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Quantifying the Association Between Diet and coronary Heart Disease Risk in the United States

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Abstract

Background

Coronary Heart Disease (CHD) is one of the most pressing medical issues facing our society today. One of the leading influencing factors in the development of CHD is diet. Notably, certain fatty acids have been shown to have a direct relationship with cholesterol levels, an important CHD risk factor. Thus, one potential CHD prevention method is diet alteration. This paper investigates how dietary change affects CHD risk on a national scale.

Methods

Multiple-linear regression was used to construct a model relating dietary habits and a 10-year CHD risk score. This model was tested on both genders combined and on each gender separately. A diet reduction scheme was then applied to the model to test the effect of dietary changes on the mean risk level of the entire sample.

Results

Significant results were obtained for the combined gender model, which indicated cardio-protective benefits from an isoenergetic increased intake of carbohydrates and polyunsaturated fats. It was predicted that diet changes would result in a one half to one percent decrease in mean risk, depending on the changes implemented. Significant results, however, were not obtained for the genders separately.

Conclusions

The combined gender model generated provided positive results, linking diet to CHD risk. However, the two separate gender models, while showing similar trends, had reduced significance. It is possible that this outcome is due to an unanticipated relationship between diet and gender. This result creates a limitation as to how conclusive the model can be. In order to overcome this limitation, further study using longitudinal data will be required. Nonetheless, the trends found in this analysis are consistent with previously noted cardio-protective diets.

1 Introduction

Coronary Heart Disease (CHD) is one of the most prevalent chronic diseases in the United States, afflicting nearly 14,740,000 adults in 2009 [17]. Consequently, it is also the most costly illness in the United States, with total annual costs reaching $286.6 billion in 2007 [16]. This amount is only expected to increase due to increasing levels of obesity [19]. Furthermore, advancements in the treatment of diseases like CHD are often cited as a leading driver in the spiraling health care costs we face today, leading to an additional increase of the burden that CHD places on society [7]. Thus, it is imperative that suitable methods of prevention be identified that can reduce the overall incidence of CHD. This report examines the effect that diet can have on the national rates of CHD. More specifically, this study will explore how the intakes of specific types of nutrients (e.g., carbohydrates and different types of fat) are associated with 10-year CHD risk.

1.1 Background

CHD is a chronic disease most often caused by the formation of atherosclerotic plaques in the arteries surrounding the heart, a condition known as atherosclerosis. These plaques can then rupture, leading to the formation of a blood clot, which can reduce or block blood flow to the heart. This loss of blood flow to the heart can result in a loss of heart function (myocardial ischemia) or the death of heart muscle (myocardial infarction), otherwise known as a heart attack. Needless to say, both these events are extremely damaging and can often be fatal. [6]

The risk factors that contribute to the formation of plaques are extremely diverse and not completely understood. Factors as wide ranging as certain genetic markers and specific personality types have been linked to CHD. However, a few, well documented indicators have become accepted as the standard, classical risk factors for atherosclerosis. Von Eckardstein lists these risk factors as being "the pre-existence of atherosclerotic vascular disease, age, male sex, a positive family history of premature atherosclerotic disease, smoking, diabetes mellitus, hypertension, hypercholesterolaemia, hypertriglyceridaemia and low high-density lipoprotein (HDL) cholesterol." [22]

Because of their preeminence as indicators for atherosclerosis, these risk factors are often used to calculate the risk an individual has of developing CHD. One such risk formula was developed

by Wilson et al. from the data of the landmark Framingham Heart Study. This formula uses age, gender, blood pressure, total cholesterol levels, HDL cholesterol levels, and the presence of diabetes mellitus and cigarette use to determine the probability that an individual will develop CHD over ten years [23]. This is the formula that will be used throughout this paper to determine CHD risk and estimate incidence.

The mechanisms by which these risk factors affect the development of plaques is complex and knowledge of them is incomplete. What is accepted, however, is that the development of CHD starts with some kind of injury or inflammation in the endothelium of the blood vessel [5]. The injury may be due to the inability of the endothelium to produce vasodilators to cope with changes in shear stress, known as endothelial dysfunction, or the introduction of low-density lipoprotein cholesterol (LDL-C) particles to the arterial intima [5, 9]. Either way, the surface of the endothelium becomes activated, binding leukocytes, introducing them into the intima, and forming an inflammatory lesion on the arterial wall. This creates what is known as a fatty streak - the first stage in plaque formation. [5]

Once the fatty streak is formed, more LDL particles are pulled into the lesion and are often oxidized. After this occurs, the particles are taken up by macrophages, which then transform into foam cells. These foam cells hold the LDL-C and secrete inflammatory cytokines, further increasing the thickening of the arterial wall. At this point, the macrophages in the lesions can exert some controlling force on its growth. The smooth muscle cells surrounding the vessel, however, are also active in the process. They begin to proliferate and secrete lipid-binding proteins that further increase the size of the lesion. This process can continue for years until the lipid concentration inside the lesion becomes so high that the foam cells at the center are destroyed. At this point, the lesion is considered to be an atherosclerotic plaque. Throughout the entire progression of the plaque, a fibrous cap is added to the top, separating the lipid rich center from the blood vessel. A coronary event occurs when this cap becomes thin, ruptures, and exposes the interior, which can then clot the blood in the vessel, causing blocked or reduced blood flow. [5, 9]

The entire process of plaque development is complex, so it has been difficult for researchers to locate a point in the process where it could be halted effectively and safely. It does seem,

however, that LDL-C is one of the main promoting factors in plaque development and any way to limit its presence in the blood stream would be beneficial. One obvious way to do this is to decrease the intake or production of LDL-C. Another way is to find some mechanism that removes LDL-C from circulation. One common mechanism for this is reverse cholesterol transport (RCT), which utilizes HDL-C to transport free cholesterol from the blood stream to the liver or other places where it cannot be incorporated into plaques [15]. Using this information, many CHD prevention methods have focused on reducing LDL-C levels and increasing HDL-C levels as a way to reduce the development of atherosclerotic plaques.

1.2 CHD Prevention

Due to the importance of cholesterol in the development of CHD, much research has been done concerning the reduction of LDL-C and total cholesterol and enhancement of HDL-C levels. Perhaps the most significant advancement has been the development of statins, a drug that blocks the rate-limiting step in cholesterol synthesis in the liver. Numerous studies have confirmed that statin use decreases the likelihood of a person dying from CHD, with the most prominent risk reduction coming when the drug is given to high-risk patients. [3]

Even with the efficacy and relative safety of statins, however, diet can also play a substantial role in CHD risk reduction [4]. A shift from saturated to unsaturated fats has been shown to have a positive effect on the HDL-C to total cholesterol ratio, an important determinant in risk. More specifically, additional saturated fat will increase both HDL-C and LDL-C levels, while additional unsaturated fats will increase HDL-C levels and decrease LDL-C levels [12]. It should be noted, however, that clinical and epidemiological trials have been inconclusive in determining that heightened saturated fat intake is *directly* linked to increased CHD risk [11]. It is fairly well determined, though, that holistic diets, such as the popular "Mediterranean Diet," do have some cardio-protective benefit. These diets tend to consist of unsaturated fats, whole grains, and fruits and vegetables. They have been shown to significantly reduce the likelihood of CHD, adjusting not only cholesterol levels, but also other factors related to the development of heart disease, such as insulin sensitivity and diabetes [8, 18]. Many studies have been conducted to determine the influ-

ence of diet on heart disease. Most of those studies however, looked at a relatively small population using a control study approach [8].

In order to inform health policy and public health officials, research also needs to be conducted on a larger scale than these small control studies. To determine the effect of different CHD prevention and treatment methods on a national level, some researchers use epidemiological data to model the progression of CHD under different conditions [20, 10]. These studies employed a variety models, usually designed to model the progression of disease states through time and in response to interventions, such as a Markov chain model. Only cross sectional data was available for the investigation described in this paper. This paper, therefore, uses the 10-year Framingham Risk Score to simulate disease progression and attempts identify the relationship, if any, between the cardio-protective diets described in previous studies and the Framingham measure of CHD risk in a nationally representative sample [23].

2 Methods

This study used data from the 2005/2006 and 2007/2008 cycles of the National Health and Nutrition Examination Survey (NHANES), conducted by the Centers for Disease Control [2]. NHANES uses a complex survey design to collect health and nutritional data from a nationally representative sample of about 10,000 people in each cycle. After examining the data collection methods and available data, the relevant datasets were extracted, including laboratory results on blood lipoprotein levels, physical examination results, prescription drug usage, and self reported diet and health history. The complete dataset provided information on 20,497 respondents. Not all respondents, however, completed all parts of the survey. Furthermore, since the Framingham Risk Score was designed for people between the ages of 30 and 74, all respondents outside those age groups were excluded [23]. After identifying all people that fit these criteria, 6,314 people were included in the analysis. It should be noted that the Framingham Risk Score was designed for people without a previous history of heart disease. This is significant because an individual's risk profile does change significantly after a cardiac event. A decision was made, however, to keep survey respondents with a history of CHD in the analysis because they represented a sizable ($\sim$ 900 respondents)

high-risk group that might add important information regarding diet trends and high CHD risk. Since the purpose of the study was not to provide clinically relevant patient level risk scores, but to provide an overview of the US population, this inclusion of CHD positive respondents should strengthen, not detract from, the significance of the model.

The 10-year CHD risk, as defined by Wilson et al. from the Framingham Heart Study, was calculated for all the included respondents [23]. (It should be noted that Wilson et al. had two possible equations for risk calculation. One used LDL-C instead of total cholesterol. The total cholesterol equation was used.) This risk score, in addition to information on age, sex, blood pressure measurements, and HDL and total cholesterol levels, required indicators on the presence of diabetes and cigarette use. In order to facilitate the analysis of as large a dataset as possible, only self-reported information was used to set these indicators. This is especially important for the diabetes variable, since fasting plasma glucose levels were available for a small subset of the population, enabling direct diagnosis of diabetes of those respondents tested. In order to maintain a uniform procedure across the entire population, however, these laboratory results were not used when available.

Various variables describing different aspects of dietary intake were also created in order to facilitate a more thorough analysis of the data. The dietary dataset from NHANES contained information on total energy intake, measured in kilocalories, and the absolute intake of various nutrients, measured in grams or milligrams. A summary of the additional variables created can be found in Table 1.

Next, various multiple-linear regression models were tested with the intent of finding a model that identified statistically significant relationships between diet and CHD risk. Various control variables were tested in the model, including, but not limited to, history of heart disease, family history of heart disease or diabetes, use of statins or other cholesterol lowering medications, and body measurements such as waist circumference, body mass index, or weight. All regressions were preformed using the *Stata* statistical software package. The complex survey model of NHANES was accounted for using the *svy* class of commands. The sampling weights were taken from the dietary section of the NHANES dataset, with each two year sampling weight divided in half to facilitate the

Table 1: Dietary Variables Generated for this Study

No.	Variable	Equation	Notes
1	Relative Nutrient Intake	$\frac{N * A}{TE}$	N is total fat, SFA, PUFA, MUFA, carbohydrate, or protein intake, measured in grams and TE is the total energy intake, measured in kilocalories. A is the Atwater factor for each of the nutrients, measured at $9\frac{kcal}{gm}$ for all fats and $4\frac{kcal}{gm}$ for carbohydrates and proteins [13].
2	Differences in fat type intake	$\frac{FA_1 - FA_2}{TFA}$	FA is SFA, PUFA, or MUFA, while TFA is the total fatty acid, all measured in grams.
3	Difference in carbohydrate and fat intake	$\frac{C * 4 - TFA * 9}{TE}$	C is carbohydrate intake and TFA is total fatty acid intake, both measured in grams. TE is total energy intake, measured in kilocalories.

combination of two survey cycles.

The method for selecting viable regression models was fairly fluid, mostly consisting of using trial and error to find the combination of right-hand variables which was reasonably well correlated with CHD risk and had at least a moderate level of statistical significance ($p < 0.1$) in each variable. There was, however, a basic method that was followed. First, all the variables with a suspected connection to CHD risk were included in a correlation matrix. (This matrix can be found in Appendix 1.) The coefficients generated were used to determine how the different variables related to one another and to the risk score. Based on this information, preliminary regression models were estimated. Models with the best R^2 values were selected and further refined by including variables that increased (or excluding variables that decreased) the significance of the dietary coefficients in the model. Some variables, such as weight and waist circumference, were significantly associated and highly correlated with CHD risk and consequently increased the fit of any model in which they were included. It could be argued, however, that those variables are partially a function of lifestyle factors, including diet. For that reason, to avoid collinear regressors in the model, such variables were omitted. Other control variables were added or removed to enhance the significance of diet

Table 2: Proposed Dietary Changes

Nutrient	Level 1	Level 2
Carbohydrate	+5% Energy Intake	+10% Energy Intake
Total Fat	−5% Energy Intake	−10% Energy Intake
PUFA	+10% Fat Intake	+20% Fat Intake
MUFA	−5% Fat Intake	−10% Fat Intake
SFA	−5% Fat Intake	−10% Fat Intake

variables and improve the overall fit of the model.

With this final model, two new risk scores were estimated based on two different levels of diet change, as detailed in Table 2. These new risk scores could then be compared to the original CHD risk scores to investigate what level of risk reduction could be achieved with diet change. In order to fully investigate the association of diet and risk this same model was also applied to each gender separately.

3 Results

The final model quantified the association between CHD risk and the following variables: difference in carbohydrate and total fat intake (Table 1, Equation 3), difference in PUFA and SFA intake (Table 1, Equation 2), difference in MUFA and SFA intake (Table 1, Equation 2), and indicator variables controlling for hypolipedemic drug use, previous history of CHD and family history of diabetes. The full results of this regression can be found in Table 3.

After predicting the effects of the diet changes described in Table 2, the mean risk (SE) for the group dropped by 0.4544% (0.1415) after the Level 1 changes and dropped by 0.9089% (0.1415) after the Level 2 changes. The mean risk scores for before and after the diet changes can be found in Table 4. To put this risk change in a human perspective, as of 2009, there were 161,590,058 people in the 30 to 74 age group for which the risk score was valid [21]. Under the original risk score, out of that population, 11,757,826 people would be predicted to get CHD within 10 years. Under the Level 1 diet change, 11,023,512 would be predicted to have CHD within 10 years, while

Table 3: Final Regression Model, $R^2 = 0.0734$

Variable	Regression Coefficient	Standard Error	p-value
Difference between PUFA and SFA intake	-0.0221	0.0114	0.063
Difference between MUFA and SFA intake	0.0241	0.0150	0.118
Difference between carbohydrate and total fat intake	-0.0124	0.0050	0.018
History of CHD	0.0498	0.0046	> 0.001
Family History of Diabetes	0.0040	0.0020	0.053
Use of Hypolipidemic Drug	0.0288	0.0030	> 0.001
Constant	0.0619	0.0029	0.000

Table 4: Predicted Risk Score for Proposed Dietary Changes

Dietary Change	Mean	Std. Err.	95% Conf. Interval
Original Risk Score	0.0728	0.0014	[0.0700, 0.0756]
Predicted Risk Score after Level 1 Adjustment	0.0682	0.0003	[0.0675, 0.0689]
Predicted Risk Score after Level 2 Adjustment	0.0637	0.0003	[0.0630, 0.0644]

under the Level 2 change 10,289,199 people would be expected to get CHD. This translates into a 734,314-person reduction for Level 1 and a 1,468,627-person reduction for Level 2. It should be noted that these are not per year reductions, which cannot be calculated from this data. Rather, these are the expected results if all 30 to 74 year olds in America today adhered to the hypothetical dietary changes and were followed as a single cohort for 10 years.

After isolating each gender and performing the same regression again, similar trends were observed but the changes lost their statistical significance (Tables 5 and 6). For this reason, the diet adjustments were not tested separately by gender.

Table 5: Final Regression Model, Females Only, $R^2 = 0.0875$

Variable	Regression Coefficient	Standard Error	p-value
Difference between PUFA and SFA intake	-0.0042	0.0131	0.749
Difference between MUFA and SFA intake	-0.0018	0.0159	0.910
Difference between carbohydrate and total fat intake	-0.0077	0.0065	0.242
History of CHD	0.0402	0.0073	0.000
Family History of Diabetes	0.0068	0.0019	0.001
Use of Hypolipidemic Drug	0.0296	0.0038	> 0.001
Constant	0.0425	0.0028	> 0.001

Table 6: Final Regression Model, Males Only, $R^2 = 0.0564$

Variable	Regression Coefficient	Standard Error	p-value
Difference between PUFA and SFA intake	0.0028	0.0166	0.865
Difference between MUFA and SFA intake	-0.0231	0.0230	0.324
Difference between carbohydrate and total fat intake	-0.0038	0.0076	0.618
History of CHD	0.0453	0.0075	0.000
Family History of Diabetes	0.0062	0.0033	0.071
Use of Hypolipidemic Drug	0.0234	0.0038	> 0.001
Constant	0.0887	0.0049	> 0.001

4 Discussion

The purpose of this project was to attempt to predict and quantify the benefit of positive diet change to the US population. The model that was developed and estimated in this project suggested that the proposed moderate diet changes could lower the mean 10-year CHD risk of Americans age 30 to 74 by about one half to one percent, depending on the level of diet reduction implemented. This reduction is small, but statistically significant, and could be a positive step towards a larger reduction in CHD rates. The results of this model, however, must be interpreted in light of the assumptions and methods upon which it was built. The first, and possibly most important, assumption in the model is that it represents a cause and effect, meaning that a change in diet will cause a quantifiable change in CHD risk as determined by the model. In reality, however, the regression used to generate the model simply quantifies the *association* between diet and CHD risk. Even with the model controlling for other contributing factors, the fact that the data used in the model is cross-sectional makes the assumption of causation unwise. Only in light of all the supporting evidence from other studies that links diet, specifically fatty acid intake, to CHD risk is it possible to assume at least some level of causation between the diet variables in the model and CHD risk.

Another important assumption made in the analysis is that the diet reduction levels applied to the model would have 100% compliance from all people in the population. This would obviously never occur. A more realistic compliance rate might be 50% or lower. As long as this assumption is considered when viewing the results, however, it is not detrimental to the model. All results just need to be seen as an upper limit of the possible benefits of specific diet changes.

One of the other major assumptions underlying the model is that the final regression coefficients account for all useful data. However, the iterative procedure used to develop the model dropped variables from the model that did not enhance the significance of diet variables, even when the deleted variables were connected to heart disease risk by previous research. For example, the final model did not include an indicator for a family history of heart disease, even though it was available. It could be argued that this amounts to "cherry picking" values to achieve the best model. In one sense, this is true, since only using the whole of the available data would produce the most

complete and transparent model possible. However, to achieve a usable model, some trade-offs must be made and it seems appropriate to forego some comprehensiveness of the model to obtain a significant estimation of how diet relates to CHD risk.

Along with this assumption, the whole model is predicated on the idea that all influencing factors of CHD risk were available in the NHANES dataset and could be used as controls to isolate the effect of diet on CHD. This is, obviously, however, not true. One example is the lack of a standard variable measuring exercise levels, which was not available across both NHANES survey cycles. Exercise has been shown to reduce CHD risk and its absence from the model might have had important consequences for its outcome [14]. Furthermore, this model assumes the bulk of the effects of diet on CHD come from the amount and composition of fats. While this is generally supported by other research, other aspects of diet, such as glycemic index, dietary antioxidants, and dietary fiber have also been shown to be significant when considering the potential benefits of a diet [18, 1].

With these assumptions, the results may still be useful when considering the question this paper was trying to answer. The results do quantify the relationship between diet and CHD risk in the US population as a whole. However, in light of the assumptions, the model cannot be more specific than that. For example, application of the regression equation in a clinical setting would probably not produce satisfactory results, since the regression equation does not take into account many of the non-diet variables that contribute to CHD risk.

Even as the original model, with both genders included, seems to produce significant and usable results, the significance all but disappears when the samples are separated by gender. This probably occurs for one of two reasons. First, it could be that the samples, when separated by gender, are reduced to a size that does not provide sufficient variation for the regression to determine a significant relationship between risk and diet. The second possible reason is that both CHD risk and the dietary variables are highly correlated with gender, with CHD risk being connected to gender independent of diet. This is the most likely reason since being male was a risk factor that contributed heavily to the Framingham Risk Score [23]. Furthermore, it can be seen in Table 7 that the mean values of the diet variables from the model differed significantly between

Table 7: Gender-Specific Means for Diet Variables

Variable		Mean	Standard Error	95% Confidence Interval
Female	Difference between PUFA and SFA intake	-0.1119	0.0048	[-0.1217, -0.1021]
	Difference between MUFA and SFA intake	0.0279	0.0031	[0.0216, 0.0343]
	Difference in carbohydrate and total fat intake	0.1509	0.0055	[0.1396, 0.1621]
Male	Difference between PUFA and SFA intake	-0.1173	0.0031	[-0.1237, -0.1109]
	Difference between MUFA and SFA intake	0.0430	0.0021	[0.0387, 0.0473]
	Difference in carbohydrate and total fat intake	0.1243	0.0061	[0.1119, 0.1366]

the two genders. In fact, the mean values show that males tended towards more atherogenic diets, with higher saturated fat and total fat intake, while females tended towards the cardio-protective polyunsaturated fats and higher carbohydrate intake. It may seem then, at this point, that the high risk found in males may be due to poor diet or, conversely, the low risk in females due to good diet. There is previous research, however, that links a significant portion of the higher risk in males and lower risk in females to physiological factors [22]. What this suggests, then, is that the poor diet characteristics of men simply track with their naturally high CHD risk, while the good diet characteristics of females track with their naturally low risk. This does not rule out a connection between CHD risk and diet, but it seems that any relationship that is there is hidden by the confounding gender variable.

Due to these results, it is clear that more study is needed to quantify how much the CHD risk of the American population could be lowered through moderate diet change. The most useful research would probably involve a large, nationally representative control study, following people through long stretches of time to determine the effect of diet change. Such a study, however, is probably not feasible. Instead, a large, nationally representative longitudinal study that focused on

diet and disease outcomes would probably be the best way to most definitively quantify the effect of diet change on the health of the US population.

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Appendix I

	CHD Risk Score	History of Angina	Weight	BMI	Waist Circumference	Family History of Heart Attack	Family History of Diabetes	History of CHD	Use of Hypolipidemic Drugs	Energy Intake (kCal)	Intake of Cholesterol (mg)	Intake of Sodium (mg)	Intake of Alcohol (gm)	Relative Protein Intake	Relative Carbohydrate Intake	Relative Sugar Intake	Relative Total Fat Intake	Relative SFA Intake	Relative MUFA Intake	Relative PUFA Intake
CHD Risk Score	1																			
History of Angina	0.0263	1																		
Weight	0.2024	0.0083	1																	
BMI	0.1289	0.0356	0.8735	1																
Waist Circumference	0.2877	0.0409	0.914	0.8827	1															
Family History of Heart Attack	-0.0031	0.0774	0.0246	0.0469	0.0503	1														
Family History of Diabetes	0.0492	0.0505	0.114	0.1551	0.1444	0.0817	1													
History of CHD	0.1711	0.1498	0.0648	0.0557	0.1167	0.0657	0.0243	1												
Use of Hypolipidemic Drugs	0.136	0.067	0.1459	0.1498	0.2057	0.0677	0.0542	0.2886	1											
Energy Intake (kCal)	0.0054	-0.0386	0.1858	0.0067	0.0882	-0.0376	-0.0444	-0.0466	-0.0563	1										
Intake of Cholesterol (mg)	0.0525	-0.0312	0.173	0.0637	0.1129	0.0096	0.0253	-0.02	-0.0284	0.5331	1									
Intake of Sodium (mg)	0.0084	-0.0394	0.2106	0.0655	0.131	-0.0332	0.005	-0.0219	-0.0093	0.757	0.4891	1								
Intake of Alcohol (gm)	-0.0107	-0.018	-0.0288	-0.1131	-0.061	-0.0299	-0.0549	-0.0319	-0.0563	0.3187	0.1197	0.1262	1							
Relative Protein Intake	0.0183	-0.0316	0.0617	0.0746	0.0565	0.0104	0.0485	-0.0026	0.0115	-0.1765	0.1993	0.047	-0.1647	1						
Relative Carbohydrate Intake	-0.0343	0.014	-0.0916	-0.0384	-0.0747	0.006	-0.0493	0.0323	-0.0156	-0.1501	-0.4043	-0.2319	-0.337	-0.4222	1					
Relative Sugar Intake	-0.0333	0.0183	-0.0628	-0.0445	-0.0624	0.0427	-0.0404	0.0078	-0.0587	-0.0322	-0.1836	-0.2426	-0.2525	-0.3802	0.7358	1				
Relative Total Fat Intake	0.0417	0.0023	0.115	0.0999	0.1153	-0.004	0.0682	-0.021	0.0576	0.1735	0.3425	0.2455	-0.1744	0.0615	-0.6948	-0.4527	1			
Relative Saturated Fat Intake	0.0272	0.0201	0.0695	0.0578	0.081	0.0249	0.0469	-0.0191	0.0215	0.1529	0.3192	0.1676	-0.1274	0.044	-0.5255	-0.2688	0.733	1		
Relative Monounsaturated Fat Intake	0.0554	-0.0055	0.1176	0.0893	0.1115	-0.0045	0.054	-0.0079	0.0539	0.1782	0.305	0.2307	-0.1343	0.0489	-0.6281	-0.4238	0.8976	0.5496	1	
Relative Polyunsaturated Fat Intake	-0.0033	-0.0195	0.0555	0.0613	0.0483	-0.036	0.0406	-0.0223	0.0537	0.0581	0.076	0.1421	-0.1197	-0.0118	-0.3593	-0.3085	0.5857	-0.0102	0.4487	1