Rank correlation (Spearman's rho,  $r_s$ ) -0.145 ( $p < 0.01$ ).

**Figure 2**

Boxplot illustrating correlation between systolic blood pressure and quartiles of polyunsaturated fatty acids in men. Rank correlation (Spearman's rho,  $r_s$ ) -0.127 ( $p < 0.01$ ).

**Figure 3**

Boxplot illustrating correlation between diastolic blood pressure and quartiles of polyunsaturated fatty acids in men. Rank correlation (Spearman's rho,  $r_s$ ) -0.131 ( $p < 0.01$ ).

## Appendix

**Table 6a.** Correlation coefficients for the association between macronutrients and MetS variables.

Males	CHO	Fat	Protein	Kcal
BMI (kg/m <sup>2</sup> )	0.03	-0.03	0.047	0.01
Waist circumference (cm)	0.014	-0.01	0.059	0.02
S-Triglycerides (mmol/l)	<b>0.104*</b>	0.083	0.07	<b>0.100*</b>
S-HDL-cholesterol (mmol/l)	<b>-0.101*</b>	-0.06	-0.063	-0.064
Systolic blood pressure (mmHg)	-0.041	<b>-0.099*</b>	-0.064	-0.073
Diastolic blood pressure (mmHg)	-0.047	<b>-0.089*</b>	-0.07	-0.074
Fasting B-glucose (mmol/l)	-0.023	-0.02	-0.028	-0.026
Females	CHO	Fat	Protein	Kcal
BMI (kg/m <sup>2</sup> )	0.024	0.007	0.055	0.021
Waist circumference (cm)	0.047	0.032	0.08	0.051
S-Triglycerides (mmol/l)	0.035	0.007	0.001	0.026
S-HDL-cholesterol (mmol/l)	-0.071	-0.06	-0.048	-0.057
Systolic blood pressure (mmHg)	0.007	-0.02	0.023	-0.002
Diastolic blood pressure (mmHg)	-0.057	-0.08	-0.026	-0.059
Fasting B-glucose (mmol/l)	<b>-0.083*</b>	-0.06	-0.018	-0.07

HDL: high-density lipoprotein, MetS: metabolic syndrome, CHO: Carbohydrates, Kcal: kilocalories, B: blood.

Calculations performed on absolute continuous macronutrients (g/day) and total energy intake (kcal/day).

All correlations expressed as Pearson correlation coefficient, r.

\* Correlation is significant at the 0.05 level.

\*\* Correlation is significant at the 0.01 level.

**Table 6b.** Correlation coefficients for the association between sugar, dietary fibre, mono-unsaturated fatty acids, poly-unsaturated fatty acids, saturated fatty acids and MetS variables.

Males	Sugar	Fibre	MUFA	PUFA	SFA
BMI (kg/m <sup>2</sup> )	<b>0.111*</b>	-0.03	-0.02	-0.061	-0.025
Waist circumference (cm)	0.08	-0.02	0.003	-0.022	-0.007
S-Triglycerides (mmol/l)	<b>0.123**</b>	<b>0.112*</b>	<b>0.093*</b>	0.082	0.06
S-HDL-cholesterol (mmol/l)	<b>-0.159**</b>	-0.05	-0.063	-0.041	-0.054
Systolic blood pressure (mmHg)	0.022	-0.02	<b>-0.109*</b>	<b>-0.161**</b>	-0.053
Diastolic blood pressure (mmHg)	0.006	-0.03	-0.086	<b>-0.145**</b>	-0.057
Fasting B-glucose (mmol/l)	-0.009	-0.03	-0.018	-0.02	-0.021
Females	Sugar	Fibre	MUFA	PUFA	SFA
BMI (kg/m <sup>2</sup> )	0.032	0.028	0.027	0.021	-0.008
Waist circumference (cm)	0.05	0.059	0.051	0.055	0.012
S-Triglycerides (mmol/l)	0.034	0.02	0.013	0.043	-0.003
S-HDL-cholesterol (mmol/l)	-0.056	-0.03	-0.072	<b>-0.083*</b>	-0.033
Systolic blood pressure (mmHg)	0.045	-0.02	-0.038	-0.031	-0.006
Diastolic blood pressure (mmHg)	-0.026	-0.07	<b>-0.083*</b>	-0.071	-0.064
Fasting B-glucose (mmol/l)	-0.063	-0.06	-0.062	-0.038	-0.052

HDL: high-density lipoprotein, MetS: metabolic syndrome, MUFA: mono-unsaturated fatty acids, PUFA: poly-unsaturated fatty acids, SFA: saturated fatty acids, B: blood.

Calculations performed on absolute continuous macronutrients (g/day).

All correlations expressed as Pearson correlation coefficient, r.

\* Correlation is significant at the 0.05 level.

\*\* Correlation is significant at the 0.01 level.



**Table 7a.** Linear regression continuous macronutrients and MetS variables.

Males									
Measure	CHO			Fat			Protein		
	R <sup>2</sup>	β	p-value	R <sup>2</sup>	β	p-value	R <sup>2</sup>	β	p-value
Waist circ. (cm)	<b>0.801</b>	<b>-0.172</b>	<b>0.002</b>	0.798	0.075	0.213	0.797	0.035	0.473
S-TG (mmol/l)	0.111	0.069	0.552	0.111	-0.061	0.626	0.116	-0.182	0.074
S-HDL-C (mmol/l)	<b>0.167</b>	<b>-0.314</b>	<b>0.006</b>	0.154	-0.015	0.902	0.154	0.04	0.688
SBP (mmHg)	0.209	0.081	0.46	0.209	-0.084	0.479	0.209	-0.074	0.444
DBP (mmHg)	0.135	0.077	0.503	0.135	-0.037	0.764	0.136	-0.094	0.35
FG (mmol/l)	0.056	-0.057	0.637	0.057	0.108	0.403	0.056	-0.076	0.469
Females									
Measure	CHO			Fat			Protein		
	R <sup>2</sup>	β	p-value	R <sup>2</sup>	β	p-value	R <sup>2</sup>	β	p-value
Waist circ.(cm)	0.769	-0.029	0.589	0.769	-0.026	0.623	0.769	0.015	0.736
S-TG (mmol/l)	0.204	0.046	0.653	0.204	-0.05	0.614	<b>0.213</b>	<b>-0.213</b>	<b>0.011</b> <sup>1</sup>
S-HDL-C (mmol/l)	0.103	-0.206	0.059	0.098	0.059	0.582	0.097	0.008	0.927
SBP (mmHg)	0.313	-0.078	0.407	0.312	0.065	0.483	0.311	-0.015	0.85
DBP (mmHg)	0.243	-0.128	0.196	0.241	0.019	0.849	0.24	0.014	0.864
FG (mmol/l)	0.07	-0.164	0.135	0.068	0.114	0.289	0.072	0.169	0.06

CHO: carbohydrates, TG: triglycerides, HDL: high-density lipoprotein, SBP: systolic blood pressure, DBP: diastolic blood pressure, FG: fasting blood glucose, MetS: metabolic syndrome, β: standardized coefficient, R<sup>2</sup>: adjusted R<sup>2</sup>.

Calculations performed on continuous macronutrients (g/day).

Adjustment made for BMI, age, physical activity (leisure time) and total energy intake (kcal/day).

p-values <0.05 is considered significant.

<sup>1</sup> does not meet requirement of normal distribution for residuals.

**Table 7b.** Linear regression total caloric intake, sugar, dietary fibre, mono-unsaturated fatty acids, poly-unsaturated fatty acids, saturated fatty acids and MetS variables.

Males									
Measure	Kcal			Sugar <sup>2</sup>			Fibre		
	R <sup>2</sup>	β	p-value	R <sup>2</sup>	β	p-value	R <sup>2</sup>	β	p-value
Waist circ. (cm)	0.798	0.016	0.43	<b>0.8</b>	<b>-0.077</b>	<b>0.018</b>	0.797	0.001	0.979
S-TG (mmol/l)	<b>0.112</b>	<b>0.087</b>	<b>0.042</b> <sup>1</sup>	0.111	0.043	0.53	<b>0.119</b>	<b>0.133</b>	<b>0.032</b> <sup>1</sup>
S-HDL-C (mmol/l)	0.156	-0.043	0.306	<b>0.169</b>	<b>-0.201</b>	<b>0.003</b>	0.156	-0.069	0.255
SBP (mmHg)	0.21	-0.034	0.4	0.211	0.093	0.15	0.208	0.016	0.787
DBP (mmHg)	0.136	-0.047	0.266	0.136	0.069	0.307	0.134	0.014	0.824
FG (mmol/l)	0.057	-0.005	0.904	0.056	-0.035	0.616	0.056	-0.048	0.451
Females									
Measure	Kcal			Sugar <sup>2</sup>			Fibre		
	R <sup>2</sup>	β	p-value	R <sup>2</sup>	β	p-value	R <sup>2</sup>	β	p-value
Waist circ. (cm)	0.769	0.032	0.101	0.769	-0.012	0.729	0.769	0.026	0.353
S-TG (mmol/l)	0.205	0.026	0.476	0.204	-0.015	0.814	0.203	0.001	0.985
S-HDL-C (mmol/l)	0.099	-0.04	0.311	0.099	-0.076	0.266	0.098	-0.009	0.874
SBP (mmHg)	0.313	0.019	0.571	0.312	0.001	0.981	0.314	-0.063	0.193
DBP (mmHg)	0.242	-0.046	0.198	0.241	-0.042	0.497	0.244	-0.082	0.105
FG (mmol/l)	0.068	-0.07	0.078	0.067	-0.05	0.465	0.067	-0.039	0.481
Males									
Measure	MUFA			PUFA			SFA		
	R <sup>2</sup>	β	p-value	R <sup>2</sup>	β	p-value	R <sup>2</sup>	β	p-value
Waist circ. (cm)	0.798	0.078	0.167	<b>0.8</b>	<b>0.093</b>	<b>0.016</b>	0.797	-0.008	0.854
S-TG (mmol/l)	0.11	0.018	0.879	0.111	0.053	0.52	0.115	-0.147	0.119
S-HDL-C (mmol/l)	0.154	-0.03	0.793	0.154	-0.017	0.828	0.154	0.015	0.874
SBP (mmHg)	0.21	-0.12	0.281	<b>0.221</b>	<b>-0.22</b>	<b>0.004</b>	0.21	0.088	0.327
DBP (mmHg)	0.134	0.011	0.921	<b>0.144</b>	<b>-0.187</b>	<b>0.02</b>	0.135	0.062	0.505
FG (mmol/l)	0.058	0.153	0.209	0.058	0.089	0.288	0.056	0.029	0.77
Females									
Measure	MUFA			PUFA			SFA		
	R <sup>2</sup>	β	p-value	R <sup>2</sup>	β	p-value	R <sup>2</sup>	β	p-value
Waist circ. (cm)	0.769	-0.007	0.88	0.769	0.034	0.34	0.769	-0.043	0.313
S-TG (mmol/l)	0.204	-0.022	0.813	0.207	0.1	0.133	0.205	-0.073	0.359
S-HDL-C (mmol/l)	0.098	0.042	0.672	0.098	-0.031	0.664	0.099	0.071	0.403
SBP (mmHg)	0.312	0.064	0.45	0.313	0.064	0.298	0.312	0.033	0.653
DBP (mmHg)	0.241	0.018	0.842	0.241	0.041	0.529	0.241	0.001	0.985
FG (mmol/l)	0.067	0.051	0.607	0.069	0.092	0.201	0.068	0.082	0.337

MUFA: mono-unsaturated fatty acids, PUFA: poly-unsaturated fatty acids, SFA: saturated fatty acids, TG: triglycerides, HDL: high-density lipoprotein, SBP: systolic blood pressure, DBP: diastolic blood pressure, FG: fasting blood glucose, MetS: metabolic syndrome, β: standardized coefficient, R<sup>2</sup>: adjusted R<sup>2</sup>.

Calculations performed on continuous macronutrients (g/day) and total energy intake (kcal/day).

Adjustment made for BMI, age, physical activity (leisure time) and total energy intake (kcal/day).

p-values <0.05 is considered significant.

<sup>1</sup>does not meet requirement of normal distribution for residuals.

<sup>2</sup> mono- and disaccharides analysed.

Figure 4

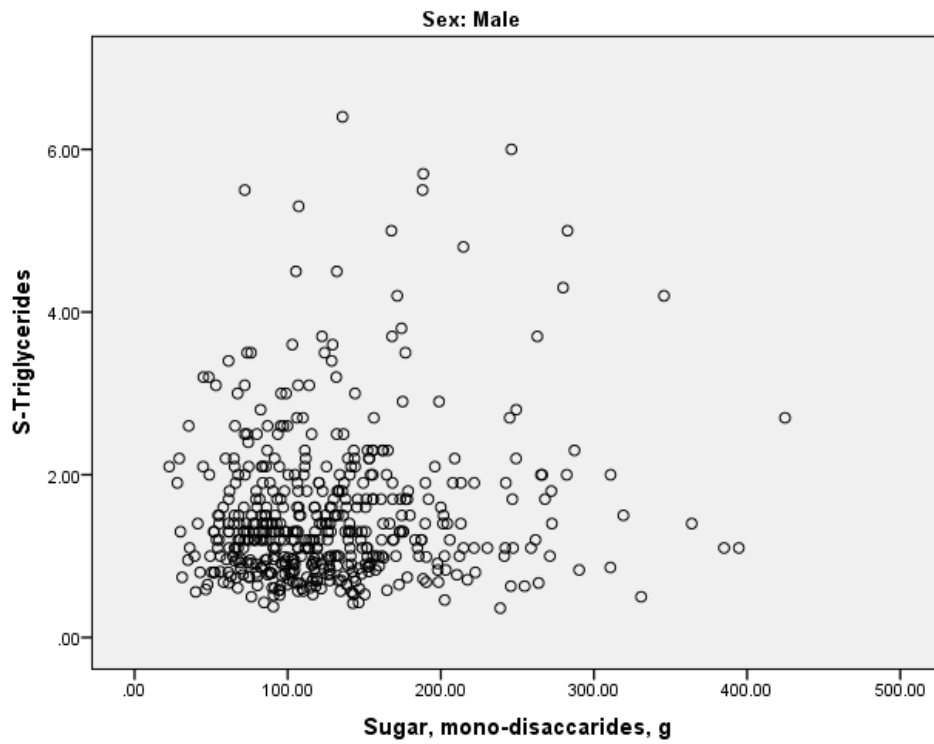


Figure 5

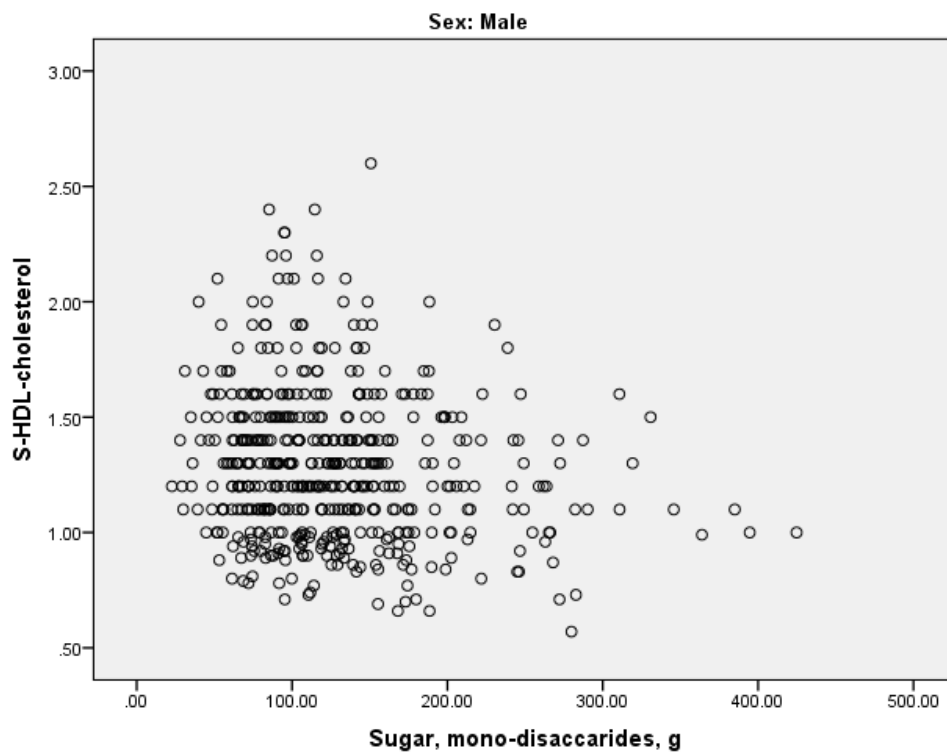


Figure 6

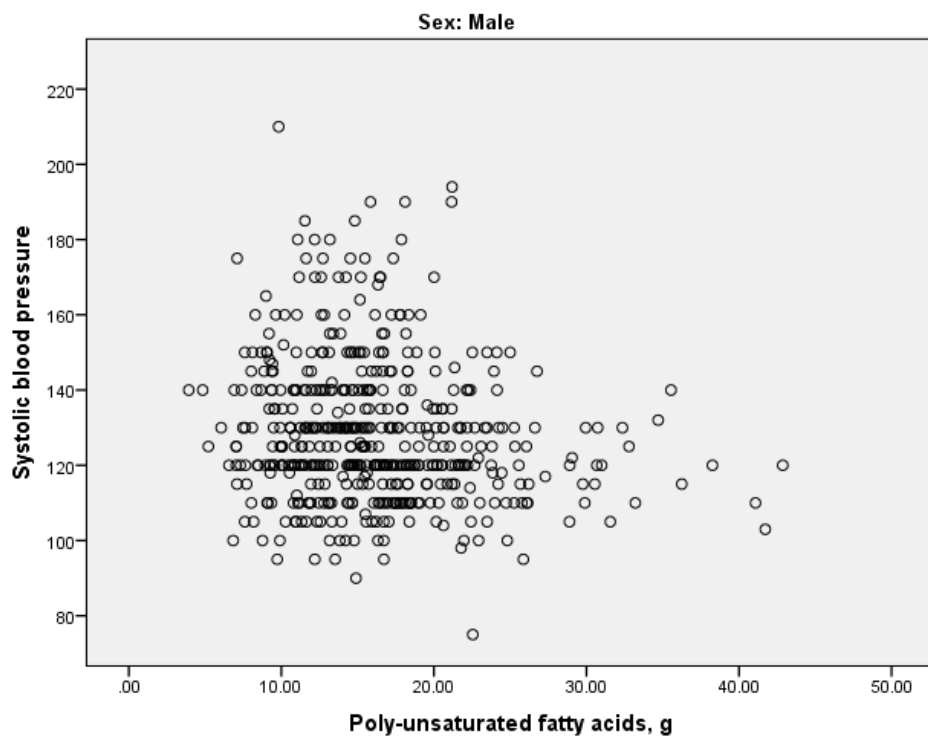
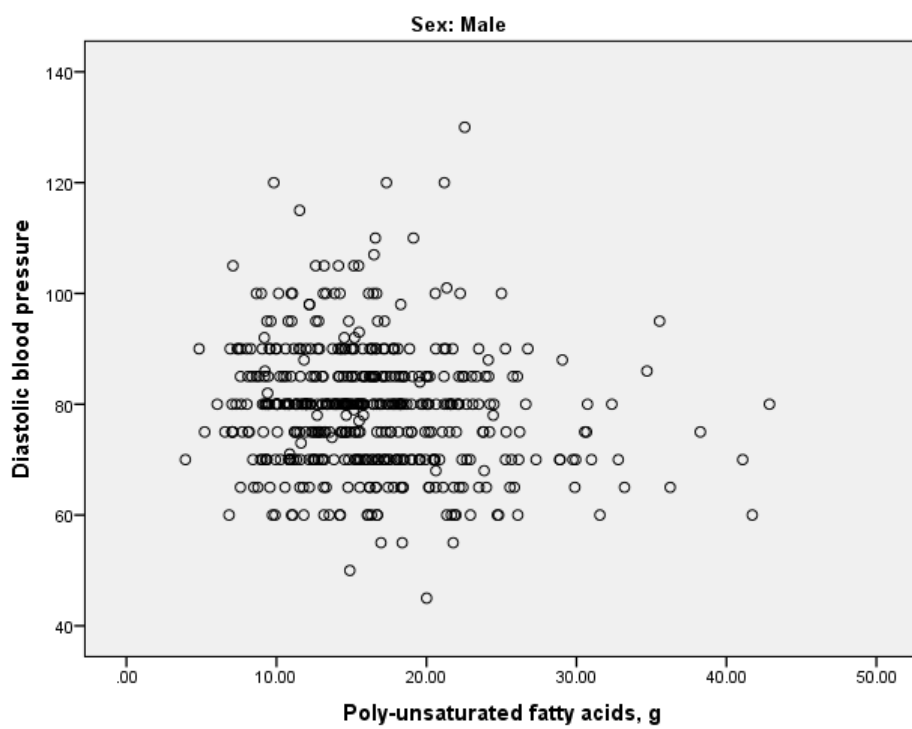


Figure 7



## *Legend*

### **Figure 4**

Scatterplot illustrating correlation between triglycerides and mono- and disaccharides in men. Pearson's correlation coefficient (r) 0.123 (p<0.01).

### **Figure 5**

Scatterplot illustrating correlation between HDL-cholesterol and mono- and disaccharides in men. Pearson's correlation coefficient (r) -0.159 (p<0.01).

### **Figure 6**

Scatterplot illustrating correlation between systolic blood pressure and polyunsaturated fatty acids in men. Pearson's correlation coefficient (r) -0.161 (p<0.01).

### **Figure 7**

Scatterplot illustrating correlation between diastolic blood pressure and polyunsaturated fatty acids in men. Pearson's correlation coefficient (r) -0.145 (p<0.01).