THE EFFECT OF SATURATED FAT ON CARDIOVASCULAR RISK (ARTERIAL BLOOD PRESSURE)

Final Year Project Scientific Academic Paper

ST20060405

Cardiff School of Health Sciences | BSc Biomedical Science (Health, Exercise & Nutrition)

Cardiff Metropolitan University | Prifysgol Metropolitan Caerdydd

Table of Contents

ABBREVIATIONS 2

ABSTRACT 2

INTRODUCTION 3

METHOD 5
- STUDY DESIGN AND POPULATION 5
- DATA COLLECTION 5
- STATISTICAL METHODS 6
- ETHICAL CONSIDERATIONS 6

RESULTS 7
- TABLE 1A 7
- TABLE 1B 7
- TABLE 2A 8
- TABLE 2B 8
- FIGURE 1A 9
- FIGURE 1B 9
- FIGURE 2A 10
- FIGURE 2B 10
- FIGURE 3A 11
- FIGURE 3B 11
- FIGURE 4A 12
- FIGURE 4B 12
- FIGURE 5A 13
- FIGURE 5B 13
- TABLE 5 14
- TABLE 6A 14
- TABLE 6B 15

DISCUSSION 15
- DIETARY SATURATED FAT, HEART RATE AND BLOOD PRESSURE 19
- BLOOD PRESSURE AND BODY FAT PERCENTAGE 20
- OVERALL STUDY OBSERVATIONS 21

CONCLUSION 22

REFERENCES 23

APPENDIX 25
- TABLE 7 25
- PARTICIPANTS' ENERGY DISTRIBUTION BY NUTRIENT 25
- ACKNOWLEDGMENTS 33
- ETHICS APPROVAL LETTER (10/08/2016) 34

ABBREVIATIONS

CVD	Cardiovascular Disease	**CHD**	Coronary Heart Disease
SFA	Saturated Fatty Acid	**PUFA**	Polyunsaturated Fatty Acid
MUFA	Monounsaturated Fatty Acid	**TC**	Total Cholesterol
HR	Heart Rate	**RCT**	Randomised Control Trial

ABSTRACT

Cardiovascular diseases (CVD), such as stroke, myocardial infarction and atherosclerosis are a major cause of morbidity and mortality throughout the western world. This cross-sectional study was conducted to investigate the relationship between dietary saturated fat intake and developing risk factors associated with the aetiology and pathogenesis of CVD.

Volunteers (13 men and 12 women) were recruited from students of Cardiff Metropolitan University. Dietary intake through a 7-day food diary was analysed through Nutrimen, and body composition, resting heart rate, blood pressure, and body mass index were assessed in relation to dietary saturated fat intake. Measures variables and their relationships were assessed by 2-sample T tests, and by linear regression analysis.

Dietary saturated fat was positively associated with systolic ($p > 0.05$) and diastolic ($p = < 0.05$) blood pressure and body fat percentage ($p > 0.05$) and negatively associated with heart rate ($p > 0.05$) in males. Body fat percentage was positively associated with diastolic blood pressure (< 0.05) in males, but not in females (> 0.05). There was almost no association between diastolic blood pressure ($p > 0.05$) and saturated fat in females, and a negative association between saturated fat and heart rate ($p > 0.05$).

The data of this study indicated gender-related differences in response to dietary saturated fat and body composition. Although the men who took part in the study were all relatively diet and exercise conscious, their body fat percentage and diastolic blood pressure readings are indicative of a risk of cardiovascular disease. More dietary recommendations and education should be provided surrounding replacement of dietary saturated fat, but customary dietary and lifestyle advice may be required for men in comparison to women who appeared unaffected in the study.

INTRODUCTION

Cardiovascular diseases (CVD), such as stroke, myocardial infarction and atherosclerosis are a major cause of morbidity and mortality throughout the western world. There were at least 2.1 million people living with a disease of the circulatory system in 2015, with 476,000 of those suffering from coronary heart disease (CHD) (British Heart Foundation, 2015). By 2030, researchers project that non-communicable diseases will account for more than three-quarters of deaths worldwide; CVD alone will be responsible for more deaths in low income countries than infectious diseases (including HIV/AIDS, tuberculosis, and malaria), maternal and perinatal conditions, and nutritional disorders combined (Beaglehole & Bonita, 2008).

The Framingham Heart Study suggested that various aspects of health can increase the risk of developing CVD, for example tobacco use, unhealthy diet, physical inactivity, obesity, raised blood pressure and raised blood cholesterol (Mendis, 2010). Elevated blood pressure and hypertension usually clusters with other cardiovascular risk factors such as ageing, being overweight, insulin resistance, diabetes, and hyperlipidaemia (Messerli et al, 2007), but as these risk factors are modifiable, through regular physical activity, eating a healthier diet and by not smoking, it is possible to profoundly reduce the risk of CVD in both sexes and all age groups (Buttar et al, 2005).

Dietary fats and cholesterol play an important role in CVD development, generally through the modulating concentrations of plasma lipoproteins (Schaefer, 2002). Saturated fatty acid (SFA) intake raises low density lipoprotein (LDL) cholesterol concentrations more than other nutrients except trans fats (Siri-Tarino et al, 2010). Replacement of SFA by polyunsaturated (PUFA) or monounsaturated fat (MUFA) has been shown to lower both LDL and HDL cholesterol (Siri-Tarino et al, 2010). Low plasma HDL and high plasma LDL concentrations are considered to cause detrimental effects on the cardiovascular system such as hyperlipidaemia and hypertension, whereas increased HDL and decreased LDL concentrations have been shown to display cardio-protective effects (Gaziano & Manson, 1996). Similarly, the amount of cholesterol consumed in the diet has been shown to modulate effects of saturated fat so that, at lower intakes of cholesterol, the effect of saturated fat on LDL cholesterol was minimal in comparison to the significant LDL-cholesterol raising effects at higher concentrations of cholesterol in the diet (Siri-Tarino et al, 2010).

Physiologically, lipids play a vital role in the proper functioning of the cardiovascular system. Although the heart is fuelled in part by glucose and lactate, it predominantly and favourably uses fatty acids to meet its energy needs (Buttar et al, 2007). Up to 90% of the energy demands of the heart are fulfilled by fatty acids during starvation (Buttar et al, 2007). During a fasting state, carbohydrates provide 15% to 20% of the energy requirements, divided between glucose (approximately 5%) and lactate (approximately 10%) (Liu, 2003). The remaining energy provided by lipids is divided between triglycerides (approximately 10%) and free fatty acids (approximately 60%) (Gaziano & Manson, 1996).

Current dietary guidelines continue to recommend restricting intake of saturated fatty acids (SFA), which follows largely from the observation that SFA can increase levels of total serum cholesterol (TC), thus supposedly increasing the risk of atherosclerosis and hypertension, and the development of further cardiovascular diseases (CVD) (DiNicolantonio et al, 2015). Some food sources of SFAs may pose no risk for CHD or possibly even be protective (DiNicolantonio et al, 2015). But by reducing saturated fat intake, a replacement nutrient or nutrients would need to be incorporated more into the diet.

Substituting polyunsaturated fat instead of saturated fat reduces LDL cholesterol and the total cholesterol to high-density lipoprotein cholesterol ratio (Siri-Tarino et al, 2010). But if this replacement nutrient were carbohydrate, (particularly refined carbohydrates and added sugars) this would increase small LDL particles and triglyceride levels and reduce HDL cholesterol (Siri-Tarino et al, 2010). These effects could exacerbate the atherogenic dyslipidaemia associated with insulin resistance and obesity which could possibly contribute to the development of hypertension and further CVD's (Siri-Tarino et al, 2010).

Consequently, this study was conducted to investigate the effects of dietary saturated fat on cardiovascular health, and to assess the possible benefits of SFA in the diet rather than the detrimental effects. By doing so, this could possibly disseminate any benefits that may come from incorporating more saturated fat into the diet in replacement of other nutrients such as refined carbohydrates and sugars. The aim of this study was to determine the relationship between consuming saturated fat and the prevalence of risk factors associated with CVD, more specifically the relationship between SFA intake and arterial blood pressure.

METHOD

STUDY DESIGN AND POPULATION

25 participants aged between 18 and 30 were recruited to attend an appointment at the Health Assessment Suite at the Cardiff Metropolitan University in November 2016. Within this appointment, participants were required to undergo an examination concentrating specifically on physiological and haematological parameters of cardiovascular health. The participants were required to sign an informed consent form upon entry of the health assessment suite and briefed on what would be taking place within their appointment, giving them an opportunity to state any last-minute queries and to ensure they were still fully certain on taking part. During this, participants' date of birth was recorded. An exclusion criteria was applied during the recruitment process of the study to try and reduce varying results. This included participants who were active smokers, participants who suffered from a reduced blood clotting ability (Hemophilia), those who may have been advised not to give blood, pregnant women, diabetic participants, and participants who have a pacemaker fitted.

DATA COLLECTION

After signing the necessary documents and ensuring the participants were allowed enough time for their heart rate to return to normal, their blood pressure was measured using a Boso Medicus Uno Automatic Blood Pressure Monitor©. Their weight and height were then collected to determine their BMI, and so it could be inputted into the Bodystat 1500© body composition analyser to determine their total body fat percentage.

Following completion of these tests, participants were then invited to provide blood samples via finger prick taken by a fully qualified phlebotomist. The size of the blood samples required were 3 x 32µl pre-measured capillary tubes, and 100µl in a separate tube which were spun to separate plasma from blood. 32µl plasma was collected from each spun sample in order to measure high density lipoproteins (HDL) using the Reflotron Plus Clinical Chemistry Analyser. Using Reflotron test reagent strips (requiring 32µl sample of blood on each strip), the individuals HDL (mmol/L), total cholesterol (mmol/L), and glucose (mmol/L) levels were also determined.

Participants were also required to complete a 7-day food diary on the application 'myfitnesspal', recording each meal and portion size they ate throughout an ordinary week. This was to obtain an understanding of their dietary habits under normal circumstances, and once completed, each diet underwent analysis through Nutrimen (Dark Green Media, 2015). This allowed for a breakdown of the dietary fat each participant consumed, and thus provided a figure of average saturated fat intake which was used for comparison in the analysis of the study.

STATISTICAL METHODS

Regarding the choice of statistical analysis with the results in this study, the Pearson Correlation coefficient (a parametric test used on normally distributed data) was used to measure any possible correlations between the various physiological and haematological parameters. To determine the significance between the various sets of results, p-values were used through the programme 'Minitab' (Minitab, 2017). Any p values <0.05 were considered significant. Due to the two groups of participants being independent and the data regarded as normal, a 2-Sample T test was carried out on several variables.

ETHICAL CONSIDERATIONS

This study was conducted in line with the Declaration of Helsinki. All participant samples were handled in accordance with the HTA and ethics approval was granted by the Cardiff Metropolitan Faculty of Health Sciences Ethics Panel. All participant data remains completely anonymous in line with the Data Protection Act 1998. No participants were harmed in this study and all consent was obtained before any data was collected.

RESULTS

TABLE 1A: MALE RESULTS

Participant No.	Weight (Kg)	Height (M)	BMI	Body fat (%)	Systolic BP	Diastolic BP	PM BP	Age	% Saturated Fat
2	73.5	1.8	23	11.60%	135	72	58	19	10.87%
7	73.4	1.72	25	17%	119	64	59	20	10.30%
8	75.5	1.77	24	14.20%	136	87	62	20	11.82%
11	76.3	1.67	27	19.30%	119	92	81	22	16.14%
14	57.5	1.74	19	15%	127	65	62	20	7.97%
16	72.4	1.78	23	14.10%	134	86	60	20	15.32%
17	70.9	1.83	21	11.60%	118	70	60	24	15.48%
18	85.1	1.87	24	9.80%	123	68	74	20	9.10%
19	75.8	1.82	23	14%	128	76	70	20	15.35%
20	64.6	1.62	25	10.20%	116	70	63	19	12.37%
22	111.5	1.89	31	29.10%	144	96	69	22	17.33%
23	88.7	1.83	26	18.70%	144	75	87	21	11.42%
26	72.5	1.83	22	13.20%	132	64	54	22	12.13%

Table 1A displays the results obtained from the male participants who took part in the study.

TABLE 1B: FEMALE RESULTS

Participant No.	Weight (Kg)	Height (M)	BMI	Body fat (%)	Systolic BP	Diastolic BP	PM BP	Age	% Saturated Fat
3	65.8	1.68	23	27.70%	102	69	62	22	11%
4	55.8	1.65	20	21.50%	122	69	69	20	17.28%
5	57.1	1.61	22	19.30%	120	82	58	22	11.35%
6	79	1.73	26	29.70%	120	74	73	22	11.68%
9	68	1.67	24	32.90%	110	69	68	20	17.50%
10	70.5	1.68	25	27.90%	121	65	77	22	11.03%
12	56.5	1.55	24	25%	118	78	70	27	16.95%
13	56.3	1.63	21	27.20%	109	78	74	22	15.19%
15	86.7	1.67	19	34.80%	130	83	64	19	12.60%
21	76.2	1.7	26	26.2	120	74	65	20	18.56%
24	81.8	1.71	28	32.50%	115	78	60	21	14.24%
25	52.2	1.51	23	31.00%	98	73	95	20	10.68%

Table 1B displays the results obtained from the female participants who took part in the study.

TABLE 2A: MALE DESCRIPTIVE STATISTICS

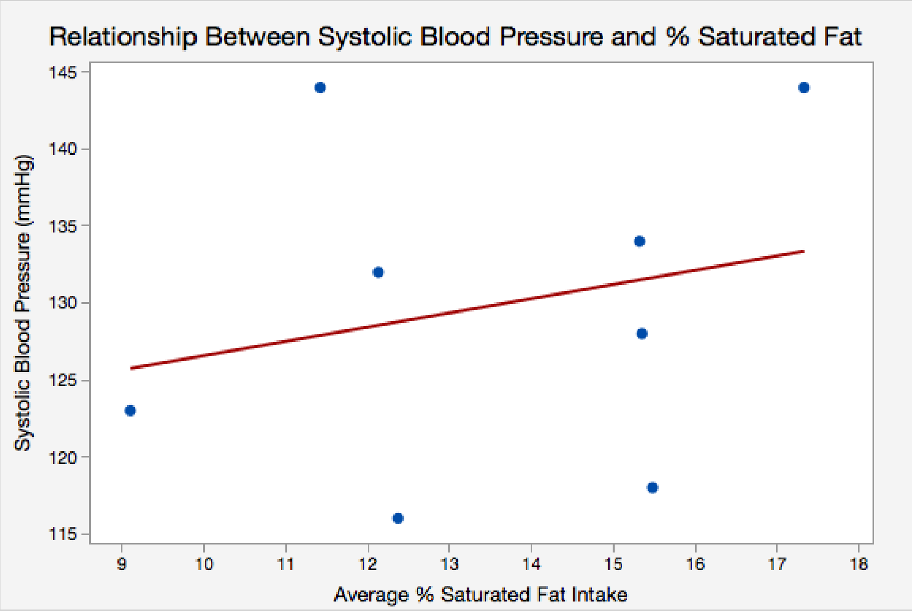
Variable	*Mean*	*SE Mean*	*ST Dev*
Age	20.69	0.3985	1.4357
Weight (kg)	76.75	3.611	13.019
Height (M)	1.78	0.02158	0.07780
Systolic BP (mmHg)	128.85	2.655	9.573
Diastolic BP(mmHg)	75.77	3.036	10.948
Heart Rate	66.08	2.685	9.682
% Dietary SFA	12.71	0.8173	2.9467
BMI	24.08	0.8201	2.9570
% Body Fat	15.21	1.417	5.109

TABLE 2B: FEMALE DESCRIPTIVE STATISTICS

Variable	*Mean*	*SE Mean*	*St Dev*
Age	21.42	0.5962	2.0652
Weight (kg)	67.16	3.390	11.745
Height (M)	1.65	0.01881	0.6515
Systolic BP (mmHg)	115.42	2.627	9.100
Diastolic BP (mmHg)	74.33	1.625	5.630
Heart Rate	69.58	2.848	9.867
% Dietary SFA	13.92	0.8527	2.9537
BMI	23.42	0.7633	2.6443
% Body Fat	27.98	1.329	4.605

Table 2A displays the descriptive statistics for male participants, and Table 2B displays the descriptive statistics for female participants. The descriptive statistics produced on Minitab Express indicate that mean systolic and diastolic blood pressure was higher in the male participants in comparison to the female participants. This was also the case for mean weight, height and BMI measurements. Mean body fat percentage was higher in females, as was participant age, heart rate, and dietary saturated fat content from the 7-day food diary analysis.

FIGURE 1A: SYSTOLIC BLOOD PRESSURE AND SATURATED FAT IN MALES

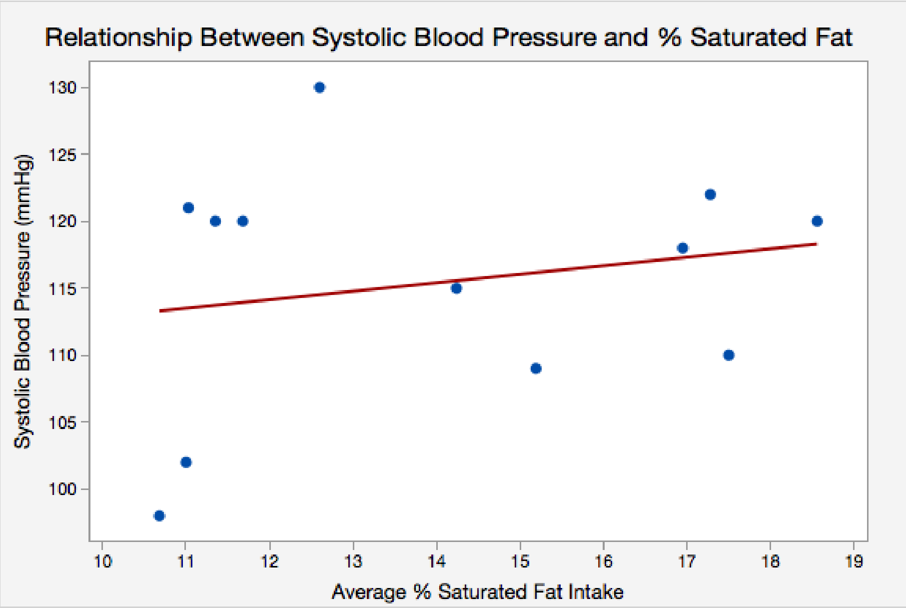


P-Value: 0.5743
R-Value: 0.235607

There is no statistical significance as P-values are >0.05

Figure 1A displays the relationship between systolic blood pressure and percentage saturated fat intake obtained from 7-day food diary analysis in male participants. The results indicate a positive linear relationship between the two variables, but with no statistical significance.

FIGURE 1B: SYSTOLIC BLOOD PRESSURE AND SATURATED FAT IN FEMALES

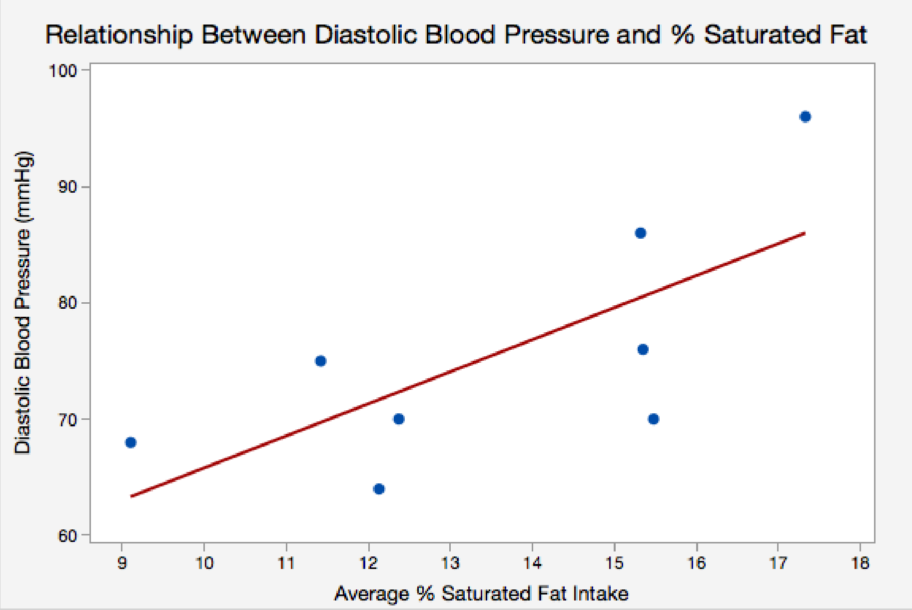


P-Value: 0.5177
R-Value: 0.207419

There is no statistical significance as P-values are >0.05

Figure 1B displays the relationship between systolic blood pressure and percentage saturated fat intake obtained from 7-day food diary analysis in female participants. The results indicate a positive linear relationship between the two variables, but with no statistical significance.

FIGURE 2A: DIASTOLIC BLOOD PRESSURE AND SATURATED FAT IN MALES

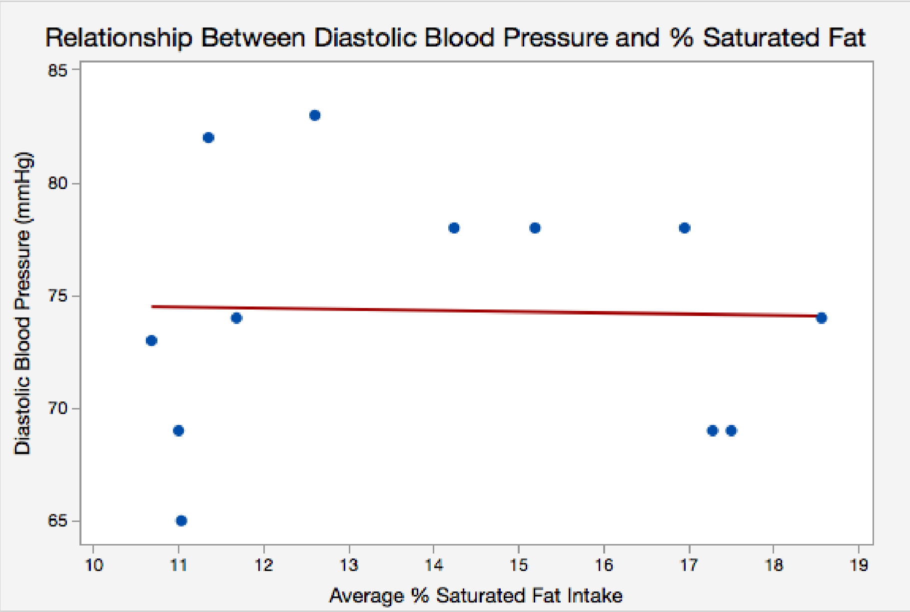


P-Value: 0.0475
R-Value: 0.712209

There is statistical significance as the P-Value is <0.05

Figure 2A displays the relationship between diastolic blood pressure and percentage saturated fat intake obtained from 7-day food diary analysis in male participants. The results indicate a positive linear relationship between the two variables, and statistical significance was found.

FIGURE 2B: DIASTOLIC BLOOD PRESSURE AND SATURATED FAT IN FEMALES

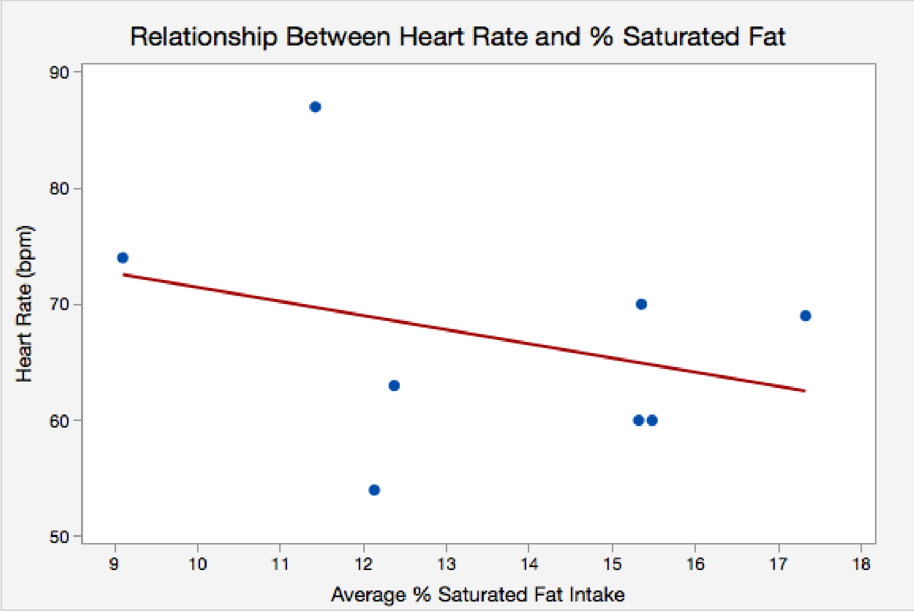


P-Value: 0.9312
R-Value: -0.027977

There is no statistical significance as the P-Value is >0.05

Figure 2B displays the relationship between diastolic blood pressure and percentage saturated fat intake obtained from 7-day food diary analysis in female participants. The results indicate a very weak, almost non-existent negative linear relationship between the two variables, and no statistical significance was found.

FIGURE 3A: HEART RATE AND SATURATED FAT IN MALES

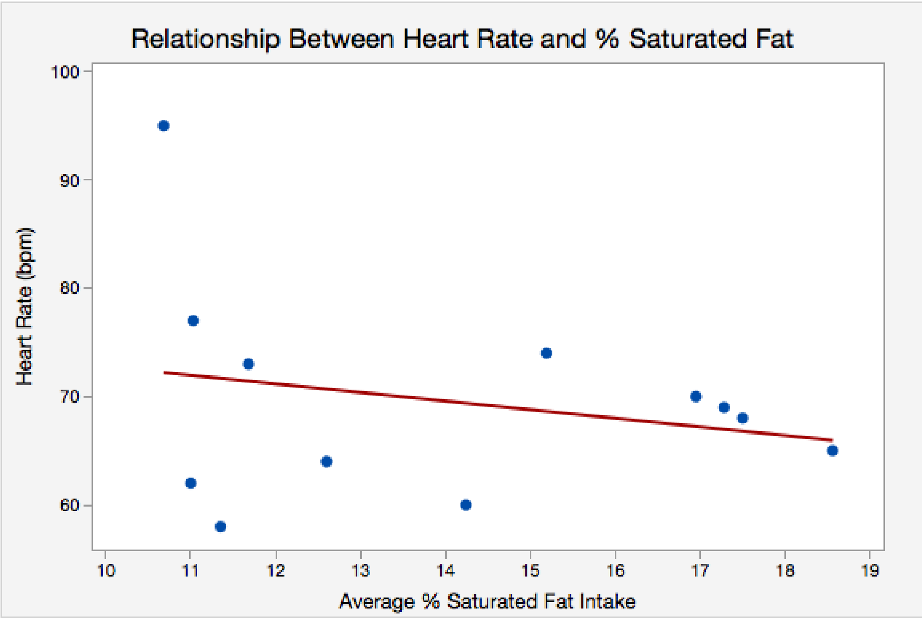


P-Value: 0.4362
R-Value: -0.322351

There is no statistical significance as the P-Value is >0.05

Figure 3A displays the relationship between heart rate and percentage saturated fat intake obtained from 7-day food diary analysis in male participants. The results indicate a negative linear relationship between the two variables, and no statistical significance was found.

FIGURE 3B: HEART RATE AND SATURATED FAT IN FEMALES

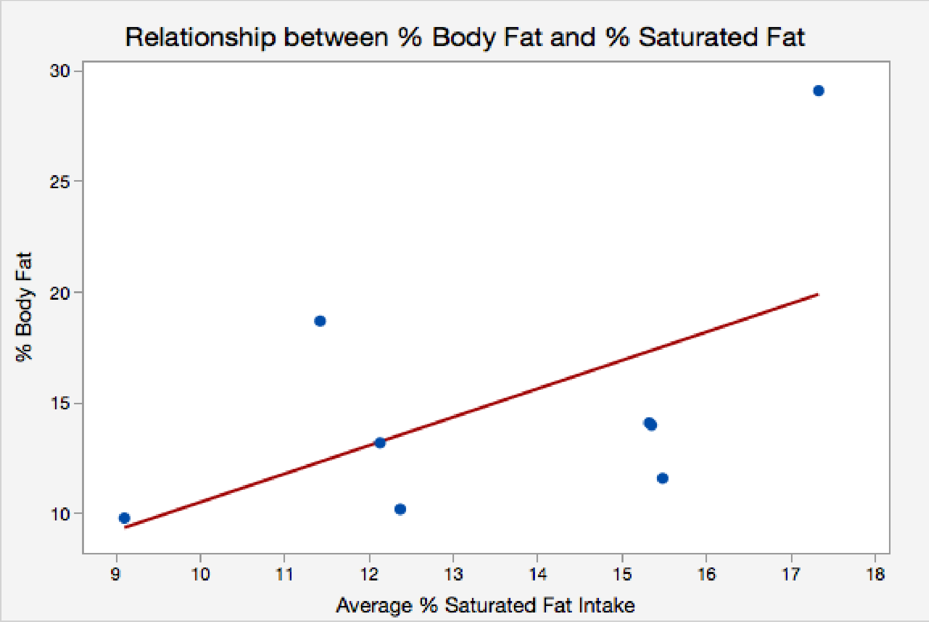


P-Value: 0.4535
R-Value: -0.239437

There is no statistical significance as the P-Value is >0.05

Figure 3B displays the relationship between heart rate and percentage saturated fat intake obtained from 7-day food diary analysis in female participants. The results indicate a negative linear relationship between the two variables, and no statistical significance was found.

FIGURE 4A: % BODY FAT AND SATURATED FAT IN MALES



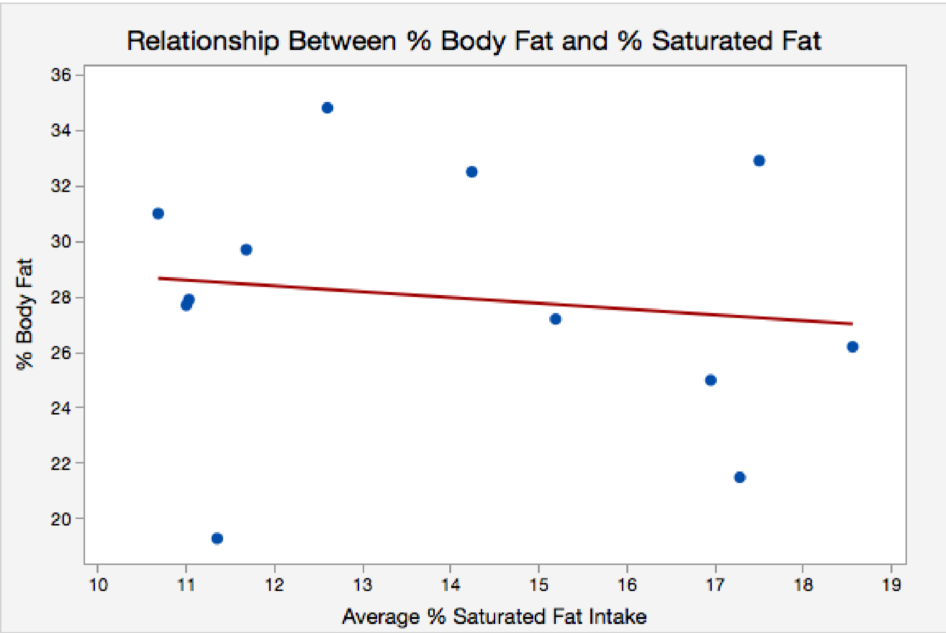
.

P-Value: 0.1543
R-Value: 0.553864

There is no statistical significance as the P-Value is >0.05

Figure 4A displays the relationship between body fat percentage in male participants and percentage saturated fat intake obtained from 7-day food diary analysis. The results indicate a positive linear relationship between the two variables, and no statistical significance was found.

FIGURE 4B: % BODY FAT AND SATURATED FAT IN FEMALES



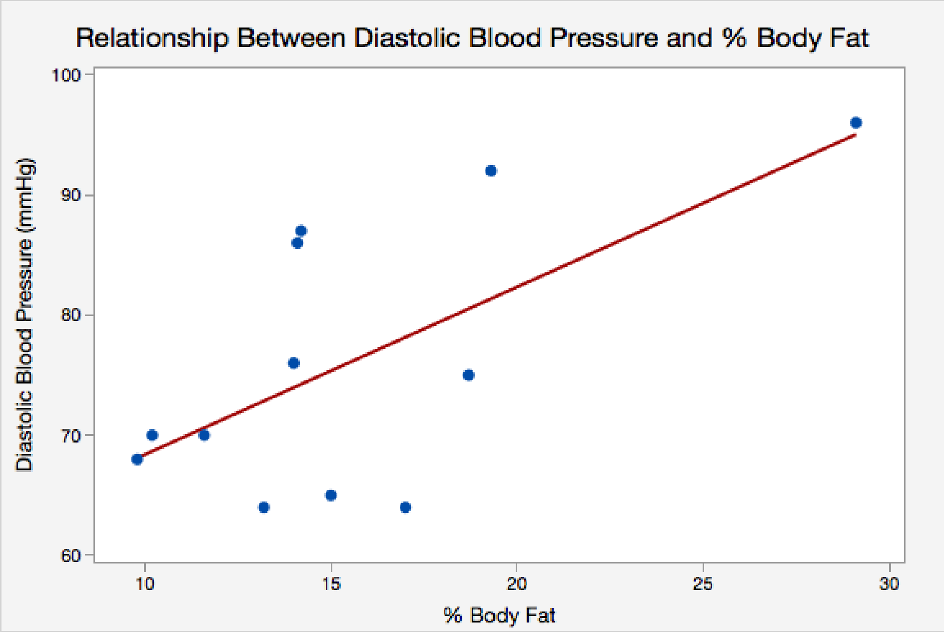
.

P-Value: 0.6766
R-Value: -0.134618

There is no statistical significance as the P-Value is >0.05

Figure 4B displays the relationship between body fat percentage in female participants and percentage saturated fat intake obtained from 7-day food diary analysis. The results indicate a negative linear relationship between the two variables, and no statistical significance was found.

FIGURE 5A: % BODY FAT AND DIASTOLIC BLOOD PRESSURE IN MALES

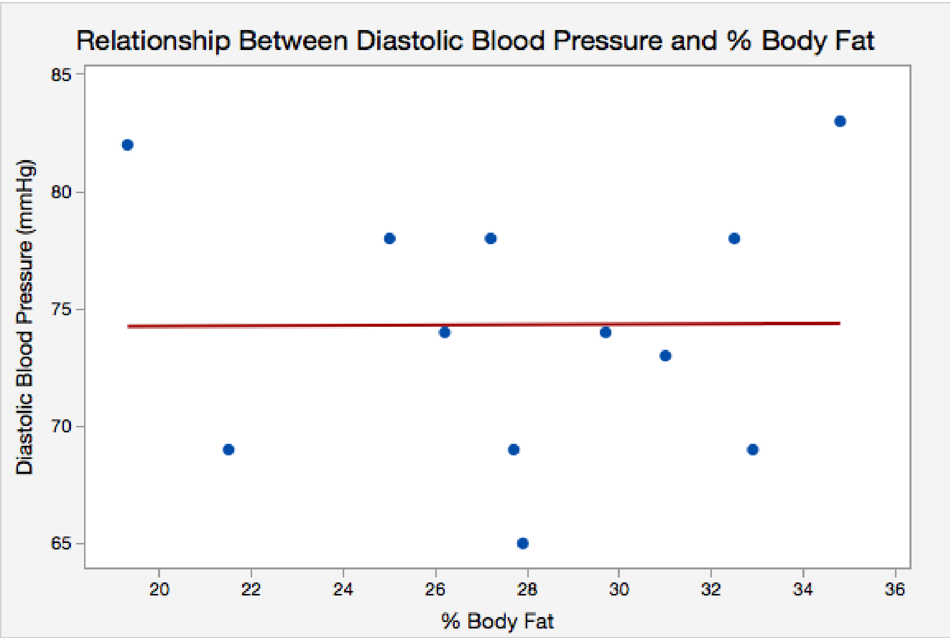


P-Value: 0.0252
R-Value: 0.639465

There is statistical significance as the P-Value is <0.05

Figure 5A displays the relationship between body fat percentage in male participants and diastolic blood pressure. This regression analysis was examined due to the findings in Figure 2A, and to see if body fat percentage in males had a similar relationship with diastolic blood pressure. The results display a positive linear relationship between the two variables, and statistical significance was found.

FIGURE 5B: % BODY FAT AND DIASTOLIC BLOOD PRESSURE IN FEMALES



P-Value: 0.9836
R-Value: 0.006662

There is no statistical significance as the P-Value is >0.05

Figure 5B displays the relationship between body fat percentage in females and diastolic blood pressure. The results indicate that there is no linear relationship between the two variables, and no statistical significance was found.

TABLE 5: P AND R VALUES

GENDER	SYST BP VS SAT FAT	DIAS BP VS % SAT FAT	HEART RATE VS % SAT FAT	% BODY FAT VS % SAT FAT	% BODY FAT VS DIAS BP
Male	P = 0.5743 R = 0.235607	P = 0.0475 R = 0.712209	P = 0.4362 R = -0.322351	P = 0.1543 R = 0.553864	P = 0.0252 R = 0.639465
Female	P =: 0.5177 R = 0.207419	P = 0.9312 R = -0.027977	P = 0.4535 R = -0.239437	P = 0.6766 R = -0.134618	P = 0.9836 R = 0.006662

Table 5 displays the P and R values which were obtained from the regression analysis and Pearson correlation coefficient analysis. The results determined that there was statistical significance between diastolic blood pressure and percentage dietary saturated fat in males, and body fat percentage and diastolic blood pressure also in males. No statistical significance was found between any of the variables regarding the female participant results.

TABLE 6A: MALE MEAN ENERGY INTAKES

PARTICIPANT NO.	*MEAN ENERGY INTAKES (KCAL)*
2	1403
7	2616
8	1915
11	2437
14	1718
16	2064
17	3429
18	1839
19	1790
20	1745
22	1357
23	2204
26	2451

Mean Energy Intakes of Male Participants

Table 6A displays the mean energy intakes calculated on Nutrimen (Dark Green Media, 2015) derived from the 7-day food diaries from male participants.

TABLE 6B: **MEAN FEMALE ENERGY INTAKES**

PARTICIPANT NO.	*MEAN ENERGY INTAKES (KCAL)*
3	1173
4	1194
5	784
6	1966
9	1585
10	1446
12	2310
13	1130
15	1582
21	1496
24	1180
25	1445

Table 6A displays the mean energy intakes calculated on Nutrimen (Dark Green Media, 2015) derived from the 7-day food diaries from female participants.

DISCUSSION

This study was conducted to investigate the effects of dietary saturated fat on cardiovascular health. The main aim was to determine if there was a relationship between consuming saturated fat and whether it affected the prevalence of risk factors associated with CVD, specifically arterial blood pressure. The findings in this study suggest that a higher saturated fat content within the diet significantly increases blood pressure, more specifically diastolic blood pressure in males. Following this, it was found that participants with a higher body fat percentage were also found to have a significantly higher diastolic blood pressure.

Figure 1A shows a positive correlation between resting systolic blood pressure and the average dietary saturated fat consumed. This could suggest that a higher amount of dietary saturated fat within a male participant's diet could increase their systolic blood pressure, but no statistical significance was found (P-Value=>0.05). With Figure 1B, a similar result was found in females, but the correlation was not as steep. The R-values of both genders (M=0.235607. F=0.207419) indicates a weak positive linear relationship within these cardiovascular risk factors, and no statistical significance was found in the female participants either due to the P value = >0.05.

The positive correlation displayed in Figure 2A shows the relationship between resting diastolic blood pressure and the average dietary saturated fat consumed in male participants. The R-value (R=0.712209) indicates that there is a strong positive linear relationship between the two variables, and the P-value suggests that there is statistical significance present due to it being <0.05 (P = 0.0475). This could suggest that saturated fat may actively elevate resting diastolic blood pressure levels in men, but other variables would have to be firstly considered. Figure 2B suggests an entirely different observation regarding the results, displaying no signs of an obvious relationship between the two variables. There was no statistical significance present (P=>0.05) between resting diastolic blood pressure and the average dietary saturated fat consumed in females, and the R-value indicates that there is a slight but almost no linear relationship (R=-0.027977) between the two variables. These findings suggest that saturated fat could possibly have a detrimental effect on diastolic blood pressure in males, but not necessarily in females.

It should be noted that participants' daily physical activity levels were not recorded. This may have affected some physiological parameters, for example a sedentary individual may naturally have had a slightly higher resting heart rate than that of a very active individual, but precautions were taken to ensure that participants' heart rate had returned to normal before blood pressure and heart rate readings were obtained. Figure 3A displays a negative correlation between heart rate and the average dietary saturated fat consumed in male participants. This relationship displayed no statistical significance (P=>0.05) and the R-value indicated that the two variables have a weak negative linear relationship (R=-0.322351). Figure 3B showed similar results, with no statistical significance (P=>0.05) and displaying a weak negative linear relationship (R=-0.239437). These findings suggest that the effect of saturated fat within the diet may have the same effect on both genders, in the sense that it may not noticeably alter or affect resting heart rate.

Body composition is very different within the two genders. Women generally have a higher body fat percentage due to fatty deposits such as their breasts in comparison to males who tend to have a naturally higher regional fatty acid metabolism. Therefore, it isn't alarming that Figure 4A and 4B display such varying results. The relationship between the two variables in the male participants suggests that the more dietary saturated fat incorporated into a male's diet, the higher their body fat percentage. The R-value (R=0.553864) indicates a moderately positive relationship between the two variables, but no statistical significance was found (P=>0.05). This differs in the female participants group, in which Figure 4B displays a slightly negative but almost no relationship. The R-Value (R=-0.134618) shows that the two variables share a weak negative linear relationship, and no statistical significance was found (P=>0.05). This gives the impression that the amount of saturated fat consumed in the diet doesn't necessarily affect body composition in females.

Due the statistical significance found in males between average dietary saturated fat consumed and diastolic blood pressure, a further test was done to investigate whether body fat percentage had a similar effect on diastolic blood pressure. Figure 5A displays an evident positive correlation between these two variables, suggesting that the higher a male's body fat percentage, the higher their diastolic blood pressure may be. The R-value (R=0.639465) indicates that there is a moderately strong positive linear relationship, and statistical significance between the two variables was also found (P=0.0252). The expected differences in results between the male and female participants also occurred within these two variables. Due to women having a naturally higher body fat percentage within their body composition, no statistical significance was found (P=>0.05) and the R-Value (0.006662) indicated that there was essentially no linear relationship.

The results of this study strongly indicate gender related differences in response to dietary SFA. It appears that dietary SFA affects men more detrimentally than women in terms of their resting blood pressure, suggesting that men are at a higher risk of developing risk factors associated with CVD. However, other variables would have to be investigated, for example their daily activity levels or other possible underlying medical problems possibly derived from genetics.

The efficiency in which the data was collected allowed for an easy and stress free experience for each participant as well as the researchers. Appointments were made a month in advance and participants were send reminders a week prior to their appointment, as well as the day before. No participants cancelled their appointments or didn't show up, and only one participant chose to withdraw from the study (participant no.1). The only questionable factor within the health assessment period was whether individuals were honest about their overnight fast, as some of the blood glucose readings were slightly higher than expected.

One of the requirements of the participants in this study included completing a 7-day food diary. This was to determine how much saturated fat the participants consumed in their daily lives, under the assumption that they recorded their diet on an ordinary week with no unusual dietary adaptations, for example dieting to lose weight. Using this method of dietary recall demonstrated a few minor issues that could have affected the overall result of the study.

The recall of diaries appeared to be somewhat of a burden to a small percentage of participants, displaying a slight lack of interest and enthusiasm when completing their entries. Some did not appear entirely honest in what they were eating, whether it was lack of detail or the fact they felt they should have omitted unhealthy food groups on purpose. For example, simply entering 'bread' as a breakfast option, but when asked in person what they truly meant, they had consumed toast with butter and jam. This can be observed by looking at table 7A and 7B which indicates that participants' mean energy intake is questionably low with some of the results. This in turn may have resulted in obtaining a lower saturated fat content that what they truly consume on a regular basis. However, this was not the case for all participants, and most of the diaries were completed with the utmost detail. Most of the diaries displayed evident thoroughness which allowed for a more accurate representation of their true dietary habits and saturated fat percentage.

Another way in which inaccuracies may have presented themselves in the study was analysing the food diaries through using Nutrimen (Dark Green Media, 2015). When inputting some of the foods, nutrimen did not offer specific brands or measurements that matched the participants' diaries. For example, when inputting a liquid, the only measurement available was in grams, so inaccuracies may have presented themselves in the sense of over compensating for how much was factually consumed. Due to this, presumptions on portion sizes were made on several items from the food diaries.

The current knowledge surrounding dietary SFA and cardiovascular risk is that dietary SFA intake increases the risk of CVD. This effect is understood to be mediated predominantly by increased LDL-cholesterol concentrations, but other variables are in question within this study, such as its effect on resting arterial blood pressure.

Past research investigated the effect of dietary SFA, MUFA's and PUFA's on resting systolic and diastolic blood pressure in healthy subjects. Within the study, two groups were assigned diets rich in either SFA or MUFA's to see if there were any mediations in blood pressure. The study concluded that both systolic and diastolic blood pressure levels in the individuals did not change with the SFA diet (Rasmussen et al, 2006). These results could indicate that the significant findings on figure 2A are not solely based on saturated fat content in diet, and other lifestyle variables may factor into why there is a positive linear relationship present. For example, the carbohydrate percentage in most the participants' diets shows that it makes up for almost half of their dietary intake.

DIETARY SATURATED FAT, HEART RATE AND BLOOD PRESSURE

Past pathophysiological studies have indicated that an elevated resting heart rate (HR) has direct detrimental effects on the development of CVD's such as coronary atherosclerosis, on the incidence of myocardial ischemia and ventricular arrhythmias, and on left ventricular function (Fox et al, 2007). This relationship appears generally stronger in males when compared to females (Perret-Guillaume et al, 2009). Literature explains that although average life span is modestly longer in women than in men, it is well documented that, when adjusted for age, HR in women averages 2 to 7 beats/min higher than in men (Fox et al, 2007). The results in this study support these facts, illustrating that the female participants have a higher mean resting HR.

Additionally, the findings in this study show no direct relationship between dietary saturated fat intake and resting heart rate in both groups of participants, suggesting that it may not necessarily affect it. However, a repeatedly elevated heart rate is generally associated with an increase in cardiovascular risk through the suggestion that it is comparable to an increase in risk observed with elevated blood pressure (Perret-Guillaume et al, 2009). The findings in this study indicated a strong association between dietary saturated fat intake and increased diastolic blood pressure in men.

Past literature argues that dietary SFA increases CVD risk through the mechanism of increasing plasma LDL-cholesterol concentrations (Siri-Tarino et al, 2010). It has been well accepted that elevated serum levels of LDL-cholesterol coupled with elevated blood pressure play an important role in the initiation and progression of developing atherosclerosis (Badimon & Vilahur, 2012).

Research investigating the effects of SFA's, MUFA's and PUFA's on blood pressure found that substituting dietary SFA for MUFA significantly lowered diastolic blood pressure, but this result was negated when total fat intake was increased (Rasmussen et al, 2006). This suggests that replacing SFA's with MUFA's within the diet could be beneficial, but would be disregarded if their total fat intake was higher than the dietary recommendation (<30% total energy intake). The blood pressure reducing effect of n-3 PUFAs (docosahexaenoic acid) has also been demonstrated in past research (Mori, 2006). However, a meta-analysis of prospective cohort studies investigating the association of SFA with CVD found that there was no association between SFA and an increased risk of developing CHD, stroke or CVD (Siri-Tarino et al, 2010).

Additionally, past research conducted on mildly hypertensive patients investigated the effects of replacing dietary SFA with PUFA (linoleic acid) and carbohydrates (Sacks et al, 1987). The findings of the study determined that there were no significant differences in blood pressure amongst the participants, suggesting that replacement of dietary SFA with carbohydrate or PUFA doesn't affect resting blood pressure (Sacks et al, 1987). This refutes the findings of this study in which Figure 2A displays statistical significance between diastolic blood pressure and dietary SFA intake. More research is required to elucidate whether CVD risks are likely to be influenced by the specific nutrients used to replace dietary SFA (Siri-Tarino et al, 2010).

BLOOD PRESSURE AND BODY FAT PERCENTAGE

Current literature explains that women generally have a higher body fat percentage than men when it comes to body composition. The predictor ranges for women when undergoing body composition analysis are <10% higher than men. The female body stores more fat in the gluteal-femoral region, in comparison to men who store more fat in their abdominal area (Blaak, 2001). This is due to the pronounced regional differences in the regulation of regional fatty acid metabolism between males and females, for example the release of catecholamine mediated lower limb free fatty acids is higher in males compared to females (Blaak, 2001). The release of fatty acids by the upper body subcutaneous fat deposits is also higher in males than in females. This indicates a higher resistance to the anti-lipolytic effect of meal ingestion in the upper body fat deposits in men (Blaak, 2001). There is also evidence to suggest that basal fat oxidation is lower in females than males, therefore contributing to a higher storage of fat in women (Blaak, 2001). The results found in this study support this current knowledge, displaying strong differences in the relationship between body fat percentage between males and females.

A recent cross-sectional study conducted on middle aged, healthy, normal-weight adults investigated the implications of having a high body fat percentage in relation to CV risk factors (Kim et al, 2013). The findings of the study found that only male subjects with a high body fat percentage had a significantly higher prevalence of developing high blood pressure (hypertension) in comparison to females (Kim et al, 2013). These findings support the results found in this current study regarding male participants, as Figure 4A displays statistical significance between increased dietary SFA intake and body fat percentage. This in turn supports the statement that high amounts of dietary SFA may have a detrimental effect on diastolic blood pressure in males. If their body fat percentage rises in accordance with increased dietary SFA intake, males appear to be more susceptible to developing hypertension as well as other CV risk factors.

OVERALL STUDY OBSERVATIONS

This current study may have indicated a relationship between dietary SFA and diastolic blood pressure, and a further link between body fat percentage and diastolic blood pressure. Both variables interlink in the complex pathogenesis of CVD, and this could point in the direction that dietary SFA should indeed be reduced in line with current recommendations. However, this study does not prove any direct causal links between CVD and dietary SFA consumption, not does it link dietary SFA consumption to hypertension.

Past research and current literature has explained how there is no direct causal link between dietary SFA intake and CVD, and that elevated blood pressure and hypertension usually clusters with other CVD risk factors such as ageing and being overweight (Messerli et al, 2007). Current dietary guidelines suggest a diet low in fat, but recommendations don't generally specify what the replacement nutrients should be, whether it be carbohydrate, protein etc. Replacing dietary SFA for PUFA's reduces LDL-cholesterol and the total cholesterol to HDL-cholesterol ratio (Siri-Tarino et al, 2010). However, replacing dietary SFA with carbohydrates (more specifically refined carbohydrates and added sugars) has been shown to increase triglyceride concentrations as well as small LDL-particles and reduce HDL-cholesterol (Siri-Tarino et al, 2010). In the context of the increased prevalence of obesity and insulin resistance, these effects are concerning for the development of CVD risk factors. Furthermore, a meta-analysis of randomised control trials (RCT) of clinical CHD events, coupled with consistent findings from both prospective cohort studies of clinical CHD events and RCTs of intermediate risk factors, offered strong evidence that replacing SFA for PUFA's does indeed lower the risk of CVD development (Mozaffarian et al, 2010).

Therefore, this study agrees with the notion that dietary recommendations should emphasize substitution of PUFA and minimally processed grains for SFA, and concentrate on spreading the message of reducing *trans* fats, refined carbohydrates and foods high in sugar. More specific research needs to be completed to find a specific recommendation for how much dietary SFA is healthy in the diet.

CONCLUSION

The results of this study strongly indicate gender related differences in response to dietary SFA. It appears that dietary SFA affects men more detrimentally than women in terms of their resting blood pressure, suggesting that men are at a higher risk of developing risk factors associated with CVD. However, other variables would have to be investigated, for example their daily activity levels or other possible underlying medical problems. Evaluation of the association of SFA with risk of CVD development requires heavy consideration of the nutrients in its place. Past research supports the notion that replacing SFA with PUFA's may result in a decreased risk of CVD development, through improved lipid profiles (Siri-Tarino et al, 2010). More observational studies need to be conducted to investigate the relationship between blood pressure and dietary SFA intake, and to determine whether there is a direct causal link between dietary SFA intake and hypertension. To summarise, this study found links between diastolic blood pressure in men and dietary SFA intake, and diastolic blood pressure and males' percentage body fat. Given the current situation in the UK regarding the obesity epidemic, public health dietary goals should concentrate more on decreasing the consumption of *trans* fats, refined carbohydrates and sugars and increase obesity prevention and dietary awareness.

REFERENCES

Badimon, L. & Vilahur, G. (2012) 'LDL-cholesterol versus HDL-cholesterol in the atherosclerotic plaque: inflammatory resolution versus thrombotic chaos', *Ann N Y Acad Sci.,* Vol.1254 pp. 18-32.

Beaglehole, R. and Bonita, R. (2008) 'Global public health: A scorecard', *Lancet (London, England).,* Vol.372(9654), pp. 1988–96.

Blaak, E. (2001) 'Gender Differences in Fat Metabolism', *Curr Opin Clin Nutr Metab Care., Vol.4(6) pp.499-502.*

British Heart Foundation (2015) *Cardiovascular disease statistics 2015*. Available at: https://www.bhf.org.uk/publications/statistics/cvd-stats-2015 (Accessed: 22 February 2017).
Buttar, H.S., Li, T. and Ravi, N. (2005) 'Prevention of cardiovascular diseases: Role of exercise, dietary interventions, obesity and smoking cessation', Vol.10(4).

DiNicolantonio, J., Lucan, S. and O'Keefe, J. (2015) 'The evidence for saturated fat and for sugar related to coronary heart disease', *Progress in cardiovascular diseases.*, Vol.58(5), pp. 464–72.

Fox, K. Borer, JS. Camm, SJ. Danchin, N. Ferrari, R. & Lopez Sendon, JL. (2007) 'Resting Heart Rate in Cardiovascular Disease' *Journal of the American College of Cardiology*., Vol.50(9) pp.823-830.

Gaziano, J. and Manson, J. (1996) 'Diet and heart disease. The role of fat, alcohol, and antioxidants', *Cardiology clinics.*, Vol.14(1), pp. 69–83.

Kim, JY. Han, SH. & Yang, BM. (2013) 'Implication of high body fat percentage on cardio metabolic risk in middle aged, healthy, normal-weight adults' *Obesity (Silver Spring),* Vol.21(8), pp. 1571-7.

Liu, R. (2003) 'Health benefits of fruit and vegetables are from additive and synergistic combinations of phytochemicals', *The American journal of clinical nutrition.*, Vol.78.
Mendis, S. (2010) 'The contribution of the Framingham heart study to the prevention of cardiovascular disease: A global perspective', *Progress in cardiovascular diseases.*, Vol.53(1), pp. 10–4.

Messerli, F., Williams, B. and Ritz, E. (2007) 'Essential hypertension', *Lancet (London, England).,* Vol.370(9587), pp. 591–603.

Mori, T. (2006) 'Omega-3 fatty acids and hypertension in humans', *Clin Exp Pharmacol Physiol.*, Vol.33(9) pp.842-6.

Mozaffarian, D. Micha, R. Wallace, S. 'E ffects on Coronary Heart Disease of Increasing Polyunsaturated Fat in Place of Saturated Fat: A Systematic Review and Meta-Analysis of Randomized Controlled Trials', *PLoS Med.*, Vol. 7(3) e1000252.

Perret-Guillaume, C. Joly, L. & Benetos, A. (2009) 'Heart rate as a risk factor for cardiovascular disease' *Progressive Cardiovscular Disease.,* Vol. 52(1) pp.6-10

Rasmussen, BM. Vessby, B. Uusitupa, M. Berglund, L. Pedersen, E. Riccardi, G. Rivellese, AA. Tapsell, L. Hermansen, K & KANWU Study Group (2006) 'Effects of dietary saturated, monounsaturated, and n-3 fatty acids on blood pressure in healthy subjects', *Am J Clin Nutr.,* Vol.83(2) pp.221-6.

Sacks, F., Rouse, I., Stampfer, M., Bishop, L., Lenherr, C. and Walther, R. (1987). Effect of dietary fats and carbohydrate on blood pressure of mildly hypertensive patients. *Hypertension,* Vol.10(4), pp.452-460.

Schaefer, E. (2002) 'Lipoproteins, nutrition, and heart disease', *The American journal of clinical nutrition.,* Vol.75(2), pp. 191–212.

Siri-Tarino, P., Sun, Q., Hu, F. and Krauss, R. (2010) 'Saturated fat, carbohydrate, and cardiovascular disease', *The American journal of clinical nutrition.,* Vol.91(3), pp. 502–9.

Siri-Tarino, P.W., Sun, Q., Hu, F.B. and Krauss, R.M. (2010) 'Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease' *The American journal of clinical nutrition*., Vol.91(3) pp.535-46.

Siri-Tarino, P.W., Sun, Q., Hu, F.B. and Krauss, R.M. (2010) 'Saturated fatty acids and risk of coronary heart disease: Modulation by replacement nutrients', *The American journal of clinical nutrition.,* Vol.12(6). pp.384-90.

APPENDIX

TABLE 7: T TESTS

Variable	*T-Value*	*DF*	*P-Value*
Systolic BP (mmHg)	-3.60	22	0.0016
Diastolic BP (mmHg)	-0.42	18	0.6817
PM BP	0.90	22	0.3801
% Saturated Fat	1.03	22	0.3161
% Body Fat	6.57	22	<0.0001

Table 6 displays the results obtained from the 2-sample T-tests carried out on Minitab.

PARTICIPANTS’ ENERGY DISTRIBUTION BY NUTRIENT

Participant no.2

Nutrient	**% contrib**	**DRV**
Total carbohydrate	40.85	50.0
Protein	31.52	-
Saturated fat	10.87	11.0
Polyunsaturated fat	4.8	6.5
Monounsaturated fat	11.36	13.0
Trans fat	0.6	2.0

Participant no. 3

Nutrient	**% contrib**	**DRV**
Total carbohydrate	39.29	50.0
Protein	24.24	-
Saturated fat	11.0	11.0
Polyunsaturated fat	7.17	6.5
Monounsaturated fat	17.97	13.0
Trans fat	0.33	2.0

Participant no. 4

Nutrient	% contrib	DRV
Total carbohydrate	45.79	50.0
Protein	17.77	-
Saturated fat	17.52	11.0
Polyunsaturated fat	3.55	6.5
Monounsaturated fat	14.53	13.0
Trans fat	0.83	2.0

Participant no. 5

Nutrient	% contrib	DRV
Total carbohydrate	49.57	50.0
Protein	18.87	-
Saturated fat	11.35	11.0
Polyunsaturated fat	7.19	6.5
Monounsaturated fat	12.6	13.0
Trans fat	0.43	2.0

Participant no. 6

Nutrient	% contrib	DRV
Total carbohydrate	36.55	50.0
Protein	33.02	-
Saturated fat	11.68	11.0
Polyunsaturated fat	5.69	6.5
Monounsaturated fat	12.62	13.0
Trans fat	0.42	2.0

Participant no. 7

Nutrient	% contrib	DRV
Total carbohydrate	46.22	50.0
Protein	27.03	-
Saturated fat	10.3	11.0
Polyunsaturated fat	5.48	6.5
Monounsaturated fat	10.71	13.0
Trans fat	0.26	2.0

Participant no. 8

Nutrient	% contrib	DRV
Total carbohydrate	46.07	50.0
Protein	19.59	-
Saturated fat	11.82	11.0
Polyunsaturated fat	8.4	6.5
Monounsaturated fat	13.81	13.0
Trans fat	0.31	2.0

Participant no. 9

Nutrient	% contrib	DRV
Total carbohydrate	41.74	50.0
Protein	17.28	-
Saturated fat	17.5	11.0
Polyunsaturated fat	7.74	6.5
Monounsaturated fat	15.15	13.0
Trans fat	0.59	2.0

Participant no. 10

Nutrient	% contrib	DRV
Total carbohydrate	50.0	50.0
Protein	17.67	-
Saturated fat	11.36	11.0
Polyunsaturated fat	6.93	6.5
Monounsaturated fat	13.61	13.0
Trans fat	0.43	2.0

Participant no. 11

Nutrient	% contrib	DRV
Total carbohydrate	37.73	50.0
Protein	28.95	-
Saturated fat	16.14	11.0
Polyunsaturated fat	5.88	6.5
Monounsaturated fat	10.96	13.0
Trans fat	0.34	2.0

Participant no. 12

Nutrient	% contrib	DRV
Total carbohydrate	43.63	50.0
Protein	11.91	-
Saturated fat	16.95	11.0
Polyunsaturated fat	9.76	6.5
Monounsaturated fat	17.68	13.0
Trans fat	0.08	2.0

Participant no. 13

Nutrient	% contrib	DRV
Total carbohydrate	40.39	50.0
Protein	19.61	-
Saturated fat	15.19	11.0
Polyunsaturated fat	7.81	6.5
Monounsaturated fat	16.48	13.0
Trans fat	0.52	2.0

Participant no. 14

Nutrient	% contrib	DRV
Total carbohydrate	52.47	50.0
Protein	23.87	-
Saturated fat	7.97	11.0
Polyunsaturated fat	4.05	6.5
Monounsaturated fat	11.26	13.0
Trans fat	0.38	2.0

Participant no. 15

Nutrient	% contrib	DRV
Total carbohydrate	49.75	50.0
Protein	15.0	-
Saturated fat	12.6	11.0
Polyunsaturated fat	6.93	6.5
Monounsaturated fat	15.2	13.0
Trans fat	0.52	2.0

Participant no. 16

Nutrient	% contrib	DRV
Total carbohydrate	48.84	50.0
Protein	11.9	-
Saturated fat	15.32	11.0
Polyunsaturated fat	6.56	6.5
Monounsaturated fat	16.84	13.0
Trans fat	0.53	2.0

Participant no. 17

Nutrient	% contrib	DRV
Total carbohydrate	47.21	50.0
Protein	16.76	-
Saturated fat	15.48	11.0
Polyunsaturated fat	5.28	6.5
Monounsaturated fat	14.74	13.0
Trans fat	0.53	2.0

Participant no. 18

Nutrient	% contrib	DRV
Total carbohydrate	39.99	50.0
Protein	25.36	-
Saturated fat	9.1	11.0
Polyunsaturated fat	8.93	6.5
Monounsaturated fat	16.32	13.0
Trans fat	0.29	2.0

Participant no. 19

Nutrient	% contrib	DRV
Total carbohydrate	42.87	50.0
Protein	19.15	-
Saturated fat	15.46	11.0
Polyunsaturated fat	6.74	6.5
Monounsaturated fat	15.61	13.0
Trans fat	0.18	2.0

Participant no. 20

Nutrient	% contrib	DRV
Total carbohydrate	40.19	50.0
Protein	19.93	-
Saturated fat	12.37	11.0
Polyunsaturated fat	9.76	6.5
Monounsaturated fat	16.92	13.0
Trans fat	0.83	2.0

Participant no. 21

Nutrient	% contrib	DRV
Total carbohydrate	46.62	50.0
Protein	18.11	-
Saturated fat	18.56	11.0
Polyunsaturated fat	4.6	6.5
Monounsaturated fat	11.82	13.0
Trans fat	0.29	2.0

Participant no. 22

Nutrient	% contrib	DRV
Total carbohydrate	35.69	50.0
Protein	20.09	-
Saturated fat	17.33	11.0
Polyunsaturated fat	9.05	6.5
Monounsaturated fat	17.19	13.0
Trans fat	0.64	2.0

Participant no. 23

Nutrient	% contrib	DRV
Total carbohydrate	46.1	50.0
Protein	23.39	-
Saturated fat	11.42	11.0
Polyunsaturated fat	6.55	6.5
Monounsaturated fat	12.27	13.0
Trans fat	0.28	2.0

Participant no. 24

Nutrient	% contrib	DRV
Total carbohydrate	42.18	47.0
Protein	25.51	-
Saturated fat	14.24	10.0
Polyunsaturated fat	4.97	6.0
Monounsaturated fat	12.27	12.0
Trans fat	0.82	2.0

Participant no. 25

Nutrient	% contrib	DRV
Total carbohydrate	50.57	50.0
Protein	21.85	-
Saturated fat	10.68	11.0
Polyunsaturated fat	5.23	6.5
Monounsaturated fat	11.15	13.0
Trans fat	0.52	2.0

Participant no. 26

Nutrient	% contrib	DRV
Total carbohydrate	44.15	50.0
Protein	19.95	-
Saturated fat	12.13	11.0
Polyunsaturated fat	6.43	6.5
Monounsaturated fat	16.95	13.0
Trans fat	0.39	2.0

ACKNOWLEDGMENTS

Thank you to the following researchers and supervisors for making this project possible.

Laura Watkeys
Fully qualified phlebotomist responsible for finger prick sample collection.
Supervisor in the health assessment suite throughout data collection.

Dr. Peter Watkins
Overall project leader

Adrian Hayward & Ben Desseta
Joint Project Researchers

Thank you to all participants that volunteered to take part in this study.

ETHICS APPROVAL LETTER (10/08/2016)

Lakin, Charlotte BSc (Hons) Biomedical Sciences Cardiff School of Health Sciences

Dear Applicant

Re: Application for Ethical Approval: Is saturated fat bad for you?

Ethics Reference Number: 8262

Your ethics application, as shown above, was considered by the Biomedical Sciences Ethics Panel on 10/5/2016. I am pleased to inform you that your application for ethical approval was **APPROVED,** subject to the conditions listed below – *please read carefully.*

Standard Conditions of Approval

Your Ethics Application has been given a Project Reference number as above. This **MUST** be quoted on all documentation relating to the project (E.g. consent forms, information sheets), together with the full project title. All documents must also have the approved University Logo and the Version number in addition to the reference and project title as above.

A full **Risk Assessment** must be undertaken for this proposal, as appropriate, and be made available to the Committee if requested. Any changes in connection to the proposal as approved must be referred to the Panel/Committee for consideration ***without delay quoting your Project Reference Number***. Changes to the proposed project may have ethical implications and so must be approved.

Any untoward incident which occurs in connection with this proposal must be reported back to the Panel/Committee ***without delay***. If your project involves the use of human samples, your approval is given on the condition that you or your supervisor notify the HTA Designated Individual of your intention to work with such material by completing the form entitled "Notification of Intention to Work with Human Samples". The form must be submitted to the PD (Sean Duggan), BEFORE any activity on this project is undertaken   This approval expires on **10/5/2017** . Please set a reminder on your Outlook calendar or equivalent if you need to continue beyond this approval date. It is your responsibility to reapply / request extension if necessary.   Yours sincerely

Dr Rachel Adams Chair of BMS Ethics Panel Cardiff School of Health Sciences

Tel : 029 20416855 E-mail : radams@cardiffmet.ac.uk Cc: Watkins, Peter