Non-Biological Factors Contribute to Increased Risk

of Cardiovascular Disease and Metabolic Syndrome in

Mexican-Americans Living in Metropolitan Phoenix

by

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#### ABSTRACT

Among the general US population, cardiovascular disease (CVD) is the main cause of mortality for Mexican-Americans. CVD is less prevalent among Mexican-Americans than non-Hispanic Whites or African Americans. However, there is limited research regarding the factors associated with increased CVD risk among Mexican-Americans. Thus, this cross-sectional study was conducted to evaluate the effects of non-biological factors (income, education, employment, acculturation) and diet on CVD risk factors in 75 Mexican-American adults (26 males, 49 females; age=37.6 $\pm$ 9.3 y, BMI=28.9 $\pm$ 5.3 kg/m<sup>2</sup>, systolic BP=117±11 mmHq, diastolic BP=73±9 mmHq, LDL  $cholesterol = 114 \pm 32 mg/dL$ , HDL  $cholesterol = 44 \pm 11 mg/dL$ , triglycerides= $115\pm61$  mg/dL, serum glucose= $92\pm7$  mg/dL). Aside from collecting anthropometric measurements, blood pressure, and measuring fasting blood lipids, glucose, and insulin, information about participants' socioeconomic status, income, employment, education, and acculturation were gathered using a survey. Diet data was collected using the Southwestern Food Frequency Questionnaire. Weight, BMI, and waist circumference were significantly greater for those with a monthly income of <\$3000 than for those earning >\$3000

i

(81±15 kg vs. 71±15 kg; 29.8±4.6 kg/m<sup>2</sup> vs. 26.5±5.1 kg/m<sup>2</sup>;  $98\pm12$  cm vs.  $89\pm14$  cm; respectively) and with an education level of high school graduate or less than for those with some college ( $84\pm16$  kg vs.  $72\pm14$  kg;  $30.6\pm4.2$  kg/m<sup>2</sup> vs.  $26.9\pm4.9$  $kg/m^2$ ; 100±11 cm vs. 91±13 cm; respectively). HDL-C was higher for those with a monthly income of >\$3000 than those earning < \$3000 (49±12 mg/dL vs. 41±10 mg/dL), those with some college education than those with high school or less  $(47\pm10 \text{ mg/dL vs. } 37\pm9 \text{ mg/dL})$ , and for those employed than those not employed ( $46\pm10 \text{ mg/dL} \text{ vs. } 40\pm12 \text{ mg/dL}$ ). There was no association between acculturation and CVD risk factors. Percent of energy consumed from fat was greater and percent of energy from carbohydrates was lower in those earning <\$3000 monthly than those earning >\$3000 (32±5% vs. 29±3%;  $52\pm8\%$  vs.  $56\pm4\%$ ; respectively). Greater acculturation to the Anglo culture was negatively correlated with body fat percentage (r=-0.238, p=0.043) and serum glucose (r=-0.265, p=0.024). Overall, these results suggest that factors related to sociocultural and socioeconomic status may affect cardiometabolic disease risk in Mexican-Americans living in the Phoenix metropolitan area.

ii

## TABLE OF CONTENTS

LIST OF TABLESvi
INTRODUCTION 1
Research Aim 6
Hypotheses
Hypothesis 1: 6
Specific Aim 1:7
Hypothesis 2:7
Specific Aim 2:7
Specific Aim 3:7
Definition of Terms
Delimitations10
Limitations10
REVIEW OF LITERATURE12
Introduction12
Cardiovascular Disease15
Lipoproteins17
Cardiovascular Disease Risk Factors21
Cardiovascular Disease Risk Factors Among Hispanics26
Metabolic Syndrome28
Health Disparities

Social Determinants of Cardiovascular Disease Risk	
Socioeconomic Status	35
Education	
Income	
Environment	41
Access to Food	41
Access to Fruits and Vegetables	43
Cost of Food and Food Quality	46
Neighborhood	47
Acculturation	51
Conclusion	54
METHODS AND MATERIALS	55
Materials	55
Participants	55
Recruitment and Consenting	56
Study Design	57
Study Protocol	58
Methods	59
Anthropometrics	59
Biological Markers	60
Diet Analysis	61

# Page

Socioeconomic Factors62
Data Analysis and Interpretation63
RESULTS66
DISCUSSION97
Income, diet and CVD risk factors in Mexican-American adults .98
Employment, diet and CVD risk in Mexican-American adults $108$
Education, diet and CVD risk in Mexican-American adults 112
Acculturation, diet and CVD risk in Mexican-American adults118
Diet and CVD risk in Mexican-American adults121
REFERENCES
APPENDIX
INSTITUTIONAL REVIEW BOARD/HUMAN SUBJECTS APPROVAL
FORM
ADVERTISEMENTS USED IN RECRUITMENT
SCREENING FORM155
SOUTHWEST FOOD FREQUENCY QUESTIONNAIRE
INFORMED CONSENT FORM181
MAIN SURVEY190
ANTHROPOMETRICS DOCUMENTATION FORM196
BLOOD SAMPLE DOCUMENTATION FORM
ACCULTURATION RATING SCALE

# LIST OF TABLES

Table 1. Sociodemographic characteristics of study
participants81
Table 2. Cardiometabolic disease risk factors of study
participants82
Table 3. Cardiometabolic disease risk factors stratified by
income83
Table 4. Cardiometabolic disease risk factors stratified by income
category84
Table 5. Cardiometabolic disease risk factors stratified by level of
education85
Table 6. Cardiometabolic disease risk factors stratified by
completion of high school86
Table 7. Cardiometabolic disease risk factors stratified by
participants' employment status87
Table 8. Cardiometabolic risk and acculturation category88
Table 9. Daily macronutrient consumption of study
participants89
Table 10. Macronutrient consumption stratified by income90
Table 11. Macronutrient consumption stratified by income
category91

Page

Table 12. Macronutrient consumption stratified by education92
Table 13. Macronutrient consumption stratified by completion of
high school93
Table 14. Macronutrient consumption stratified by
employment94
Table 15. Macronutrient consumption stratified by acculturation
category95
Table 16. Correlations between cardiometabolic risk factors and
macronutrient consumption96

#### INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death among Americans [1]. In 2005, CVD-related deaths accounted for 35% of all deaths in the United States [2]. According to the Centers for Disease Control and Prevention, over 28,000 Mexican-Americans have CVD, which is higher than any other cause of death in this ethnic group [1]. Despite the fact that this group does not have a higher prevalence of CVD than White Americans or African Americans, it is still the number 1 cause of death among Mexican-Americans [1, 2]. Within the Mexican-American population, women aged 20 years or older have a greater prevalence of CVD than do men of the same age group [2]. Some of the known risk factors for CVD include the presence of diabetes, hypertension, smoking, old age, and dyslipidemia [3, 4]. It has been found in several studies that Hispanics have higher levels of several CVD risk factors when compared with non-Hispanic Whites [5, 7-9]. However, relative to non-Hispanic Whites, Mexican-Americans are 10% less likely to have heart disease and 30% less likely to die from heart disease [6]. Moreover, it is estimated that 31% of Mexican-Americans have metabolic syndrome [8] and 40% are obese [9].

The metabolic syndrome is a cluster of metabolic abnormalities associated with increased risk for diabetes and CVD [10]. According to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) the metabolic syndrome can be defined as the presence of three or more of the following risk factors: abdominal obesity, hypertriglyceridemia, low HDL cholesterol, hypertension, and/or fasting hyperglycemia [10]. The metabolic mechanisms leading to a clustering of the above mentioned risk factors is unknown, but it is often related with having excess body fat and metabolic susceptibility [11]. Moreover, increased adiposity is related to insulin resistance and associated metabolic abnormalities [12].

Data from the National Health and Nutrition Examination Survey (NHANES) shows that the prevalence of the metabolic syndrome in the Hispanic population in the United States is 31% [13], which is higher than that of non-Hispanic whites (22%) [8]. Among Mexican-American adolescents, the prevalence of metabolic syndrome is 5.6% [14]. Regarding individual components of the metabolic syndrome, a study by Ford et al. [13] revealed that Mexican-Americans, when compared with Whites and African Americans, have the highest prevalence of abdominal obesity (45.7%), hypertriglyceridemia (37.7%), and fasting hyperglycemia (20.0%).

Health disparities refer to differences in the prevalence of a given disease among ethnic groups that cannot be attributed to biological factors [15]. Among Mexican Americans, these nonbiological factors include poverty, neighborhood environment, food access, level of education, acculturation, racism, discrimination, and stress [16-20].

Lower income and education have been associated with a greater risk of coronary heart disease [18]. Moreover, inhabitants of lower socioeconomic communities tend to have a higher prevalence of obesity and obesity related disorders [17-20]. One study found a negative correlation between individual socioeconomic status and BMI [17]. Furthermore, racially and ethnically diverse communities have a greater potential for lower dietary quality and its associated health risks and outcomes including obesity and obesity related disorders such as diabetes and CVD [18]. Larson et al. [19] suggested that individuals with better neighborhood access to supermarkets and a more limited access to convenience stores tend to have healthier diets and

lower levels of obesity. Low socioeconomic status has been suggested to increase diabetes risk due to a variety of factors including poor access to care, neglect of preventive strategies, a lower ability to exercise or an unhealthy diet [21]. The underlying question to these studies is whether the increased susceptibility to CVD or type 2 diabetes is caused by genetic or social factors or a combination of both.

Acculturation and length of residence in the U.S. have also been associated with increased risk for CVD and metabolic abnormalities [22]. Greater acculturation has been reported to negatively affect diet. For example, among Mexican-Americans, greater acculturation has been correlated with lower fruit and vegetable consumption [23]. One study found that Latinos consumed significantly more fruits and vegetables (33 times/week) compared with non-Latinos (28 times/week) suggesting that the American culture does not support a diet high in fruits and vegetables [23]. Therefore, as the Mexican-American population becomes more acculturated their newly acquired dietary habits may increase their risk for CVD.

The metabolic abnormalities associated with non-biological risk factors for chronic disease are in part a result of their effects on dietary habits. Research has suggested that living in low socioeconomic and ethnically diverse communities contribute to the difficulty of obtaining nutritious foods that can counteract the effects and reduce the risk of CVD [24]. The mean quality of the fresh fruit and vegetable produce available in lower socioeconomic areas has been reported to be lower than in higher socioeconomic areas [25]. Lower income communities also tend to have fewer stores and markets that sell fresh fruits and vegetables [26]. Without having access to healthy foods such as fruits and vegetables, individuals cannot positively change their diets [27, 28].

Despite some indication that non-biological risk factors negatively affect diet and chronic disease risk, specific information among Mexican-Americans is scarce. Moreover, it is difficult to generalize the findings of prior studies to the Mexican-American population of the Phoenix metropolitan area due to differing environmental factors that may have contributed to the results of prior studies. Therefore, the purpose of this preliminary study is to conduct an initial assessment of the

effects of non-biological factors and diet on cardiometabolic risk among Mexican-Americans living in metropolitan Phoenix. This will be done by estimating the associations between biological contributors of cardiometabolic risk (waist circumference, BMI, glucose, lipids, high sensitivity C-reactive protein [hsCRP]), diet composition, and non-biological risk factors (income, employment, acculturation, education).

#### **Research Aim**

The purpose of this work was to perform a cross-sectional evaluation of self-reported individual and family dietary and lifestyle habits, sociocultural factors (income, employment, acculturation, and education) and biological markers of cardiometabolic risk among Mexican-American adults living in the Phoenix metropolitan area.

#### Hypotheses

#### Hypothesis 1:

Income, employment, and education, are negatively associated and acculturation is positively associated with cardiometabolic risk factors among Mexican-American adults living in the Phoenix metropolitan area.

#### Specific Aim 1:

To explore whether income, employment, education and acculturation affect cardiometabolic disease risk factors (lipids, glucose, blood pressure, insulin resistance, hsCRP, waist circumference) in Mexican-American adults living in the Phoenix metropolitan area.

#### **Hypothesis 2:**

Income, employment, and education, will be negatively associated and acculturation will be positively associated with intake of macronutrients known to adversely affect cardiometabolic risk (total fat, saturated fat, *trans* fat, simple carbohydrates) in Mexican-American adults living in the Phoenix metropolitan area.

#### Specific Aim 2:

To examine whether income, employment, education and acculturation affect dietary intake of macronutrients known to adversely affect cardiometabolic risk among Mexican-American adults living in the Phoenix metropolitan area.

#### Specific Aim 3:

To examine whether macronutrient intake is associated with cardiometabolic disease risk factors.

#### **Definition of Terms**

- <u>abdominal obesity</u>: the accumulation of visceral fat in the trunk region of the body, resulting in an increased waist circumference. The measurements of waist circumference indicating abdominal obesity are >102 cm (men) and >88 cm (women)
- <u>acculturation</u>: the exchange or adaptation of different cultures resulting from living or associating with others from different cultures
- dyslipidemia: an elevation of lipids in the blood
- <u>fasting hyperglycemia</u>: blood glucose concentrations after fasting for 8-14 hours >100mg/dl
- <u>health disparities</u>: differences between populations in the prevalence of diseases, access to health care, and final health outcomes
- <u>hsCRP</u>: high sensitivity C-reactive protein used as a marker for inflammation and infection. Normal values: <1.0 mg/L, moderately high values: 1.0-3.0 mg/L, elevated values: >3.0 mg/L.
- <u>hypertension</u>: elevated blood pressure indicated by > 130 mm Hg for systolic and > 85 mm Hg for diastolic

hypertriglyceridemia: fasting triglyceride concentrations exceeding 150 mg/dl

<u>low HDL</u>: the concentration of cholesterol in high density lipoprotein <40 mg/dl (men) and <50 mg/dl (women)

<u>metabolic syndrome</u>: The metabolic syndrome is a cluster of various risk factors of metabolic origin that contribute to the development of atherosclerotic cardiovascular disease. As defined by NECP ATP III, it occurs when there is the presence of three or more of the following risk factors:

abdominal obesity defined as waist circumference >102 cm
men and >88 cm in women,

2) hypertriglyceridemia: >150 mg/dl,

3) low HDL: <40 mg/dl in men and <50 mg/dl in women,

 hypertension: > 130 mm Hg systolic and > 85 mm Hg diastolic, and

5) fasting hyperglycemia: >100mg/dl.

<u>socioeconomic status</u>: a person's standing within the community based on monthly income, education level, employment, and social status.

#### Delimitations

This cross-sectional study focused on the non-biological factors that may contribute to increased CVD risk among Mexican-Americans. These include income, employment, acculturation, and education. This study was limited to Mexican-American adults free of chronic diseases living in the Phoenix metropolitan area.

#### Limitations

This study had a cross-sectional design which does not explore the long term effects of today's lifestyles. The study involved 75 adults, which may limit the statistical power relative to larger observational studies.

Limitations include the possibility of the participants having selfreported incorrect data on the food frequency questionnaire and survey. Study procedures took approximately 1.5 hours, which may have been tiring, resulting in participants replying inaccurately due to the fact that they lost interest in the survey. This study did not include the study of children and adolescents and the affect of their lifestyle on CVD or metabolic risks later in life.

There is always risk of bias since information was gathered by multiple people. However, training of interviewers prior to data collection decreased this risk. Our study only included those contacted via newspaper, flyers, e-mail, and phone since these were the methods used for recruitment. Therefore, the results may not truly represent all of the Mexican-Americans in the Phoenix metropolitan area.

Furthermore, this study only focused on the Phoenix metropolitan area and may not be able to be generalized to other areas of the country. This study was limited in the number of non-biological factors evaluated. For example, non-biological factors that were not focused on in this study include environment of residence (access to food, recreation, etc.), racism, stress, and perceived discrimination.

#### **REVIEW OF LITERATURE**

#### Introduction

Hispanic is a general term to describe the ethnicity of those from Mexico, Central America, or South America. Mexican-American is a more specific term referring to those Hispanics who either originated from Mexico themselves or their parents, grandparents, or ancestors originated from Mexico. Some studies group all Hispanics together and evaluate them as a whole. Others look specifically at Mexican-Americans. Therefore, in citing studies the term that has been used in the study to describe their participants has also been used here in this document.

Hispanics are the largest minority in the United States and continue to grow [29]. According to the 2005 Census Bureau Report, 14% of the US population is Hispanic. Hispanics are responsible for half of the US population growth for that year [29].

Cardiovascular disease (CVD) is the number one cause of death in the United States [2]. The American Heart Association [2] estimates that in 2007 over 80 million American adults have one or more forms of cardiovascular disease (CVD), and that 33.6% of all deaths were related to CVD. Among Mexican-Americans, it is estimated that 26.9% of all male deaths and 31.1% of all female deaths are caused by CVD [2]. Even though non-Hispanic White males and females have a higher prevalence of deaths from CVD (32.7% vs. 26.9%, 34.5% vs. 31.1%; respectively) it is still the number one cause of death among Mexican-Americans [2].

Unlike among other ethnic groups, Mexican-American women have a higher prevalence as well as a higher rate of mortality from CVD than men [2]. This may be due to the fact that many Mexican-American men have low skill jobs requiring more manual labor which allow them to be more physically active during the day versus a non-Hispanic White man who may have a high skill job working at a desk doing little or no physical activity at work [30].

It has been found in several studies that Hispanics have higher concentrations of CVD risk factors when compared with non-Hispanic Whites [8, 31]. Similar observations have been reported in Mexican-Americans relative to other Hispanic groups [5]. Relative to Dominican Americans, Puerto Rican Americans, and all other Hispanic Americans combined, Mexican-Americans had the highest fasting glucose, insulin, triglyceride, and Creactive protein (CRP) concentrations as well as the greatest body mass index (BMI) [5]. This group also had the highest prevalence of metabolic syndrome, diabetes mellitus, and dyslipidemia. Furthermore, Mexican-Americans had the second highest concentration of LDL cholesterol and the lowest concentration of HDL cholesterol [5].

When examining the studies that have been done on CVD, those involving Mexican-Americans specifically are very meager. There is even concern that studies done with Hispanics in general may be misleading due to inaccurate instrumentation or data collection [32]. Furthermore, studies that included multiple ethnicities of Hispanics (e.g. Cuban, Mexican, Puerto Rican, etc) found differences in CVD risk among different ethnic groups [5, 33, 34]. Whether these differences can be in part attributed to the individual sociocultural characteristics of these individual Hispanic groups remains to be better understood. Therefore, more research is needed with regard to Mexican-Americans specifically in order to better understand the factors that contribute to CVD risk in this group.

#### **Cardiovascular Disease**

Cardiovascular disease (CVD) is a term used to define conditions that affect the heart and blood vessels [2]. CVD can be classified as one or more of five different forms: hypertension, coronary heart disease (CHD), stroke, rheumatic heart disease, or congestive heart failure. CVD has been the number one factor for mortality for men and women in the US for the last nine decades [2]. As such, it is a growing concern as to why in the last nine decades and with the medical technology of today we are unable to decrease its prevalence and what changes must be made in order to have a healthier nation.

CHD results from a slowed, impeded, or blocked blood vessel that prevents adequate blood flow to the heart. Most commonly this is caused by atherosclerosis [35, 36]. Atherosclerosis is the hardening and narrowing of blood vessels. The physical characteristics of the inner linings of the arteries are modified and change as a result of an inflammatory response, formation of a connective tissue matrix, or the buildup of lipid and

cholesterol, also known as plaque [35, 37-39]. If an artery is damaged or injured, platelets attach to the wall of the artery and release a growth hormone. This growth hormone also promotes buildup on the artery wall and the development of lesions on the inner lining of the artery [35].

The development of atherosclerosis has been divided into 5 phases [37-39]. Phase 1 consists of a fatty streak. Fatty streaks do not block or impede the blood flow and are often the result of injury as aforementioned. Phase 2 is defined by plaque containing a high concentration of lipids. Often this plaque makes the artery more susceptible to rupture. Phase 3 is characterized by lesions that rupture. The plaque has not reached the size that threatens blood flow. When plaque does reach the state of impeding blood flow it is considered Phase 4. These lesions are also prone to rupture. Phase 4 is often also associated with chest pain, myocardial infarction (MI), or even death. If the plaque lesion progresses to almost completely or fully obstructed, it is considered Phase 5. This phase is also associated with chest pain, MI, and death [35, 37, 38].

Atherosclerosis often leads to endothelial dysfunction,

characterized by the inability of the blood vessels to constrict and dilate properly in order to meet demands due to the buildup of plaque and injury to blood vessels [35, 36, 39, 40]. This can result in increased myocardial stress [41, 42], increased risk for myocardial infarction or stroke [43], increased vascular stress [41], ischemic and hemorrhagic complications such as rupture [44], an increased severity of sleep apnea [45, 46], and organ damage [47, 48].

#### Lipoproteins

Blood lipids such as cholesterol, triglycerides, and phospholipids are transported in the blood as part of three-dimensional structures called lipoproteins. Each lipoprotein varies in its size, composition, and density. The composition consists of a center core, made up of triglycerides and cholesterol, and surface, made up of phospholipids and apoproteins [49]. The functions of apoproteins are to ensure structural stability, establish the metabolic outcome of the particles on which they are located, or act as cofactors for enzymes that function to metabolize plasma lipids and lipoproteins [49].

The density is determined by the amount of protein or lipids in the particles. Therefore, a high-density lipoprotein (HDL) has more protein than lipids in its composition. On the other hand, low-density lipoproteins (LDL) are made up of less protein and more lipids [35].

In humans, LDL cholesterol carries the majority of the cholesterol in the blood. Since total cholesterol measurements reflect the total amount of cholesterol being carried by all lipoproteins (HDL, LDL, and triglycerides), total cholesterol and LDL cholesterol are positively correlated. Furthermore, LDL cholesterol can be oxidized and taken up into the arterial wall by macrophages and endothelial cells. When LDL is taken up into the wall it promotes lesion and plaque formation. As a result, LDL contributes to atherosclerosis advancement [35].

HDL cholesterol originates from precursors which have been synthesized in the liver and small intestine. Commonly, the main function of other lipoproteins is to transport lipids to the cells. However, HDL cholesterol removes excess cholesterol from peripheral cells and transports it to the liver in order to maintain the cellular cholesterol homeostasis [50, 51]. Macrophages are phagocytes that take up dead and dying cells as well as aggregated and modified lipoproteins. Both types of lipoproteins and cells contain profuse cholesterol. However, too much cholesterol can be toxic, resulting in a need to efflux the cholesterol into the extracellular environment. The most commonly studied pathway of cholesterol efflux is through the ABCA1 transporter which causes cholesterol efflux to the lipidpoor apoA-1 [52].

Enterocytes and hepatocytes synthesize apoA-1, which is synthesized and secreted by the liver and intestine in a lipidpoor form, and then immediately engage additional phospholipids and free cholesterol via the ABCA1 pathway, forming pre-β HDL [52, 53]. This nascent HDL obtains more lipid from other peripheral tissues and from lipoproteins, after which lecithin-cholesterol acyltransferase (LCAT) transforms it into cholesterol ester. The result is mature HDL [52]. HDL transports the cholesterol that has been effluxed to the liver. After the cholesterol is delivered to the liver it is catabolized and excreted. This process is known as the reverse cholesterol transport (RCT) [52-55]. There are three stages of the reverse cholesterol transport: extravascular, intravascular, and intrahepatic. The extravascular phase involves unesterified cholesterol being removed from cell membranes by the apoprotein A-1 (apo A-1) found in the interstitial fluid [54]. The unesterified cholesterol enters the blood via the peripheral lymph which begins the intravascular phase. During this phase the cholesterol is esterified by lecithincholesterol acyltransferase (LCAT). Once esterified, some of the cholesterol is transferred to chylomicrons and VLDL by a transfer protein [54]. The rest are integrated into the center of the HDL particles causing them to increase in size and decrease in density. During the intrahepatic phase the cholesterol esters are removed from the circulation through the direct transfer of the esters from the HDL particles into the liver cells. Cholesterol is then eliminated by hepatocytes by secretion into the bile [54].

High HDL concentrations are correlated with lower CVD and atherosclerosis risk [35]. Other anti-atherogenic properties of HDL include the inhibition of LDL oxidation, inhibition of endothelial inflammation, promotion of endothelial nitric oxide

production, promotion of prostacyclin bioavailability, and inhibition of platelet aggregation and coagulation [52].

Considering that LDL is directly involved in plague formation and atherosclerosis, LDL cholesterol concentrations are a better indicator of CVD risk than total cholesterol [10, 56-59]. Several studies look specifically at LDL and its relationship with CVD or atherosclerosis [10, 56-59]. Few studies explore the relationship between total cholesterol and CVD. One reason is due to the fact that concentrations of HDL cholesterol and triglycerides will also affect total cholesterol. For example, an elevated HDL cholesterol concentration is desirable due to its protecting properties. However, an elevated HDL concentration may also elevate the total cholesterol. Furthermore, studies have shown that LDL-lowering treatments reduce the risk for CVD [10, 56-59]. Seeing the effect that one lipid has on another reiterates the fact that it is important to analyze CVD risk based on all of the lipid profiles and not just one [60].

#### **Cardiovascular Disease Risk Factors**

CVD risk can be associated with either lifestyle factors or biological factors. Some risk factors include those that have

proven interventions that result in a decrease in risk. Examples include high LDL cholesterol concentrations, hypertension, and cigarette smoking [34, 61-63]. Others include risk factors for which interventions are likely to decrease risk such as diabetes, physical inactivity, low HDL cholesterol concentration, and obesity [7, 8, 31, 33, 61-66]. Risk factors where more research is needed in order to determine if the intervention will lower the risk are risk factors such as psychosocial factors, elevated triglyceride concentrations, C-reactive protein, and oxidative stress [5, 7, 31, 33, 61, 62, 66, 67]. Lastly, risk factors that cannot be changed or modified with interventions consist of age, gender, ethnicity, and family history [5, 7, 31, 33-35, 61, 62, 65-71].

Additional biological factors that increase the risk for developing CVD include elevated concentrations of total cholesterol and fasting blood glucose because these factors contribute to the buildup of plaque in the arteries that can lead to atherosclerosis [36, 39, 40, 72].

Total cholesterol concentration is an independent risk factor of atherosclerotic plaque development [8, 13, 65, 73]. According

to a study by Kerenyi et al. [73], people with lower total cholesterol concentrations had significantly less plaque when compared with those who had any amount of carotid artery plaque buildup or lesions [74, 75].

As cholesterol attaches to the inner lining of the artery and plaque and lesions form, an inflammation response occurs. Damage occurs in the cells due to the inflammation. C-reactive protein (CRP) is then stimulated since its primary function is to attach to the plasma membrane of a damaged cell and cause death to that cell through the complement cascade [76]. Thus, CRP is a good indicator of inflammation and possible atherosclerosis progression. Elevated CRP concentrations (> 3.0 mg/L) have been found to be positively associated with having a high risk of developing CVD and a risk of future cardiovascular events such as myocardial infarction and stroke. [77-79].

Weight, specifically the body mass index (BMI), is positively associated with an increased risk for CVD and CHD [31, 65, 69]. Obese participants (BMI  $\geq$  30 kg/m<sup>2</sup>) have more CVD risk factors when compared to persons with a normal BMI (18.5-24.9 kg/m<sup>2</sup>) [7, 13, 33, 65, 69]. Similarly, persons considered obese had significantly higher blood pressure than those considered to have a normal BMI [8, 13, 31, 33, 34, 65, 80]. HDL cholesterol is negatively correlated with BMI, and triglycerides have a significantly positive correlation with BMI [33, 69]. Obese persons are 1.5 times more likely to die from complications due to cardiovascular disease than persons who are normal weight [8, 31, 69, 80].

Abdominal obesity is also associated with increased risk for CVD [81-85]. There is a positive association with waist circumference and CVD [83, 84, 86, 87]. Those who have a waist circumference greater than the recommended measurement (>102/88cm for men/women) were 1.25 times more likely to develop CVD [81, 82, 87]. A clustering of 1-2 factors for metabolic syndrome including waist circumference above recommendations doubled the risk for CVD [86]. Persons with the metabolic syndrome (clustering of 3 or more factors) including waist circumference above recommendations had 2.5 times the risk for develop CVD [86].

Elevated blood glucose concentrations have also been associated with an increased risk for developing CVD [13, 70, 80].

According to Nakagami et al. [70], 2-hour plasma glucose and fasting plasma glucose concentrations are positively associated with all-cause mortality and CVD. A person with an elevated 2hour plasma glucose or fasting plasma glucose concentration has a 1.14 and 1.24 increased risk for all-cause mortality or CVD mortality [70]. The results from one study show that persons dying from coronary heart disease or stroke have a significantly higher serum glucose concentration than those not dying from CVD (p=0.024) [13]. Another study reports that impaired fasting or impaired 2-hour glucose concentrations resulted in a 1.31 increased risk of mortality [80].

During a prolonged hyperglycemic state, there is a conversion of the reversible Schiff base adducts to more stable Amadori rearrangement products [87, 88]. With time, these Amadori products continue to experience further rearrangement reactions and ultimately form the permanently bound advanced glycation end products (AGE). AGEs form as a result of the non-enzymatic reaction between reducing sugars and biological proteins [88, 89]. AGEs have been found within atherosclerotic lesions in both extra and intracellular sites [90]. Furthermore, high concentrations of the AGE receptor (RAGE) have been associated with an increased inflammatory reaction in the plaque macrophages of atherosclerosis [91]. In a study by Semba et al. [92], 17% of those who died from CVD had the highest concentration of plasma AGE (p=0.001).

AGEs that form on the extracellular matrix cause a decrease in the elasticity of the vasculatures [88]. The AGEs also reduce the nitric oxide. As a result, the endothelium vasodilation is compromised and unable to perform accurately as needed [88]. Furthermore, the reduced or diminished nitric oxide, resulting from the oxidative stress caused by the AGEs, forms peroxynitrite. This in turn causes more endothelial cell damage and even platelet activation [88]. In addition, AGEs are capable of stimulating osteoblastic differentiation of microvascular pericytes. These ultimately add to the progression of vascular calcification in atherosclerosis [88].

#### Cardiovascular Disease Risk Factors Among Hispanics

As aforementioned, it is estimated that 30% of Mexican-American males and females have at least one form of CVD [2]. Biological risk factors include but are not limited to a person's lipid profile, fasting glucose concentrations, and insulin concentrations, as well as a person's BMI, waist circumference, and blood pressure. Mexican-Americans have been shown to have more biological and anthropometric risk factors when compared to both non-Hispanic Whites and other Hispanics.

Regarding CVD risk, Hispanics have 15% higher concentrations of triglycerides when compared to non-Hispanic Whites (p<0.001) [7, 31, 61, 67]. Furthermore, Hispanics have significantly lower HDL cholesterol concentrations (49.5 mg/dL vs. 55.3 mg/dL, p<0.001) [7, 31, 61, 65] and significantly higher glucose concentrations (107 mg/dL vs. 104 mg/dL; p=0.04) [7, 67] relative to non-Hispanic Whites. Similarly, insulin concentrations are also higher in Hispanics when compared to non-Hispanics (11.2 µU/mL vs. 9.3 µU/mL) [7, 65].

In comparing Mexican-Americans with other Hispanic-American ethnicities (Dominican Americans, Puerto Rican Americans, Other Hispanic Americans), Mexican-Americans have a higher mean value when exploring various risk factors for developing CVD, such as BMI (30.0 kg/m<sup>2</sup> vs. 27.9 kg/m<sup>2</sup>, 29.7 kg/m<sup>2</sup>, or 28.6 kg/m<sup>2</sup>, respectively), glucose (112.9 mg/dL vs. 105.1 mg/dL, 110.6 mg/dL, or 110.3 mg/dL, respectively), insulin (8.7 mU/L vs. 6.5 mU/L, 7.7 mU/L, or 7.7 mU/L, respectively), percent of participants having the metabolic syndrome (49.1% vs. 33.2%, 37.9%, or 37.6%, respectively), percent of participants having diabetes (22.3% vs. 16.1%, 19.7%, or 15.2%, respectively), triglyceride (173 mg/dL vs. 125 mg/dL, 134 mg/dL, or 147 mg/dL, respectively), low HDL cholesterol (46 mg/dL vs. 48 md/dL, 49 mg/dL, or 50 mg/dL, respectively), percent of participants having dyslipidemia (41.1% vs 36.9%, 33.4%, or 33.0%, respectively), and hsCRP (4.33 mg/L vs. 3.41 mg/L, 4.14 mg/dL, or 3.92 mg/dL, respectively) [5, 34]. All non-Mexican-American Hispanics had a significantly lower prevalence of atherosclerosis (OR=0.63, 95% CI: 0.45, 0.90) [5]. Furthermore, Mexican-Americans were twice as likely to develop aortic plague buildup when compared with other non-Mexican-American ethnicities [5].

#### Metabolic Syndrome

The metabolic syndrome is a cluster of metabolic abnormalities associated with an increased risk for diabetes and CVD [10]. According to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) the metabolic syndrome can be defined as the presence of three or more of the following risk
factors: abdominal obesity, hypertriglyceridemia, low HDL cholesterol, hypertension, and/or fasting hyperglycemia [10]. Abdominal obesity is the accumulation of visceral fat in the trunk region of the body, resulting in an increased waist circumference. Per NCEP ATP III definition [10], abdominal obesity is classified as waist circumference measurements >102 cm (men) and >88 cm (women). Hypertriglyceridemia is defined as fasting triglyceride concentrations ≥150 mg/dL. Low HDL cholesterol is considered a concentration of cholesterol in high density lipoprotein <40 mg/dL (men) and <50 mg/dL (women). Hypertension, or elevated blood pressure, is indicated by ≥130 mm Hg for systolic and ≥85 mm Hg for diastolic. Fasting hyperglycemia consists of blood glucose concentrations after fasting for 8-14 hours that are ≥100 mg/dL [93, 94].

The metabolic mechanisms leading to a clustering of the above mentioned risk factors are unknown, but it is often related with having excess body fat [11]. Moreover, increased abdominal adiposity is related to insulin resistance [7, 8, 12, 31, 65-67]. Insulin resistance, which ultimately results in hyperglycemia, is a major factor in the development of type 2 diabetes.

A frequent or constant hyperglycemic state can result in damage to arteries, veins, and organs, including the heart. High concentrations of glucose in arteries, both macrovascular and microvascular, result in a significant decrease in a cell's function and also their growth ability [95]. Furthermore, hyperglycemia will also significantly increase the occurrence of cell apoptosis when compared to normal glucose concentrations [95]. As a result, uncontrolled levels of glucose concentration in the blood are positively associated with CVD and macrovascular events [96, 97].

National surveillance data indicates that individuals with the metabolic syndrome have three times greater incidence of CVD than those without the syndrome [8, 13, 98]. Cardiovascular mortality is also increased in people with the metabolic syndrome [13, 98], and those with the metabolic syndrome are found to be two times more likely to die from CVD or complications related to CVD than those without the metabolic syndrome [8, 80]. Among people with the metabolic syndrome, the highest percent of people (10%) had a combination of obesity and dyslipidemia or obesity and hypertension [13, 98]. Furthermore, Hispanics have a significantly higher prevalence of

clustering of the metabolic syndrome factors (63% in Hispanics vs. 30% in non-Hispanic Whites) when compared to non-Hispanic Whites [67, 99]. A person's risk for developing CVD increases as the number of clustering factors increases [8, 13, 80, 100].

#### **Health Disparities**

Many ethnicities, including Mexican-Americans, have a higher prevalence for certain diseases that cannot be explained biologically. Furthermore, some barriers may also prevent them from receiving treatment that they need and deserve [7, 100-112]. However, it is important to realize the prevalence of disease for different ethnicities in order to prevent the disease and not just treat the disease. In regard to the prevalence of CVD risk factors among Hispanics or Latinos, 21.5% have hypertension, 8.5% have heart disease, 5.8% have coronary heart disease, and 2.0% have had a stroke [2].

Health care, in all its forms, is an important factor in determining a person's risk of developing chronic disease and also quality of life. It is also important to evaluate the type and quality of care a person may receive, their understanding of any problems and/or treatment, and their satisfaction with the care. Non-Hispanics have a greater utilization of health care services such as general physical examinations, eye exams, and dental checkups than Hispanics [101, 102]. Mexican-Americans have several barriers that may prevent them from receiving the quality of care that they both want and deserve. These barriers include: income to pay for health care visits, health insurance, language, location/neighborhood, cultural beliefs, ethnicity, and lower expectations of health care [7, 100, 101, 103-112].

Language may be a significant barrier since the patient needs to explain to the health care provider their concerns, symptoms, and observations. Likewise the health care provider needs to be able to communicate back to the patient in order to explain diagnosis and treatment options as well as answer questions that may arise. There is a negative association between language spoken and satisfaction such that those who speak primarily Spanish have a less satisfying health care experience than those who speak English [101, 103, 104].

Mexican-Americans are more likely to live in neighborhoods that have a high population of Hispanics. When comparing

communities that have comparable socioeconomic statuses, communities that are primarily Hispanic had significantly less physicians than those neighborhoods that were predominantly non-Hispanic White [107]. Being an ethnicity that is considered a minority is also associated with a higher prevalence of health problems and a poorer health status [113-115].

When compared to non-Hispanic Whites, Mexican-Americans are more than two times as likely to have a lower socioeconomic status and lower income level [7, 100, 108-110]. They are also more likely to have jobs that do not offer health insurance [110-112]. A greater number of Hispanics are uninsured when compared to non-Hispanics, a number that is associated with income [108, 110-112, 115]. Lack of health insurance creates a barrier to receiving health care services and also needed prescription medications [102, 104, 110, 115-118].

Another barrier that Mexican-Americans have is their cultural beliefs and traditions that may affect their reception of health care and treatment. For example, there are certain holidays, foods, and traditions that Mexican-Americans have celebrated for decades or even centuries and consider important. A healthcare provider belittling or degrading such traditions in the name of health, no matter how right they may be that the tradition is unhealthy, may offend the patient and discourage them from returning for more treatment later. On the other hand, healthcare providers that respect Mexican-American traditions and culture may impress the patient who is more likely to return for more treatment later [104, 118-120]. Some Hispanics also have beliefs that self-medication and other "home remedies" can be more effective at treating disease than prescriptions or treatments they could receive from their doctor or health care provider [119, 120].

#### Social Determinants of Cardiovascular Disease Risk

Aside from the known biological conditions leading to chronic diseases such as CVD and diabetes, research has shown that non-biological factors also contribute to these diseases. These non-biological factors include poverty, neighborhood environment, food access, level of education, acculturation, racism, discrimination, access to healthcare, language barriers, and stress [16-20, 121]. Lower income and education have been associated with a greater risk of coronary heart disease [18, 34, 61, 71, 122-124]. Moreover, inhabitants of lower socioeconomic communities tend to have a higher prevalence of obesity and obesity related disorders [7, 17-20, 125-130].

Mexican-Americans have a significantly lower level of education and income than non-Hispanics [7, 100, 109]. As a result, they are more likely to live in neighborhoods that are not conducive to physical activity and that do not have access to healthy food [125, 130-132]. Furthermore, lower income individuals or persons living in poverty may not have health insurance or funds in order to receive proper healthcare from a qualified professional [109, 133, 134]. As aforementioned, Mexican-Americans who have recently migrated to the United States may face a language barrier that prevents them from receiving desired services or from knowing where to find healthy food options.

## **Socioeconomic Status**

One predictor of a person's quality of life and a person's risk for developing chronic diseases such as CVD is their socioeconomic status [122, 123, 125, 127-130, 135, 136]. Socioeconomic status influences the likelihood that a person may or may not have the funds to buy healthy foods, receive medical treatment, or pay for needed medications. It can also affect where a person lives and whether or not that environment has access to healthy foods, healthcare facilities, pharmacies, or areas conducive for physical activity. Since socioeconomic status has significant effects on the prevention, diagnosis, and treatment of disease, and because Mexican-Americans are more likely to live in lower SES areas, it is important to explore its impact on Mexican-Americans [7, 125, 135]. Although there are several factors to consider when exploring socioeconomic status, the only areas that will be covered in this section are education and income.

## Education

Education is an ideal socioeconomic variable for use in studies because it is a factor that all participants will have regardless of their employment, age, gender, or marital status [135]. It also has a high level of validity and reliability. Furthermore, education is less likely to change after early adulthood, is easily reported, and can be a continuous variable as opposed to other variables such as income, age, and marital status that may vary during life [135]. Education can also affect a healthy lifestyle in that it influences life-style behaviors, access to health services, and higher self-esteem and self-efficacy [135]. Persons with lower education have more associations with risk factors for developing cardiovascular disease when compared with those who have higher education [71, 122, 123, 127-130, 135, 137]. Persons with higher education are more likely to be literate and use print media to learn about risks for CVD [70]. Education is positively associated with income [122, 130] which has an effect on food purchases, places of residence, and finances to pay for healthcare.

Education has been negatively associated with CVD risk factors [34, 71, 99, 121, 127-129]. Hispanics who are high school graduates when compared to Hispanics who have graduated from college have higher systolic blood pressure ( $126\pm14$  mm Hg vs.  $120\pm15$  mm Hg, p=0.004, respectively), higher percent who are hypertensive (39.6% vs. 18.4%, p<0.001, respectively), and higher mean total cholesterol ( $214\pm50$  mg/dL vs.  $196\pm39$  mg/dL, p=0.01, respectively) [71]. Fifty percent of Hispanics with some high school or less have metabolic syndrome while 9.5% of those who are high school graduates have metabolic syndrome [99].

In addition, there is a positive correlation between education and motivation to change health behavior and reduce CVD risk [71, 138]. Education is also positively associated with use of health-related print media [71] or attending community-based programs intended to help people improve their lifestyle [138]. Hispanics, when compared to non-Hispanic Whites, are more likely to have less than 12 years of education [61, 71]. In particular, Mexican-Americans are 3 times more likely to have at least one risk factor for developing CVD when their education level is <12 years relative to Mexican-Americans who have >12 years of education [71, 100, 122, 123, 127, 129, 130].

#### Income

Regardless of race or ethnicity, the risk for developing CVD has been reported to be higher among those individuals with lower income levels [122, 124, 136]. According to the National Health Interview Survey (NHIS), heart disease, ischemic heart disease, and hypertension were negatively correlated with poverty status and income [9, 135]. Poverty status was scored based on the family/household's total income with regard to the family/household size and then compared to the US Census Bureau's poverty thresholds.

Total cholesterol concentrations are significantly higher in persons with a lower income level than those persons with a higher income level [136, 139, 140]. Furthermore, income is negatively associated with waist circumference, blood pressure, serum glucose levels, and triglyceride concentrations [127, 140-142]. In addition, LDL cholesterol concentrations and C-reactive protein are negatively associated with income while HDL cholesterol concentrations are positively correlated with income [129, 140, 143].

Moreover, neighborhoods with a higher median income may have better access to parks, better maintained roads and walkways, and more food options, thus resulting in more physical activity and decreased caloric intake [63, 121, 131, 134]. Furthermore, households with a higher income may have more access to health care and medical insurance which could lower their risk for obesity [109, 133, 134]. Lastly, for each \$1000 increase in median household income there is a resulted 0.6% decrease in obesity risk [134, 137].

As previously mentioned, one predictor of a person's risk for developing CVD is their socioeconomic status, including income [122, 123, 125, 127-130, 135, 136]. When compared to non-Hispanics, Mexican-Americans have a significantly lower income [7, 100, 109]. Among Hispanics, income has been negatively associated with BMI [130, 137]. One study looking at percent body fat and obesity in low-income children found that the mean percent body fat is greater in Mexican-American children when compared to non-Hispanic White children (24% vs. 17%, respectively; p=0.003) [144].

Another study focused on prevalence of the metabolic syndrome among Mexican-Americans, non-Hispanic Whites, and African Americans [145]. The participants were compared by economic category (low, middle, high) and race/ethnicity. Only 12% of Mexican-Americans were grouped in the highest economic category. Significantly more women in the low economic category (34%) were classified as having the metabolic syndrome when compared to the middle (28%) or high (21%) categories (p=0.0009) [145]. When comparing women in the various race/ethnic groups, Mexican-Americans had a significantly higher prevalence of elevated glucose (19%, p=0.0044), waist circumference >88cm (67%, p=0.0002), low HDL cholesterol (57%, p=0.0092), and elevated triglycerides (30%, p=0.0042) than non-Hispanic Whites or African Americans. Among men, there was no significant difference among economic category. Mexican-American men, when compared to the non-Hispanic Whites and African Americans, had a significantly higher prevalence of low HDL (46%, p=0.0006) and elevated triglycerides (42%, p<0.0001) [145].

#### Environment

#### Access to Food

A large contributing factor to the ability of people to have healthful diets and thus reduce their risk for CVD is their ability to access healthy foods. Several factors can affect food access and fruit and vegetable consumption. Studies have found a relationship between income and food access [146, 147]. The neighborhood where someone lives influences the number and type of supermarket, the quality of the products, and the cost of the food available [148-150].

Not only does access to food involve the food environment but also how people adapt and live within that environment. One

example is that if neighborhoods are unsafe for walking, people will be less likely to walk no matter the distance to their destination. Furthermore, whether a household owns a car might have a greater importance in determining access to food than actual distance to the store indicating that time may have as much of an impact on food access as does distance and transportation [146].

Food environments differ by community [151]. Physical access to healthy food choices differs by location, and these differences are often patterned according to socio-demographic characteristics of the residence of the community [151]. For example, Powell et al. [152] found that all food store types are significantly less available among rural areas than in urban areas. Furthermore, their study showed that lower income neighborhoods had greater access to non-chain supermarkets and grocery stores. On the other hand, the urban low-income neighborhoods had access to more convenience stores [152]. Another study reported that areas where predominantly minorities resided had a lower socioeconomic status and had four times more liquor stores and fewer grocery stores when compared to the middle socioeconomic position residents [25]. Additionally, chain supermarkets have lower food prices and higher quality food products when compared to non-chain supermarkets and smaller grocery stores [152].

## Access to Fruits and Vegetables

Diets consisting of a variety of fruits, vegetables, whole grains, and low-fat dairy foods can reduce risk for metabolic syndrome and CVD [153-155]. Such diets have been shown to raise HDL cholesterol, lower triglycerides, lower systolic and diastolic blood pressure, lower weight, and lower blood glucose concentrations [154]. One study found that less-acculturated, low-income Hispanics eat more fruits and vegetables than bicultural or moreacculturated peers. Despite this fact, 75% of all the participants in the study still fell short of the dietary recommendations of a minimum of 5 servings of fruits and vegetables per day [156].

One factor affecting the consumption of fruits and vegetables is distance to the supermarket. One study found a negative correlation between distance to the supermarket and fruit and vegetable consumption [157]. However, the results were only significant for metropolitan areas and not for rural areas. One factor may be that to travel the same distance may take longer in a busy and congested urban area versus a less busy rural area. Thus, supermarket accessibility measured by distance may be different in urban versus rural dominated environments [157].

In general, although people may have access to foods, the selection may be limited in fruits and vegetables. Research showed that non-chain grocery stores were less likely to sell foods classified as healthy (e.g. whole wheat bread, skinless chicken) than chain supermarkets, and that the quality of produce was lower in non-chain grocery stores [158, 159]. Results show that the store type was related to the food choices and the availability of those choices [159]. Chain supermarkets have more availability of fresh, canned, and frozen fruits and vegetables than independent groceries, drug stores, or convenience stores. Furthermore, supermarkets (chain, independent, and discount) had three times as many fruits and vegetables available as smaller grocery stores, drug stores, and convenience stores [159].

Block and Kouba [159] found that the grocery stores, as opposed to larger supermarkets, provided produce at a

competitive price. They also found that produce quality varied greatly between store types. Supermarkets had higher quality produce whereas grocery store produce was not satisfactory quality. They concluded that access to healthy food choices is related more to the type of store than to the number of stores in a given area [159].

Another study by Bodor and Rose found that mean space dedicated for fruits and vegetables was considerably larger for supermarkets than for small food stores [28]. In addition, supermarkets had 70% of fruit and vegetable shelf space available for fresh produce, where as small food stores allotted only 32% of their fruit and vegetable shelf space to fresh produce. Moreover, supermarkets also offered a substantially larger variety of fresh produce than did small food stores. Therefore, a greater fresh vegetable availability was a positive predictor of vegetable intake. However, fresh fruit availability was not associated with intake [28].

Individuals with a high school education or less are reported to have decreased access to a large selection of fruits and vegetables [160]. This same group was also reported to be more likely to shop at convenience stores [160]. Thus, those with a high school education or less may not have access to foods that allow them to follow the aforementioned recommendation of consuming a diet consisting of a variety of fruits, vegetables, whole grains, and low-fat dairy foods which may reduce risk for metabolic syndrome and CVD [153-155]. Such diets have been shown to raise HDL cholesterol, lower triglycerides, lower systolic and diastolic blood pressure, lower weight, and lower blood glucose concentrations [154].

## **Cost of Food and Food Quality**

Another contributing factor besides access to food is the cost of healthy foods when compared with energy dense foods. Lipsky et al. [161] found an inverse relationship between energy density (defined as kcal/gram) and energy cost (defined as cost/kcal). Although the total package price and unit price of produce were lower than those of snacks, the average per serving price for produce was higher than for snack foods ( $0.70\pm0.10$ /serving produce vs.  $0.40\pm0.04$ /serving of snacks, p=0.02). Furthermore, inner-city markets were found to charge more for their food than do larger chain grocery stores found in suburban communities [161]. Rural areas have reported disadvantages when examining availability, accessibility, and adequacy of healthy foods [162, 163].

The Changing Individuals' Purchase of Snacks (CHIPS) study [164] found that by reducing the price of low-fat snacks sold in vending machines by 10%, 20%, and 50%, there was an associated increase in low-fat snack sales. Similarly, a study done in a supermarket found that by using verbal prompts, product sampling, and price reduction there was an increase in sales for lower-fat products [165].

#### Neighborhood

The layout and accessibility within a neighborhood can have an effect on the health outcome of people living within that neighborhood. Factors that have an effect include availability of supermarkets or other food stores that supply healthy food choices, parks and other recreational facilities, well maintained sidewalks and walkways, walkability, and street connectivity [134]. Walkability is the ease with which a person can walk to their destination, such as a store or park. This involves the condition of the sidewalk, the number of busy streets to cross, and how close to the traffic the sidewalk is placed. The greater

the walkability, the more inviting it will be for pedestrians. Street connectivity refers to the size of blocks, the number of intersections and roads to cross, and the ease or ability to get from one part of a community to another either in a direct manner or through multiple pathways [134].

There is a positive association between obesity risk and urban spacing [134]. Urban spacing involves the distances between residences and the layout of the streets [134]. Furthermore, there is also a positive relationship between neighborhood structure, including street connectivity and physical activity, and obesity risk [134].

In looking at different ethnic groups, Lopez [134] hypothesized four different neighborhood-related associations that could be possible factors for increasing the risk of obesity within the Hispanic population: 1) the potential lack of, or poorly maintained, infrastructure, including street lights, sidewalks, public transportation, or parks, might reduce physical activity; 2) public safety conditions or perceptions may decrease physical activity; 3) socioeconomic conditions or lack of private investment may reduce quality or availability of recreation facilities or places that sell nutritious food; 4) targeted advertising may include less healthy consumer products.

Evidence suggests that the environment in which people live can influence their likeliness to walk [166]. People tend to walk more in communities that have sidewalks in good condition with few obstructions, provide destinations and facilities that can be reached within walking distance and are free from physical disarray such as trash or abandoned buildings [167]. Moreover, individuals who live in low income and racial and ethnic minority communities tend to experience having less of an access to environmental features that support physical activity when compared with higher income communities [166].

Neighborhood areas that are reported by residents as safe have been significantly related to walking activity [167]. There is also a significant correlation between the number of street intersections and the perceptions of safety from traffic [167]. Furthermore, residents in communities with more street intersections who reported being safer from traffic tended to also report more walking activity within their neighborhood [167]. Another study reports that individuals who perceived that they had no place to walk were significantly less healthy than persons who thought they had at least one place to walk [168].

Additionally, another study explored racial and ethnic disparities in response to direct-to-consumer advertising (DTCA) [169]. Non-Hispanic Whites were more likely than Hispanics and African Americans to be exposed to DTCA. However, Hispanics and African Americans are more likely to be influenced by DTCA than Whites [169]. Moreover, Hispanics and African Americans are more positive about the health benefits they are exposed to through DTCA [169].

Evidence from the aforementioned article by Lopez also shows that an increase in population density results in a decrease in obesity risk [134]. In addition, an increase in population density may be associated with greater walkability, more destinations for walking, increased likelihood that a person will use alternatives to driving, or amenities that increase the possibility that physical activity will take place [134]. Another study by Stark-Casagrande et al. [170] found that individuals living in high socioeconomic status areas with highly walkable neighborhoods

had a lower prevalence of obesity when compared with people living in poorly walkable neighborhoods.

Rundle et al. [171] conducted a study that examined the association of neighborhood environments with BMI and obesity. The results of this study were that the highest density of healthy food stores was located in higher-income neighborhoods [171]. On the other hand there was a higher concentration of food stores that did not supply a large selection of healthy food choices in the lower-income neighborhoods. Therefore, it can be concluded that a lower BMI and lower prevalence of obesity may be associated with access to healthy food stores [171].

## Acculturation

Acculturation refers to the extent that an immigrant accepts and adopts the beliefs, customs, and practices of the country to where they have relocated [172]. Acculturation and length of residence in the U.S. have been associated with increased risk for CVD and metabolic abnormalities [22]. Greater acculturation has been reported to negatively affect diet. For example, among Mexican-Americans, greater acculturation has been correlated with lower fruit and vegetable consumption [23, 156]. When comparing non-Hispanic Whites with Hispanics, non-Hispanic Whites had a mean consumption of 0.87 servings of fruit and 0.31 servings of vegetables less than that of Hispanics [173-176]. Furthermore, non-U.S. born Hispanics eat significantly more fruits and vegetables than U.S. born Hispanics [173, 174]. Mexican-Americans with a lower acculturation score had a significantly higher daily intake of fruits and vegetables than Hispanics that are more acculturated (5.07 servings/day vs. 4.70 servings per day, p<0.05, respectively) [176].

In a study comparing fruit and vegetable intake of Mexican-American and non-Hispanic White women, there was a lower consumption of fruits and vegetables among Mexican-American women with greater acculturation [22]. Furthermore, less acculturated Mexican-American participants had significantly higher servings of fruits and vegetables each day than Mexican-Americans who were more acculturated to American customs [22]. This suggests that acculturation could be an independent predictor of diet. It can also be suggested that the American culture does not support a diet high in fruits and vegetables and

that there is an association between level of acculturation and fruit and vegetable consumption among Hispanics.

Another study reports that Hispanics, regardless of their level of acculturation, were significantly less likely than non-Hispanic Whites to prefer the "fruit and breakfast cereal" diet and more likely to prefer the "high starchy foods" diet [177]. Accordingly, the rice and starchy food diet, common among Hispanics, was associated with a significantly higher BMI and waist circumference when compared with the fruits and breakfast cereals diet [178].

Acculturation appears to also be positively associated with participating in leisure-time physical activity [178]. This may be due to the aforementioned neighborhood environment and socioeconomic status of Hispanics resulting in environmental and economic barriers to accessing fitness facilities, such as gyms, and safe recreational areas where they can participate in physical activities [178].

## Conclusion

Several non-biological risk factors positively affect cardiovascular disease risk. These factors are connected in such a way that one factor may have an effect on another factor which results in an increase in cardiovascular or metabolic disease risk. Therefore, due to multiple risk factors and their interlinking tendencies, it is difficult to know which of the factors causes an increased risk for cardiovascular disease the most.

Specific information among Mexican-Americans is scarce, and it is difficult to generalize the findings of prior studies to the Mexican-American population of the Phoenix metropolitan area due to differing environmental factors, known or unknown, which may have contributed to the results of prior studies. Therefore, the purpose of this preliminary study is to conduct an initial assessment of the effects of non-biological factors on cardiometabolic risk among Mexican-Americans living in metropolitan Phoenix.

### **METHODS AND MATERIALS**

This study was approved by the Institutional Review Board at Arizona State University (IRB Protocol # 0910004426; PI: Sonia Vega-López). The approval notice is attached in Appendix I.

## Materials

Reagents for measuring total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, glucose and hsCRP as well as deionized water were purchased from Roche Diagnostics (Indianapolis, IN). The reagent for measuring insulin was purchased from Siemens Medical Solutions Diagnostics (Los Angeles, CA).

## Participants

Seventy-five self reported Mexican-American adults were recruited free from diabetes and chronic diseases between the ages of 21 and 60. Participants were excluded if they followed a specific diet regime (veganism, very low carbohydrate diet, etc.), or if they were participating in any other research study in which diet was assessed or manipulated. Further exclusion criteria included:

- Inability to walk for exercise
- History of difficult vein access

- Fear of needles or blood drawing, or adverse reactions to blood drawing (fainting)
- Body weight less than 110 lb
- Pregnancy or breastfeeding
- Use of cholesterol lowering medications (cholestyramine, colestipol, niacin, HMG CoA reductase inhibitors, gemfibrozil, fenofibrate, clofibrate, thiazide diuretics, ezetimibe, probucol, colesevelam, ciprofibrate, diphenylhydantoin)
- Heart disease
- Diabetes
- Renal disease
- Liver disease or hepatitis
- Cancer
- Thyroid disease unless controlled with medication for at least 6 months

# **Recruitment and Consenting**

Participants were recruited from Maricopa County using flyers and advertisements placed in local community centers and local stores, Spanish language community newsletters and newspapers, through the Maricopa Insulin Resistance Initiative database, and through electronic distribution list (Appendix II). Individuals interested in participation in the study had a phone interview in which a trained research assistant explained the details of the study and answered any questions the participant may have had. The interview had a threefold purpose: first, to explain study purpose and procedures; second, to obtain verbal consent for screening to assess if the potential participant qualified for the study; and third, to verify eligibility via a short questionnaire including questions about exclusion criteria (Appendix III). A study visit was scheduled for those participants who meet the inclusion criteria. Participants were mailed a food frequency questionnaire (Appendix IV) to complete prior to their visit. At the beginning of the study visit, time was allotted to answer questions participants may have had after which written informed consent was obtained. Participants were assured that participation was voluntary and that they were able to discontinue at any point if they chose to. The informed consent form is attached in Appendix V.

# **Study Design**

This study had a cross-sectional design with one visit per participant unless the participant failed to fast prior to their visit for at least 8 hours at which point another appointment was scheduled for a blood draw only. Data regarding socioeconomic factors including education, income, and employment, and lifestyle factors such as diet were gathered using a survey (Appendix VI). Anthropometrics gathered included height, weight, body fat percentage, waist circumference, and blood pressure. Biomarkers examined were total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, hsCRP, fasting blood glucose, and insulin.

## **Study Protocol**

On the day scheduled for their visit, participants arrived between 7:30 – 9:30 am after fasting for 12 hours. The study procedures were explained and participants were allowed to ask questions prior to consenting to participate in the study. Once written consent was collected, the completed food frequency questionnaire was retrieved and anthropometric measurements were taken including their weight, height, percent body fat, and waist and hip circumferences. After a 5 minute rest blood pressure was also taken. These measurements were taken in triplicate and were recorded using an Anthropometrics Documentation Form (Appendix VII). Following the measurements, a venous blood sample was collected for the measurement of fasting chronic disease risk factors. Record of fasting status and the successful completion of blood withdrawal were kept using a Blood Draw Documentation Form (Appendix VIII).

Thereafter, the survey designed to collect socio-demographic, dietary, socioeconomic, and acculturation information was administered by a trained bilingual research assistant for survey administration consistency. All survey instruments were available in English and Spanish and were administered to study participants in their language of preference (Appendix VI).

#### Methods

#### Anthropometrics

Weight was measured in kilograms using a Tanita body composition analyzer (Tanita Corporation, Tokyo, Japan) which also measured percent body fat. Height was measured in centimeters using a wall mounted stadiometer. Waist and hip circumferences were measured using a flexible tape measure. Waist was measured at the belly button, and hip was measured at the largest portion of the hips. Blood pressure was taken following a 5 minute rest from the participant's left arm using an electronic sphygmomanometer (IntelliSense Blood Pressure Monitor HEM-907XL, Omron Healthcare, Kyoto City, Japan). All measurements were taken in triplicate to ensure validity. Body mass index (BMI) was calculated as mean weight in kilograms divided by mean height in meters squared (kg/m<sup>2</sup>).

### **Biological Markers**

A 40 mL sample of fasting blood was drawn for the measurement of biological markers of CVD risk including glucose, insulin, hsCRP, and lipids (total cholesterol, HDL cholesterol, triglycerides). Serum/plasma was separated by centrifugation at 1,100 x g at 4°C for 20 minutes. Samples were aliquoted and frozen at -80 °C until analyzed.

A complete lipid panel (total cholesterol, HDL cholesterol and triglycerides) and glucose were measured in serum with colorimetric enzymatic assays using an automated chemistry analyzer (Cobas C111; Roche Diagnostics, Indianapolis, IN). LDL cholesterol was calculated using the Friedewald equation [179]. Homeostatic model assessment (HOMA) is a method used in order to compare insulin resistance and beta-cell function. It was calculated as: fasting plasma glucose (mM) x insulin  $(\mu U/ml)/22.5$  [180, 181].

Serum hsCRP was measured with a turbidimetric assay using an automated chemistry analyzer (Cobas C111; Roche Diagnostics, Indianapolis, IN). The automated chemistry analyzer was calibrated and tested for quality control for each analysis. Insulin was measured by immunoassay using an automated instrument (Immulite, Siemens Medical Solutions Diagnostics, Los Angeles, CA).

## **Diet Analysis**

Diets were assessed using the semi-quantitative Southwestern Food Frequency Questionnaire (Appendix IV) [182, 183]. This food frequency questionnaire was chosen because it is a bilingual questionnaire widely used in the United States and includes foods that are culturally appropriate for the Mexican diet. Information derived from this questionnaire focused on dietary macronutrient composition, namely total fat, saturated fat, *trans* fat, complex carbohydrates, and protein. Data was also analyzed to reflect the percent of energy in kilocalories that came from total fat, saturated fat, *trans* fat, monounsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA), carbohydrates, and protein.

### **Socioeconomic Factors**

Participants completed a survey that included questions regarding last level of education completed, monthly total household income, acculturation, and employment status (Appendix VI). Acculturation was measured using the Acculturation Rating Scale for Mexican-Americans – II (ARSMA-II; Appendix IX) [184], a multidimensional scale designed to measure acculturation related to language, ethnic identity, and ethnic interaction. The factor structure, reliability, and validity of the ARSMA-II have been well established in English and Spanish [184].

The acculturation part of the survey included questions that asked the participant about certain daily activities (e.g. speaking, reading, watching TV, listening to music, friends, etc.) done in either English or Spanish. The participant responded with how often they did the certain activity in either English or Spanish. The questions are classified as either an Anglo Orientation Scale (AOS) or Mexican Orientation Scale (MOS). Each participant is scored (1-5) according to their answers to the AOS and MOS questions.

The mean score for the MOS questions is subtracted from the mean score for the AOS questions in order to obtain an acculturation score. A scale is then used to determine, from the participant's acculturation score, what their acculturation level/categorization is. An acculturation level of 1 is a person who is very Mexican oriented with an acculturation score of <-1.33. An acculturation level of 2 means the person is between being Mexican oriented and being approximately balanced biculturally. Level 2 includes acculturation scores  $\geq$  -1.33 to  $\leq$  -0.07. Level 3 are those Mexicans with a slightly Anglo oriented biculture and a score between >-0.07 and <1.19. Level 4 includes scores of  $\geq$ 1.19 and <2.45 and defines Mexicans as being strongly Anglo oriented. Lastly, Level 5 are those Mexicans who are very assimilated and anglicized with a score  $\geq$ 2.45 (Appendix IX).

## **Data Analysis and Interpretation**

Statistical analyses were conducted using SPSS 17.0 for Windows (SPSS, Inc., Chicago, IL). Data was examined for

normality and, if necessary, was transformed. The biological data that was transformed includes triglycerides (inverse), hsCRP (log), and insulin (log). The only data from macronutrient consumption that was transformed was total energy (log). Correlation analyses were used to explore the relation among acculturation, diet macronutrient composition, and anthropometric measurements with cardiometabolic risk factors. In addition to descriptive tables and plots, explanatory variables were tested using analysis of variance (ANOVA). ANOVA stratified participants based on income, employment, education, and acculturation categories. The post-hoc LSD was used for analyzing biological markers and macronutrient consumption with income, education, and acculturation.

Outliers were defined as data that was more than 3 standard deviations from the mean. Biological data containing outliers that were removed for the statistical analysis included: BMI, diastolic blood pressure, total cholesterol, LDL cholesterol, LDL/HDL cholesterol ratio, total/HDL cholesterol ratio, triglycerides, high sensitivity C-reactive protein (hsCRP), and fasting plasma glucose. One participant had an extremely elevated triglyceride concentration which made it impossible to
calculate the LDL cholesterol and the LDL/HDL cholesterol ratio using the Friedewald calculation. The macronutrient data containing outliers that were removed included: total energy, total fat, saturated fat, *trans* fat, carbohydrates, sugar, and protein.

#### RESULTS

### **Participant Sociodemographic Characteristics**

A total of 75 individuals participated in this study. A majority of the participants were female (n= 49, 65.3% female; n=26, 34.7% male). All of the participants self identified themselves as Mexican (n=37, 49%), Mexican-American (n=28, 37%), or Hispanic/Latino (n=10, 13%). The mean residence time in the United States was 23.8 years (SD= $\pm$ 14.8) [Table 1].

The level of education that was completed by the participants are as follows: 5% completed elementary school, 15% completed middle school, 15% completed high school, 36% completed some college, and 29% were college graduates or higher. Sixty-seven percent (n=50) of the participants spoke both Spanish and English, 25% (n=19) of the participants spoke Spanish only, and 8% (n=6) spoke English only [Table 1].

Sixty-one percent (n=45) of the participants were employed at the time of the study. Of those, 35 participants (47% of total) were working full time ( $\geq$ 35 hrs/week) and 10 participants (14% of total) were working part time (<35 hrs/week). Thirty-nine percent (n=29) of the participants were not employed. Seventeen participants (23%) had a household monthly income of \$0-1000, 18 participants (24%) had a household monthly income of \$1001-2000, 15 participants (20%) had a household monthly income of \$2001-3000, 9 participants (13%) had a household monthly income of \$3001-4000, and 15 participants (20%) had a household monthly income of >\$4000 [Table 1].

Acculturation level was ranked based on their calculated acculturation score on a scale from 1 to 5 (ARSMA-II) [184]. Those with an acculturation level of 1 are least acculturated to the American culture, and those with an acculturation score of 5 are most acculturated to the American culture. The mean acculturation level for the participants was  $2.27\pm1.11$ . The majority of participants had an acculturation level of 1 (n=25, 34%). Fifteen participants (21%) had an acculturation level of 2, and 21 participants (29%) had an acculturation level of 3. The least number of participants had an acculturation level of 4 (n=12, 16%). Two participants had incomplete data which prevented the calculation of their acculturation level. Therefore, they were not included in this data [Table 1].

67

Participants were classified according to the Mexican-American generation they belong to as follows:  $1^{st}$  generation=born in Mexico or other country;  $2^{nd}$  generation=born in the US, at least one parent born in Mexico or other country;  $3^{rd}$  generation=born in the US, both parents born in the US, all grandparents born in Mexico or other country;  $4^{th}$  generation=born in the US, both parents born in the US, all grandparents born in Mexico or other country;  $5^{th}$  generation=born in US, both parents born in US, at least one grandparent born in Mexico or other country;  $5^{th}$  generation=born in US, both parents born in US, all grandparents born in US, all grandparents born in US. Fifty-three percent of the participants were  $1^{st}$  generation (n=40), 28% of the participants were  $2^{nd}$  generation (n=21), the rest were  $3^{rd}$ ,  $4^{th}$ , or  $5^{th}$  generation (n=1, 1%; n=9, 12%, n=4, 5%, respectively) [Table 1].

## **Cardiometabolic disease risk factors**

The cardiometabolic disease risk factors for the male and female participants were compared using an independent sample t-test [Table 2]. One participant had an extremely elevated triglyceride level which made it impossible to calculate LDL using the Friedewald equation. Thus, the ratio between cholesterol in LDL and HDL also could not be calculated for that participant. The mean participant age was 37.6±9.3 years. Mean weight was 78 $\pm$ 16 kg. Mean BMI was 28.9 $\pm$ 5.3 kg/m<sup>2</sup>. Male participants had 15% greater weight than female participants  $(85\pm13 \text{ kg vs. } 74\pm16 \text{ kg, } p=0.005)$ , but BMI did not significantly differ between males and females. Mean waist circumference was  $95\pm13$  cm. Mean percent body fat was  $34\pm9.1$  and was 40% greater in females than males (37.7±8.7% vs. 26.9±4.8%, p < 0.0001). Mean systolic blood pressure was  $117 \pm 11$  mm Hg and mean diastolic blood pressure was 73±9 mm Hg, with no differences between male and female participants. Mean total, LDL-, and HDL-cholesterol concentrations were 183±39 mg/dL, 114±32 mg/dL, and 44±11 mg/dL, respectively. HDL cholesterol was 24% higher in females than in males (47±11 mg/dL vs.  $38\pm11$  mg/dL, p=0.001). Mean total/HDL cholesterol ratio was 4.5±2.0. Male participants had 38% higher total/HDL cholesterol ratio than female participants  $(5.5\pm2.7 \text{ vs. } 4.0\pm1.2,$ p=0.015). Mean LDL/HDL cholesterol ratio was 2.8±1.1 and was 32% higher for males than females  $(3.3\pm1.2 \text{ vs. } 2.5\pm0.9,$ p=0.002). Mean triglycerides were 115±61 mg/dL and mean hsCRP was  $4.2\pm6.7$  mg/L. Mean fasting plasma glucose was 94±14 mg/dL, and male participants had 9% higher glucose concentrations than female participants  $(99\pm21 \text{ mg/dL vs. } 91\pm7)$ 

mg/dL, p=0.018). Mean insulin was  $8.7\pm5.8 \mu$ IU/mL. Mean HOMA score was  $2.0\pm1.4$ .

# Association of cardiometabolic disease risk factors with non-biological factors

Cardiometabolic disease risk factors after stratifying participants based on non-biological factors (income, education, employment, acculturation level) are displayed in Tables 3-6. When stratifying participants based on income [Table 3], those with a household income between \$2001 and \$3000 per month had 20% greater waist circumference than those participants with a household monthly income between \$3001 and \$4000 (103±11 cm vs. 86±11 cm, p=0.017). Similarly, weight and BMI were significantly higher among participants with a monthly income of \$2001-3000 relative to those earning \$3001-4000 per month (89±15 kg vs. 65±13 kg, p=0.037; 31.4±3.6 kg/m<sup>2</sup> vs. 25.6±3.9 kg/m<sup>2</sup>, p=0.005; respectively). The rest of the cardiometabolic disease risk factors did not differ by income level [Table 3].

Participants were stratified into income categories of those who earn  $\leq$ \$3000 per month and those who earn >\$3000 per month.

Those earning  $\leq$ \$3000 per month had significantly higher weight, BMI, and waist circumferences than those earning >\$3000 per month (81±15 kg vs. 71±15 kg, p=0.005; 29.8±4.6 kg/m<sup>2</sup> vs. 26.5±5.1 kg/m<sup>2</sup>, p=0.007; 98±12 cm vs. 89±14 cm, p=0.008; respectively). Furthermore, HDL cholesterol concentrations were significantly higher for those earning>\$3000 than those earning  $\leq$ \$3000 per month (49±12 mg/dL vs. 41±10 mg/dL, p=0.003). When compared to those earning >\$3000 per month, glucose, insulin, and HOMA were significantly higher than those earning  $\leq$ \$3000 per month (93±7 mg/dL vs. 90±5 mg/dL, p=0.048; 9.7±5.9 µIU/mL vs. 6.4±51 µIU/mL, p=0.023; 2.2±1.4 vs. 1.5±1.2, p=0.019; respectively) [Table 4].

After stratifying participants according to education, weight was significantly higher for those who completed elementary school or those who completed high school than for those who completed some college ( $93\pm19$  kg,  $87\pm17$ , and  $72\pm14$  kg, respectively; p=0.023). HDL cholesterol was significantly higher in participants who had completed some college or graduated from college than those who completed elementary school ( $47\pm10$  mg/dL &  $47\pm12$  mg/dL vs.  $33\pm10$  mg/dL, p=0.005; respectively). Similarly, the total/HDL cholesterol ratio was significantly higher for participants who completed elementary school when compared with all other levels of education (Elementary,  $6.7\pm1.3$  vs. Middle School,  $5.0\pm1.3$ ; High School,  $4.9\pm0.8$ ; Some College,  $3.6\pm0.9$ ; College Graduate  $4.3\pm1.5$ ; p<0.0001). The LDL/HDL cholesterol ratio was significantly lower for participants who had completed some college or graduated from college than those who completed elementary school ( $2.2\pm0.8 \& 2.7\pm1.1 vs. 3.9\pm1.1$ , respectively; p=0.002). Triglycerides were significantly higher for participants who completed elementary school than for those who completed high school, some college, or graduated from college ( $195\pm68 mg/dL$ vs.  $115\pm40 mg/dL$ ,  $89\pm32 mg/dL$ , and  $116\pm70 mg/dL$ , respectively; p=0.005) [Table 5].

The cardiometabolic disease risk factors were also compared among participants when education was also grouped into three categories: (1) those that completed elementary, middle, and high school, (2) those that completed some college, and (3) those who were college graduates or higher [Table 6]. Compared to those who completed some college, those who completed elementary, middle, and high school had a

significantly higher weight ( $84\pm16$  kg vs.  $72\pm14$  kg, p=0.024), significantly higher BMI ( $30.6\pm4.2$  kg/m<sup>2</sup> vs.  $26.9\pm4.9$  kg/m<sup>2</sup>, p=0.024), and significantly larger waist circumference (100±11) cm vs.  $91\pm13$  cm, p=0.029). Participants who completed only elementary, middle or high school had significantly lower HDL cholesterol concentrations  $(37\pm9 \text{ mg/dL vs. } 47\pm10 \text{ mg/dL \&})$  $47\pm12$  mg/dL, respectively; p=0.001). Compared to those who completed some college and those who were college graduates or higher, those who completed elementary, middle, and high school had a significantly higher total/HDL cholesterol ratio  $(5.2\pm1.2 \text{ vs. } 3.6\pm1.9 \& 4.3\pm1.5, \text{ respectively; } p<0.0001)$  and significantly higher LDL/HDL cholesterol ratio (3.3±0.8 vs.  $2.2\pm0.8 \& 2.7\pm1.1$ , respectively; p=0.001). Participants who completed elementary, middle, and high school had the highest triglycerides concentrations  $(138\pm67 \text{ mg/dL vs. } 89\pm32 \text{ mg/dL } \&$ 116 $\pm$ 70 mg/dL, respectively; p=0.011) and fasting plasma glucose (94±7 mg/dL vs. 92±7 mg/dL & 89±5 mg/dL, respectively; p=0.030) [Table 6]. There were no other significant differences between the groups.

When comparing participants based on employment status, those who were employed had 15% greater HDL cholesterol concentrations than participants not employed ( $46\pm10 \text{ mg/dL}$ vs.  $40\pm12 \text{ mg/dL}$ , p=0.024) [Table 7]. Among participants who were working, cardiometabolic disease risk factors were not significantly different when comparing those employed full time vs. those employed part time [Table 7]. Despite the lack of significance, HDL cholesterol was 20% higher in full-time employees relative to part-time workers ( $48\pm10 \text{ mg/dL}$  vs.  $40\pm9 \text{ mg/dL}$ ; p=0.051).

There were no significant differences in cardiometabolic disease risk factors when stratifying participants based on their acculturation level [Table 8].

# **Dietary Macronutrients**

Participants' daily macronutrient composition of the diet is displayed in Table 9. Mean participant energy consumption was 2447±1371 kcal. Men consumed 20% more energy than women (2761±1542 kcal vs. 2293±1267 kcal), but this difference did not reach statistical significance (p=0.172). Mean total fat consumption was 89±56 grams. Male participants consumed 37% more grams of total fat than female participants (108±66 g/d vs. 79±48 g/d, p=0.037). Mean saturated fatty acid intake was 30±19 grams. The mean consumption of *trans* fatty acid was 1.5±1.2 grams. Mean participant carbohydrate consumption was  $345\pm224$  grams of which 44% ( $151\pm126$  g) corresponded to sugar. Mean daily protein intake was 109±70 grams, with men consuming 40% more protein than women  $(134\pm89 \text{ g vs. } 96\pm54 \text{ g; } p=0.056)$ . The contribution of macronutrients towards energy intake is as follows:  $31\pm5\%$  of energy from total fat,  $53\pm7\%$  from carbohydrates, and  $17\pm3\%$ from protein, with no significant differences in macronutrient composition of the diet between men and women despite the difference in total fat intake noted above. Participants consumed  $10.3\pm2.2\%$  of energy,  $12.1\pm1.9\%$  of energy, and  $6.1\pm1.2\%$  of energy from saturated, monounsaturated, and polyunsaturated fatty acids, respectively. Of the 53% of energy consumed from carbohydrates, almost half  $(23\pm6\% \text{ of total energy})$  was consumed as sugar [Table 9].

### Macronutrient consumption and non-biological factors

The dietary macronutrient composition after stratifying participants based on non-biological factors (income, education, employment, acculturation level) are displayed in Tables 10-15. When stratifying participants based on income, those earning between \$2001 and \$3000 per month had a significantly greater intake of *trans* fatty acids (2.1±1.5 g/d) than participants in the other subgroups (p=0.047). Participants in this subgroup consumed 110% more *trans* fatty acids than participants in the income bracket of \$0-1000, 62% more than those with an income of \$1001-2000, 75% more than those with an income of \$3001-4000, and 50% more than those with an income of >\$4000 (1.0±0.6 g/d, 1.3±0.9 g/d, 1.2±0.8 g/d, and 1.4±1.0 g/d, respectively; p=0.047) [Table 10]. There were no other macronutrient intake differences when stratifying participants by income.

After stratifying participants according to income ( $\leq$ \$3000 or >\$3000 per month) [Table 11], percent of energy from total fat and from monounsaturated fatty acids was significantly higher for those earning  $\leq$ \$3000 than for those earning >\$3000 per month (32±5% vs. 29±3%, p=0.042; 12.4±2.1% vs. 11.4±1.4%, p=0.031; respectively). Participants with an income of >\$3000 per month had a higher consumption of energy from carbohydrates than those with an income  $\leq$ \$3000 per month (56±4% vs. 52±8%, p=0.027) [Table 11]. When stratifying participants based on education level, the only significant differences detected were related to percent of energy from polyunsaturated fatty acids. Participants with some college education consumed 40% more energy from polyunsaturated fatty acids relative to those who completed high school  $(6.5\pm1.1\% \text{ energy vs. } 5.1\pm0.8\% \text{ energy, } p=0.007)$  [Table 12].

Macronutrient consumption was also compared among participants when education was also grouped into three categories: (1) those who completed elementary, middle, and high school, (2) those who completed some college, and (3) those who were college graduates or higher. These groups were compared according to macronutrient consumption of the participants. The only significant difference was that those who completed some college consumed a significantly higher percentage of energy from polyunsaturated fatty acids than those who completed elementary, middle, and high school  $(6.5\pm1.1\% vs. 5.6\pm1.0\%, p=0.010)$  [Table 13].

Diet data based on employment status was analyzed using an independent samples t-test. Employed individuals consumed 27% less grams of polyunsaturated fatty acids than unemployed

77

individuals (14.5 $\pm$ 7.4 g vs. 19.8 $\pm$ 12.6 g, p=0.049). Relative to employed participants, unemployed individuals had a 12% significantly greater energy intake from saturated fatty acids (11.0 $\pm$ 2.6% energy vs. 9.8 $\pm$ 1.8% energy, p=0.023). No significant differences were found between macronutrient consumption and full- or part-time employment status [Table 14].

No significant differences in macronutrient consumption were observed when stratifying participants based on acculturation category [Table 15].

# Cardiometabolic disease risk factors and macronutrient consumption

The associations between cardiometabolic disease risk factors and macronutrient consumption were analyzed using Pearson and Spearman correlations. This data is displayed in Table 16. Weight was positively correlated with percent energy from total fat (r=0.330, p=0.004), percent energy from monounsaturated fatty acids (r=0.412, p=0.000), and percent energy from polyunsaturated fatty acids (r=0.241, 0.037), and was negatively correlated with percent energy from carbohydrates (r=-0.311, p=0.007).

BMI was positively correlated with percent energy from total fat (r=0.231, p=0.048), and percent energy from monounsaturated fatty acid (r=0.293, p=0.011) [Table 16]. Waist circumference was positively associated with almost all of the macronutrients including percent energy from total fat (r=0.300, p=0.009), percent energy from *trans* fatty acid (r=0.242, p=0.037), percent energy from monounsaturated fatty acid (r=0.396, p=0.000), and percent energy from polyunsaturated fatty acid (r=0.239, p=0.039). Waist circumference was also negatively associated with percent energy from carbohydrates (r=-0.248, p=0.032).

There was a positive correlation between percent energy from monounsaturated fatty acids and systolic (r=0.247, p=0.033) and diastolic (r=0.261, p=0.025) blood pressure [Table 16]. There was a positive correlation between hsCRP and percent energy from monounsaturated fatty acid (r=0.244, p=0.036). Insulin was positively correlated with percent of energy from total fat (r=0.258, p=0.026), percent energy from *trans* fatty acid (r=0.311, p=0.007), and percent of energy from monounsaturated fatty acids (r=0.238, p=0.040). HOMA was positively correlated with percent energy from *trans* fatty acid (r=0.254, p=0.030) [Table 16].

Characteristics	n	Percent of Total	Mean ±	Characteristics	n	Percent of Total
Gender	75	orrotar	50	Employment	74 <sup>1</sup>	orrotar
Male	26	34.7	-	Employed	45	60.8
Female	49	65.3	-	Full-Time	35	47.3
Age	75	-	37.6±9.3	Part-Time	10	13.5
Self Identification	75			Not Employed	29	39.2
Mexican	37	49.3	-	Language Spoken	75	
Mexican- American	28	37.3	-	Spanish	19	25.3
Hispanic/ Latino	10	13.3	-	English	6	8.0
Time in the US (years)	75	-	23.8± 14.8 (1-56)	Both	50	66.7
Education (Completed)	75			Acculturation Level	73 <sup>1</sup>	
Elementary	4	5.3	-	1-Mexican	25	34.2
Middle School	11	14.7	-	2	15	20.5
High School	11	14.7	-	3	21	28.8
Some College	27	36.0	-	4	12	16.4
College Graduate or Higher	22	29.3	-	5-Anglo	0	0.0
Monthly Income	74 <sup>1</sup>			Generation	75	
\$0-1000	17	23.0	-	1 <sup>st</sup>	40	53.3
\$1001-2000	18	24.3	-	2 <sup>nd</sup>	21	28.0
\$2001-3000	15	20.3	-	3 <sup>rd</sup>	1	1.3
\$3001-4000	9	12.2	-	4 <sup>th</sup>	9	12.0
>\$4000	15	20.3	-	5 <sup>th</sup>	4	5.3

Table 1. Sociodemographic characteristics of study participants

<sup>1</sup> - Unable to show representation of total sample due to incomplete data.

Characteristics	All (n=75)	Men (n=26)	Women (n=49)	p value <sup>2</sup>
Age (years)	37.6±9.3	39.5±1.6	36.6±1.4	0.179
Weight (kg)	78±16	85±13	74±16	0.005
BMI (kg/m²)	28.9±5.3	28.9±3.5	28.9±6.1	0.945
Waist Circumference (cm)	95±13	98±10	93±14	0.061
Percent Body Fat (%)	34.0±9.1	26.9±4.8	37.7±8.7	0.000
Systolic Blood Pressure (mm Ha)	117±11	120±10	115±12	0.050
Diastolic Blood Pressure (mm Hg)	73±9	76±9	72±9	0.148
Total Cholesterol (mg/dL)	183±39	189±51	179±30	0.390
HDL Cholesterol (mg/dL)	44±11	38±11	47±11	0.001
LDL Cholesterol (mg/dL) <sup>3</sup>	114±32	121±42	111±25	0.253
Total/HDL Cholesterol Ratio	4.5±2.0	5.5±2.7	4.0±1.2	0.015
LDL/HDL Cholesterol Ratio <sup>3</sup>	2.8±1.1	3.3±1.2	2.5±0.9	0.002
Triglycerides (mg/dL) <sup>3</sup>	115±61	123±68	110±57	0.378
hsCRP (mg/L)	4.2±6.7	4.3±10.3	4.2±3.9	0.940
Fasting Plasma Glucose (mg/dL)	94±14	99±21	91±7	0.018
Insulin (µIU/mL)	8.7±5.8	8.0±4.9	9.1±6.3	0.449
НОМА	2.0±1.4	1.9±1.1	2.1±1.5	0.633

Table 2. Cardiometabolic disease risk factors of study participants<sup>1</sup>

<sup>1</sup> - Data displayed as mean  $\pm$  SD.

<sup>2</sup> - Mean values for women and men were compared using an independent samples t-test.

<sup>3</sup> - n=74; one participant excluded due to extremely high triglyceride levels. Unable to calculate LDL Cholesterol and LDL/HDL Cholesterol Ratio.

	\$0-1000 (n=17)	\$1001- 2000 (n=18)	\$2001- 3000 (n=15)	\$3001- 4000 (n=9)	>\$4000 (n=15)	p- value²
Weight (kg)	80±16 <sup>ab</sup> (60-120)	77±14 <sup>bc</sup> (53-95)	89±15ª (69-127)	65±13° (50-86)	74±16 <sup>bc</sup> (52-102)	0.005
BMI (kg/m²) <sup>3</sup>	29.7±4.7 <sup>ab</sup> (21.4-37.0)	28.7±5.1 <sup>abc</sup> (20.5-38.7)	31.4±3.6ª (26.7-38.5)	25.6±3.9 <sup>c</sup> (21.2-30.5)	27.1±5.8 <sup>bc</sup> (19.9-38.8)	0.037
Waist Circumference (cm)	97±11 <sup>ab</sup> (82-117)	94±12 <sup>abc</sup> (67-113)	103±11ª (86-124)	86±11 <sup>c</sup> (71-107)	92±13 <sup>bc</sup> (73-130)	0.017
Percent Body Fat (%)	34±9 (19-49)	33±10 (18-47)	36±9 (24-53)	32±7 (24-43)	35±10 (21-49)	0.866
Systolic Blood Pressure (mm Hg)	114±10 (98-133)	118±11 (100-137)	119±11 (102-136)	113±14 (87-134)	119±13 (100-138)	0.539
Diastolic Blood Pressure (mm Hg) <sup>3</sup>	72±9 (61-89)	75±9 (56-95)	75±8 (60-90)	72±10 (44-86)	75±9 (64-96)	0.808
Total Cholesterol (mg/dL) <sup>3</sup>	181±47 (104-274)	175±32 (117-249)	183±40 (115-250)	171±32 (133-237)	192±30 (153-243)	0.661
HDL Cholesterol (mg/dL)	39±9 (20-55)	42±12 (18-70)	41±8 (28-54)	48±14 (21-71)	50±12 (31-67)	0.054
LDL Cholesterol (mg/dL) <sup>3</sup>	109±31 (50-163)	108±27 (56-174)	120±36 (62-188)	105±29 (67-158)	119±28 (79-161)	0.609
Total/HDL Cholesterol Ratio <sup>3</sup>	4.8±1.3 (2.7-7.2)	4.3±1.3 (2.5-7.1)	4.5±1.2 (3.1-6.9)	4.0±1.8 (2.4-8.2)	4.2±1.5 (2.5-7.8)	0.562
LDL/HDL Cholesterol Ratio <sup>3</sup>	3.1±1.1 (1.3-5.2)	2.6±0.9 (1.2-4.4)	3.0±0.9 (1.7-4.6)	2.1±0.8 (1.3-3.3)	2.6±1.1 (1.3-5.2)	0.131
Triglycerides (mg/dL) <sup>3</sup>	131±69 (50-266)	117±69 (54-273)	105±50 (40-195)	91±36 (43-139)	118±67 (53-259)	0.554
hsCRP (mg/L) <sup>3</sup>	3.5±3.9 (0.2-13.9)	2.5±2.1 (0.3-6.9)	4.7±4.3 (0.2-14.1)	2.0±2.0 (0.1-5.7)	3.0±2.7 (0.2-8.1)	0.287
Fasting Plasma Glucose (mg/dL) <sup>3</sup>	95±6 (85-107)	94±8 (81-109)	90±6 (80-103)	88±4 (82-95)	91±6 (81-101)	0.054
Insulin (µIU/mL)	10.6±7.0 (3.6-25.1)	8.9±5.4 (2.0-25.3)	9.8±5.3 (2.0-16.9)	6.0±4.5 (2.0-15.5)	6.7±5.5 (2.0-22.6)	0.203
НОМА	2.6±1.6 (0.8-5.9)	2.0±1.2 (0.5-5.5)	2.2±1.2 (0.5-3.8)	1.3±1.0 (0.4-3.4)	1.5±1.3 (0.4-5.5)	0.137

Table 3. Cardiometabolic disease risk factors stratified by  $\underline{income^{1}}$ 

 $^{1}$  - Data displayed as Mean ± SD (Range). Mean values with different superscripts are <sup>2</sup> - Mean values were compared using an ANOVA test with LSD post-hoc
 <sup>3</sup> - Data more than 3 SD from the mean were excluded

	і бу пісоп	le categor	у
	≤\$3000 (n=50)	>\$3000 (n=24) <sup>2</sup>	p-value <sup>3</sup>
Weight (kg)	81±15 (53-127)	71±15 (50-102)	0.005
BMI (kg/m²) <sup>4</sup>	29.8±4.6 (20.5-38.7)	26.5±5.1 (19.9-38.8)	0.007
Waist Circumference (cm)	98±12 (67-124)	89±14 (71-130)	0.008
Percent Body Fat (%)	34.1±9.4 (18.1-53.3)	33.5±8.9 (21.1-48.9)	0.789
Systolic Blood Pressure (mm Hg)	117±10 (98-137)	117±13 (87-138)	0.929
Diastolic Blood Pressure (mm Hg) <sup>4</sup>	74±9 (56-95)	73±9 (57-96)	0.771
Total Cholesterol (mg/dL) <sup>4</sup>	179±39 (104-274)	183±31 (133-243)	0.629
HDL Cholesterol (mg/dL)	41±10 (18-70)	49±12 (21-71)	0.003
LDL Cholesterol (mg/dL) <sup>4</sup>	112±31 (50-188)	113±28 (67-161)	0.907
Total/HDL Cholesterol Ratio <sup>4</sup>	4.5±1.3 (2.5-7.2)	4.1±1.6 (2.4-8.2)	0.199
LDL/HDL Cholesterol Ratio <sup>4</sup>	2.9±1.0 (1.2-5.2)	2.4±1.0 (1.3-5.2)	0.057
Triglycerides (mg/dL) ⁴	118±63 (40-273)	108±58 (43-259)	0.488
hsCRP (mg/L) <sup>4</sup>	3.5±3.5 (0.2-14.1)	2.7±2.4 (0.1-8.1)	0.300
Fasting Plasma Glucose (mg/dL) <sup>4</sup>	93±7 (80-109)	90±5 (81-101)	0.048
Insulin (µIU/mL)	9.7±5.9 (2.0-25.3)	6.4±5.1 (2.0-22.6)	0.023
НОМА	2.2±1.4 (0.5-5.9)	1.5±1.2 (0.4-5.5)	0.019

Table 4. Cardiometabolic disease risk factors stratified by income category<sup>1</sup>

<sup>1</sup> - Data displayed as Mean ± SD (Range).
 <sup>2</sup> - Data for one participant not available
 <sup>3</sup> - Mean values for income categories were compared using an independent samples *t*-test
 <sup>4</sup> Determinent them 2 CD for the mean mean available

<sup>4</sup> - Data more than 3 SD from the mean were excluded

	Completed Elementary School (n=4)	Completed Middle School (n=11)	Completed High School (n=11)	Some College (n=27)	College Grad or Higher (n=22)	p- value <sup>2</sup>
Weight (kg)	93±19ª (75-120)	77±11 <sup>ab</sup> (57-99)	87±17ª (70-127)	72±14⁵ (50-93)	76±17 <sup>ab</sup> (53-106)	0.023
BMI (kg/m²) <sup>3</sup>	32.2±3.3 (29.4-36.8)	29.8±4.6 (23.0-38.5)	30.9±4.2 (24.3-38.7)	26.9±4.9 (19.9-38.8)	28.6±5.2 (20.2-37.0)	0.087
Waist Circumference (cm)	104±12 (87-117)	96±10 (81-110)	102±12 (85-124)	91±13 (67-130)	95±13 (73-117)	0.071
Percent Body Fat (%)	34.7±6.3 (29.0-42.5)	34.3±8.8 (22.0-50.4)	36.2±11.3 (19.4-53.3)	32.1±8.8 (18.1-48.9)	34.8±9.3 (21.1-48.5)	0.739
Systolic Blood Pressure (mm Hg)	122±13 (104-133)	113±10 (98-127)	115±9 (106-136)	119±15 (87-138)	116±8 (103-133)	0.521
Diastolic Blood Pressure (mm Hg) <sup>3</sup>	75±9 (63-82)	70±9 (60-86)	76±7 (66-89)	76±10 (56-96)	72±7 (57-84)	0.303
Total Cholesterol (mg/dL) <sup>3</sup>	210±40 (176-262)	195±30 (153-248)	170±41 (105-274)	171±36 (104-249)	187±33 (128-250)	0.095
HDL Cholesterol (mg/dL)	33±10 <sup>b</sup> (21-46)	41±10 <sup>ab</sup> (28-58)	36±8 <sup>b</sup> (20-44)	47±10ª (18-67)	47±12ª (30-71)	0.005
LDL Cholesterol (mg/dL) <sup>3</sup>	138±28 (102-163)	126±27 (93-184)	101±19 (62-136)	105±32 (50-174)	117±29 (67-188)	0.065
Total/HDL Cholesterol Ratio <sup>3</sup>	6.7±1.3ª (5.6-8.2)	5.0±1.3 <sup>b</sup> (3.5-6.9)	4.9±0.8 <sup>b</sup> (3.8-6.3)	3.6±0.9° (2.4-6.3)	4.3±1.5 <sup>bc</sup> (2.5-7.8)	0.000
LDL/HDL Cholesterol Ratio <sup>3</sup>	3.9±1.1ª (3.1-5.2)	3.2±0.8 <sup>ab</sup> (2.2-4.5)	3.2±0.7 <sup>ab</sup> (2.4-4.9)	2.2±0.8 <sup>c</sup> (1.2-4.4)	2.7±1.1 <sup>bc</sup> (1.3-5.2)	0.002
Triglycerides (mg/dL) <sup>3</sup>	195±68ª (119-266)	142±80 <sup>ab</sup> (50-258)	115±40 <sup>bc</sup> (47-167)	89±32 <sup>c</sup> (48-178)	116±70 <sup>bc</sup> (40-273)	0.005
hsCRP (mg/L) <sup>3</sup>	3.7±4.4 (0.2-9.7)	4.7±4.3 (0.5-13.9)	2.7±2.0 (0.2-6.8)	3.0±3.3 (0.1-14.1)	2.9±2.8 (0.2-10.3)	0.594
Fasting Plasma Glucose (mg/dL) <sup>3</sup>	95±6 (86-101)	95±9 (80-107)	93±6 (85-104)	92±7 (82-109)	89±5 (81-98)	0.107
Insulin (µIU/mL)	12.8±7.2 (6.1-22.4)	8.7±6.4 (2.0-24.7)	11.3±7.9 (3.7-25.3)	8.4±5.3 (2.0-22.6)	7.2±4.4 (2.0-16.7)	0.215
НОМА	2.9±1.5 (1.5-4.8)	2.0±1.6 (0.5-5.7)	2.8±1.8 (0.8-5.9)	1.9±1.3 (0.4-5.5)	1.6±1.0 (0.4-3.5)	0.137

Table 5. Cardiometabolic disease risk factors stratified by level of  $\underline{education^{1}}$ 

<sup>1</sup> - Data displayed as Mean ± SD (Range). Mean values with different superscripts are significantly different
 <sup>2</sup> - Mean values were compared using an ANOVA test with LSD post-hoc
 <sup>3</sup> - Data more than 3 SD from the mean were excluded

	Completed Elementary, Middle, or High School (n=26)	Some College (n=27)	College Graduate or Higher (n=22)	p- value²
Weight (kg)	84±16ª (57-127)	72±14 <sup>b</sup> (50-93)	78±17 <sup>ab</sup> (53-106)	0.024
BMI (kg/m²) <sup>3</sup>	30.6±4.2ª (23.0-38.7)	26.9±4.9 <sup>b</sup> (19.9-38.8)	28.6±5.2 <sup>ab</sup> (20.2-37.0)	0.024
Waist Circumference (cm)	100±11ª (81-124)	91±13 <sup>b</sup> (67-130)	95±13 <sup>ab</sup> (73-117)	0.029
Percent Body Fat (%)	35.2±9.3 (19.4-53.3)	32.1±8.8 (18.1-48.9)	34.8±9.3 (21.1-48.5)	0.413
Systolic Blood Pressure (mm Hg)	115±10 (98-136)	119±15 (87-138)	116±8 (103-133)	0.503
Diastolic Blood Pressure (mm Hg) <sup>3</sup>	73±8 (60-89)	76±10 (56-96)	72±7 (57-84)	0.305
Total Cholesterol (mg/dL) <sup>3</sup>	187±38 (105-274)	171±36 (104-249)	187±33 (128-250)	0.196
HDL Cholesterol (mg/dL)	37±9 <sup>b</sup> (20-58)	47±10ª (18-67)	47±12ª (30-71)	0.001
LDL Cholesterol (mg/dL) <sup>3</sup>	118±27 (62-184)	105±31 (50-174)	117±29 (67-188)	0.230
Total/HDL Cholesterol Ratio <sup>3</sup>	5.2±1.2ª (3.5-8.2)	3.6±1.9 <sup>b</sup> (2.4-6.3)	4.3±1.5 <sup>b</sup> (2.5-7.8)	0.000
LDL/HDL Cholesterol Ratio <sup>3</sup>	3.3±0.8ª (2.2-5.2)	2.2±0.8 <sup>b</sup> (1.2-4.4)	2.7±1.1 <sup>b</sup> (1.3-5.2)	0.001
Triglycerides (mg/dL) <sup>3</sup>	138±67ª (47-266)	89±32 <sup>b</sup> (48-178)	116±70 <sup>ab</sup> (40-273)	0.011
hsCRP (mg/L) <sup>3</sup>	3.7±3.5 (0.2-13.9)	3.0±3.3 (0.1-14.1)	2.8±2.8 (0.2-10.3)	0.625
Fasting Plasma Glucose (mg/dL) <sup>3</sup>	94±7ª (80-107)	92±7 <sup>ab</sup> (82-109)	89±5 <sup>b</sup> (81-98)	0.030
Insulin (µIU/mL)	10.4±7.1 (2.0-25.3)	8.4±5.3 (2.0-22.6)	7.2±4.4 (2.0-16.7)	0.145
НОМА	2.5±1.6 (0.5-5.9)	1.9±1.3 (0.4-5.5)	1.6±1.0 (0.4-3.5)	0.088

Table 6. Cardiometabolic disease risk factors stratified by completion of high school<sup>1</sup>

<sup>1</sup> - Data displayed as Mean ± SD (Range). Mean values with different superscripts are significantly different
 <sup>2</sup> - Mean values were compared using an ANOVA test with LSD post-hoc

<sup>3</sup> - Data more than 3 SD from the mean were excluded

· · · ·	Employed (n=45)	Not Employed (n=28)	p- value²	Employed Full Time (n=35)	Employed Part Time (n=10)	p- value²
Weight (kg)	78±17 (50-127)	77±15 (53-120)	0.777	78±17 (50-127)	79±17 (53-103)	0.859
BMI (kg/m²) <sup>3</sup>	28.5±5.1 (19.9-38.8)	28.9±4.7 (20.5-38.7)	0.753	28.3±5.2 (19.9-38.8)	29.4±5.2 (21.2-37.0)	0.568
Waist Circumference (cm)	95±14 (73-130)	95±12 (67-117)	0.812	95±15 (73-130)	97±11 (80-113)	0.611
Percent Body Fat (%)	33.9±9.3 (19.4-53.3)	34.1±9.1 (18.1-50.4)	0.936	33.7±9.3 (19.4-53.3)	34.4±9.6 (23.7-48.5)	0.857
Systolic Blood Pressure (mm Hg)	118±11 (100-138)	115±12 (87-137)	0.304	118±11 (100-138)	115±7 (102-126)	0.391
Diastolic Blood Pressure (mm Hg) <sup>3</sup>	74±8 (60-96)	73±10 (56-95)	0.661	74±8 (60-96)	74±6 (62-89)	0.916
Total Cholesterol (mg/dL) <sup>3</sup>	186±34 (115-274)	172±39 (104-262)	0.134	189±38 (115-274)	177±17 (153-209)	0.149
HDL Cholesterol (mg/dL)	46±10 (25-71)	40±12 (18-70)	0.024	48±10 (25-71)	40±9 (30-56)	0.051
LDL Cholesterol (mg/dL) <sup>3</sup>	116±28 (62-188)	107±32 (50-174)	0.205	119±31 (62-188)	108±10 (91-125)	0.085
Total/HDL Cholesterol Ratio <sup>3</sup>	4.2±1.2 (2.5-7.8)	4.6±1.6 (2.4-8.2)	0.310	4.1±1.2 (2.5-7.8)	4.6±1.3 (3.1-7.1)	0.278
LDL/HDL Cholesterol Ratio <sup>3</sup>	2.7±0.9 (1.3-5.2)	2.8±1.1 (1.2-5.2)	0.754	2.7±0.9 (1.3-5.2)	2.8±0.8 (1.8-4.2)	0.679
Triglycerides (mg/dL) <sup>3</sup>	108±61 (40-273)	125±61 (48-266)	0.240	99±51 (40-256)	142±83 (62-273)	0.144
hsCRP (mg/L) <sup>3</sup>	3.4±3.5 (0.2-14.1)	2.8±2.6 (0.1-9.7)	0.447	3.3±3.3 (0.2-14.1)	4.3±1.4 (0.3-13.9)	0.805
Fasting Plasma Glucose (mg/dL) <sup>3</sup>	91±6 (81-104)	93±8 (80-109)	0.262	91±6 (82-104)	91±6 (81-97)	0.974
Insulin (µIU/mL)	8.3±5.7 (2.0-25.1)	9.5±6.1 (2.0-25.3)	0.387	8.4±5.2 (2.0-22.6)	7.9±7.2 (2.0-25.1)	0.814
НОМА	1.9±1.3 (0.4-5.9)	2.2±1.4 (0.4-5.7)	0.301	1.9±1.2 (0.4-5.5)	1.8±1.8 (0.4-5.9)	0.767

Table 7. Cardiometabolic disease risk factors stratified by participants' employment status  $^{\rm 1}$ 

 $^1$  - Data displayed as Mean  $\pm$  SD (Range)  $^2$  - Mean values for employment were compared using an independent samples *t*-test.  $^3$  - Data more than 3 SD from the mean were excluded

	Mexican				Anglo	
	1	2	3	4	5	p-
	(n=25)	(n=15)	(n=21)	(n=12)	(n=0)	value
Weight (kg)	77±13 (53-103)	79±15 (53-99)	81±16 (58-127)	72±17 (50-102)	N/A	0.409
BMI (kg/m²) <sup>3</sup>	29.4±5.0 (20.5-38.5)	28.9±5.3 (21.2-38.7)	28.7±4.1 (22.1-36.3)	26.4±5.6 (19.9-38.8)	N/A	0.398
Waist Circumference (cm)	94±12 (67-113)	95±13 (71-115)	97±13 (73-124)	91±16 (73-130)	N/A	0.593
Percent Body Fat (%)	37.3±8.9 (20.4-50.4)	30.5±9.4 (18.1-47.0)	33.7±9.1 (21.1-53.3)	31.3±8.6 (21.4-48.9)	N/A	0.091
Systolic Blood Pressure (mm Hg)	115±11 (98-137)	115±14 (87-136)	117±10 (100-135)	120±12 (101-138)	N/A	0.677
Diastolic Blood Pressure (mm Hg) <sup>3</sup>	73±10 (56-95)	74±9 (62-90)	74±6 (62-85)	77±9 (64-96)	N/A	0.691
Total Cholesterol (mg/dL) <sup>3</sup>	193±33 (153-262)	175±39 (117-274)	166±38 (104-229)	186±33 (153-250)	N/A	0.089
HDL Cholesterol (mg/dL)	42±7 (29-58)	44±12 (25-70)	44±14 (18-71)	47±12 (31-66)	N/A	0.730
LDL Cholesterol (mg/dL) <sup>3</sup>	123±27 (93-184)	102±27 (56-151)	103±29 (50-159)	115±34 (79-188)	N/A	0.062
Total/HDL Cholesterol Ratio <sup>3</sup>	4.7±1.1 (3.2-7.1)	4.3±1.4 (2.4-6.3)	4.0±1.4 (2.6-8.2)	4.3±1.7 (2.5-7.8)	N/A	0.348
LDL/HDL Cholesterol Ratio <sup>3</sup>	3.0±0.7 (1.9-4.5)	2.7±1.1 (1.2-4.9)	2.3±0.7 (1.3-4.1)	2.7±1.3 (1.3-5.2)	N/A	0.126
Triglycerides (mg/dL) <sup>3</sup>	135±67 (50-273)	108±63 (48-245)	90±40 (40-174)	119±69 (53-259)	N/A	0.092
hsCRP (mg/L) <sup>3</sup>	3.7±3.6 (0.3-13.9)	2.2±2.1 (0.1-6.8)	3.8±3.8 (0.2-14.1)	2.6±2.6 (0.2-8.1)	N/A	0.402
Fasting Plasma Glucose (mg/dL) <sup>3</sup>	95±8 (80-109)	90±6 (81-102)	92±6 (82-103)	90±5 (81-99)	N/A	0.093
Insulin (µIU/mL)	9.0±6.0 (2.0-25.1)	7.7±5.8 (2.0-25.3)	8.3±4.7 (2.0-16.9)	8.6±6.6 (2.0-22.6)	N/A	0.915
НОМА	2.1±1.4 (0.5-5.9)	1.7±1.3 (0.4-5.5)	1.9±1.1 (0.4-3.8)	1.9±1.6 (0.4-5.5)	N/A	0.829

Table 8. Cardiometabolic risk and acculturation category<sup>1</sup>

<sup>1</sup> - Data displayed as Mean ± SD (Range). Mean values with different superscripts are significantly different
 <sup>2</sup> - Mean values were compared using an ANOVA test with LSD post-hoc
 <sup>3</sup> - Data more than 3 SD from the mean were excluded

N/A – No participant is categorized as a 5

Characteristics	All (n=75)	Men (n=26)	Women (n=49)	p value <sup>2</sup>				
Total Energy (kcal)	2587±1598	3140±1997	2293±1267	0.057				
Macronutrient amounts (g)								
Total Fat <sup>3</sup>	89±56	108±66	79±48	0.037				
SFA <sup>3</sup>	30±19	35±22	32±17	0.114				
MUFA <sup>3</sup>	34.2±20.8	40.0±22.0	31.3±19.7	0.089				
PUFA <sup>3</sup>	16.6±10.0	19.7±11.9	15.0±8.6	0.059				
Trans Fatty Acids <sup>3</sup>	1.5±1.2	1.8±1.5	1.3±1.0	0.137				
Carbohydrates <sup>3</sup>	345±224	416±292	307±169	0.089				
Sugar <sup>3</sup>	151±126	191±174	129±85	0.097				
Protein <sup>3</sup>	109±70	134±89	96±54	0.056				
Energy contribution from macronutrients (% energy)								
Total Fat	31±5	32±5	30±5	0.356				
SFA	10.3±2.2	10.2±2.3	10.3±2.1	0.801				
MUFA	12.1±1.9	12.5±2.0	11.9±1.9	0.173				
PUFA	6.1±1.2	6.4±1.3	5.9±1.1	0.078				
Trans Fatty Acids	0.51±0.2	0.50±0.2	0.52±0.2	0.688				
Carbohydrates	53±7	52±8	54±6	0.153				
Sugar	23±6	22±6	23±6	0.660				
Protein	17±3	17±3	17±2	0.496				

Table 9. Daily macronutrient consumption of study participants<sup>1</sup>

 $^1$  - Data displayed as Mean  $\pm$  SD  $^2$  - Mean values for men and women were compared using an independent samples *t*-test.

<sup>3</sup> - Data more than 3 SD from the mean were excluded

SFA=Saturated Fatty Acids, MUFA=Monounsaturated Fatty Acids, PUFA=Polyunsaturated Fatty Acids

	\$0-1000 (n=17)	\$1001- 2000 (n=18)	\$2001- 3000 (n=15)	\$3001- 4000 (n=9)	>\$4000 (n=15)	p- value <sup>2</sup>
Total Energy (kcal) <sup>3</sup>	2387±1360 (498-5036)	2506±1144 (495-4512)	2934±1946 (884-6495)	2179±1513 (1070-5575)	2210±929 (852-3997)	0.645
Macronutrient an	nounts (g)					
Total Fat <sup>3</sup>	84±50 (11-192)	88±48 (16-191)	103±62 (25-213)	87±76 (34-238)	75±34 (30-143)	0.737
SFA <sup>3</sup>	28±16 (4-57)	30±18 (5-78)	35±20 (9-69)	27±24 (11-69)	25±12 (10-50)	0.661
MUFA <sup>3</sup>	33.4±20.0 (4.1-75.6)	34.9±18.6 (6.0-72.7)	40.8±25.2 (9.0-82.4)	33.8±29.6 (13.9-92.8)	29.5±13.9 (11.3-58.8)	0.711
PUFA <sup>3</sup>	16.8±10.7 (2.6-43.7)	17.0±9.5 (2.7-36.9)	17.6±9.9 (4.7-34.3)	17.6±15.3 (5.7-52.3)	14.7±6.5 (6.2-30.1)	0.947
<i>Trans</i> Fatty Acids <sup>3</sup>	1.0±0.6 <sup>b</sup> (0.2-2.1)	1.3±0.9 <sup>b</sup> (0.2-3.8)	2.1±1.5ª (0.4-4.9)	1.2±0.8 <sup>b</sup> (0.6-3.0)	1.4±1.0 <sup>b</sup> (0.3-4.2)	0.047
Carbohydrates <sup>3</sup>	314±198 (89-862)	331±152 (64-650)	417±307 (125-974)	309±206 (145-771)	299±123 (110-527)	0.532
Sugar <sup>3</sup>	127±83 (45-320)	145±90 (25-378)	184±155 (60-461)	129±96 (54-355)	125±63 (44-290)	0.481
Protein <sup>3</sup>	98±54 (15-195)	106±49 (27-198)	122±77 (40-286)	116±93 (48-292)	94±41 (35-159)	0.714
Energy contribut	ion from ma	cronutrients	s (% energy)	)		
Total Fat	31±6 (20-42)	31±5 (25-39)	32±5 (25-41)	27±2 (24-31)	31±4 (23-37)	0.144
SFA	10.4±2.3 (6-14)	10.3±2.4 (6-16)	11.1±2.0 (9-15)	8.7±1.4 (7-11)	10.2±2.1 (8-15)	0.120
MUFA	12.4±2.6 (7-18)	12.3±1.9 (9-16)	12.7±1.8 (9-15)	10.6±0.9 (9-12)	11.9±1.5 (9-14)	0.104
PUFA	6.2±1.3 (4-9)	5.9±1.2 (4-9)	6.3±1.3 (4-8)	5.6±1.1 (4-8)	6.1±1.2 (4-9)	0.720
<i>Trans</i> Fatty Acids	0.5±0.3 (0.2-1.4)	0.5±0.2 (0.3-1.0)	0.6±0.3 (0.2-1.2)	0.4±0.2 (0.2-0.8)	0.6±0.3 (0.2-1.1)	0.238
Carbohydrates	53±9 (38-72)	53±7 (45-66)	50±7 (40-61)	58±4 (54-67)	54±4 (46-63)	0.158
Sugar	21±6 (13-36)	23±7 (13-37)	22±6 (10-35)	26±8 (13-40)	23±6 (16-35)	0.491
Protein	17±3 (12-22)	17±3 (12-25)	18±3 (14-25)	17±2 (12-19)	17±1 (14-20)	0.758

Table 10. Macronutrient consumption stratified by income<sup>1</sup>

<sup>1</sup> - Data displayed as Mean ± SD (Range). Mean values with different superscripts are significantly different
 <sup>2</sup> - Mean values were compared using an ANOVA test with LSD post-hoc
 <sup>3</sup> - Data more than 3 SD from the mean were excluded
 SFA=Saturated Fatty Acids, MUFA=Monounsaturated Fatty Acids, PUFA=Polyunsaturated

Fatty Acids

	≤\$3000 (n=50)	>\$3000 (n=24) <sup>2</sup>	p- value <sup>3</sup>
Total Energy (kcal) <sup>4</sup>	2587±1469 (495-6495)	2199±1130 (852-5575)	0.267
Macronutrient an	nounts (g)		
Total Fat <sup>4</sup>	91±52 (12-213)	80±52 (30-238)	0.382
SFA <sup>4</sup>	31±18 (4-78)	26±17 (10-69)	0.287
MUFA <sup>4</sup>	36±21 (4-82)	31±21 (11-93)	0.516
PUFA ⁴	17±10 (3-44)	16±10 (6-52)	0.341
<i>Trans</i> Fatty Acids ⁴	1.5±1.2 (0.2-4.9)	1.3±0.9 (0.3-4.2)	0.621
Carbohydrates <sup>4</sup>	351±223 (64-974)	302±152 (110-771)	0.343
Sugar <sup>4</sup>	151±111 (25-461)	126±74 (44-355)	0.340
Protein <sup>4</sup>	108±59 (15-286)	102±65 (35-292)	0.710
Energy contribut (% energy)	ion from ma	cronutrients	5
Total Fat	32±5 (20-42)	29±3 (23-37)	0.042
SFA	10.6±2.2 (6.1-15.5)	9.6±2.0 (6.8-15.1)	0.073
MUFA	12.4±2.1 (7.4-17.6)	11.4±1.4 (9.0-13.7)	0.031
PUFA	6.1±1.2 (4.1-8.6)	5.9±1.2 (3.9-9.2)	0.520
<i>Trans</i> Fatty Acids	0.5±0.2 (0.2-1.4)	0.5±0.2 (0.2-1.1)	0.937
Carbohydrates	52±8 (38-72)	56±4 (46-67)	0.027
Sugar	22±6 (10-37)	24±7 (13-40)	0.244
Protein	17±3 (11-25)	17±2 (12-20)	0.625

Table 11. Macronutrient consumption stratified by income category<sup>1</sup>

<sup>1</sup> - Data displayed as Mean  $\pm$  SD (Range)

<sup>2</sup> - Data for one participant not available

<sup>3</sup> - Mean values for income categories were

compared using an independent samples t-test

 <sup>4</sup> - Data more than 3 SD from the mean were excluded

SFA=Saturated Fatty Acids, MUFA=Monounsaturated Fatty Acids, PUFA=Polyunsaturated Fatty Acids

		Completed	Completed			///
	Completed Elementary (n=4)	Middle Sch. (n=11)	High School (n=11)	Some College (n=27)	Grad or Higher (n=22)	p- value <sup>2</sup>
Total Energy (kcal) <sup>3</sup>	1476±71 (1401-1542)	2733±1604 (498-5232)	2508±1828 (495-6395)	2312±1151 (852-4561)	2585±1376 (959-6495)	0.666
Macronutrient am	iounts (g)					
Total Fat <sup>3</sup>	99±93 (43-238)	90±59 (11-198)	76±50 (16-183)	84±48 (25-192)	91±50 (30-213)	0.927
SFA <sup>3</sup>	30±26 (13-68)	30±21 (4-69)	26±17 (5-61)	28±17 (9-78)	31±17 (9-69)	0.969
MUFA <sup>3</sup>	39.4±35.9 (17.4-92.8)	37.5±25.2 (4.1-82.4)	31.1±21.3 (6.0-78.0)	32.8±18.9 (9.0-75.6)	35.1±19.2 (11.9-76.7)	0.930
PUFA <sup>3</sup>	21.2±20.7 (9.6-52.3)	17.0±10.1 (2.6-34.3)	13.9±10.0 (2.7-34.2)	17.1±9.8 (4.7-43.7)	16.1±8.0 (5.7-32.6)	0.783
<i>Trans</i> Fatty Acids <sup>3</sup>	1.3±1.1 (0.4-3.0)	1.7±1.6 (0.2-4.9)	1.2±1.0 (0.2-3.8)	1.3±0.8 (0.4-4.2)	1.5±1.0 (0.3-4.3)	0.822
Carbohydrates <sup>3</sup>	192±57 (132-245)	429±269 (89-961)	363±299 (64-974)	295±145 (110-650)	337±173 (135-792)	0.283
Sugar <sup>3</sup>	66±22 (45-90)	171±136 (45-448)	159±123 (25-419)	131±85 (44-378)	142±95 (46-461)	0.524
Protein <sup>3</sup>	117±116 (54-292)	128±82 (15-286)	105±66 (27-240)	95±48 (35-198)	104±50 (33-249)	0.674
Energy contributi	on from macr	onutrients (	% energy)			
Total Fat	31±8 (25-42)	29±5 (21-36)	29±5 (20-36)	32±4 (24-38)	31±5 (23-41)	0.256
SFA	9.7±2.5 (7-13)	9.3±2.1 (6-12)	10.0±2.2 (6-13)	10.6±2.1 (7-16)	10.5±2.3 (8-15)	0.477
MUFA	12.6±3.4 (10-18)	11.8±2.0 (7-15)	11.6±1.7 (9-15)	12.4±1.7 (9-15)	12.1±2.0 (9-16)	0.737
PUFA	6.5±1.2 <sup>ab</sup> (6-8)	5.7±0.8 <sup>bc</sup> (4-7)	5.1±0.8 <sup>c</sup> (4-6)	6.5±1.1ª (5-9)	6.1±1.4 <sup>ab</sup> (4-9)	0.007
<i>Trans</i> Fatty Acids	0.4±0.2 (0.2-0.7)	0.4±0.2 (0.2-0.8)	0.4±0.2 (0.3-0.9)	0.6±0.3 (0.3-1.4)	0.5±0.2 (0.2-1.2)	0.252
Carbohydrates	54±11 (38-64)	56±8 (44-72)	55±8 (40-68)	52±6 (42-67)	53±6 (42-63)	0.436
Sugar	23±12 (13-40)	22±8 (10-36)	24±5 (18-34)	22±6 (13-37)	22±6 (15-35)	0.876
Protein	16±2 (14-18)	16±3 (12-25)	18±4 (11-24)	17±2 (12-20)	18±2 (14-25)	0.185

Table 12 Macronutrient consumption stratified by education<sup>1</sup>

<sup>1</sup> - Data displayed as Mean ± SD (Range). Mean values with different superscripts are significantly different
 <sup>2</sup> - Mean values were compared using an ANOVA test with LSD post-hoc
 <sup>3</sup> - Data more than 3 SD from the mean were excluded

SFA=Saturated Fatty Acids, MUFA=Monounsaturated Fatty Acids, PUFA=Polyunsaturated Fatty Acids

	Completed			
	Elementary, Middle, or High School (n=26)	Some College (n=27)	College Graduate or Higher (n=22)	p- value <sup>2</sup>
Total Energy (kcal) <sup>3</sup>	2473±1619 (495-6395)	2312±1151 (852-4561)	2585±1376 (959-6495)	0.786
Macronutrient amou	nts (g)			
Total Fat <sup>3</sup>	86±59 (11-238)	84±48 (25-192)	91±50 (30-213)	0.913
SFA <sup>3</sup>	28±19 (4-69)	28±17 (9-78)	31±17 (9-69)	0.869
MUFA <sup>3</sup>	35.0±34.5 (4.1-92.8)	32.8±18.9 (9.0-75.6)	35.1±19.2 (11.9-76.7)	0.908
PUFA <sup>3</sup>	16.3±11.9 (2.6-52.3)	17.1±9.8 (4.7-43.7)	16.1±8.0 (5.7-32.6)	0.939
Trans Fatty Acids <sup>3</sup>	1.4±1.3 (0.2-4.9)	1.3±0.8 (0.4-4.2)	$1.5\pm1.0$ (0.3-4.3)	0.868
Carbohydrates <sup>3</sup>	371±271 (64-974)	295±145 (110-650)	337±173 (135-792)	0.404
Sugar <sup>3</sup>	153±123 (25-448)	131±85 (44-378)	142±95 (46-461)	0.734
Protein <sup>3</sup>	117±78 (15-292)	95±48 (35-198)	104±50 (33-249)	0.444
Energy contribution	from macron	utrients (%	energy)	
Total Fat	29±5 (20-42)	32±4 (24-38)	31±5 (23-41)	0.104
SFA	9.7±2.1 (6.1-13.1)	10.6±2.1 (6.8-15.5)	10.5±2.3 (7.7-15.2)	0.219
MUFA	11.8±2.1 (7.4-17.6)	12.4±1.7 (9.2-14.9)	12.1±2.0 (9.0-16.1)	0.543
PUFA	$5.6 \pm 1.0^{b}$ (4.1-8.3)	6.5±1.1ª (4.8-8.6)	6.1±1.4 <sup>ab</sup> (3.9-9.2)	0.010
<i>Trans</i> Fatty Acids	0.4±0.2 (0.2-0.9)	0.6±0.3 (0.3-1.4)	0.5±0.2 (0.2-1.2)	0.067
Carbohydrates	55±8 (38-72)	52±6 (42-67)	53±6 (42-63)	0.181
Sugar	23±7 (10-40)	22±6 (13-37)	22±6 (15-35)	0.806
Protein	17±4 (11-25)	17±2 (12-20)	18±2 (14-25)	0.381

Table 13. Macronutrient consumption stratified by  $completion of high school^{1}$ 

<sup>1</sup> - Data displayed as Mean  $\pm$  SD (Range). Mean values with different <sup>2</sup> - Mean values were compared using an ANOVA test with LSD post-hoc

 $^{3}$  - Data more than 3 SD from the mean were excluded

SFA=Saturated Fatty Acids, MUFA=Monounsaturated Fatty Acids,

PUFA=Polyunsaturated Fatty Acids

	0.01.001.00	ie eenean			, emple,	
	Employed (n=45)	Not Employed (n=28)	p- value²	Employed Full Time (n=35)	Employed Part Time (n=10)	p- value²
Total Energy (kcal) <sup>3</sup>	2431±1423 (495-6495)	2389±1303 (498-6395)	0.901	2414±1401 (495-6495)	2488±1572 (1300-5575)	0.887
Macronutrient an	nounts (g)					
Total Fat <sup>3</sup>	82±52 (16-213)	91±53 (11-238)	0.461	82±50 (16-213)	83±60 (37-198)	0.941
SFA <sup>3</sup>	27±17 (5-69)	31±18 (4-78)	0.312	26±16 (5-62)	29±22 (11-69)	0.614
MUFA <sup>3</sup>	30.5±17.6 (9.0-78.0)	40.3±24.2 (4.1-92.8)	0.068	30.3±18.2 (9.0-78.0)	31.2±16.4 (13.9-59.8)	0.889
PUFA <sup>3</sup>	14.5±7.4 (4.7-34.2)	19.8±12.6 (2.6-52.3)	0.049	14.2±7.3 (4.7-34.2)	15.6±8.0 (5.7-27.5)	0.615
<i>Trans</i> Fatty Acids <sup>3</sup>	1.3±1.1 (0.2-4.9)	1.5±1.0 (0.2-4.3)	0.468	1.4±1.1 (0.2-4.9)	1.2±1.2 (0.4-4.4)	0.598
Carbohydrates <sup>3</sup>	341±210 (64-974)	309±194 (89-974)	0.523	342±215 (64-961)	338±200 (186-771)	0.966
Sugar <sup>3</sup>	145±107 (25-461)	131±93 (45-419)	0.587	149±113 (25-461)	131±85 (59-355)	0.649
Protein <sup>3</sup>	102±62 (27-286)	108±60 (15-292)	0.668	99±60 (27-286)	110±70 (47-249)	0.635
Energy contribut	ion from mac	ronutrients (	% energy	y)		
Total Fat	30±4 (20-38)	32±6 (21-42)	0.071	30±4 (20-38)	29±3 (24-34)	0.433
SFA	9.8±1.8 (6.1-13.0)	11.0±2.6 (6.4-15.5)	0.023	9.7±1.8 (6.1-12.7)	10.1±1.9 (7.2-13.0)	0.639
MUFA	11.8±1.6 (8.6-14.9)	12.6±2.4 (7.4-17.6)	0.093	11.9±1.6 (8.6-14.9)	11.1±1.5 (9.3-14.2)	0.175
PUFA	6.0±1.2 (3.9-8.6)	6.2±1.3 (4.3-9.2)	0.536	6.1±1.1 (3.9-8.6)	5.6±1.3 (4.1-8.2)	0.229
<i>Trans</i> Fatty Acids	0.5±0.2 (0.2-1.2)	0.5±0.2 (0.2-1.4)	0.241	0.6±0.2 (0.2-1.2)	0.5±0.2 (0.2-1.0)	0.202
Carbohydrates	54±6 (42-68)	51±8 (38-72)	0.080	54±7 (42-68)	56±5 (49-65)	0.514
Sugar	23±6 (10-37)	22±7 (13-40)	0.877	23±6 (13-37)	22±7 (10-31)	0.794
Protein	17±3 (11-25)	17±2 (12-24)	0.624	17±3 (11-25)	18±2 (13-22)	0.446

Table 14. Macronutrient consumption stratified by employment<sup>1</sup>

 $^1$  - Data displayed as Mean ± SD (Range)  $^2$  - Mean values for employment were compared using an independent samples *t*-test.  $^3$  - Data more than 3 SD from the mean were excluded

SFA=Saturated Fatty Acids, MUFA=Monounsaturated Fatty Acids, PUFA=Polyunsaturated Fatty Acids

	Mexican	2	3	4	Anglo	p-
	(n=25)	(n=15)	(n=21)		(n=0)	value
Total Energy (kcal) <sup>3</sup>	2570±1258 (495-5232)	2560±1723 (959-6395)	2542±1515 (884-6495)	2082±941 (852-3997)	N/A	0.761
Macronutrient am	nounts (g)					
Total Fat <sup>3</sup>	89±47 (11-198)	85±61 (30-191)	99±61 (25-238)	70±36 (30-143)	N/A	0.530
SFA <sup>3</sup>	30±16 (4-69)	29±23 (9-78)	32±18 (9-68)	23±12 (10-44)	N/A	0.613
MUFA <sup>3</sup>	35.4±19.6 (4.1-82.4)	34.4±24.3 (11.9-78.0)	38.2±23.3 (9.0-92.8)	26.9±14.5 (11.4-58.8)	N/A	0.532
PUFA <sup>3</sup>	16.5±8.6 (2.6-34.3)	16.4±11.2 (5.7-36.9)	19.0±12.4 (4.7-52.3)	14.0±7.6 (6.2-30.1)	N/A	0.617
<i>Trans</i> Fatty Acids <sup>3</sup>	1.4±1.0 (1.2-4.4)	1.1±0.9 (0.4-3.8)	1.7±1.2 (0.4-4.9)	1.3±1.0 (0.3-4.2)	N/A	0.454
Carbohydrates <sup>3</sup>	344±173 (64-650)	354±243 (135-974)	356±249 (125-961)	280±123 (110-527)	N/A	0.752
Sugar <sup>3</sup>	141±89 (25-378)	158±119 (46-419)	149±120 (60-461)	125±72 (44-290)	N/A	0.858
Protein <sup>3</sup>	108±52 (15-221)	108±73 (33-249)	114±72 (34-292)	89±43 (36-159)	N/A	0.741
Energy contributi	on from mac	ronutrients	(% energy)			
Total Fat	30±4 (21-40)	29±4 (25-38)	32±5 (20-41)	30±5 (23-37)	N/A	0.210
SFA	10.2±1.8 (6.4-13.7)	9.8±2.4 (6.1-15.5)	10.7±2.3 (6.7-15.2)	10.0±2.6 (6.8-15.1)	N/A	0.602
MUFA	12.0±2.0 (7.4-16.1)	11.8±1.3 (9.9-14.5)	12.5±2.1 (8.6-15.2)	11.5±1.6 (9.0-13.8)	N/A	0.399
PUFA	5.7±0.9 (4.3-8.0)	5.8±1.3 (4.1-8.6)	6.6±1.1 (4.3-8.6)	6.0±1.4 (3.9-9.2)	N/A	0.050
<i>Trans</i> Fatty Acids	0.5±0.2 (0.2-1.0)	0.4±0.1 (0.2-0.6)	0.6±0.3 (0.3-1.4)	0.6±0.3 (0.2-1.1)	N/A	0.071
Carbohydrates	54±7 (44-72)	56±6 (47-66)	51±7 (40-68)	54±6 (46-67)	N/A	0.241
Sugar	22±7 (10-37)	24±5 (13-34)	22±6 (15-40)	24±7 (13-35)	N/A	0.791
Protein	17±3 (12-25)	17±3 (11-22)	17±3 (13-25)	17±2 (12-20)	N/A	0.991

Table 15. Macronutrient consumption stratified by acculturation categorv<sup>1</sup>

<sup>1</sup> - Data displayed as Mean  $\pm$  SD (Range). Mean values with different superscripts are significantly different

<sup>2</sup> - Mean values were compared using an ANOVA test with LSD post-hoc

 $^{3}$  - Data more than 3 SD from the mean were excluded N/A – No participant is categorized as a 5

SFA=Saturated Fatty Acids, MUFA=Monounsaturated Fatty Acids, PUFA=Polyunsaturated Fatty Acids

Table 16. Correlations betv	veen car	diometab	olic risk fi	actors a	nd macı	ronutrient o	consum	ption
	Total Fat (%)	Saturated Fatty Acid (%)	<i>Trans</i> Fatty Acid (%)	MUFA (%)	PUFA (%)	Carbohydrate (%)	Sugar (%)	Protein (%)
Weight (kg)	r=0.330	r=0.214	r=0.182	r=0.412	r=0.241	r=-0.311	r=-0.172	r=0.132
	p=0.004	p=0.065	p=0.118	p=0.000	p=0.037	p=0.007	p=0.140	p=0.260
BMI (kg/m²) ²	r=0.231	r=0.146	r=0.154	r=0.293	r=0.155	r=-0.131	r=-0.125	r=-0.072
	p=0.048	p=0.216	p=0.190	p=0.011	p=0.189	p=0.265	p=0.287	p=0.540
Waist Circumference (cm)	r=0.300	r=0.161	r=0.242	r=0.396	r=0.239	r=-0.248	r=-0.158	r=0.051
	p=0.009	p=0.167	p=0.037	p=0.000	p=0.039	p=0.032	p=0.177	p=0.665
Percent Body Fat (%)	r=0.146	r=0.169	r=0.170	r=0.175	r=-0.028	r=-0.060	r=-0.085	r=-0.028
	p=0.212	p=0.148	p=0.146	p=0.133	p=0.812	p=0.610	p=0.466	p=0.811
Systolic Blood Pressure	r=0.211	r=0.123	r=0.204	r=0.247	r=0.175	r=-0.201	r=0.076	r=0.071
(mm Hg)	p=0.070	p=0.293	p=0.079	p=0.033	p=0.132	p=0.083	p=0.519	p=0.545
Diastolic Blood Pressure (mm Hg) <sup>2</sup>	r=0.216	r=0.162	r=0.127	r=0.261	r=0.191	r=-0.215	r=0.046	r=0.027
	p=0.064	p=0.168	p=0.282	p=0.025	p=0.103	p=0.065	p=0.695	p=0.822
Total Cholesterol (mg/dL) <sup>2</sup>	r=0.134	r=0.157	r=0.106	r=0.126	r=-0.069	r=-0.129	r=-0.198	r=0.076
	p=0.255	p=0.183	p=0.367	p=0.284	p=0.557	p=0.272	p=0.091	p=0.519
HDL Cholesterol (mg/dL)	r=0.029	r=0.073	r=-0.002	r=-0.066	r=0.075	r=-0.084	r=-0.124	r=0.087
	p=0.804	p=0.534	p=0.989	p=0.575	p=0.525	p=0.475	p=0.288	p=0.458
LDL Cholesterol (mg/dL) <sup>2</sup>	r=0.130	r=0.154	r=0.107	r=0.129	r=-0.082	r=-0.128	r=-0.132	r=0.088
	p=0.274	p=0.193	p=0.369	p=0.278	p=0.489	p=0.281	p=0.267	p=0.460
Total/HDL Cholesterol Ratio <sup>1</sup>	r=-0.004	r=-0.026	r=-0.127	r=-0.062	r=0.113	r=-0.022	r=0.030	r=0.033
	p=0.974	p=0.828	p=0.278	p=0.598	p=0.333	p=0.849	p=0.798	p=0.778
LDL/HDL Cholesterol Ratio <sup>1</sup>	r=-0.006	r=0.025	r=0.128	r=0.040	r=-0.133	r=0.019	r=-0.018	r=0.026
	p=0.961	p=0.835	p=0.277	p=0.738	p=0.260	p=0.872	p=0.880	p=0.825
Triglycerides (mg/dL) <sup>1</sup>	r=0.014	r=0.006	r=-0.102	r=-0.067	r=0.090	r=-0.059	r=0.054	r=0.124
	p=0.908	p=0.961	p=0.388	p=0.573	p=0.444	p=0.617	p=0.650	p=0.292
hsCRP (mg/L) <sup>1</sup>	r=0.184	r=0.157	r=0.162	r=0.244	r=0.092	r=-0.088	r=-0.159	r=-0.106
	p=0.116	p=0.181	p=0.169	p=0.036	p=0.434	p=0.453	p=0.177	p=0.367
Fasting Plasma Glucose (mg/dL) <sup>2</sup>	r=0.197	r=0.142	r=0.099	r=0.166	r=0.133	r=-0.156	r=-0.090	r=0.018
	p=0.094	p=0.231	p=0.404	p=0.161	p=0.262	p=0.188	p=0.448	p=0.879
Insulin (µIU/mL) <sup>1</sup>	r=0.258	r=0.197	r=0.311	r=0.238	r=0.162	r=-0.219	r=-0.015	r=-0.034
	p=0.026	p=0.091	p=0.007	p=0.040	p=0.164	p=0.059	p=0.898	p=0.774
НОМА	r=0.140	r=0.094	r=0.254	r=0.147	r=0.074	r=-0.059	r=0.068	r=-0.032
	p=0.238	p=0.428	p=0.030	p=0.213	p=0.534	p=0.618	p=0.568	p=0.790

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<sup>1</sup>-Data not normally distributed and transformed; Spearman used. <sup>2</sup>-Data more than 3 SD from the mean excluded in analysis.

### DISCUSSION

Cardiovascular disease is a rising health problem and the main cause of death among Mexican-Americans. There are multiple factors that can contribute to the risk of developing CVD that include, but are not limited to, dyslipidemia, elevated glucose, elevated hsCRP, hypertension, increased adiposity, and macronutrient consumption. Despite some indication that nonbiological risk factors negatively affect diet and chronic disease risk [16-20, 34, 61, 71, 121-124], specific information among Mexican Americans is scarce. Therefore, the purpose of this work was to perform a cross-sectional evaluation of self-reported individual and family dietary and lifestyle habits, sociocultural factors (income, employment, acculturation, and education) and biological markers of cardiometabolic risk among Mexican-American adults living in the Phoenix metropolitan area. The relevance of this work lies on the pressing need to aid in the elimination of health disparities by focusing on chronic disease risk reduction and prevention, and not just on disease treatment.

97

# Income, diet and CVD risk factors in Mexican-American adults

Several studies have documented greater CVD risk among individuals with a lower socioeconomic status [7, 17, 122, 126, 127, 129, 130]. In the current study, participants with higher household income (greater than \$3000 per month) had 27% lower weight, 18% lower BMI, and 17% smaller waist circumference than those with lower household incomes. In particular, these indicators of adiposity were significantly greater among participants with a household income lower than \$1000 per month or between \$2001 and \$3000 per month. Interestingly, weight, BMI and waist circumference of participants earning between \$1001 and \$2000 per month did not differ from those of individuals earning more than \$3000 per month. In agreement with our findings, Appel et al. [122] explored the relationship between sociodemographics and CVD risk in 1,110 participants and found a significant negative association between income and CVD risk. Similarly, Robert et al. [17] reported a negative association between income and BMI. Kavanagh et al. [127] also reported a negative association between income and waist circumference, dyslipidemia, and blood pressure. Unlike Appel et al. [122] CVD risk was not

categorized according to the various risk factors associated with CVD.

Regarding biomarkers of cardiometabolic risk, after grouping the participants in our study into two income categories ( $\leq$ \$3000 and >\$3000), those earning less than \$3000 per month had 3% significantly higher fasting glucose, 52% higher insulin, and 47% higher HOMA than those earning more than \$3000 per month. As previously established, having greater HOMA scores is associated with greater CVD risk. One study by Strufaldi et al. [185] looked at the relationship between HOMA values and CVD risk factors. Participants were divided into tertiles based on HOMA values (Tertile 1, 0.32-0.53; Tertile 2, 0.55-1.10; Tertile 3, 1.16-6.01). Those in the lowest tertile of HOMA score had a significantly lower number of risk factors for CVD than those in the highest tertile. No participants in Tertile 1 had increased insulin compared to 83% of participants in Tertile 3. Participants from Tertile 1 also had lower prevalence of high BMI (25% vs. 48%, p=0.007), high blood pressure (13% vs. 61%, p=0.008), and low HDL cholesterol (22% vs. 51%, p=0.020) than those in Tertile 3. Furthermore, Tertile 3 had higher incidence of elevated triglyceride concentration (concentrations  $\geq$ 130 mg/dL,

27% vs. 50%) and elevated glucose concentrations (concentrations ≥100 mg/dL, 0% vs. 43%) than Tertile 1, although this data was not statistically significant.

Barr et al. [186] documented that participants who were the least insulin sensitive according to HOMA were more than twice as likely to experience a CVD event as those who were the most insulin sensitive. Smith et al. [187] documented that participants with a fasting serum glucose concentration  $\leq 109$ mg/dL but who had an elevated 2-hour glucose concentration of  $\geq$ 200 mg/dL were 1.3 times as likely to have a CVD event than those who had a fasting serum glucose concentration  $\leq 109$ mg/dL and a 2-hour glucose concentration  $\leq$ 139 mg/dL. They also reported that participants with an impaired fasting serum glucose concentration (110-125 mg/dL) coupled with an elevated 2-hour glucose concentration ( $\geq$ 200 mg/dL) were 2.2 times as likely to have a CVD event than those who had impaired fasting serum glucose concentrations and normal 2-hour glucose concentrations ( $\leq$ 139 mg/dL).

Orencia et al. [188] explored the effect of high glucose concentrations of 39,573 non-diabetic participants following a 100
50-gram glucose load. They reported that only 16% of those in the lowest quintile of glucose concentration 1-hour postprandial (39-121 mg/dL) had CVD whereas 27% of those in the highest quintile (144-700 mg/dL) had CVD. Although these studies did not find a relationship between income and most CVD risk factors, our study found that those with a lower income had higher HOMA, insulin, and glucose. Therefore, it is possible that having a lower income is associated with an increased risk for CVD since higher HOMA, insulin, and glucose concentrations are associated with increased CVD risk or CVD events [185-188].

The present study also found that HDL cholesterol was lower for those earning \$3000 per month than those earning more than \$3000 per month. Kavanagh et al. [127] reported a positive association between income and HDL cholesterol concentrations ( $\beta$ =-0.09, p<0.05), and Muennig et al. [129] documented that participants earning less than \$20,000 annually have a lower HDL cholesterol concentration than those earning more than \$20,000 annually (50.8 mg/dL vs. 51.6 mg/dL). Although this shows that those with a lower income are more likely to also have a lower HDL cholesterol concentration, the difference is relatively small from a physiological point of view, and may not dramatically change CVD risk. It should also be noted that the income cutoff point used by Muennig et al. [129] was lower than that used in our study (\$1667/month vs. \$3000/month). However, it shows the positive association between income and HDL cholesterol concentrations.

Steptoe et al. [140] explored the relationship between socioeconomic status and CVD. After grouping participants based on their grade of employment (higher, intermediate, lower), they reported that higher socioeconomic status was related to a significantly smaller waist/hip ratio (0.90 vs, 0.93), higher HDL cholesterol concentrations (1.84 mmol/L vs. 1.53) mmol/L), lower total/HDL cholesterol ratio (3.48 vs. 3.91), and lower fasting glucose concentrations (5.26 mmol/L vs. 5.76 mmol/L). Kavanagh et al. [127] documented a significant difference in blood pressure between high and low income participants ( $\beta$ =-2.09, p≤0.05). Friedman and Herd [189] also explored the relationship between income and CVD risk. Income was considered all pretax income and was adjusted for household size. They also grouped participants into guintiles (Q1: ≤\$17,838; Q2: \$17,839-\$35,037; Q3: \$35,038-\$50,161; Q4: \$50,162-\$76,809; Q5: ≥\$76,810). Friedman and Herd

found that hsCRP was significantly higher in the lowest income group than in the highest group (1.9  $\mu$ g/mL vs. 1.1  $\mu$ g/mL;  $\beta$ =0.19, p<0.001). The income grouping used in these studies [127, 140, 189] was different than that used in our study, but it shows the positive association between income and CVD risk.

The findings of our study concur with previous findings suggesting that persons with a higher income have fewer biological risk factors for CVD than those with a lower income. Even after grouping income into different categories our study was able to find negative associations between income and CVD risk factors. However, our study did not find significant differences between total cholesterol, LDL cholesterol, triglycerides, total/HDL cholesterol ratio, LDL/HDL cholesterol ratios, or hsCRP when stratified by income. Significant relationships were not found between income and percent body fat or blood pressure.

In addition to relationships between income and cardiovascular disease risk factors, the present study found that income is associated with some aspects of the diet. Participants earning less than \$3000 per month had a higher percent of their energy intake from total fat and monounsaturated fatty acids and a lower percent of their energy intake from carbohydrates than those earning more than \$3000 per month (32% vs. 29%) energy from total fat; 12.4% vs. 11.4% energy from monounsaturated fatty acids; 52% vs. 56% energy from carbohydrates). Moreover, participants earning between \$2001 and \$3000 per month had a significantly greater intake of *trans* fatty acids than those in the lowest income bracket (2.1g vs. 1.0g). Although this difference in percent energy from total fat, monounsaturated fatty acids, and *trans* fatty acid intake is statistically significant, it may not be physiologically relevant. For example, considering that our study participants' mean energy intake was 2587±1598 kilocalories per day, the difference between 32% and 29% energy from fat is equivalent to consuming 78 kilocalories (8.7 g) more from fat.

The differences noted above could be attributed to eating out more frequently rather than eating homemade meals. Another factor with eating out of the home is where someone eats and what they order. Additionally, one speculation might be that those earning less than \$3000 per month felt they were too busy to make homemade meals and found eating out more convenient. Another contributing factor to the larger intake of *trans* fatty acids is the cost of healthy foods when compared with energy dense foods. There is an inverse relationship between energy density (kcal/gram) and energy cost (cost/kcal) [161]. Although the total package price and unit price of produce may be lower than those of snacks, the average per serving price for produce can be higher than for snack foods that are potentially higher in *trans* fatty acids [161]. Thus, those with a higher income are better able to pay the higher price for produce and healthy food options.

One study reported that in a particular low-income community there were only 2 stores that provided enough variety of foods for consumers to be able to meet the recommended dietary guidelines [190]. This suggests that low-income areas may not have access to healthy food options even if they can afford them.

Simon et al. [191] explored the number of fast food restaurants within a close proximity to schools of varying socioeconomic neighborhoods. They also reported that 38% of schools in lower income neighborhoods (income range of \$0-32,832) had at least one fast food restaurant within 400 meters as opposed to higher income neighborhoods (income range of \$58,319+) in which only 12% of the schools had at least one fast food restaurant within 400 meters. Furthermore, 77% of schools in the lower socioeconomic neighborhood had one or more fast food restaurants within 800 meters of the school instead of 47% of schools in the higher income neighborhood [191]. It can be supposed that a greater exposure to fast food would result in an increased consumption.

Our study found a relationship between income and BMI, weight, waist circumference, HDL cholesterol, fasting glucose, insulin, and HOMA. In contrast to previous work, our results did not indicate a relationship between household income and cardiometabolic disease risk factors such as serum lipids, hsCRP, blood pressure, or percent body fat. One reason that the present study may not have resulted in the same conclusions as previous studies is that in this study income was not corrected based on the number of people who lived in the household. When comparing those in the same income category, households with more people have less of a financial advantage than households with less people. Another contributing factor could be the demographics of our study participants. Based on where participants were recruited from, several of our participants might be students. This can affect household size and income. They may have reported their income as individual but then included roommates as part of the household. It should also be noted that college students often have a different eating schedule than others. They may end up having classes during meal times. This can create an obstacle to meal time, and instead of possibly eating a more balanced meal at home, they may eat at a restaurant or may skip the meal all together. Instead of trying to carry a meal with them, students may also resort to eating something smaller and more convenient that may not be as healthy as a meal.

The association between income and CVD risk (increased weight, BMI, waist circumference, fasting glucose, insulin, and HOMA; decreased HDL cholesterol concentration) within the present study has caused several speculations as potential explanations. One possibility could relate to how much disposable income a person has to spend on medical treatment or preventive measures. It is also possible that individuals with lower income may not own a vehicle and therefore may have limited access to medical care, be limited to shopping at stores with a small selection of fresh foods, and may not be able to buy groceries very often and search for more of the shelf stable foods rather than produce that does not last as long. Also, lack of groceries in the home may force people to buy food on the street at vendors or fast food establishments.

# Employment, diet and CVD risk in Mexican-American adults

Employment status was not observed to affect CVD risk factors in this cohort of Mexican-Americans except for HDL cholesterol concentrations, which were 15% greater among participants who were employed than among unemployed participants. It can be speculated that employed participants had a lower risk for CVD than those not employed.

Haertel et al. [192] examined the HDL cholesterol concentrations of employed women and homemakers and found that employed women had significantly higher HDL cholesterol concentrations than homemakers. Moreover, employed women who became homemakers had a decrease in their HDL cholesterol concentrations, but homemakers who became employed showed no change in HDL cholesterol [192].

Several studies document that physical activity raises HDL cholesterol concentrations. Martin et al. [193], Wood et al. [194], Rotkis et al. [195], and Hagan and Gettman [196] found that runners had higher HDL cholesterol concentrations than sedentary non-runner participants (14.2% higher; 30% higher for women and 49% higher for men; 59% higher; and 9.5% higher; respectively). Enger et al. [197] reports a 21% higher concentration of HDL for skiers than the controls. Hartung et al. [198] reported that joggers have 35% higher HDL cholesterol concentrations and marathoners have 51% higher concentrations of HDL cholesterol than inactive participants. Some studies also suggest that active lifestyles, more active jobs, and employment may beneficially affect HDL cholesterol concentrations. Lehtonen and Viikari [199] found that lumberjacks have 36% higher concentrations of HDL cholesterol than electricians.

The aforementioned studies also reported significantly lower total cholesterol (4-12%) [193, 194, 198], lower LDL cholesterol 109 (9-22%) [193, 194, 198], and lower triglyceride concentrations (20-54%) [193, 194, 198, 199] for participants who had more active jobs or lifestyles than those who were more sedentary. Hagan and Gettman [196] and Enger et al. [197] also reported that more active participants have lower total cholesterol, LDL cholesterol, and triglyceride concentrations but their data was not significant.

Unlike the studies mentioned above [192-199], our study failed to find any relationship with employment and CVD risk factors except HDL cholesterol. Furthermore, this study did not evaluate the effect of physical activity on CVD risk factors.

After exploring the relationship between employment and diet it was found that unemployed individuals had a greater energy intake from saturated fatty acids than employed participants. An interesting study by Rathnayake and Weerahewa [200] documented that when women work and their household receives the extra income, the caloric intake of all individuals within the household increases. As a person's caloric intake increases their consumption of macronutrients also increases, including total fat, saturated fatty acids, and *trans* fatty acids. In contrast, our study did not show a significant relationship between employment and caloric, total fat, or *trans* fatty acid intake.

One causal factor may be the high cost of unsaturated fatty acid rich foods such as fish and the low cost of foods containing saturated fatty acids such as fast food, marbled red meat, or high fat dairy products like whole milk. Those who are not employed may seek the cheaper foods that are more likely to have saturated fatty acids. One study found that a person's neighborhood socioeconomic status was positively associated with intake of fruits and vegetables [201]. Interestingly, Morris et al. [202] found that unemployed men were more likely to gain weight than men who remained continuously employed.

According to the National Health Interview Survey (NHIS), heart disease, ischemic heart disease, and hypertension were negatively correlated with poverty status and income [9, 135]. Since employment directly affects income and poverty level, it can be hypothesized that employment status is associated with CVD risk. Another factor related to employment that could affect CVD risk is the location of employment. Those working in a restaurant or convenience store have greater access to calorically dense foods that do not offer many nutrients [171]. However, this information was not gathered in the survey used for the present study.

In this study, by categorizing participants who are employed as either employed full- or part-time it was easier compare participants in the same employment category and gather more detailed analyses. Similar to the study by Haertel et al. [192], the present study found that employed individuals have higher HDL cholesterol than those who are not employed, which in turn could result in lower CVD risk.

#### Education, diet and CVD risk in Mexican-American adults

Education attainment has been negatively associated with CVD risk [18, 34, 61, 71, 122-124, 126-129, 203]. In this study, participants with a lower level of education (completed high school or less) had 17% greater weight, 14% greater BMI, and 10% larger waist circumference than participants who only completed some college but did not complete college or greater education. In addition, they had a less favorable metabolic profile, characterized by having a 27% lower HDL cholesterol concentration and concurrently 44% higher total/HDL cholesterol and 50% higher LDL/HDL cholesterol ratios, in addition to having 55% and 24% greater concentrations of triglycerides and fasting plasma glucose, respectively.

In agreement with our findings, education has been shown to have a significant negative association with CVD risk factors [71, 99, 123, 127-129, 203]. Several other trials have found that education level is negatively associated with coronary heart disease risk [61, 71, 122, 123]. Multiple studies report that persons with lower education are more likely to have a clustering of more risk factors for developing CVD when compared with those who have higher education [71, 122, 123, 127-130, 135, 137].

Ribisl et al. [71] reported that 39.6% of Hispanics who completed 12 years of education were hypertensive as compared to only 18.4% of those who completed 16+ years (p<0.001). Ribisl et al. also documented that Hispanics with 16+ years of education had lower mean plasma cholesterol concentrations than Hispanics who completed 12 years (196.2±39.0 mg/dL vs 213.7±49.7 mg/dL, p=0.01) [71]. A study by Van Minh et al. [123] reported that individuals with no formal education are 4.5 times more likely to die from CVD than those with a formal education. Winkleby et al. [203] found that Hispanics who had less than 12 years of education had an average BMI greater than those who completed only 12 years, and those who completed only 12 years of education had an average BMI greater than those who completed more than 12 years of education.

Yala et al. [99] reported that within the group of participants with an education level of some high school or less 69% had the metabolic syndrome. On the other hand, only 27% of participants with a postgraduate education had the metabolic syndrome. As aforementioned, since the metabolic syndrome is the clustering of three or more risk factors that are also risk factors for CVD, it is possible that in the study by Yala et al. the participants with the lowest education level, who also had the highest prevalence of the metabolic syndrome, were the most at risk for developing CVD.

Kavanagh et al. [127] reported that participants who completed high school had a lower waist circumference measurement than those who completed some or no high school (96.2 cm vs. 98.9 cm & 98.8 cm, respectively). A study by Millar et al. [128] shows that 6% of men and 5% of women ages 20-69 who had a university/college level education were obese while 13% of men and 16% of women in the same age groups but who only had an elementary level education were obese. Millar et al. also reported a negative association between education level and diastolic blood pressure and cholesterol concentrations [128]. Those with an elementary level education had a prevalence of 23% for elevated diastolic blood pressure while those with a secondary or university/college level education both had a prevalence of 21% for elevated diastolic blood pressure. When looking at serum cholesterol, 7.7% of individuals with an elementary education had elevated concentrations of serum cholesterol, in contrast to only 4.4% of those with a secondary education. No data was available for prevalence of elevated serum cholesterol for those with a university/college education. Similarly, Muennig et al. [129] documented that participants with less than a high school education have a lower HDL

115

cholesterol concentration than those who completed high school or more (50.5 mg/dL vs. 52.4 mg/dL).

In regard to diet, participants of the present study with some college education consumed more energy from polyunsaturated fatty acids relative to those who completed high school. Lasheras et al. [204] explored the relationship between education and diet. They categorized education as low (primary education or less) and high (partial secondary education to completed university education). Those with a low education consumed a lower percentage of energy from fat, a higher percentage of energy from carbohydrates, and a lower percentage of energy from protein than those with a high education level (39.2% vs. 41.2%, p<0.001; 49.1% vs. 46.8%, p<0.05; 13.6% vs. 14.2%, p<0.05, respectively). Low education level participants also consumed a lower percentage of energy from monounsaturated fatty acids than high education participants (12.5% vs. 13.6%, p<0.01) [204].

Other studies have also found a positive correlation between education and motivation to change behavior related to health in order to reduce CVD risk [71, 138]. Education was positively associated with use of health-related print media [71] or attending community-based programs intended to help people improve their lifestyle [138].

The present study supports studies previously performed in that education has a negative association with CVD risk. Our study, as well as others previously mentioned, sustains the hypothesis that education is negatively associated with weight, BMI, and waist circumference, and positively associated with HDL cholesterol. However, this study was unable to provide significant evidence that education affects macronutrient consumption other than the significant relationship with polyunsaturated fatty acids.

One speculation as to why education may affect CVD could be that a required class before graduating high school in the United States is health education. Those who do not complete high school or who do not complete high school in the United States may not have taken this class that explains the basic guidelines for healthy eating. Another reason may be that those who are less educated might not understand more complex details or wording that explains health conditions and preventive measures. If healthcare providers do not talk to patients at their level of understanding, the patient may be confused and may not know how to treat, control, or prevent the simplest of health conditions [109]. Moreover, those with less education may not qualify for higher paying jobs. Depending on the person's financial responsibilities, a lower paying job may not give that person the financial freedom to seek medical help or receive preventive treatment. In relation to diet, those who do not understand or know how to read the nutrition food label may not be able to make healthy choices or may not know that what they currently eat is not as healthy as other options.

# Acculturation, diet and CVD risk in Mexican-American adults

In contrast to prior findings [22, 23, 156, 173, 174, 176], this study found no significant differences in cardiometabolic disease risk factors when stratifying participants based on their acculturation level. However, there is an upward trend for both systolic and diastolic blood pressure showing that those whose lifestyles reflect more of the traditions and customs of Anglos/Americans have higher systolic and diastolic blood pressure values than those whose lifestyles reflect the Mexican traditions and customs, although this data is not significant. Likewise, HDL has an upward trend from those who practice the Mexican culture to those who practice more of the Anglo/American culture, but this data is not significant.

Other studies have found that acculturation and length of residence in the U.S. are associated with increased risk for CVD and metabolic abnormalities [22, 205]. Gordon-Larsen et al. [206] reported that longer U.S. residency was associated with increased overweight among participants. They also found that the prevalence of being overweight was higher for those Hispanics born in the U.S. versus foreign-born immigrants [206]. Sandquist and Winkleby [207] documented that the country of birth (United States or Mexico) and acculturation status were associated with waist circumference and abdominal obesity. Those born in Mexico had smaller waist circumferences when compared to U.S.-born Mexican-Americans [207].

This study found no significant differences in macronutrient consumption when stratifying participants based on acculturation category despite the fact that other studies have reported that greater acculturation negatively affects diet [23, 156]. Winham and Florian [156] report that participants characterized as Hispanic dominant ate more servings of green salad per week than their Bicultural/English dominant counterparts ( $4.2\pm4.0$ servings vs.  $2.7\pm2.8$  servings, p=0.042). They also found that compared to the Bicultural/English dominant participants, Hispanic dominant participants ate less servings of hamburgers, French fries, and pizza per week ( $0.7\pm0.7$  servings vs.  $1.2\pm1.0$ servings hamburgers, p=0.023;  $0.9\pm1.0$  servings vs.  $1.4\pm1.3$ servings French fries, p=0.001;  $0.5\pm0.8$  servings vs.  $0.8\pm0.8$ servings pizza, p=0.043) [156].

The present study was unable to support previous studies that reported the positive relationship between acculturation and CVD risk. One reason for not finding any significant differences in cardiometabolic disease risk factors in this study could be directly related to the study population. The majority of our participants were educated, employed, and more acculturated to the Anglo culture than the minority of the study participants. Some of our participants were students studying some aspect of health.

#### Diet and CVD risk in Mexican-American adults

The percent of energy consumed from total fat and monounsaturated fatty acids had a positive effect on weight, BMI, waist circumference, and insulin. This signifies that a person who consumes a large amount of their energy from fat sources is more likely to have a higher weight, BMI, waist circumference, and insulin level than a person who consumes a smaller amount of energy from fat sources. The different types of fatty acids may also affect anthropometrics and biological risk factors. Weight and waist circumference also had a positive association with percent of energy consumed from polyunsaturated fatty acids. Furthermore, percent energy from *trans* fatty acids had a positive association with waist circumference, insulin, and HOMA. This suggests that the type of fatty acid has an effect on a person's CVD risk.

On the other hand, weight and waist circumference had a negative association with percent energy from carbohydrates. This supports the national dietary guidelines that a diet lower in fat and higher in fruits, vegetables, low fat dairy foods, and whole grains, all of which are low in fat and contain some carbohydrates, helps to reduce risk for certain diseases such as CVD [153-155]. Azadbakht et al. [154] reports that diets consisting of a variety of fruits, vegetables, whole grains, and low-fat dairy foods have been shown to raise HDL cholesterol, lower triglycerides, lower systolic and diastolic blood pressure, lower weight, and lower blood glucose concentrations.

One explanation for the fat content (total fat, monounsaturated fatty acids, polyunsaturated fatty acids, and *trans* fatty acids) of the diet of the study participants in this study could be the culture and traditional foods that Mexicans and Mexican-Americans consume. One study found that over the past two decades, Mexican-American consumption of legumes, tortillas, corn bread, tomatoes, and hot red chili peppers has significantly increased [208, 209]. On the other hand, there has been a significant decrease in the consumption of melons, fruits other than melon, carrots, spinach, greens, collards, kale, and broccoli [208, 209]. Since diet has an effect on CVD, this change in diet seen over the last two decades could be a contributing factor to the increased risk for CVD seen among Mexican-Americans. Another factor affecting the change in diet is the country where a Mexican-American is born. Those born in the United States have a diet lower in foods considered healthy (high fiber, fruits,

vegetables, etc.) and a higher intake of foods containing large amounts of fat than those born in Mexico [210].

In conclusion, the results of our study support previous research that income, education, and employment are negatively associated with some cardiometabolic risk factors. Our study cannot support studies claiming that acculturation is positively associated with cardiometabolic risk factors since we did not find any relationship between acculturation and cardiometabolic risk factors or diet.

A limitation to this study is that we did not ask people about their current insurance status or how often they visit the doctor for check-ups. People who visit the doctor may be able to better prevent or receive treatment for diseases such as CVD. We also did not ask about current medications or estimations on monthly medical expenses.

Another limitation to the study can be related to the sample size. Our study had only 75 participants which may have been too small to find a strong relationship between socioeconomic factors or acculturation and CVD risk. Furthermore, our research sites were located in East Phoenix which deterred some people who live in West Phoenix from participating due to the distance to the sites.

In this study, body fat percentage was not found to be significant in any stratification or correlation of data. One possible reason for this is that the equipment used (Tanita Corporation, Tokyo, Japan) measured body fat percentage using bio-electrical impedance [211], a method that can result in different measurements based on the participant's hydration status and location of fat stores.

One aspect of this study was to collect a food frequency questionnaire. This questionnaire was sent to the participant's home where they were asked to fill it out and bring it when they came for their appointment. The questionnaire may have been confusing for some of the participants causing them to guess or leave a question blank. The questionnaire asked participants about the portion size and how frequently they ate particular foods. Participants may not have known the portion size or inaccurately reported how frequently they eat a food. Therefore,

124

the self reported diet via the food frequency questionnaire may not correctly reflect the participants' diets.

Self reported data may also affect other parts of the survey as well. Incorrect data reporting may affect the data analysis and create false positive or negative associations. For example, if a participant was unsure of their household monthly income and guessed, the actual monthly income may be in a different income group than what the participant reported.

More research on the Mexican-American population living in the Phoenix metropolitan area is needed. Perhaps future studies can include a larger sample including people from all areas of Phoenix. Depending on the hypothesis and aims of future researchers, it may be beneficial to tailor the survey to include or exclude certain data. For example, data that might help determine socioeconomic status would be to gather information on monthly bills (rent, utilities, groceries, etc.) or ask for their zip code to determine area of residency. It also might be beneficial to include a section to determine literacy, especially health literacy.

### REFERENCES

- Centers for Disease Control and Prevention. Death rates for heart disease by sex, race, Hispanic origin, and age. Health Statistics, 2008. http://www.cdc.gov/nchs/data/hus/hus08.pdf#035 -Accessed Nov5, 2009.
- 2. Roger VL et al. Heart Disease and Stroke Statistics—2011 Update: A report from the American Heart Association. *Circulation*. 2011;123:e18-e209.
- 3. Adolphe A, Cook LS, Huang X. A cross-sectional study of intima-media thickness, ethnicity, metabolic syndrome, and cardiovascular risk in 2268 study participants. Mayo clinic proceedings. March 2009; 84(3): 221-228.
- Kullo J, Ballantyne M. Conditional risk factors for atherosclerosis. Mayo clinic proceedings. February 2005; 80(2): 219-230.
- Allison MA, Budoff MJ, Wong ND, Blumenthal RS, Schreiner PJ, Criqui MH. Prevalence of risk factors for subclinical cardiovascular disease in selected US Hispanic ethnic groups. Am J Epidemiol. 2008;167:962-969.
- U.S. Department of Health and Human Services. The Office of Minority Health. http://minorityhealth.hhs.gov/templates/content.aspx?ID= 3325. Accessed July 21, 2011.
- Lindeman RD, Romero LJ, Hundley R, et al. Prevalences of type 2 diabetes, the insulin resistance syndrome, and coronary heart disease in an elderly, biethnic population. Diabetes Care 1998;21:959–66.
- Meigs JB, Wilson PWF, Nathan DM, et al. Prevalence and characteristics of the metabolic syndrome in the San Antonio Heart and Framingham Offspring studies. Diabetes 2003;52: 2160–7.
- 9. Mensah GA, Mokdad AH, Ford ES, et al. State of disparities in cardiovascular health in the United States. Circulation 2005; 111:1233–41.

- NCEP Expert Panel. Executive summary of the third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Choleterol in Adults (Adult Treatment Panel III). J Am Med Assoc. 2001; 285:2486-2497.
- 11. Grundy SM. Metabolic syndrome: a multiplex cardiovascular risk factor. J Clin Endocrinol Metab. 2007;92:399-404.
- 12. Reaven GM. Insulin resistance: the link between obesity and cardiovascular disease. Endocrinol Metab Clin N Am. 2008;37:581-601.
- 13. Ford ES, Giles WH. Prevalence of metabolic syndrome among U.S. adults. J Am Med Assoc. 2002;287:356-359.
- Weigensberg MJ, Toledo-Corral CM, Goran MI. Association between the metabolic syndrome and serum cortisol in overweight latino youth. J Clin Endocrinol Metab. 2008;93:1372-1378.
- 15. Alder NE, Rehkopf DH. U.S. disparities in health: Descriptions, causes, and mechanisms. *Annu Rev Public Health*. 2008;29:235-252.
- Diez Rouz AV, Merkin SS. Neighborhood of residence and incidence of coronary heart disease. N Engl J Med. 2001;345:99-106.
- 17. Robert SA, Reither EN. A multilevel analysis of race, community disadvantage, and body mass index among adults in the U.S. Soc Sci Med. 2004;59:2421-2434.
- Galvez MP, Morland K. Race and food store availability in an inner-city neighborhood. Public Health Nutr. 2007;11:624-631.
- 19. Larson NI, Story MT. Neighborhood environments: disparities in access to healthy foods in the U.S. Am J Prev Med. 2009;36:74-81.
- Liese AD, Weis KE. Food store types, availability, and cost of foods in a rural environment. J Am Diet Assoc. 2007;107:1916-1923.

- 21. Florez JC, Price AL, Campbell D, et al. Strong association of socioeconomic status with genetic ancestry in Latinos: implications for admixture studies of type 2 diabetes. Diabetologia. 2009;52:1528-1536.
- 22. Gregory-Mercado KY, Staten LK. Fruit and vegetable consumption of older Mexican-American women is associated with their acculturation level. Ethn Dis. 2006;16:89-95.
- 23. Perez-Escamilla R, Putnik P. The role of acculturation in nutrition, lifestyle, and incidence of type 2 diabetes among Latinos. J Nutr. 2007;137:860-870.
- 24. Zenk SN, Schulz AJ. Neighborhood racial composition, neighborhood poverty, and the spatial accessibility of supermarkets in metropolitan Detroit. Am J Public Health. 2005;95:660-667.
- 25. Zenk SN, Schulz AJ. Fruit and vegetable access differs by community racial composition and socioeconomic position in Detroit, Michigan. Ethn Dis. 2006;16:275-280.
- 26. Moore LV, Diez Roux AV. Associations of neighborhood characteristics with the location and type of food stores. Am J Public Health. 2006;96:325-331.
- 27. Baker EA, Schootman M. The role of race and poverty in access to foods that enable individuals to adhere to dietary guidelines. Prev Chronic Dis. 2006;3:1-11.
- Bodor JN, Rose D. Neighborhood fruit and vegetable availability and consumption: the role of small food stores in an urban environment. Public Health Nutr. 2007;11:413-420.
- 29. Census Bureau Statistics. America.gov: engaging the world. http://www.america.gov/st/washfileenglish/2006/July/20060707160631jmnamdeirf0.2887079. html. Accessed November 19, 2009.
- 30. Pew Hispanic Center. *Hispanics: A people in motion*. 2005, Washington, DC.

http://pewhispanic.org/files/reports/40.pdf. Accessed July 27, 2011.

- 31. Mitchell BD, Stern MP, Haffner SM, et al. Risk factors for cardiovascular mortality in Mexican Americans and non-Hispanic Whites. San Antonio Heart Study. *Am J Epidemiol* 1990;131:423–33.
- 32. Lange JW. Methodological concerns for non-Hispanic investigators conducting research with Hispanic Americans. Res Nurs Health. 2002;25:411-419.
- 33. Alexander H, Lockwood LP, Harris MA, Melby CL. Risk factors for cardiovascular disease and diabetes in two groups of Hispanic Americans with differing dietary habits. J Amer College Nutr. 1999;18:127-136.
- 34. Crespo CJ, Loria CM, Burt VL. Hypertension and other cardiovascular disease risk factors among Mexican-Americans, Cuban Americans, and Puerto Ricans from the Hispanic Health and Nutrition Examination Survey. Public Health Reports. 1996;111:7-10.
- 35. Mahan LK, Escott-Stump S. Krause's food, nutrition, and diet therapy. 11th edition. Elsever, USA. 2004.
- 36. Graham MR, Evans P, Thomas NE, Davies B, Baker JS. Changes in endothelial dysfunction and associated cardiovascular disease morbidity markers in GH-IGF axis pathology. Am J Cardiovasc Drugs. 2009;9:371-381.
- 37. Fuster V. Atherothrombosis: mechanisms and clinical therapeutic approaches. Vasc Med. 1998;3:231-239.
- 38. Fuster V. Understanding the coronary disease process and the potential for prevention: a summary. Prev Med. 1999;29:S9-S10.
- 39. Moreno PR. Vulnerable plaque: definition, diagnosis, and treatment. Cardiol Clin. 2010;28:1-30.
- Hirata Y, Nagata D, Suzuki E, Nishimatsu H, Suzuki J, Nagai R. Diagnosis and treatment of endothelial dysfunction in cardiovascular disease. Int Heart J. 2010;51:1-6.

- 41. Grassi D, Desideri G, Ferri C. Cardiovascular risk and endothelial dysfunction: the preferential route for atherosclerosis. Curr Pharm Biotechnol. 2011 Jan 11 [Epub ahead of print].
- 42. Ari H, Ari S, Erdogan E, Tiryakioglu O, Huysal K, Koca V, Bozat T. The effects of endothelial dysfunction and inflammation on slow coronary flow. Arch Turk Soc Cardiol. 2010;38:327-333.
- 43. Chen SM, Tsai TH, Hang CL, Yip HK, Fang CY, Wu CJ, Guo GBF. Endothelial dysfunction in young patients with acute ST-elevation myocardial infarction. Heart Vessels. 2011;26:2-9.
- 44. Van de Born BH, Lowenberg EC, van der Hoeven NV, de Laat B, Meijers JCM, Levi M, van Montfrans GA. Endothelial dysfunction, platelet activation, thrombogenesis and fibrinolysis in patients with hypertensive crisis. J Hypertens. 2011;29:000-000.
- 45. Kadohira T, Kobayashi Y, Iwata Y, Kitahara H, Komuro I. Coronary artery endothelial dysfunction associated with sleep apnea. Angiology. Feb 2011 [Epub ahead of print].
- 46. Gozal D, Kheirandish-Gozal L, Bhattacharjee R, Spruyt K. Neurocognitive and endothelial dysfunction in children with obstructive sleep apnea. Pediatrics. 2010;126:e1161-1167.
- 47. Liu FQ, Mu JJ, Liu ZQ, Shi DC, Huang Q, Yuan ZY, Lian QF, Zheng SH. Endothelial dysfunction in normotensive saltsensitive subjects. J Human Hyper. Feb 2011 [Epub ahead of print].
- 48. Nakagawa T, Tanabe K, Croker BP, Johnson RJ, Grant MB, Kosugi T, Li Q. Endothelial dysfunction as a potential contributor in diabetic nephropathy. Nat Rev Nephrol. 2011;7:36-44.
- 49. Ginsberg HN. Lipoprotein physiology and its relationship to atherogenesis. Endocrin Met Clinics North Am. 1990;19:211-228.

- 50. Assmann G, Funke H. HDL metabolism and atherosclerosis. J Cardio Pharm. 1990;16(Suppl. 9):S15-20.
- 51. Lewis GF, Rader DJ. New insights into the regulation of HDL metabolism and reverse cholesterol transport. Circ Res. 2005;96:1221-1232.
- 52. Rader DJ. Molecular regulation of HDL metabolism and function: implications for novel therapies. J Clin Invest. 2006;116:3090-3100.
- 53. Brewer HB. HDL metabolism and the role of HDL in the treatment of high-risk patients with cardiovascular disease. Curr Cardiol Reports. 2007;9:486-492.
- 54. Miller NE. HDL metabolism and its role in lipid transport. Eur Heart J. 1990;11 Suppl H:1-3.
- 55. Ye D, Lammers B, Zhao Y, Meurs I, Van Berkel T, Van Eck M. ATP-binding cassette transporters A1 and G1, HDL metabolism, cholesterol efflux, and inflammation: important targets for the treatment of atherosclerosis. Curr Drug Targets. 2010 Nov [Epub ahead of print].
- 56. Kawamoto R, Tomita H, Oka Y, Kodama A, Kamitani A. Metabolic syndrome amplifies the LDL-cholesterol associated increases in carotid atherosclerosis. Internal Medicine. 2005;44:1232-1238.
- 57. Baigent C, Keech A, Kearney PM, et al. Efficacy and safety of cholesterol lowering treatment: prospective metaanalysis of data from 90, 056 participants in 14 randomised trials of statins. Lancet. 2005;366:1267-78.
- 58. Graham J, Atar D, Borch-Johnsen K, et al. European guidelines on cardiovascular disease prevention in clinical practice: executive summary: Fourth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of nine societies and by invited experts). Eur Heart J. 2007;28:2375-2414.
- 59. Grundy SM, Cleeman JI, Merz CNB, et al. Implication of recent clinical trials for the National Cholesterol Education

Program Adult Treatment Panel III Guidelines. Circulation. 2004;110:227-239.

- 60. Bachorik PS. Interpretation of plasma lipid and lipoprotein measurements. Clin Chem. 1985;31:1107-1108.
- 61. Swenson CJ, Trepka MJ, Rewers MJ, Scarbro S, Hiatt WR, Hamman RF. Cardiovascular disease mortality in Hispanics and non-Hispanic Whites. Am J Emidemiol. 2002;156:919-928.
- 62. Toprak A, Wang H, Chen W, Paul T, Ruan L, Srinivasan S, et al. Prehypertension and black-white contrasts in cardiovascular risk in young adults: Bogalusa Heart Study. J Hypertens. 2009;27:243-250.
- 63. Chakraborty BM, Mueller WH, Reeves R, Poston WSC, Holscher DM, Quill B, Hanis CL, Foreyt JP. Migration history, health behaviors, and cardiovascular disease risk factors in overweight Mexican-American women. Ethn Dis. 2003;13:94-108.
- 64. Fischer BA, Liao M, Mosca L. Physical activity as a potential mechanism through which social support may reduce cardiovascular disease risk. J Cardiovasc Nurs. 2008;23:90-96.
- 65. Howard BV, Criqui MH, Curb JD, Rodabough R, Safford MM, Santoro N, Wilson AC, Wylie-Rosett J. Risk factor clustering in the Insulin Resistance Syndrome and its relationship to cardiovascular disease in postmenopausal White, Black, Hispanic, and Asian/Pacific Islander women. Metabolism. 2003;52:362-371.
- 66. Sharp TA, Grunwald GK, Giltinan KEK, King DL, Jatkauskas CJ, Hill JO. Association of anthropometric measures with risk of diabetes and cardiovascular disease in Hispanic and Caucasian adolescents. Prev Med. 2003;37:611-616.
- 67. Okosun IS, Liao Y, Rotimi CN, Prewitt TE, Cooper RS. Abdominal adiposity and clustering of multiple metabolic syndrome in White, Black, and Hispanic Americans. Ann Epidemiol. 2000;10:263-270.

- 68. Pasternak RC, Grundy SM, Levy D, Thompson PD. Task Force 3. Spectrum of risk factors for coronary heart disease. *J Am Coll Cardiol*. 1996;27:964-1047.
- 69. Murphy NF, MacIntyre K, Stewart S, Hart CL, Hole D, McMurray JJV. Long-term cardiovascular consequences of obesity: 20-year follow-up of more than 15, 000 middleaged men and women (the Renfrew—Paisley study). Eur Heart J. 2006;27:96-106.
- Nakagami T, DECODA Study Group. Hyperglycaemia and mortality from all causes and from cardiovascular disease in five populations of Asian origin. Diabetologia. 2004;47:385-394.
- Ribisl KM, Winkleby MA, Fortmann SP, Flora JA. The interplay of socioeconomic status and ethnicity on Hispanic and White men's cardiovascular disease risk and health communication patterns. Health Educ Res. 1998;13:407-417.
- 72. Wasserman BA, Sharrett AR, Lai S, et al. Risk factor associations with the presence of a lipid core in carotid plaque of asymptomatic individuals using high-resolution MRI: the Multi-Ethnic Study of Atherosclerosis (MESA). Stroke: J Amer Heart Assoc. 2008;39:329-335.
- 73. Kerenyi L, Mihalka L, Csiba L, Bacso H, Bereczki D. Role of hyperlipidemia in atherosclerotic plaque formation in the internal carotid artery. J Clin Ultrasound. 2006;34:283-288.
- 74. Lewington S, Clarke R. Combined effects of systolic blood pressure and total cholesterol on cardiovascular disease risk. Circulation. 2005;112:3373-3374.
- 75. Anderson P, Voss A, Horder M. Distribution of serum total cholesterol in a population with varying risks of cardiovascular disease. Ugeskr Laeger. 1990;152:523-526.
- Wang Z, Nakayama T. Inflammation, a link between obesity and cardiovascular disease. Mediators Inflamm. 2010;2010:535918.

- Rosvall M, Engstrom G, Berglund G, Hedblad B. C-reactive protein, established risk factors and social inequalities in cardiovascular disease – the significance of absolute versus relative measures of disease. BMC Public Health. 2008;8:189-198.
- 78. Dhingra R, Gona P, Nam BH, D'Agostino RB, Wilson PWF, Benjamin EJ, O'Donnell CJ. C-reactive protein, inflammatory conditions and cardiovascular disease risk. Am J Med. 2007;120:1054-1062.
- 79. Park CS, Ihm SH, Yoo KD, Kim DB, Lee JM, Kim HY, Chung WS, Seung KB, Kim JH. Relation between c-reactive protein, homocysteine levels, fibrinogen, and lipoprotein levels and leukocyte and platelet counts, and 10-year risk for cardiovascular disease among healthy adults in the USA. Am J Cardiol. 2010;105:1284-1288.
- Malik S, Wong ND, Franklin SS, Kamath TV, L'Italien GJ, Pio JR, Williams GR. Impact of the metabolic syndrome on mortality from coronary heart disease, cardiovascular disease, and all causes in United States adults. Circulation. 2004;110:1245-1250.
- 81. Balkau B, Deanfield JE, Despres JP, Bassand JP, Fox KAA, Smith SC, et al. International day for the evaluation of abdominal obesity (IDEA): a study of waist circumference, cardiovascular disease, and diabetes mellitus in 168 000 primary care patients in 63 countries. Circulation. 2007;116:1942-1951.
- 82. Dagenais GR, Yi Q, Mann JFE, Bosch J, Pogue J, Yusuf S. Prognostic impact of body weight and abdominal obesity in women and men with cardiovascular disease. Am Heart J. 2005;149:54-60.
- 83. Reeder BA, Senthilselvan A, Despres JP, Angel A, Liu L, Wang H, Rabkin SW. The association of cardiovascular disease risk factors with abdominal obesity in Canada. Can Med Assoc. 1997;157(1 suppl):S39-45.
- 84. Lee YH, Pratley RE. Abdominal obesity and cardiovascular disease risk: the emerging role of the adipocyte. J Cardiopulm Rehabil Prev. 2007;27:2-10.

- 85. Chrostowska M, Szyndler A, Paczwa P, Narkiewicz K. Impact of abdominal obesity on the frequency of hypertension and cardiovascular disease in Poland – results from the IDEA study (International Day for the Evaluation of Abdominal Obesity). Blood Pressure. 2010 [Epub ahead of print].
- 86. Wildman RP, McGinn AP, Lin J, Wang D, Muntner P, Cohen HW, Reynolds K, Fonseca V, Sowers MR. Cardiovascular disease risk of abdominal obesity vs. metabolic abnormalities. Obesity (Silver Spring). 2011;19:853-860.
- Casanueva FF, Moreno B, Rodriguez-Azeredo R, Massien C, Conthe P, Formiguera X, Barrios V, Balkau B. Relationship of abdominal obesity with cardiovascular disease, diabetes and hyperlipidemia in Spain. Clin Endocrin. 2010;73:35-40.
- Yamagishi S, Nakamura K, Matsui T. Regulation of advanced glycation end product (AGE)-receptor (RAGE) system by PPAR-gamma agonists and its implication in cardiovascular disease. Pharm Res. 2009;60:174-178.
- Zieman SJ, Kass DA. Advanced glycation end product cross-linking: pathophysiologic role and therapeutic target in cardiovascular disease. Congest Heart Fail. 2004;10:144-151.
- Nakamura Y, Horii Y, Nishino T, Shiiki H, Sakaguchi Y, Kagoshima T, et al. Immunohistochemical localization of advanced glycosylation end products in coronary atheroma and cardiac tissue in diabetes mellitus. Am J Pathol 1993;43:1649–56.
- 91. Cipollone F, Iezzi A, Fazia M, Zucchelli M, Pini B, Cuccurullo C, et al. The receptor RAGEas a progression factor amplifying arachidonate-dependent inflammatory and proteolytic response in human atherosclerotic plaques: role of glycemic control. Circulation 2003;108:1070–7.
- 92. Semba RD, Bandinelli S, Sun K, Guralnik JM, Ferrucci L. Plasma carboxymethyl-lysine, an advanced glycation end product, and all-cause and cardiovascular disease mortality

in older community-dwelling adults. J Am Geriatr Soc. 2009;57:1874-1880.

- 93. Eckel RH, Alberti KFMM, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet. 2010;375:181-183.
- 94. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. Circulation. 2005;112:2735-2752.
- Duffy A, Liew A, O'Sullivan J, Avalos G, Samali A, O'Brien T. Distinct effects of high-glucose conditions on endothelial cells of macrovascular and microvascular origins. Endothelium. 2006;13:9-16.
- 96. Zoungas S, de Galan BE, Ninomiya T, Grobbee D, Hamet P, Heller S, MacMahon S, Marre M, Neal B, Patel A, Woodward M, Chalmers J. Combined effects of routine blood pressure lowering and intensive glucose control on macrovascular and microvascular outcomes in patients with type 2 diabetes. Diabetes Care. 2009;32:2068-2074.
- 97. Turnbull FM, Abraira C, Anderson RJ, Byington RP, Chalmers JP, Duckworth WC, Evans GW, Gerstein HC, Holman RR, Moritz TE, Neal BC, Ninomiya T, Patel AA, Paul SK, Travert F, Woodward M. Intensive glucose control and macrovascular outcomes in type 2 diabetes. Diabetologia. 2009;52:2288-2298.
- Isomaa B, Almgren P, Tuomi T, Forsen B, Lahti K, Nissen M, Taskinen MR, Groop L. Cardiovascular morbidity and mortality associated with the metabolic syndrome. Diabetes Care. 2001;24:683-689.
- 99. Yala SM, Fleck EM, Sciacca R, Castro D, Joseph Z, Giardina EG. Metabolic syndrome and the burden of cardiovascular disease in Caribbean Hispanic women living in northern Manhattan: a red flag for education. Met Syn Rel Dis. 2009;7:315-322.
- 100. Sharma S, Malarcher AM, Giles WH, Myers G. Racial, ethnic, and socioeconomic disparities in the clustering of
cardiovascular disease risk factors. Ethn Dis. 2004;14:43-48.

- 101. Hu DJ, Covell RM. Health care usage by Hispanic outpatients as a function of primary language. West J Med. 1986;144:490-493.
- 102. Beaton SJ, Robinson SB, Von Worley A, Davis HT, Boscoe AN, Ben-Joseph R, Okamoto LJ. Cardiometabolic risk and health care utilization and cost for Hispanic and non-Hispanic women. Pop Health Manage. 2009;12:177-183.
- 103. David RA, Rhee M. The impact of language as a barrier to effective health care in an underserved urban Hispanic community. Mt Sinai J Med. 1998;65:393-397.
- 104. Askim-Lovseth MK, Aldana A. Looking beyond "affordable" health care: cultural understanding and sensitivity necessities in addressing the health care disparities of the US Hispanic population. Health Mark Q. 2010;27:354-387.
- 105. Johnson RA, Somnath S, Arbelaez JJ, Beach MC, Cooper LA. Racial and ethnic differences in patient perceptions of bias and cultural competence in health care. J Gen Inter Med. 2004;19:101–110.
- 106. DuBard CA, Gizlice Z. Language spoken and differences in health status, access to care, and receipt of preventive services among U.S. Hispanics. Am J Public Health. 2008;98:2021–2028.
- 107. Komaromy M, Grumbach K, Michael D, Vranizan K, Lurie N, Keane D, Bindman AB. The role of black and Hispanic physicians in providing health care for underserved populations. N Engl J Med. 1996;3334:1305-1310.
- Freeman G, Lethbridge-Cejku M. Access to health care among Hispanic or Latino women: United States, 2000-2002. Adv Data. 2006;368:1-26.
- 109. Davidson JA, Moreno PR, Badimon JJ, Lopez-Candales A, Giachello ALM, Ovalle F, Rodriguez CJ, Rosenson RS, Rodbard HW, Kannel WB. Cardiovascular disease

prevention and care in Latino and Hispanic subjects. Endocr Pract. 2007;13:77-85.

- 110. Lillie-Blanton M, Hoffman C. The role of health insurance coverage in reducing racial/ethnic disparities in health care. Health Affairs. 2005;24:398–402.
- 111. U.S. Census Bureau. The American community—Hispanics: 2004 (ACS-03). Washington, DC: Author. 2007. Retrieved from http://www.census.gov/prod/2007pubs/acs-03.pdf. Accessed February 10, 2011.
- 112. DeNavas-Walt C, Proctor BD, Smith JC. Income, poverty, and health insurance coverage in the United States: 2007 (U.S. Census Bureau, Current Population Reports, P60– 235). Washington, DC: U.S. Government Printing Office. 2008. Retrieved from http://www.census.gov/prod/2008pubs/p60–235.pdf. Accessed February 10, 2011.
- 113. Livingston G, Minushkin S, Cohn D. Hispanics and health care in the United States: Access, information and knowledge. Washington, DC: Pew Hispanic Center, and Princeton, NJ: Robert Wood Johnson Foundation. 2008. Retrieved from http://pewhispanic.org/files/reports/91.pdf. Accessed February 10, 2011.
- 114. James C, Thomas M, Lillie-Blanton M, Garfield R. Key facts—race, ethnicity & medical care (No. 6069–02). Menlo Park, CA: The Henry J. Kaiser Family Foundation. 2007. Retrieved from http://www.kff.org/minorityhealth/upload/6069–02.pdf. Accessed February 10, 2011.
- 115. Agency for Healthcare Research and Quality. National health disparities report, 2007 (AHRQ Publication No. 08– 0041). Rockville, MD: U.S. Department of Health and Human Services. 2008. Retrieved from http://www.ahrq.gov/qual/nhdr07/nhdr07.pdf. Accessed February 10, 2011.
- 116. Reyes C, Van de Putte L, Falco ´n AP, Levy RA. Genes, culture, and medicines: bridging gaps in treatment for Hispanic Americans. Washington, DC: National Alliance for

Hispanic Health. 2004. Retrieved from http://www.hispanichealth.org/pdf/hispanic\_report04.pdf. Accessed February 10, 2011.

- 117. Briesacher B, Limcangco R, Gaskin D. Racial and ethnic disparities in prescription coverage and medication use. Health Care Financ Rev. 2003;25:63–76.
- 118. Cristancho S, Garces DM, Peters KE, Mueller BC. Listening to rural Hispanic immigrants in the Midwest: a communitybased participatory assessment of major barriers to health care access and use. Qual Health Res. 2008;18:633-646.
- 119. La Clinica del Pueblo. Central American immigrants in the Washington region: A manual for health providers. Washington, DC: Author. 1998. Retrieved from http://www.gwumc.edu/partners/laclinicamanual.doc. Accessed February 10, 2011.
- 120. Pylypa J. Latino immigrants: Self-medication practices in two California Mexican communities. *J Immigr Health*. 2001;3:59–75.
- Mobley LR, Root ED, Finkelstein EA, Khavjou O, Farris RP, Will JC. Environment, obesity, and cardiovascular disease risk in low-income women. Am J Prev Med. 2006;30:327-332.
- 122. Appel SJ, Harrell JS, Deng S. Racial and socioeconomic differences in risk factors for cardiovascular disease among southern rural women. Nurs Res. 2002;51:140-147.
- 123. Minh HV, Huong DL, Wall S, Chuc NTK, Byass P. Cardiovascular disease mortality and its association with socioeconomic status: findings from a population-based cohort study in rural Vietnam, 1999-2003. Prev Chronic Dis. 2006;3:1-11.
- 124. Thomas AJ, Eberly LE, Smith GD, Neaton JD, Stamler J. Race/ethnicity, income, major risk factors, and cardiovascular disease mortality. Am J Public Health. 2005;95:1417-1423.

- 125. Winkleby MA, Cubbin C. Influence of individual and neighbourhood socioeconomic status on mortality among black, Mexican-American, and white women and men in the United States. J Epidemiol Community Health. 2003;57:444-452.
- 126. Fisher-Hoch SP, Rentfro AR, Salinas JJ, Perez A, Brown HS, Reininger BM, Restrepo BI, Wilson JG, Hossain MM, Rahbar MH, Hanis CM, McCormick JB. Socioeconomic status and prevalence of obesity and diabetes in a Mexican-American community, Cameron County, Texas, 2004-2007. Prev Chronic Dis. 2010;7:1-10.
- 127. Kavanagh A, Bentley RJ, Turrell G, Shaw J, Dunstan D, Subramanian SV. Socioeconomic position, gender, health behaviours and biomarkers of cardiovascular disease and diabetes. Soc Sci Med. 2010;71:1150-1160.
- 128. Millar WJ, Wigle DT. Socioeconomic disparities in risk factors for cardiovascular disease. Can Med Assoc J. 1986;134:127-132.
- 129. Muennig P, Sohler N, Mahato B. Socioeconomic status as an independent predictor of physiological biomarkers of cardiovascular disease: evidence from NHANES. Prev Med. 2007;45:35-40.
- 130. Tarasiuk A, Greenberg-Dotan S, Simon T, Tal A, Oksenberg A, Reuveni H. Low socioeconomic status is a risk factor for cardiovascular disease among adult obstructive sleep apnea syndrome patients requiring treatment. Chest. 2006;130:766-773.
- 131. Katzmarzyk PT, Church TS, Craig CL, Bouchard C. Sitting time and mortality from all causes, cardiovascular disease, and cancer. Med Sci Sports Exerc. 2009;41:998-1005.
- 132. Giardina EGV, Laudano M, Hurstak E, Saroff A, Fleck E, Sciacca R, Boden-Albala B, Cassetta J. Physical activity participation among Caribbean Hispanic women living in New York: relation to education, income, and age. J Women Health. 2009;18:187-193.

- 133. Leybas-Amedia V, Nuno T, Garcia F. Effect of acculturation and income on Hispanic women's health. J Health Care Poor Underserved. 2005;16:128-141.
- 134. Lopez RP. Neighborhood risk factors for obesity. Obesity. 2007;15:2111-2119.
- 135. Winkleby MA, Jatulis DE, Frank E, Fortmann SP. Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. Am J Public Health. 1992;82:816-820.
- 136. Kanjilal S, Gregg EW, Cheng YJ, Zhang P, Nelson DE, Mensah G, Beckles GLA. Socioeconomic status and trends in disparities in 4 major risk factors for cardiovascular disease among US adults, 1971-2002. Arch Intern Med. 2006;166:2348-2355.
- 137. Balistreri KS, Van Hook J. Socioeconomic status and body mass index among Hispanic children of immigrants and children of natives. Am J Public Health. 2009;99:2238-2246.
- 138. Archer SL, Greenlund KJ, Casper ML, Rith-Najarian S, Croft JB. Associations of community-based health education programs with food habits and cardiovascular disease risk factors among Native Americans with diabetes: The Inter-Tribal Heart Project, 1992 to 1994. J Am Diet Assoc. 2002;102:1132-1135.
- 139. Cunningham J. Socioeconomic disparities in self-reported cardiovascular disease for Indigenous and non-Indigenous Australian adults: analysis of national survey data. Popul Health Metr. 2010;24;8:31.
- 140. Steptoe A, Shamaei-Tousi A, Gylfe A, Henderson B, Bergstrom S, Marmot M. Socioeconomic status, pathogen burden and cardiovascular disease risk. Heart. 2007;93:1567-1570.
- 141. Quibrera-Infante R, Hernandez Rodriguez HG, Aradillas Garcia C, Gonzalez Rodriguez S, Calles-Escandon J. Prevalences of diabetes, glucose intolerance, hyperlipemia

and risk factors as a function of socioeconomic level. Rev Invest Clin. 1994;46:25-36.

- 142. Chen R, Tunstall-Pedoe H. Socioeconomic deprivation and waist circumference in men and women: The Scottish MONICA Surveys 1989-1995. Eur J Epidemiol. 2005;20:141-147.
- 143. Linn S, Fulwood R, Rifkind B, Carroll M, Muesing R, Williams OD, Johnson C. High density lipoprotein cholesterol levels among US adults by selected demographic and socioeconomic variables. The Second National Health and Nutrition Examination Survey 1976-1980. Am J Epidemiol. 1989;129:281-294.
- 144. Treviño RP, Marshall RM, Hale DE, Rodriguez R, Baker G, Gomez J. Diabetes risk factors in low-income Mexican-American children. Diabetes Care. 1999;22:202-207.
- 145. Salsberry PJ, Corwin E, Reagan PB. A complex web of risks for metabolic syndrome: race/ethnicity, economics, and gender. Am J Prev Med. 2007;33:114-120.
- 146. Rose D, Rickelle R. Food store access and household fruit and vegetable use among participants in the US Food Stamp Program. Public Health Nutr. 2004;7:1081-1088.
- 147. Krebs-Smith SM, Kantor LS. Choose a variety of fruits and vegetables daily: understanding the complexities. J Nutr. 2001;131:487S-501S.
- 148. Kaufman PR, MacDonald JM, Lutz SM, Smallwood DM. Do the poor pay more for food? Item selection and price differences affect low-income household food costs. Agricultural Economic Report No. 759. Washington, DC: Economic Research Service, US Department of Agriculture, 1997.
- 149. Morland K, Wing S, Diez Roux A. The contextual effect of the local food environment on residents' diets: the Atherosclerosis Risk in Communities Study. Am J Public Health. 2002;92:1761-1767.

- 150. Edmonds J, Baranowski T, Baranowski J, Cullen KW, Myres D. Ecological and socioeconomic correlates of fruit, juice, and vegetable consumption among African-American boys. Preventative Med. 2001;32:476-81.
- 151. Freedman DA. Local food environments: they're all stocked differently. Am J Community Psychol. Published online October 17, 2009. [Epub ahead of print]
- 152. Powell LM, Slater S, Mirtcheva D, Bao Y, Chaloupka FJ. Food store availability and neighborhood characteristics in the United States. Prev Med. 2009;44:189-195.
- 153. American Heart Association. Healthy diet goals. http://www.heart.org/HEARTORG/GettingHealthy/Nutrition Center/HealthyDiet%20Goals/Healthy-Diet-Goals\_UCM\_001152\_Article.jsp. Accessed July 10, 2010.
- 154. Azadbakht L, Mirmiran P, Esmaillzadeh A, Azizi T, Azizi F. Beneficial effects of a diet approach to stop hypertension eating plan features of the metabolic syndrome. Diabetes Care. 2005;28:2823-2831.
- 155. United States Department of Agriculture. MyPyramid dietary guidelines. www.mypyramid.gov. Accessed July 12, 2010.
- 156. Winham DM, Florian TA. Hispanic women in EFNEP have low adherence with dietary guidelines regardless of acculturation level. J Hunger Envir Nutr. 2010;5:498-509.
- 157. Michimi A, Wimberly MC. Associations of supermarket accessibility with obesity and fruit and vegetable consumption in the conterminous United States. Int J Health Geograph. 2010;9:49-62.
- Jetter KM, Cassady DL. The availability and cost of healthier food alternatives. Am J Prev Med. 2006;30:38-44.
- 159. Block D, Kouba J. A comparison of the availability and affordability of a market basket in two communities in the Chicago area. Public Health Nutr. 2005;9:837-845.

- 160. Casey AA, Elliot M. Impact of the food environment and physical activity environment on behaviors and weight status in rural U.S. communities. Prev Med. 2008;47:600-604.
- 161. Lipsky LM. Are energy-dense foods really cheaper? Reexamining the relation between food price and energy density. Am J Clin Nutr. 2009;90:1397-1401.
- 162. Sharkey JR, Johnson CM, Dean WR. Food access and perceptions of the community and household food environment as correlates of fruit and vegetable intake among rural seniors. BMC Geriatrics. 2010;10:32-41.
- 163. Morton LW, Blanchard TC. Starved for access: life in rural America's food deserts. Rural Realities. 2007;1:1-10.
- 164. French SA, Jeffery RW, Story M, et al. Pricing and promotion effects on low-fat vending snack purchases: the CHIPS study. Am J Public Health. 2001;91:112-117.
- 165. Paine-Andrews A, Francisco VT, Fawcett SB, Johnston J, Coen S. Health marketing in the supermarket: using prompting, product sampling, and price reduction to increase customer purchases of lower-fat items. Market Quart. 1996;14:85-99.
- 166. Kelly CM, Schootman M, Baker EA, Barnidge EK, Lemes A. The association of sidewalk walkability and physical disorder with area-level race and poverty. J Epidemiol Community Health. 2007;61:978-983.
- 167. Li F, Fisher KJ, Brownson RC, Bosworth M. Multilevel modeling of built environment characteristics related to neighbourhood walking activity in older adults. J Epidemiol Community Health. 2005;59:558-564.
- 168. Rohrer J, Pierce JR, Denison A. Walkability and self-related health in primary care patients. BMC Family Practice. 2004;5:29-35.
- 169. Lee D, Begley CE. Racial and ethnic disparities in response to direct-to-consumer advertising. Am J Health Syst Pharm. 2010;67:1185-1190.

- 170. Stark-Casagrande S, Gittelsohn J, Zonderman AB, Evans MK, Gary-Webb TL. Association of walkability with obesity in Baltimore City, Maryland. Am J Public Health. 2010. (Epub ahead of print)
- 171. Rundle A, Neckerman KM, Freeman L, Lovasi GS, Purciel M, Quinn J, Richards C, Sircar N, Weiss C. Neighborhood food environment and walkability predict obesity in New York City. Environ Health Perspect. 2009;117:442-447.
- 172. Schaefer SE, Salazar M, Bruhn C, Saviano D, Boushey C, Van Loan MD. Influence of race, acculturation, and socioeconomic status on tendency toward overweight in Asian-American and Mexican-American early adolescent females. J Immigrant Minority Health. 2009;11:188-197.
- 173. Sharma S, Murphy SP, Wilkens LR, Shen L, Hankin JH, Henderson B, Kolonel LN. Adherence to the Food Guide Pyramid recommendations among Japanese Americans, Native Hawaiians, and whites: results from the Multiethnic Cohort Study. J Am Diet Assoc. 2003;103:1195–1198.
- 174. Sharma S, Murphy SP, Wilkens LR, Shen L, Hankin JH, Monroe KR, Henderson B, Kolonel LN. Adherence to the food guide pyramid recommendations among African Americans and Latinos: results from the Multiethnic Cohort. J Am Diet Assoc. 2004;104:1873–1877.
- 175. Bermudez OI, Ribaya-Mercado JD, Talegawkar SA. Tucker KL. Hispanic and non-Hispanic white elders from Massachusetts have different patterns of carotenoid intake and plasma concentrations. J Nutr. 2005;135:1496–502.
- 176. Neuhouser ML, Thompson B, Coronado GD, Solomon CC. Higher fat intake and lower fruit and vegetables intakes are associated with greater acculturation among Mexicans living in Washington State. J Am Diet Assoc. 2004;104:51-57.
- 177. Lin H, Bermudez OI, Tucker KL. Dietary patterns of Hispanic elders are associated with acculturation and obesity. J Nutr. 2003;133:3651-3657.

- 178. Crespo CJ, Smit E, Carter-Pokras O, Andersen R. Acculturation and leisure-time physical inactivity in Mexican American adults: results from NHANES III, 1988-1994. Am J Public Health. 2001;91:1254-1257.
- 179. Fukuyama N, Homma K, Wakana N, Kudo K, Suyama A, Ohazama H, Tsuji C, Ishiwata K, Egochi Y, Nakazawa H, Tanaka E. Validation of the Friedewald Equation for evaluation of plasma LDL cholesterol. J Clin Biochem Nutr. 2008;43:1-5.
- 180. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985;28:412-419.
- 181. Wallace TM, Matthews DR. The assessment of insulin resistance in man. Diabet Med. 2002;19:527-534.
- 182. Martinez ME, Marshall JR, Graver E, et al. Reliability and Validity of a Self-Administered Food Frequency Questionnaire in a Chemoprevention Trial of Adenoma Recurrence. Cancer Epidemiol Biomarkers Prev. October 1, 1999 1999;8(10):941-946.
- 183. Thomson CA, Giuliano A, Rock CL, et al. Measuring Dietary Change in a Diet Intervention Trial: Comparing Food Frequency Questionnaire and Dietary Recalls. Am. J. Epidemiol. April 15, 2003 2003;157(8):754-762.
- 184. Cuellar I, Arnold B, et al. Acculturation rating scale for Mexican Americans-II: A revision of the original ARSMA Scale. Hispanic Journal of Behavioral Sciences. 1995;17(3):275-304.
- 185. Strufaldi MWL, da Silva EMK, Puccini RF. Insulin resistance among Brazilian schoolchildren: association with risk factors for cardiovascular diseases. Acta Paediatr. 2009;98:1646-1650.
- 186. Barr ELM, Cameron AJ, Balkau B, Zimmet PZ, Welborn TA, Tonkin AM, Shaw JE. HOMA insulin sensitivity index and the risk of all-cause mortality and cardiovascular disease

events in the general population: the Australian Diabetes, Obesity and Lifestyle Study (AusDiab) study. Diabetologia. 2010;53:79-88.

- 187. Smith NL, Barzilay JI, Shaffer D, Savage PJ, Heckbert SR, Kuller LH, Kronmal RA, Resnick HE, Psaty BM. Fasting and 2-hour postchallenge serum glucose measures and risk of incident cardiovascular events in the elderly: The Cardiovascular Health Study. Arch Intern Med. 2002;162:209-216.
- 188. Orencia AJ, Daviglus ML, Dyer AR, Walsh M, Greenland P, Stamler J. One-hour post plasma glucose and risks of fatal coronary heart disease and stroke among nondiabetic men and women: The Chicago Heart Association Detection Project in Industry (CHA) Study. J Clin Epidemiol. 1997;50:1369-1376.
- 189. Friedman EM, Herd P. Income, education, and inflammation: Differential Association in a National Probability Sample (The MIDUS Study). Psychosom Med. 2010;72:290-300.
- 190. Sheldon M, Gans KM, Tai R, George T, Lawson E, Pearlman DN. Availability, affordability, and accessibility of a healthful diet in a low-income community, Central Falls, Rhode Island, 2007-2008. Prev Chronic Dis. 2010;7:1-7.
- 191. Simon PA, Kwan D, Angelescu A, Shih M, Fielding JE. Proximity of fast food restaurants to schools: Do neighborhood income and type of school matter? Prev Med. 2008;47:284-288.
- 192. Haertel U, Heiss G, Filipiak B, Doering A. Cross-sectional and longitudinal associations between high density lipoprotein cholesterol and women's employment. Am J Epidemiol. 1992;135:68-78.
- 193. Martin R, Haskell W, Wood P. Blood chemistry and lipid profiles of elite distance runners. Acad Sci. 1977;301:346-360.

- 194. Wood P, Haskell W, Stern M, et al. Plasma lipoprotein distributions in male and female runners. Acad Sci. 1977;301:748-763.
- 195. Rotkis T, Cote R, Coyle E, et al. Relationship between high density lipoprotein cholesterol and weekly running mileage. J Cardiac Rehab. 1982;2:109-112.
- 196. Hagan R, Gettman L. Maximal aerobic power, body fat, and serum lipoproteins in male distance runners. J Cardiac Rehab. 1983;3:331-337.
- 197. Enger S, Herbjornsen K, Erikssen J, et al. High density lipoproteins (HDL) and physical activity: the influence of physical exercise, age, and smoking on HDL-cholesterol and the HDL-/total cholesterol ratio. Scand J Clin Lab Invest. 1977;37:251-255.
- 198. Hartung G, Foreyt J, Mitchell R, et al. Relation of diet to high density-lipoprotein cholesterol in middle-aged marathon runners, joggers, and inactive men. N Engl J Med. 1980;302:357-361.
- 199. Lehtonen A, Viikari J. The effects of vigorous physical activity at work on serum lipids with a special reference to serum high-density lipoprotein cholesterol. Acta Physiol Scand. 1978;104:117-121.
- 200. Rathnayake IM, Weerahewa J. Maternal employment and income affect dietary calorie adequacy in households in Sri Lanka. Food Nutr Bull. 2005;26:222-9.
- 201. Dubowitz T, Heron M, Bird CE, Lurie N, Finch BK, Basurto-Davila R, Hale L, Escarce JJ. Neighborhood socioeconomic status and fruit and vegetable intake among whites, blacks, and Mexican Americans in the United States. Am J Clin Nutr. 2008;87:1883-1891.
- 202. Morris JK, Cook DG, Shaper AG. Non-employment and changes in smoking, drinking, and body weight. BMJ. 1992;304:536-541.
- 203. Winkleby MA, Gardner CD, Taylor CB. The influence of gender and socioeconomic factors on Hispanic/White

differences in body mass index. Prev Med. 1996;25:203-211.

- 204. Lasheras C, Patterson AM, Casado C, Fernandez S. Effects of education on the quality of life, diet, and cardiovascular risk factors in an elderly Spanish community population. Exp Aging Res. 2001;27:257-270.
- 205. Lutsey PL, Diez Roux AV, Jacobs DR, Burke GL, Harman J, Shea S, Folsom AR. Associations of acculturation and socioeconomic status with subclinical cardiovascular disease in the Multi-Ethnic Study of Atherosclerosis. Am J Public Health. 2008;98:1963-1970.
- 206. Gordon-Larsen P, Harris KH, Ward DS, Popkin BM. Acculturation and overweight-related behaviors among Hispanic immigrants to the US: the National Longitudinal Study of Adolescent Health. Soc Sci Med. 2003;57:2023-2034.
- 207. Sandquist J, Winkleby M. Country of birth, acculturation status and abdominal obesity in a national sample of Mexican-American women and men. Int J Epidemiol. 2000;29:470-477.
- 208. Reyes-Ortiz CA, Hyunsu J, Eschbach K, Kuo YF, Goodwin JS. Neighbourhood ethnic composition and diet among Mexican-Americans. Public Health Nutr. 2009;12:2293-2301.
- 209. Reyes-Ortiz CA, Hyunsu J, Inniss A, Eschbach K, Kuo YF, Goodwin JS. Acculturation and serum nutrients thought to be involved with cancer prevention among Mexican American men in the United States. Cancer Control. 2008;15:169-175.
- 210. Montez JK, Eschbach K. Country of birth and language are uniquely associated with intakes of fat, fiber, and fruits and vegetables among Mexican-American women in the United States. *J Am Diet Assoc.* 2008;108:473-480.
- 211. Tanita, Monitoring Your Health Website. Professional Products – Body Composition Analyzer.

http://www.tanita.com/en/tbf-300a/. Accessed July 26, 2011.

#### APPENDIX I

## INSTITUTIONAL REVIEW BOARD/HUMAN SUBJECTS APPROVAL FORM

	RESEARCH AND ECONOMIC AFFAIRS	
	<u>na na n</u>	Office of Research Integrity and Assurance
	То:	Sonia Vega-Lopez 6950 E. Wi
P	- From:	Carol Johnston, Chair Biosci IRB
	Date:	11/09/2009
	Committee Action:	Expedited Approval
	Approval Date:	11/09/2009
	Review Type:	Expedited F2 F7
	IRB Protocol #:	0910004426
	Study Title:	Factors Related to Obesity in Latinos Living in the Phoenix Area
	Expiration Date:	11/08/2010
	The above-referenced proto	col was approved following expedited review by the Institutional Review Board.

It is the Principal Investigator's responsibility to obtain review and continued approval before the expiration date. You may not continue any research activity beyond the expiration date without approval by the Institutional Review Board.

Adverse Reactions: If any untoward incidents or severe reactions should develop as a result of this study, you are required to notify the Biosci IRB immediately. If necessary a member of the IRB will be assigned to look into the matter. If the problem is serious, approval may be withdrawn pending IRB review.

Amendments: If you wish to change any aspect of this study, such as the procedures, the consent forms, or the investigators, please communicate your requested changes to the Biosci IRB. The new procedure is not to be initiated until the IRB approval has been given.

Please retain a copy of this letter with your approved protocol.

#### APPENDIX II

ADVERTISEMENTS USED IN RECRUITMENT

#### Participants needed for Mexican and Mexican-American health study

We want to understand how diet and lifestyle affect health in Mexicans and Mexican-Americans

You could qualify if you:

- Are of Mexican descent
- Are 21-60 years old
- Live in Maricopa county
- Do not have diabetes or heart disease

Please contact Kristin Hunt at kristin.j.hunt@asu.edu or call 480-727-1731.

You will receive a \$20 gift card in compensation for your time.



# Buscamos participantes para un estudio de la salud de Mexicanos y Mexicano Americanos

Queremos entender cómo la dieta y el estilo de vida afectan la salud de los mexicanos y mexicanos americanos

Usted puede calificar si:

- Es de ascendencia mexicana
- Tiene 21-60 años
- Vive en el distrito de Maricopa
- No tiene diabetes o enfermedades del corazón

Comuníquese con Kristin Hunt <u>kristin.j.hunt@asu.edu</u> o llame al 480-727-1731.

Recibirá una tarjeta de \$20 por su tiempo.



#### APPENDIX III

#### SCREENING FORM

Screening ID#:	_ Date of Phone Call:	Recruiter:
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## Recruiter: Obtain verbal consent to ask eligibility criteria questions by reading and asking the following:

In order to determine whether you qualify or not for the study I need to ask a few questions about you and some general health information. This will take about 15 minutes. Can I ask these questions at this time?

Para saber si Usted califica para participar en este estudio tengo que hacerle algunas preguntas acerca de usted y de su estado general de salud. Esto tomará alrededor de 15 minutos. ¿Puedo hacerle estas preguntas en este momento?

YES 🗌 🛛 🛛	
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If YES, continue asking eligibility verification questions.

If NO, inform participant that you cannot proceed and thank him/her for their time. (STOP)

(Do not read) Participant's gender:	MALE 🗌	FEMALE
<b>ELIGIBILITY VERIFICATION</b> / VERIFICACION DE ELEGIBILIDAD	CTITERIOS L	DE
How old are you? / ¿Cuántos años tiene?		
(Do not read) Is age between 21 and 60 years?	YES [	] NO []
Do you consider yourself as Hispano(a)/Latino(a)? ¿Usted se considera a si mismo(a) como Hispano (	YES (a)/Latino(a)?	] NO []
Do you live in the Phoenix area? / ¿Vive en el área	de Phoenix? YES [	] NO []
How long have you lived in the Phoenix area? ¿Durante cuánto tiempo ha vivido en el área de Pho	oenix?	
(Do not read) Is time residing in Phoenix greater the	nan 12 months YES [	? ] NO []
What is your body weight? / ¿Cuánto pesa? (Do not read) Is weight at least 110 pounds (50 kg)	? YES [	NO

Are you able to walk without assistance? / ¿Puede camina	r sin ayuda? YES 🗌	NO 🗌
If the answer to any of these questions is NO, read:	(STOP	)
At this point you do not qualify for this study. Thank you ve En este momento usted no califica para este estudio. Muci tiempo.	ery much for you has gracias por	ur time. <sup>.</sup> su
If the answer to all of these questions is YES, continue.		
Are you afraid of needles or blood drawing? ¿Le dan miedo las agujas o que le saquen sangre?	YES 🗌	NO 🗌
Do you faint when you have your blood drawn? ¿Se desmaya cuando le sacan sangre?	YES 🗌	NO 🗌
Is it usually hard for medical personnel to draw your blood? ¿Regularmente le cuesta trabajo al personal médico saca	? le sangre?	
	YES 🗌	NO 🗌
If participant is a woman <50 y old please ask:		
Are you/¿Está Pregnant? / Embarazada ? Breastfeeding? / Lactando?	YES 🗌 YES 🗍	NO 🗌 NO 🗌
Are you following any of the following diets? / ¿Esta llevan siguientes dietas?	do alguna de la	S
Vegan / <i>vegetariano(a) estricto(a)</i> Very low carbohydrate / <i>Muy baja en carbohidratos</i> Atkins <i>/ Atkins</i>	YES YES YES	NO NO NO
Are you following any other specific diet? What type ¿Esta siguiendo alguna otra dieta específica? ¿De	e? qué tipo?	
(Do not read) Is this a restrictive diet?	YES 🗌	NO 🗌
<b>Recruiter:</b> consider any extreme diet or any diet that restri group, except for regular vegetarian diets, as a restrictive of	cts a major food liet.	d
Are you enrolled in any other research study anywhere? ¿Está participando en cualquier otro estudio de investigaci	YES 🗌 ión?	NO 🗌

Has a doctor or health care provider ever told you that you have... ¿Alguna vez le ha dicho su doctor o personal médico que usted tiene....

Is	patient eligible for participation? YES	NO 🗌 (S	STOP)
If the At this En est tiempo	answer to any of these questions is YES, read: point you do not qualify for this study. Thank you te momento usted no califica para este estudio. Mu o.	<b>(ST(</b> very much for <i>ichas gracias µ</i>	<b>DP)</b> your time por su
Are yo ¿ <i>Tom</i>	ou taking any cholesterol-lowering medications? a medicina para bajar el colesterol?	YES 🗌	NO 🗌
	Heart disease? / <i>Enfermedad del corazón?</i> Diabetes? Kidney disease? / Enfermedad de los riñones? Liver disease? / Enfermedad del hígado? Cancer? Hepatitis?	YES YES YES YES YES YES YES	NO    NO    NO    NO    NO

As part of this research study we will ask you to one of our ASU study sites for a visit in which we will measure your height, weight, waist and hip circumferences, and blood pressure, we will draw your blood, and we will ask you to complete a survey and a diet questionnaire. You will have to fast for 12 hours before your study visit. Are you willing to participate in this study?

Como parte de este estudio de investigación le vamos a pedir que venga a uno de nuestros sitios de investigación en ASU en donde le vamos a medir su estatura, peso, circunferencia de cintura y cadera y presión sanguínea, le vamos a sacar una muestra de sangre, y le vamos a pedir que complete una encuesta y un cuestionario de la dieta. Va a tener que ayunar por 12 horas antes de la visita. ¿Está dispuesto(a) a participar en este estudio?

			NO, STOP
Have you donated blood in the past 4 weeks?	YE	ES 🗌	NO 🗌
¿Ha donado sangre en las últimas 4 semanas	?		
If YES, when? / ¿cuándo?			

**Recruiter:** Schedule study visit at least 4 weeks after the blood donation date.

Study visit date and time: Study ID:	
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When you come to your study visit we will ask you to sign a consent form before we conduct any study-related activities. Would you like to receive a copy of this form in the mail prior to your visit for you to review it?

Cuando venga a la visita del estudio le vamos a pedir que firme una forma de consentimiento antes de realizar cualquiera de las actividades relacionadas con el estudio. ¿Le gustaría recibir una copia de esta forma por correo antes de su visita para que la revise?

You have the option to complete the diet questionnaire in your home prior to your study visit and bring it with you at the time of your appointment. This will shorten your study visit by about 45 minutes. Would you like to fill out the diet questionnaire ahead of time?

Tiene la opción de completar el cuestionario de la dieta en su casa antes de la visita del estudio, y traerla el día de su cita. Esto acortará su visita alrededor de 45 minutos. ¿Quiere llenar el cuestionario por adelantado?

	YES 🗌	NO 🗌
Are you allergic to LATEX? / ¿Es alérgico(a) al LÁTEX?	YES 🗌	NO 🗌

Thank you for your time. / Gracias por su tiempo.

#### APPENDIX IV

#### SOUTHWEST FOOD FREQUENCY QUESTIONNAIRE



No dobl	e, ron	ipa, er	ngrap	e, perfo	ore o s	separe	las pá	ginas.		
Please d	lo not	fold, o	cut, st	aple, p	unch,	or sep	arate	pages.		
Este cuestionario se re	fiere a	sus h	abitos	alime	nticio	s USA	LES.			
The questionnaire asks	s you :	about	your l	USUAI	eatin	ng hab	its.			
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NUNCA. Si selecciona porción. Fill in the circle that de food, fill in the circle u no serving size is neces EN PROMEDIO, ¿QUE TAN On the average, how often do	RAR escrib nder l sary. SEGU you ea	A VEZ es you Rarely <u>EI</u> IDO C t the fo	EMPI OME Illowing	UNCA ERAGE r. How LOS Ex LOS SIG	, no e E USE ever, ample GUIEN	s neces . If you if you souther wres A	u rare select LIMEN	ly or n RARE WTOS?	tama ever a CLY/N	no de nte the EVER
NUNCA. Si selecciona porción. Fill in the circle that de food, fill in the circle u no serving size is neces EN PROMEDIO, ¿QUE TAN On the average, how often do	RAR escrib nder l sary. SEGU you ea Po	A VEZ es you Rarely HDO C t the fo ORCIO	Z O N Tr AVH Neve EMPI OME Illowing	UNCA ERAGE r. How LOS Ex LOS SIG g foods?	, no e E USE ever, ample GUIEN	s neces . If you if you <u>es</u> vtES A U	u rare select LIMEN SO PRO Avera	IVTOS?	tama ever a 2LY/N	no de nte the EVER
NUNCA. Si selecciona porción. Fill in the circle that de food, fill in the circle u no serving size is neces EN PROMEDIO, ¿QUE TAN On the average, how often do GUISADOS Y SOPAS	RAR escrib nder I sary. SEGU you ea P( Po	A VE2 es you Rarely EI DO C t the fo ORCIO ortion S	Z O N T AVI Neve EMPI OME I Ilowing DN ize G	UNCA ERAGH T. How COS Ex LOS SIG 30 MAS VECS AL	use ever, ample guien	s neces . If you if you s vtES A VECES POR Van 6 Veces POR	u rare select LIMEN SO PR( Avera 2A 3 VECES POR	VTOS?	tama ever a CLY/N O	MENOS DE UNA VEZ
NUNCA. Si selecciona porción. Fill in the circle that de food, fill in the circle u no serving size is neces EN PROMEDIO, ¿QUE TAN On the average, how often do GUISADOS Y SOPAS Side, Mixed Dishes, and Souns	RAR escrib nder I sary. SEGU you ea Po Po S	A VE2 es you Rarely EI DIDO C t the fo ORCIO ortion S M	Z O N Tr AVE Neve EMPI OME I Illowing N ize G	UNCA ERACH F. How COS Ex LOS SIG 3 of MAS VECES AL DIA 3 of more Tormore	USE ever, ample GUIEN	s neces . If you if you if you vres A veces POR SEMANA 406 times a	LIMEN SO PRC Avera VECES POR SEMANA 2103 times a	VTOS? OMEDI ge Use VEZ PORA 1 ime	tama ever a CLY/N 2LY/N VECES AL MES 2103 times a	MENOS DE UNA VEZ ALES Less than 1 time
NUNCA. Si selecciona porción. Fill in the circle that de food, fill in the circle u no serving size is neces EN PROMEDIO, ¿QUE TAN On the average, how often do GUISADOS Y SOPAS Side, Mixed Dishes, and Soups FRIJOLES REFRITOS. Befried Bears	RAR escrib nder l sary. SEGU you ea Po Po S	A VE2 es you Rarely EI TIDO C t the fo ORCIO ortion S M M	EMPI OME I Ilowing SN ize G L	UNCA ERACI F. How COS Ex LOS SIG foods?	, no e e USE ever, ample GUIEN 10 <sup>2</sup> veces AL DIA 10 <sup>2</sup> tinday	s neces . If you if you if you SE WTES A US 4A6 VECES POR POR SEMANA 4 406 US 4 406 VECES VECES VI SE SE VI SE SE VI SE SE VI SE SE VI SE SE SE SE SE SE SE SE SE SE	LIMEN SO PRC Avera 2A 3 VECS SEMANA 210 3 times a we we we we we we we we	IVTOS?	CLY/N	MENOS DE UNA VEZ AL MES Less than 1 time a month
NUNCA. Si selecciona porción. Fill in the circle that de food, fill in the circle u no serving size is neces EN PROMEDIO, ¿QUE TAN On the average, how often do GUISADOS Y SOPAS Side, Mixed Dishes, and Soups FRLJOLES REFRITOS. Refried Beans FRLIOLES DE LA OLLA.	RAR escrib nder l sary. SEGU you ea P(Po Po S	A VE2 es you Rarely HDO C t the fo ORCIO ortion S M M	EMPI OME Neve OME Noving	UNCA ERACH F. How OS Ex LOS SIC 3 of MAS VECES AL 3 of more inday	, no e c USE ever, ample GUIEN 102 VECES AL 107 VECES AL 107 VECES aday	s neces s neces if you if you set veces por set veces por set veces por set veces por set veces set set veces set veces set veces set veces set set veces set set set set set set set s	LIMEN SO PRC Avera 24.3 VECES POR SEMANA 210.3 times a week	VTOS? OMEDI ge Use UNA VEZ POR SEMANA 1 time a week	CLY/N	MENOS DE UNA VEZ MENOS DE UNA VEZ S Less than 1 time a month
NUNCA. Si selecciona porción. Fill in the circle that de food, fill in the circle u no serving size is neces EN PROMEDIO, ¿QUE TAN On the average, how often do GUISADOS Y SOPAS Side, Mixed Dishes, and Soups FRIJOLES REFRITOS. Refried Beans FRIJOLES DE LA OLLA, CHARROS, BAYOS, NEGROS, PINTOS, ALUBIAS.	RAR escrib ader l sary. SEGU you ea Po Po S	EI EI EI DDO C t the fo ORCIO ortion S M M	Z O N T AVH Neve EMPI OME I Ilowing N ize C L	UNCA ERAGE F. How COS Ex LOS SIG g foods?	, no e E USE ever, ample GUIEN 102 VECES AL 102 a day	s neces s neces if you if you set vtes A Us vtes A Us vtes A us set vtes A vtes A	LIMEN SO PRC Avera SEMANA 2 to 3 times a week	VTOS?	CLY/N	MENOS DE UNA VEZ AL MES Less time a month
NUNCA. Si selecciona porción. Fill in the circle that de food, fill in the circle u no serving size is neces EN PROMEDIO, ¿QUE TAN On the average, how often do GUISADOS Y SOPAS Side, Mixed Dishes, and Soups FRIJOLES REFRITOS. Refried Beans FRIJOLES DE LA OLLA, CHARROS, BAYOS, NEGROS, PINTOS, ALUBIAS. Baked / Cooked Beans, "Charro-Style" Beans, Black, Pinto, and Kidney Beans	RAR escrib nder l sary. SEGU you ea Po Po S	EI Rarely EI DDO C t the fo ORCIO ortion S M M	EMPI OME Illowing C L O	UNCA ERAGE F. How COS Ex LOS Ex LOS SIG g foods?	, no e E USE ever, ample GUIEN 102 VECES AL DIA 107 dimes a day	s neces s neces if you if you set vites A us vites A us vites A us vites A us vites A vites A vi	LIMEN SO PRC Avera SEMANA 2 to 3 VECES POR SEMANA 2 to 3 VECES VEC	VTOS? VTOS? OMEDI ge Use VEZ VEZ SEMANA 1 ime a week	CO CO CO CO CO CO CO CO CO CO CO CO CO C	MENOS DE UNA VEZ AL MES Les MES Les MES O
NUNCA. Si selecciona porción. Fill in the circle that de food, fill in the circle u no serving size is neces EN PROMEDIO, ¿QUE TAN On the average, how often do GUISADOS Y SOPAS Side, Mixed Dishes, and Soups FRIJOLES REFRITOS. Refried Beans FRIJOLES REFRITOS. Refried Beans FRIJOLES DE LA OLLA, CHARROS, BAYOS, NEGROS, PINTOS, ALUBIAS. Baked / Cooked Beans, "Charro-Sityle" Beans, Black, Pinto, and Kidney Beans	RAR escrib nder l sary. SEGU you ea Po Po S	A VEZ es you Rarely EI TIDO C t the fo ORCIO ortion S M M	EMPI OME I Ilowing N ize G L O	UNCA ERACIE T. How OS Ex LOS SIG g foods?	, no e 2 USE ever, ample GUIEN 102 VECES AL DIA DIA 2 times o 0	A A 6 VICES A VITES A UN VITES A UN VITES A UN VITES A UN VITES A VICES A VICE	LIMEN SO PRO Avera SE Avera SE	VTOS? VTOS? OMEDI DE Use UNA VEZ POR SEMANA 1 time a week	CLY/N	MENOS DE UNA VEZ AL MES Less than 1 time a month

	Po Po	ORCIC ortion S	)N ize			U	SO PRO Avera	OMEDI ge Use	0		
GUISADOS Y SOPAS Side, Mixed Dishes, and Soups	P S	M M	G L	3 O MAS VECES AL DIA 3 or more times a day	1 O 2 VECES AL DIA 1 or 2 times a day	4A6 VECES POR SEMANA 4 to 6 times a week	2 A 3 VECES POR SEMANA 2 to 3 times a week	UNA VEZ POR SEMANA 1 time a week	2 A 3 VECES AL MES 2 to 3 times a month	MENOS DE UNA VEZ AL MES Less than 1 time a month	RARA VEZ O NUNCA Rarely or never
RIJOLES REFRITOS. Refried Beans	0	0	0	0	0	0	0	0	0	0	0
RIJOLES DE LA OLLA, CHARROS BAYOS, NEGROS, INTOS, ALUBIAS. Baked / Cooked Beans, C'harro-Style" Beans, Black, Pinto, and Kidney Beans	0	0	0	0	0	0	0	0	0	0	0
ARROZ BLANCO. Plain Rice	0	0	0	0	0	0	0	0	0	0	0
ARROZ A LA MEXICANA. Jexican Rice	0	0	0	0	0	0	0	0	0	0	0
OPAS DE PASTA / FIDEO SIN QUESO Y SIN CARNE). Koodle Soup, Pastas without Cheese or Meat	0	0	0	Q	6	PE	10	0	0	0	0
ENTEJAS, GARBANZOS, HABAS COCIDAS, EN SOPAS, ETC.). .entils, Garbanzo Beans (Cooked, a Soups, etc.)	00	8	Q		0	0	0	6	0	0	0
APAS FRITAS, PAPITAS, O APAS A LA FRANCESA. French Fries and Fried Potatoes	6	0	6	0	0	0	0	0	0	0	0
APAS HERVIDAS, AL HORNO, URE DE PAPA, OTRAS PAPAS. ther Potatoes, including Boiled, aked, Mashed	0	0	0	0	0	0	0	6	0	0	0
MOLE ROJO O VERDE, CON OLLO, PUERCO, CABRA U OVEJA. Red or Green Mole, with Chicken,	0	0	0		0	0	0	0	0	0	0
PORK, GOAT, OF LAMD	0	0	0	0	0	0	0	0	0	0	0
Cucchini with Cheese CHILES RELLENOS CON QUESO D PICADILLO.	0	0	0	0	0	0	0	0	0	0	0
TAMALES DE CARNE.	0	0	0	0	0	0	0	0	0	0	0
Teat Tamales AMALES DE ELOTE.	0	0	0	0	0	0	0	0	0	0	0
UESADILLAS DE HARINA O	0	0	0	0	0	0	0	0	0	0	0
DE MAIZ. Flour or Corn Quesadillas						1		-	-	1000	

### EN PROMEDIO : OUE TAN SECUIDO COME LOS SIGUENTES ALIMENTOS?

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EN PROMEDIO, ¿QUE TAN SEGUIDO COME LOS SIGUIENTES ALIMENTOS? On the average, how often do you eat the following foods?

1	(CONTINUACION) (continued)	Po Po	ORCIO ortion S	)N lize	1		U	SO PRO Avera	OMEDI ge Use	10		
	GUISADOS Y SOPAS Side, Mixed Dishes, and Soups	P S	M M	G L	3 O MAS VECES AL DIA 3 or more times	1 O 2 VECES AL DIA 1 or 2 times	4A 6 VECES POR SEMANA 4 to 6 times a	2 A 3 VECES POR SEMANA 2 to 3 times a	UNA VEZ POR SEMANA 1 time	2 A 3 VECES AL MES 2 to 3 times a	MENOS DE UNA VEZ AL MES Less than 1 time	
Ì	TACOS SUAVES. Soft Tacos	0	0	0	a day	a day	week O	O	a week	month	a month	
ľ	TOSTADAS	0	0	0	0	0	0	0	0	0	0	
	BURRITOS	0	0	0	0	0	0	0	0	0	0	
ľ	ENCHILADAS, CHILAQUILES, PASTEL AZTECA	0	0	0	0	0	0	0	G	0	0	I
	FLAUTAS / TACOS DORADOS. Crispy Tacos (Fried)	0	0	0	0	0	0	0	0	0	0	1
	SALSA MEXICANA, SALSA PARA TACOS, OTRAS SALSAS. Mexican Sauce, Taco Sauce, Other	0	0	0	0	0	O	10	0	0	0	
	POZOLE, MENUDO, GALLINA PINTA	0	0	0	2	Q	10	P	0	0	0	
	CAZUELA, SOPA DE ALBONDIGAS. Cazuela Soup, Meatball Soup	91	Q	6	0	0	0	6	0	0	0	
	SOPA DE TORTILLA. Tortilla Soup	19	O	-0	6	10	0	0	0	0	0	
	CALDO DE QUESO. Cheese Soup	0	0	10	0	0	0	0	6	0	0	
	SOFA DE VERDURAS, SOFA DE VERDURAS CON CARNE, COCIDO, MINESTRONE, Y SOPA DE TOMATE. Vegetable Soup, Vegetable Beef, Cocido, Minestrone, and Tomato Soup	0	0	0	0	0	0	0	0	0	0	
	OTRAS SOPAS. Other Soups	0	0	0	0	Ø	0	0	0	0	0	
	ESPAGUETI, LASAGNA, OTRAS PASTAS CON PURE O SALSA DE TOMATE. Spaghetti, Lasagna, Other Pasta with Tomato Sauce	0	0	0	0	0	0	0	0	0	0	
	PIZZA	0	0	0	0	0	0	0	0	0	0	
	PLATILLOS QUE CONTENGAN QUESO COMO MACARRONES CON QUESO. Mixed Dishes with Cheese, like Macaroni and Cheese	0	0	0	0	0	0	0	6	0	0	

	Po Po	ORCIO ortion S	)N ize			U	SO PRO Avera	OMEDI ge Use	0		
CARNES Y HUEVOS	Р	М	G	3 O MAS VECES AL DIA	1 O 2 VECES AL DIA	4 A 6 VECES POR SEMANA	2 A 3 VECES POR SEMANA	UNA VEZ POR SEMANA	2 A 3 VECES AL MES	MENOS DE UNA VEZ AL MES	RARA VEZ O NUNCA Basely
vieats and Eggs	S	М	L	times a day	times a day	times a week	times a week	time a week	times a month	1 time a month	or
IUEVOS. Sggs	0	0	0	0	0	0	0	0	0	0	0
OCINO.	0	0	0	0	0	0	0	0	0	0	0
THORIZO. Aexican Sausage	0	0	0	0	0	0	0	0	0	0	0
ALCHICHON.	0	0	0	0	0	0	0	G	0	0	0
ALCHICHAS. Hot dogs	0	0	0	0	0	0	0	0	0	0	0
IAMBURGUESAS, IAMBURGUESAS CON QUESO, ASTEL DE CARNE, MILANESA DE TERNERA, PICADILLO. Iamburgers, Cheeseburgers, Meat .oaf, Veal Dishes	0	0	0	Q	6	PE	20	0	0	0	0
AMON, MORTADELA, BOLOGNA SPAM. lam, Lunch Meats, and Spam	70	8	b	0	d	b	0	6	0	0	0
HSTEC DE RES, ASADO AL IORNO, CARNE ASADA. Geef-Steaks, Roasts, Carne Asada		0	6	S	0	0	0	0	0	0	0
CUISADOS DE CARNE CON CANAHORIA Y OTRAS TERDURAS.	7	1	-								
Beef Stew or Pot Pie with Carrots and Other Vegetables	0	0	0	0	0	0	0	0	0	0	0
CARNE CON CHILE (ESTILO ONORA), CHILE CON CARNE DE ES, DE PUERCO, O DE TERNERA, HRIA. eef with Chile ("Sonoran-style"), ork or Veal with Chile, Birria.	0	0	0	0	0	0	0	6	0	0	0
MACHACA.	0	0	0	0	0	0	0	0	0	0	0
HIGADO DE RES, POLLO, 'ERNERA. .iver: Beef, Chicken, Veal	0	0	0	0	0	0	0	0	0	0	0
PUERCO: INCLUYENDO CARNITAS CHULETAS, AL IORNO, MILANESAS. York, including Carnitas Chops, Roasts, Fried	0	0	0	0	0	0	0	0	0	0	0

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EN PROMEDIO, ¿QUE TAN SEGUIDO COME LOS SIGUIENTES ALIMENTOS? On the average, how often do you eat the following foods?

	Po	ORCIC ortion S	)N lize	1		U	SO PRO	OMEDI ge Use	10		
AVES, PESCADOS, Y MARISCOS	P	M	G	3 O MAS VECES AL DIA 3 or more	102 VECES AL DIA 1 or 2	4A 6 VECES POR SEMANA 4 to 6	2 A 3 VECES POR SEMANA 2 to 3	UNA VEZ POR SEMANA 1	2 A 3 VECES AL MES 2 to 3	MENOS DE UNA VEZ AL MES Less than	
POLLO EMPANIZADO O ERITO	5	M	L	a day	a day	week	week	a week	month	a month	
Breaded or Fried Chicken	0	0	0	0	Ο	0	0	0	0	0	
COCIDO O A LA PARRILLA. Chicken or Turkey: Baked, Stewed, or Broiled	0	0	0	0	0	0	0	6	0	0	
PESCADO FRITO, EMPANIZADO O SANDWICH DE FILETE DE PESCADO. Breaded or Fried Fish or Fish Sandwich	0	0	0	0	0	0	0	0	0	0	
ATUN, ENSALADA DE ATUN, ATUN AL HORNO O GUISADO. Tuna Fish, Tuna Salad, Tuna Casserole	0	0	0	Q	0	6	1	6	0	0	
MARISCOS: CAMARONES, LANGOSTA, JAIBA, OSTIONES, ETC. Shell Fish: Shrimp, Lobster, Crab; Oysters, etc.	P	P	26	6	0	0	50	0	0	0	
CEVICHE, ESCABECHE DE PESCADO. Ceviche, Pickled Herring	6	þ	10	0	0	0	0	0	0	0	
OTRO TIPO DE PESCADOS: ASADOS, A LAS BRASAS, ETC. Other Fish: Broiled, Baked, etc.	6	0	0	0	0	0	0	O	0	0	
DERIVADOS DE LA Dairy Products	LECH	IE									
QUESOS COMO CHEDDAR Y SWISS. Cheeses such as Cheddar and Swiss	0	0	0	0	0	0	0	6	0	0	
QUESO FRESCO Y PANELA. Farmers/Fresh Cheese	0	0	0	0	0	0	0	0	0	0	l
OTROS QUESOS Y QUESOS PARA UNTAR. Other Cheeses and cheese spreads	0	0	0	0	0	0	0	0	0	0	
QUESO CUAJADA O REQUESON. Cottage Cheese	0	0	0	0	0	0	0	0	0	0	
YOGURT	0	0	0	0	0	0	0	6	0	0	
CREMA / ACIDA. Creams (Sour, Semi-Sweet, Sweet)	0	0	0	0	0	0	0	3	0	0	
		NO	ESCRIE	IA EN ES	EA ZON	4					
		PLEASE	DONO	WRITE	IN THIS	AREA					

	Po Po	ORCIO rtion S	)N lize	S		U	SO PRO Avera	OMEDI ge Use	0		
CEREALES Cereals	P	M	G	3 O MAS VECES AL DIA 3 or more	1 O 2 VECES AL DIA 1 or 2	4 A 6 VECES POR SEMANA 4 to 6	2 A 3 VECES POR SEMANA 2 to 3 times o	UNA VEZ POR SEMANA 1	2 A 3 VECES AL MES 2 to 3	MENOS DE UNA VEZ AL MES Less than	RARA VEZ O NUNCA Rarely
	3	M	L	a day	a day	week	week	a week	month	a month	never
AVENA O CEREALES COCIDOS COMO ATOLE, CHAMPURRADO. Oatmeal/Other Cooked Cereals	0	0	0	0	o	0	0	0	0	0	0
; CEREALES DE DESAYUNO INSTANEOS? Eat Cold Cereals?	0	0	0	0	0	0	0	3	0	0	0
CUALES SON LOS CEREALES QUE COME CON MAS FRECUENCIA? Which cereals do you usually eat?											
1.	0	0	0	0	0	0	0	0	0	0	0
2.	0	0	0	0	0	0	0	0	0	0	0
LE AÑADE LECHE A LOS CEREALES QUE COME? Do you add milk to cereal?	0	0	Ø	Q	Q	p	20	0	0	0	0
LE AÑADE AZUCAR A LOS CEREALES QUE COME? Do you add sugar to cereal?	To	8	10	0	d	a	10	0	0	0	0
VERDURAS Vegetables	T		L	9							
Zucchini	0	0	0	0	0	0	0	0	0	0	0
CALABAZA COCIDA, AL HORNO. Winter Squash, Baked Squash	0	0	0	0	0	0	0	0	0	0	0
ELOTES. Corn	0	0	0	0	0	0	0	0	0	0	0
ZANAHORIAS. Carrots	0	0	0	0	0	0	0	0	0	0	0
CAMOTES. Sweet Potatos, Yams	0	0	0	0	0	0	0	6	0	0	0
EJOTES. String Beans	0	0	0	0	0	0	0	6	0	0	0
CHICHAROS. Peas	0	0	0	0	0	0	0	O	0	0	0
ESPARRAGO. Asparagus	0	0	0	0	0	0	0	0	0	0	0
COLIFLOR O COLES DE BRUSELAS. Cauliflower or Brussels Sprouts	0	0	0	0	0	0	0	0	0	0	0
SETAS. Mushrooms	0	0	0	0	0	0	0	6	0	0	0
ESPINACAS COCIDAS. Cooked Spinach	0	0	0	0	0	0	0	0	0	0	0
NO ESCRIBA EN ES		- PLEAS	E DO NO	T WRITE I		REA		SE	RIAL	#	

EN PROMEDIO, ¿QUE TAN SEGUIDO COME LOS SIGUIENTES ALIMENTOS? On the average, how often do you eat the following foods?

(	(CONTINUACION) (continued)	Po Po	ORCIO	)N lize	land.		U	SO PRO Avera	OMEDI ge Use	10		
	VERDURAS Vegetables	P S	M M	G L	3 O MAS VECES AL DIA 3 or more times	102 VECES AL DIA 1 or 2 times	4A6 VECES POR SEMANA 4 to 6 times a	2 A 3 VECES POR SEMANA 2 to 3 times a	UNA VEZ POR SEMANA 1 time	2 A 3 VECES AL MES 2 to 3 times a month	MENOS DE UNA VEZ AL MES Less than 1 time	RAF VE O NUN Raro
1010	ACELGAS, VERDOLAGAS, QUELITES. Mustard Greens, Turnip Greens, Collards	0	0	0	o	a day	o	o	a week	month	a month	nev
1	VEGETALES MIXTOS QUE CONTENGAN ZANAHORIAS. Mixed vegetables containing Carrots, canned or frozen	0	0	0	0	0	0	0	6	0	0	0
	CHILES VERDES, JALAPEÑOS, POBLANOS, SERRANOS, EN RAJAS, CHILE PIMIENTO / MORRON. Chiles: Jalapeño, Serrano, etc., including Bell Peppers	0	0	0	0	0	6	20	0	0	0	0
1	AGUACATE, GUACAMOLE. Avocado, Guacamole	0	0	Ø	O	Ø	0	e,	0	0	0	C
-	NOPALES. Cactus Leaves	ar	Q	10	10	0	0	0	0	0	0	0
115	REPOLLO O COL, COL AGRIA V ENSALADA DE COL. Cabbage, Sauerkraut and Cole Slaw	B	10	a	6	5	6	0	0	0	0	(
1	BROCOLI. Broccoli	Q	Q	10	0	0	0	0	0	0	0	(
1	ESPINACAS CRUDAS, BERROS. Raw Sninach, Watercress	6	0	0	0	0	0	0	6	0	0	(
1	LECHUGA.	0	0	0	0	0	0	0	0	0	0	(
1	TOMATE CRUDO. Raw Tomato	0	0	0	0	0	0	0	6	0	0	(
-	IICAMA.	0	0	0	0	0	0	0	0	0	0	(
1	PEPINO.	0	0	0	0	0	0	0	6	0	0	(
-	CEBOLLA.	0	0	0	0	0	0	0	0	0	0	(
1	AJO.	0	0	0	0	0	0	0	0	0	0	(
-	CILANTRO.	0	0	0	0	0	0	0	6	0	0	(

	Po Po	ORCIO ortion S	N ize			U	SO PRO Avera	OMEDI ge Use	0		
FRUTAS	Р	М	G	3 O MAS VECES AL DIA	1 O 2 VECES AL DIA	4A6 VECES POR SEMANA	2 A 3 VECES POR SEMANA	UNA VEZ POR SEMANA	2 A 3 VECES AL MES	MENOS DE UNA VEZ AL MES	RARA VEZ O NUNC/
Fruits	s	М	L	3 or more times a day	1 or 2 times a day	4 to 6 times a week	2 to 3 times a week	1 time a week	2 to 3 times a month	Less than 1 time a month	Rarely or never
NARANJAS, MANDARINAS. Oranges, Tangerines	0	0	0	0	0	0	0	0	0	0	0
LIMONES Y JUGO DE LIMON. Lemon/Limes and Lime Juice	0	0	0	0	0	0	0	0	0	0	0
PLATANOS. Bananas	0	0	0	0	0	0	0	0	0	0	0
PIÑA. Pineapple	0	0	0	0	0	0	0	0	0	0	0
MANZANAS, PERAS, GUAYABAS. Apples, Pears, Guavas	0	0	0	0	0	0	0	0	0	0	0
MANGOS. Mangoes	0	0	0	0	0	0	0	0	0	0	0
DURAZNOS, CHABACANOS, ALBARICOQUES, Y NECTARINAS. Peaches, Apricots, and Nectarines	0	0	0	Q	6	P	20	0	0	0	0
SANDIA. Watermelon	0	0	00	0	d	d	10	0	0	0	0
MELÓN. Cantaloupe and other melons	Vo	9	A	6	0	0	0	0	0	0	0
FRESAS. Strawberries	p	0	6	0	0	0	0	G	0	0	0
OTRAS MORAS Frambuesas, zarzamoras). Other berries blueberries/raspberries)	0	0	0	0	0	0	0	0	0	0	0
UVAS.	0	0	0	0	0	0	0	0	0	0	0
CEREZAS. Cherries	0	0	0	0	0	0	0	0	0	0	0
PASAS, CIRUELAS PASAS, HIGOS. Raisins, Prunes, Figs	0	0	0	0	0	0	0	0	0	0	0
CIRUELAS FRESCAS. Fresh Plums	0	0	0	0	0	0	0	0	0	0	0
FORONJAS.	0	0	0	0	0	0	0	0	0	0	0

\_ ]

EN LAS SIGUIENTES SECCIONES AGRUPAMOS ALIMENTOS QUE PUEDEN CONSUMIRSE EN GRANDES CANTIDADES. POR ESA RAZON LA <u>FRECUENCIA DE CONSUMO</u> VA AHORA DESDE 6 O MAS VECES AL DIA HASTA RARA VEZ O NUNCA. EN EL CASO DE LAS TORTILLAS QUEREMOS QUE NOS DIGA APROXIMADAMENTE <u>CUANTAS</u> COME REGULARMENTE Y DE QUE <u>TAMAÑO</u> EN EL CASO DE LAS TORTILLAS DE HARINA. POR EJEMPLO, 2 TORTILLAS

DE HARINA MEDIANITAS (APROXIMADAMENTE 10 PULGADAS DE DIAMETRO).

In the following sections, we have grouped food items that can be eaten in large quantities. For that reason, the <u>frequency of consumption</u> now goes from 6 or more times a day to rarely or never. In regards to tortillas, we want you to specify approximately <u>how many</u> you usually eat and the <u>size</u> in the case of flour tortillas. For example, 2 medium size flour tortillas (about 10 inches in diameter).

= EN PROMEDIO, ¿QUE TAN SEGUIDO Y CUANTAS TORTILLAS COME?

On the average, how often and how many tortillas do you eat?

F



EN PROMEDIO, ¿QUE TAN SEGUIDO COME LOS SIGUIENTES ALIMENTOS?
On the average, how often do you eat the following foods?

	P Po	ORCIO ortion S	N ize				USO A	PROM verage l	EDIO Use			
PANES Breads	P S	M M	G L	6 O MAS VECES AL DIA 6 or more times a day	3 O 5 VECES AL DIA 3 or 5 times a day	2 VECES AL DIA 2 times a day	UNA VEZ AL DIA 1 time a day	506 VECES POR SEMANA 5 or 6 times a week	2A4 VECES POR SEMANA 2 to 4 times a week	UNA VEZ POR SEMANA 1 time a week	1A 3 VECES AL MES 1 to 3 times a month	RAR/ VEZ O NUNC Rarel or never
PAN BLANCO, BIROTE/ BOLILLO, GALLETAS SALADAS, ETC., INCLUYENDO EN SANDWICHES. White Bread, Rolls, Crackers, Mexican Bread (including sandwich bread)	0	0	0	0	0	0	0	0	0	6	0	0
PAN O PANECILLOS DE TRIGO ENTERO INTEGRAL. Whole Wheat Bread / Bolls	0	0	0	0	0	0	0	0	0	6	0	0

(continued)	Po Po	ORCIO	DN Size				USO A	PROM verage	EDIO Use			
PANES	Р	М	G	6 O MAS VECES AL DIA	305 VECES AL DIA	2 VECES AL DIA	UNA VEZ AL DIA	506 VECES POR SEMANA	2 A 4 VECES POR SEMANA	UNA VEZ POR SEMANA	1 A 3 VECES AL MES	RARA VEZ O NUNCA
Breads	s	М	L	6 or more times a day	3 or 5 times a day	2 times a day	1 time a day	5 or 6 times a week	2 to 4 times a week	1 time a week	1 to 3 times	Rarely or
PAN DE MAIZ / ELOTE. Corn Bread	0	0	0	0	0	0	0	0	0	0	0	0
ANECITOS / BIZCOCHOS DE SALVADO, O NTEGRALES. Gran or whole wheat muffin	0	0	0	0	0	0	0	0	0	6	0	0
PANCAKES Y WAFFLES. Pancakes and Waffles	0	0	0	0	0	0	0	0	0	0	0	0
PAN DULCE. Sweet Bread	0	0	0	0	0	0	0	0	0	0	0	0
POSTRES Sweets	5	0)	D	3	5	U		1				
NIEVE DE LECHE O HELADO.	1	5		1	-	0	0		-	0	~	-
ce Cream NIEVE DE AGUA, PALETAS.	0	2	0	0	0	0	0	0	0	0	0	0
herbet, Popsicles ATILLA O FLAN, BUDIN. Justard or Pudding	0	0	0	0	0	0	0	0	0	6	0	0
ARROZ CON LECHE Y ASAS. Rice Pudding with Raisins	0	0	0	0	0	0	0	0	0	0	0	0
DONAS.	0	0	0	0	0	0	0	0	0	0	0	0
GALLETAS.	0	0	0	0	0	0	0	0	0	0	0	0
Cookies	0	0	0	0	0	0	0	0	0	0	0	0
Cookies PASTEL. Cake					0	0	0	0	0	6	0	0
Cookies PASTEL. Cake PASTEL, O DULCE DE CALABAZA, CAMOTE DE DULCE. Pumpkin Pic, Sweet Potato Pic	0	0	0									
Cookies PASTEL, Cake PASTEL, O DULCE DE CALABAZA, CAMOTE DE DULCE. Pumpkin Pic, Sweet Potato Pie BUÑUELOS, SOPAPILLAS, PASTELILLOS,	0	0	0	0	0	0	0	0	0	0	0	0

(continued)	P Pe	ORCIC ortion S	)N Size				USO A	PROM verage l	EDIO Use			
POSTRES Sweets	P	м	G	6 O MAS VECES AL DIA 6 or more timor	3 O 5 VECES AL DIA 3 or 5 times	2 VECES AL DIA 2 times	UNA VEZ AL DIA 1	5 O 6 VECES POR SEMANA 5 or 6 times a	2 A 4 VECES POR SEMANA 2 to 4	UNA VEZ POR SEMANA 1	1A3 VECES AL MES 1 to 3 times	RA V NU Ra
CHOCOLATES.	•	0	0	a day	a day	a day	a day	week	a week	a week	a month	ne
OTROS DULCES, JALEA, MERMELADA, MIEL, PANOCHA. Other Candy, Jelly, Honey, Molasses	0	0	0	0	0	0	0	0	0	0	0	
BOTANAS Salty Snacks and S	Spread	ds										
PALOMITAS DE MAIZ O ESQUITE. Popcorn	0	0	0	0	0	0	6	10	0	6	0	(
PAPITAS FRITAS DE BOLSA, CUALQUIER TIPO DE "CHIPS". Chips, all types	0	ar	q	6	0	0	0	6	0	0	0	
CHICHARRONES DE PUERCO. Pork Rinds	0	B	0	a	6	6	0	0	0	G	0	(
NUECES, INCLUYENDO CACAHUATES. Shelled Nuts, including Peanuts	0	6	0	0	0	0	0	0	0	0	0	
MANTEQUILLA DE CACAHUATE. Peanut Butter	0	0	0	0	0	0	0	0	0	6	0	
ACEITUNAS. Olives	0	0	0	0	0	0	0	0	0	0	0	(
ADEREZOS PARA ENSALADA. Salad Dressing	0	0	0	0	0	0	0	0	0	6	0	1
MAYONESA. Mayonnaise	0	0	0	0	0	0	0	0	0	0	0	(
SALSA DE TOMATE Y SALSA DE BARBACOA. Tomato Ketchup and BBQ	0	0	0	0	0	0	0	0	0	6	0	(
Sauce			0	0	0	0	0	0	0	6	0	
CONTINUACION)	PORCION Portion Size			USO PROMEDIO Average Use								
--	-------------------------	--------	--------	--	---	--	---	--	--	--	---	--
BOTANAS Salty Snacks and Spreads	P S	M M	G L	6 O MAS VECES AL DIA 6 or more times a day	3 O 5 VECES AL DIA 3 or 5 times a day	2 VECES AL DIA 2 times a day	UNA VEZ AL DIA 1 time a day	506 VECES POR SEMANA 5 or 6 times a week	2A4 VECES POR SEMANA 2 to 4 times a week	UNA VEZ POR SEMANA 1 time a week	1A3 VECES AL MES 1 to 3 times a month	RARA VEZ O NUNCA Rarely or never
MOSTAZA, RABANO PICANTE. Mustard, Horseradish	0	0	0	0	0	0	0	0	0	6	0	0
JUGO Y/O GRASA DE CARNE CON HARINA (Gravy). Gravies made with Meat Drippings or White Sauce	0	0	0	0	0	0	0	0	0	6	0	0
BEBIDAS												
Beverages												
AGUA. Water	0	0	0	0	0	0	0	0	0	0	0	0
JUGO DE NARANJA O TORONJA. Orange or Grapefruit Juice	0	0	0	0	Q	6	0	Po	0	6	0	0
LIMONADA. Lemonade/Limeade	0	0	Q	0	O	0	a	0	0	6	0	0
HORCHATA. Rice-Based	6	holl	8	Ko	0	6	10-	0	0	0	0	0
JAMAICA.	d	e	O	6	0	0	0	0	0	0	0	0
JUGO DE UVA. Grane Inice	o	6	6	0	0	0	0	0	0	0	0	0
JUGO DE TOMATE.	0	0	0	0	0	0	0	0	0	0	0	0
TANG, JUGOS EN POLVO INSTANTANEOS. Tang, Start Breakfast Drinks, Juice Drinks	0	0	0	0	0	0	0	0	0	6	0	0
REFRESCOS / SODAS NO DIETETICAS. Regular Soft Drinks	0	0	0	0	0	0	0	0	0	0	0	0
REFRESCOS / SODAS DIETETICAS. Diet Soft Drinks	0	0	0	0	0	0	0	0	0	0	0	0
CERVEZA. Beer	0	0	0	0	0	0	0	0	0	0	0	0
LICOR. Liquor / Alcohol	0	0	0	0	0	0	0	0	0	0	0	0
VINO. Wine	0	0	0	0	0	0	0	0	0	0	0	0
CAFE REGULAR. Regular Coffee	0	0	0	0	0	0	0	0	0	0	0	0
CAFE DESCAFEINADO. Decaffeinated Coffee	0	0	0	0	0	0	0	0	0	0	0	0

(CONTINUACION) (continued)	PORCION Portion Size			USO PROMEDIO Average Use								
BEBIDAS Beverages	P S	M M	G L	6 O MAS VECES AL DIA 6 or more times a day	3 O 5 VECES AL DIA 3 or 5 times a day	2 VECES AL DIA 2 times a day	UNA VEZ AL DIA 1 time a day	5 O 6 VECES POR SEMANA 5 or 6 times a week	2 A 4 VECES POR SEMANA 2 to 4 times a week	UNA VEZ POR SEMANA 1 time a week	1A 3 VECES AL MES 1 to 3 times a month	R. V NU Ra
TE DE HIERBAS. Herbal Tea	0	0	0	0	0	0	0	0	0	(3)	0	(
TE CON CAFEINA (NEGRO / VERDE) HELADO O CALIENTE. Tea, Hot or Iced	0	0	0	0	0	0	0	0	0	0	0	
A SU CAFE O TE, ¿LE AÑADE: To your coffee or tea, do you add:												
CREMA EN POLVO PARA CAFE? Non-Dairy Creamer?	0	0	0	0	0	0	10	10	0	0	0	
LECHE? Milk?	0	0	0	0	0	Q	0	0	0	6	0	
O CREMA? or Real Cream?	0	0	0	6	10	a	0	6	0	0	0	1
;A SU CAFE O TE, LE AÑADE: To your coffee or tea, do you add:	5	D		3	1			4				
AZUCAR? Sugar	0	0	Q	10	0	0	0	0	0	0	0	
O ENDULZANTE ARTIFICIAL (AZUCAR DE DIETA, AZUCAR ARTIFICIAL)? Diet Sugar or Artificial Sweetener?	0	0	0	0	0	0	0	0	0	6	0	
LECHE ENTERA Y BEBIDAS CON LECHE ENTERA (SIN INCLUIR EN CEREALES). Whole Milk and Beverages with Whole Milk (excluding milk in cereals)	0	0	0	0	0	0	0	0	0	0	0	
LECHE DESCREMADA, LECHE AL 1% O LECHE EN POLVO RECONSTITUIDA (SIN INCLUIR EN CEREALES). Skim Milk, 1% Milk or Buttermilk, Reconstituted Milk (excluding milk in cereals)	0	0	0	0	0	0	0	0	0	6	0	

CONTINUACION)	PORCION Portion Size				USO PROMEDIO Average Use								
BEBIDAS Beverages	P S	M M	G L	6 O MAS VECES AL DIA 6 or more times a day	3 O 5 VECES AL DIA 3 or 5 times a day	2 VECES AL DIA 2 times a day	UNA VEZ AL DIA 1 time a day	506 VECES POR SEMANA 5 or 6 times a week	2A4 VECES POR SEMANA 2 to 4 times a week	UNA VEZ POR SEMANA 1 time a week	1A3 VECES AL MES 1 to 3 times a month	RARA VEZ O NUNCA Rarely or	
ECHE AL 2% Y BEBIDAS CON LECHE AL 2% (SIN NCLUIR EN CEREALES). % Milk and Beverages with % Milk (excluding milk in ereals)	0	0	0	0	0	0	0	0	0	6	0	0	
LECHE CONDENSADA. Condensed Milk	0	0	0	0	0	0	0	0	0	0	0	0	
LECHE EVAPORADA. Evaporated Milk	0	0	0	0	0	0	0	0	0	0	0	0	
Additional Foods 1. ¿HAY ALGUN OTRO ESTE EN EL CUEST Are there any foods no EN PROMEDIO, ¿QU	D ALIN TONA ot liste	MENTO RIO? d on the	QUE e quest	COME 7 onnaire COME I	THAT YOU	KOS UN I eat at l UIENT	A VEZ east on ES AE	AL ME ce a moi	S Y QU nth? DS?	E NO			
Additional Foods 1. ¿HAY ALGUN OTRO ESTE EN EL CUEST Are there any foods no EN PROMEDIO, ¿QI Please list any additio	O ALIN TONA ot liste UE TA	MENTC RIO? d on the N SEG ods and Po Po	QUE e quest UIDO how of ORCIO	COME 7 formaire COME I ften on a N Size G	that you OS SIC verage y	NOS UN I eat at l UIENT ou eat e VECES AL DIA	A VEZ east on ES AE ach foo U	AL ME ce a moi IMENTO d? SO PRO Avera UNA VEZ AL DIA	S Y QU nth? OS? DMED ge Use 506 VECES POR SEMANA	E NO	UNA VEZ POR SEMANA	1A3 VECES AL MES	
Additional Foods 1. ¿HAY ALGUN OTRO ESTE EN EL CUEST Are there any foods no EN PROMEDIO, ¿QU Please list any additio	O ALIN IONA ot liste UE TA	MENTC RIO? d on the N SEG ods and Po Po S	P QUE e quest UIDO how of ORCIO rtion M M	COME 7 ionnaire COME I COME I iten on a Size G L	that you OS SIG verage y 60 MAS VECES AL DIA 6 or more times a day	NOS UN a eat at l UIENT ou eat e VECES AL DIA 3 or 5 times a day	A VEZ east on ES AE ach foo U VECES AL DIA 2 veces AL DIA 2 a day	AL ME ce a mon IMENTO d? SO PRO Avera VEZ AL DIA 1 intime intime a day	S Y QU nth? OS? OMED ge Use 506 VECES POR SEMANA 5 or 6 times week	E NO 2A4 VECES POR SEMANA 2104 times a week	UNA VEZ POR SEMANA 1 time a week	1A 3 VECES AL MES 1 to 3 a month	
Additional Foods 1. ¿HAY ALGUN OTRO ESTE EN EL CUEST Are there any foods no EN PROMEDIO, ¿Q Please list any additio	O ALIN TONA ot liste UE TA onal foo	MENTC RIO? d on the N SEG ods and Po Po S	P QUE e quest UIDO how of ORCIO ortion M M	COME 7 ionnaire COME I iten on a N Size G L	that you OS SIC verage y 60 MAS VECES AL DIA 6 or more a day	NOS UN I eat at l UIENT ou eat et UIENT ou eat et DIA 3 or 5 times a day	A VEZ east on ES AE ach foo U VECES AL DIA 2 times a day	AL ME ce a mou IMENTO d? SO PRC Avera UNA VEZ AL DIA 1 time a day	SYQU nth? DS? DMEDD ge Use 506 VECES POR SEMANA 5 or 6 times a week	E NO IO 2A4 VECES POR SEMANA 2104 times a week	UNA VEZ POR SEMANA I time a week	1A 3 VECES AL MES 1 to 3 times a month	
Additional Foods 1. ¿HAY ALGUN OTRO ESTE EN EL CUEST Are there any foods nd EN PROMEDIO, ¿QI Please list any additio	) ALIN TONA tot liste UE TA nal foo	MENTC RIO? dd on thin N SEG ods and Po Po S	P QUE e quest UIDO how of ORCI ortion	COME 7 ionnaire COME I iten on a N Size G L C	AL MEN that you OS SIC verage 5 AL DIA 6 or more a day	305 VECES AL DIA 305 VECES AL DIA 305 times a day	A VEZ east on ES AE ach foo U VECES AL DIA 2 times a day	AL ME ce a mou IMENTO d? SO PRO Avera VEZ AL DIA 1 time a day	S Y QU nth? OS? OMED ge Use ge Use POR SEMANA 5 or 6 times a week 3 3 3 3 3 3 3 3 3 3 3 3 3	E NO 2A4 VECES SEMANA 2io4 2io4 2io4 2io4 2io4 2io4 2io4 2io4	UNA VEZ POR SEMANA I time a week	IA3 VECES AL MES I to3 times a month	
Additional Foods 1. ¿HAY ALGUN OTRO ESTE EN EL CUEST Are there any foods nd EN PROMEDIO, ¿QI Please list any additio	) ALIM TONA ot liste UE TA mai foo	MENTC RIO? d on the N SEC ods and Po Po P S	O QUE e quest unbo howood ORCI( m M M	COME 7 formaire COME I Ten on a N Size G L C O O	that you os SIC verage y 60 MAS VECES AL DIA 6 or more times a day	305 VECS UN eat at at l UIENT our eat e VECES AL DIA JA Suff a day	A VEZ east on ES AL DIA UU VECES AL DIA 2 times a day	AL ME ce a mou d? SO PRC Avera. UNA VEZ AL DIA 1 time a day	S Y QU nth? DS? DMEDD ge Use 506 VECES POR SEMANA 5 or 6 times a week 3 3 3 3 3 3 3 3 3 3 4 5 3 5 4 5 5 5 6 5 5 6 5 5 6 5 5 6 5 7 6 5 7 6 5 7 6 5 7 6 5 7 6 5 7 6 5 7 6 5 7 6 5 7 6 5 7 6 5 7 6 5 7 6 5 7 6 5 7 6 5 7 6 5 7 6 5 7 7 7 7 7 7 7 7 7 7 7 7 7	E NO	UNA VEZ POR SEMANA 1 time a week	1A3 VECES AL MES a month	
Additional Foods 1. ¿HAY ALGUN OTRO ESTE EN EL CUEST Are there any foods nd EN PROMEDIO, ¿QI Please list any additio	) ALIN TIONA to liste TA mai foo	MENTC RIO? d on the N SEG ods and Po Po Po S	D QUE e quest UIDO D ORCIO M M M	COME 7 ionnaire COME I ten on a DN izze G L Q Q Q Q Q Q Q Q Q Q Q Q Q Q Q Q Q Q	that you OS SIC verage y 60 Mas veces a day 6 or more times a day 0 0	SOS UN n eat at 11 CUIENT our eat e VECES AL DIA Jor 5 Unes a day a day o O	A VEZ east on ES AE: VECES ALA 2 times a aby O	AL ME ce a mou tMENTO d? SO PRC Avera: UNA VEZ AL DIA UNA VEZ AL DIA 1 time a day	S Y QU nth? DS? DMEDI ge Use \$06 YEOS SEMANA \$076 times a (0) (0) (0) (0) (0) (0)	E NO	UNA VEZ POR SEMANA 1 time a week 0 0	1A3 VECES AL MES 1 tímes a month	
Additional Foods 1. ¿HAY ALGUN OTRO ESTE EN EL CUEST Are there any foods nd EN PROMEDIO, ¿QI Please list any additio	) ALIN TONA to liste TA mal for	MENTC RIO? d on the N SEG ods and Po Po P S O O O	D QUE e quest UIDO Dowol	COME 7 ionnaire COME I Iten on a N Size G L O O O O	that you os SIC verage y 60 Mas veces aday 0 0 0 0	SOS UN a cat at 11 CUIENT our cat ce 30 5 VECES AL 3 or 5 times a day 3 0 0 0 0 0 0 0 0 0 0 0 0 0	A VEZ east on ES AE: ach foo U U VECES ALA DIA DIA DIA DIA DIA DIA DIA DIA DIA DI	AL ME ce a mou timento d? SO PRC Avera: UNA VEZ AL DIA 1 time a day	S Y QU nth? DS? DMED ge Use 506 VECES VECES SMANA 5 or 6 times a 5 or 6 times a times	E NO	UNA VEZ POR SEMANA 1 time a week	IA3 VECES AL MES Itimes a month	

	REGUNTAS SEGUN A SUS HABITOS LIMENTARIOS DURANTE EL PERIODO DE EMPO QUE LE PIDIERON CONSIDERAR.	yoi we	ur eating habits during the period of time you re asked to consider
2.	¿QUE TAN SEGUIDO SE COME EL PELLEJO DEL POLLO?	2.	How often do you eat the skin on chicken?
			G Fraquently or Always
-	O ALCUNAS VECES	-	Sometimes
Ξ.	RARA VEZ O NUNCA		Rarely or Never
3.	¿QUE TAN SEGUIDO SE COME LA GRASA DE	3.	How often do you eat the fat on meat?
	LA CARNE?		
-	O SEGUIDO O SIEMPRE		Frequently or Always
-	ALGUNAS VECES		O Sometimes
-	O RARA VEZ O NUNCA		Rarely or Never
4.	POR LO GENERAL, CUANDO COMIÓ HAMBURGUESA U OTRA CARNE MOLIDA, ¿QUE TIPO COMIÓ?	4.	When you ate hamburger or other ground mea what type did you usually eat?
-	O NO COMIÓ HAMBURGUESA U OTRA CARNE MOLIDA	1	Did not eat hamburger or other ground meat
100	O REGULAR	1	O Regular
-	O MAGRO (80-89% MAGRO)	$\left( \right)$	Dean (80-89%)
-	O EXTRA MAGRO (90% O MÁS)	11	Extra lean (90% or greater lean)
-	O NO SE	11	Don't know
-		//	
-		11	0) =
5.	POR LO GENERAL, CUANDO COMIÓ LAS	5.	When you ate canned tuna, what type did you
-	CONSERVAS DE ATÚN, ¿QUE TIPO COMIÓ?		usually eat?
-	O NO COMIÓ CONSERVAS DE ATÚN		O Did not eat canned tuna
-	O ENVASADO EN AGUA		O Water-packed
-	O ENVASADO EN ACEITE		O Oil-packed
-	O NO SE		O Don't know
6.	POR LO GENERAL, CUANDO SE COMIÓ	6.	When you ate fruit, was it usually
-	FRUTA, FUE		
-	🔘 NO COMIÓ FRUTA		O Did not eat fruit
-	FRESCO, CONGELADO		O Fresh, frozen
-	O CONSERVADO EN JUGOS NATURALES		Canned in natural juices
-	O CONSERVADO EN JARABE LIGERO		O Canned in light syrup
2	O CONSERVADO EN JARABE PESADO		Canned in heavy syrup
7.	POR LO GENERAL, CUANDO USÓ ADEREZOS PARA ENSALADA, ¿QUE TIPO USÓ?	7.	When you used salad dressing, what type did you usually use?
-	O NO USO ADEREZOS PARA ENSALADA		O Did not use salad dressing
-	O REGULAR	-	U Regular
-	BAJO EN GRASA O REDUCIDO EN CALORIAS		U Low Fat or Reduced Calorie
Ξ	U SIN GRASA		• Fat-Free
=			
=	NO ESCRIBA EN ESTA ZONA - PLEASE DO NOT WRIT	OOC	SERIAL #

	POR LO GENERAL, CUANDO USO MAYONESA, ¿QUE TIPO USÓ?	8.	When you used mayonnaise, what type did you usually use?
	O NO USO MAYONESA		Did not use mayonnaise     Regular
	BAIO EN GRASA O REDUCIDO EN CALORÍAS		Regular     Low Fat or Reduced Calorie
	SIN GRASA		Fat-Free
	POR LO GENERAL, CUANDO COMIÓ PALOMITAS DE MAÍZ O ESQUITE, ¿COMO FUE PREPARADO?	9.	When you ate popcorn, how was it usually prepared?
	O NO COMIÓ PALOMITAS DE MAIZ O ESQUITE		O Did not eat popcom
	O HECHO EN ACEITE O COMPRADO DEL ALMACEN		O Popped in oil or pre-popped
	O MICROONDA REGULAR		O Regular microwave
	MICROONDA LIGERO     HECHO EN AIRE		Light microwave     Air-popped
	POR LO GENERAL, ¿QUE TIPO DE GRASA USA?	10.	What kind of fat do you usually use?
	O NINGUNA		O Don't add fat
	O MARGARINA PARA UNTAR		O Soft Margarine
	MARGARINA DE BARRA		O Stick Margarine
	O MANTEQUILLA	1	Butter
	MITAD MANTEQUILLA, MITAD MARGARINA	(	Half Butter, Half Margarine
	ACEITE VEGETAL	11	Vezetable Oil
		11	
•	POR LO GENERAL, ¿CON QUE TIPO DE GRASA O ACEITE COCINA?	11.	What kind of fat or oil do you usually cook with?
	O NO SE O "YO NO COCINO"		O Don't know or don't cook
	O MARGARINA PARA UNTAR		O Soft Margarine
	MARGARINA EN BARRA		O Stick Margarine
	O MANTEQUILLA		Butter
	MANTECA MANTECA DE GRASA O TOCINO		Lard Fatback or Bacon Fat
	PAM O "NO USO ACEITE"		Pam or "no oil"
	¿LLEVA ALGUNA DIETA ESPECIAL?	12.	Are you currently on a special diet?
	O NO		O No
	O SI, DIETA PARA BAJAR PESO		Yes, Weight Loss
	SI, DIETA POR PROBLEMA DE SALUD		Ves, for Medical Condition
	SI, DIETA VEGETARIANA		Ves, Vegetarian
	SI, DIETA BAJA EN SAL		Ves Low Cholesterol
	SI, DIETA PARA SUBIR DE PESO		Yes, Weight Gain
	NA PROPERTY	NEST	A 20NA
	PLEASE DO NOT W	RITE	NTHISAREA







## APPENDIX V

## INFORMED CONSENT FORM

Factors related to obesity in Latinos living in the Phoenix area PI: Vega-López

#### ARIZONA STATE UNIVERSITY INFORMED CONSENT FORM Factors related to obesity in Latinos living in the Phoenix area

Principal Investigator:

Co-Investigators

Gabriel Shaibi, PhD Barbara Ainsworth, PhD, MPH Keith Martin, PhD

Sonia Vega-López, PhD

	ASU IRB
sign_SM	Approved
Date109	09-110610

#### INTRODUCTION

The purposes of this form are to provide you (as a prospective research study participant) information that may affect your decision as to whether or not to participate in this research and to record the consent of those who agree to be involved in the study.

#### RESEARCHERS

Dr. Sonia Vega-López, Assistant Professor, College of Nursing and Health Innovation and her co-investigators have invited your participation in a research study.

#### STUDY PURPOSE

The purpose of the research is to identify diet, lifestyle, environmental and biological factors that affect the risk of developing diabetes and heart disease in Latino adults.

#### DESCRIPTION OF RESEARCH STUDY

If you decide to participate, then as a study participant you will join a study involving research of different diet, lifestyle, environmental and biological factors that affect whether people develop diabetes and heart disease. All study procedures described below will be done for research purposes only.

We will use a brief questionnaire over the phone to determine if you are eligible for the study. If you qualify for the study and you decide to participate, we will schedule an appointment for you to come to one of our three study sites at Arizona State University: the Health Sciences Center at the Polytechnic campus, the Clinical Research Unit at the Tempe campus, or ASU Health Center at the Downtown campus. You can choose the site that is more convenient for you. This appointment will be scheduled early in the morning so that you can come before you have breakfast.

Before the study visit, we will mail you a food frequency questionnaire. This is a questionnaire that asks questions about how often you eat several foods and takes about 45 minutes to complete. You can complete this questionnaire before you come to the study visit, or you can choose to fill it out while you are at the visit, especially if you want help completing it. If you fill out the questionnaire ahead of time, we will ask you to bring it with you to the study visit.

You will need to fast for 12 hours prior to your study visit. This means that you should not eat or drink anything but water starting 12 hours before your appointment.

At the time of your visit we will give you a chance to ask any additional questions you may have about this study. We will ask you to use the restroom to empty your bladder after which we will measure your height, weight and the measurement around your waist and hips. We will then ask you to sit down for a few minutes after which we will measure your blood pressure three times.

We will collect a blood sample to measure your blood cholesterol and triglycerides, sugar and other indicators of how cholesterol and sugar are transported and processed in your vessels and removed from the blood. We will also measure indicators of the type of fat, vitamins, and other compounds that you normally eat in the diet, and indicators of how your

10/15/09

Version 1

Page 1 of 4

## Factors related to obesity in Latinos living in the Phoenix area PI: Vega-López

antioxidant system responds. The total amount of blood that we will draw will be 40 ml (about 3 tablespoons). We will store some of the blood we collect (15 ml or about 1 tablespoon) for the future measurement of additional indicators of diabetes and heart disease risk, diet quality and response to oxidation. You will be given the option to decide whether you want us to store your blood for future use. If you agree to have your blood stored for future use, you give us permission to share this blood with other investigators without notifying you.

After we draw your blood, we will give you a light snack and will ask you to complete a survey. This survey will ask questions about yourself, your diet and physical activity habits, your neighborhood and about what you know about diabetes and heart disease. You can skip questions of this survey if you do not feel comfortable answering specific questions. It will take about 1 hour to complete the survey, and up to 1 hour and 45 minutes if you decide to complete the food frequency questionnaire at the time of the study visit.

If you say YES, then your participation will last for up to 2.5 hours at either the Health Sciences Center at the Polytechnic campus or the Clinical Research Unit at the Tempe campus. Approximately 100 people from the Phoenix area will be participating in this study.

#### RISKS

If you decide to participate in this study, then you may face a risk of bruising and discomfort, dizziness and fainting associated with blood drawing. However, this risk is small. The research team will minimize these risks by using trained personnel to draw your blood and by giving you a snack after the blood draw. You might experience mild discomfort during blood pressure testing as the cuff inflates. However, this risk is small, and the discomfort will go away after the cuff is deflated. There is also a small risk that you may not feel comfortable answering some of the questions in the survey, in which case you can decide not to answer. As with any research, there is some possibility that you may be subject to risks that have not yet been identified. There are no feasible alternative procedures available for this study. You can ask your doctor for a health exam.

#### BENEFITS

Although there may be no direct benefits to you, knowledge may be gained by your participation, which may benefit the health of others.

#### NEW INFORMATION

If the researchers find new information during the study that would reasonably change your decision about participating, then they will provide this information to you.

#### CONFIDENTIALITY

All information obtained in this study is strictly confidential unless disclosure is required by law. The results of this research study may be used in reports, presentations, and publications, but the researchers will not identify you. In order to maintain confidentiality of your records, Dr. Vega-López will code all the data and blood samples so that they do not contain any information that could identify you. All confidential information will be kept in a locked filing cabinet in Dr. Vega-López' office or in a password-protected computer, and will only be available to members of the research team. All samples and study materials will be destroyed 10 years after the study has been completed or upon your withdrawal from the study. Blood samples will be discarded and study-related documents will be shredded.

#### WITHDRAWAL PRIVILEGE

Taking part in this research study is totally your choice. It is ok for you to say no. Even if you say yes now, you are free to say no later. You can decide to stop taking part in this research study at any time for any reason.

10/15/09

Version 1

Page 2 of 4

Factors related to obesity in Latinos living in the Phoenix area PI: Vega-López

#### COSTS AND COMPENSATION

The researchers want your decision about participating in the study to be absolutely voluntary. Yet they recognize that your participation may pose some inconvenience due to the time needed to complete the research activities and because we will draw a blood sample from you. In order to compensate for your time and discomfort, you may receive a \$20 gift card to a local store at the end of the study visit even if the study procedures were not entirely completed.

There is no cost to you for participating in this research study. We will pay your parking costs using validation stamps or temporary parking passes.

#### COMPENSATION FOR ILLNESS AND INJURY

If you agree to participate in the study, then your consent does not waive any of your legal rights. However, no funds have been set aside to compensate you in the event of injury.

#### VOLUNTARY CONSENT

Any questions you have concerning the research study or your participation in the study, before or after your consent, will be answered by Dr. Vega-López. You can contact her at 6950 E. Williams Field Road, Mesa, Arizona, 85212, sonia.vega.lopez@asu.edu, or 480-727-5016.

If you have questions about your rights as a subject/participant in this research, or if you feel you have been placed at risk, you can contact the Chair of the Human Subjects Institutional Review Board, through the ASU Office of Research Integrity and Assurance, at 480-965-6788.

This form explains the nature, demands, benefits and any risk of the project. By signing this form you agree knowingly to assume any risks involved. Remember, your participation is voluntary. You may choose not to participate or to withdraw your consent and discontinue participation at any time without penalty or loss of benefit. In signing this consent form, you are not waiving any legal claims, rights, or remedies. A copy of this consent form will be given (offered) to you.

Your signature below indicates that you consent to participate in the above study.

Subject's Signature	Printed Name	Date
Other Signature (if appropriate)	Printed Name	Date
Your initials here indicate whe blood for future use.	ther you give us permission to :	store 15 ml (1 tablespoon) of you
I 🗌 DO consent to have my b	ood stored for future analyses.	

I DO NOT consent to have my blood stored for future analyses.

Subject's initials

10/15/09

Version 1

Page 3 of 4

Factors related to obesity in Latinos living in the Phoenix area PI: Vega-López

### INVESTIGATOR'S STATEMENT

"I certify that I have explained to the above individual the nature and purpose, the potential benefits and possible risks associated with participation in this research study, have answered any questions that have been raised, and have witnessed the above signature. These elements of Informed Consent conform to the Assurance given by Arizona State University to the Office for Human Research Protections to protect the rights of human subjects. I have provided (offered) the subject/participant a copy of this signed consent document."

Signature of Investigator\_\_\_\_\_

Date\_\_\_\_

10/15/09

Version 1

Page 4 of 4

Spanish

Factors related to obesity in Latinos living in the Phoenix area PI: Vega-López

#### ARIZONA STATE UNIVERSITY FORMA DE CONSENTIMIENTO INFORMADO Factores relacionados con la obesidad en Latinos que viven en el área de Phoenix

Investigador Principal: Sonia Vega-López, PhD Co-Investigadores: Gabriel Shaibi, PhD Barbara Ainsworth, PhD, MPH Keith Martin, PhD

### INTRODUCCION

Esta forma tiene como propósito: (1) darle (como participante potencial en un estudio de investigación) la información que pueda afectar su decisión de participar en este estudio, y (2) documentar su consentimiento si usted acepta participar en este estudio.

#### INVESTIGADORES

La Dra. Sonia Vega-López, del Colegio de Innovación en Enfermería y Salud de Arizona State University (ASU) y sus co-investigadores le están invitando a participar en este estudio.

#### PROPOSITO DEL ESTUDIO

El propósito de este estudio es identificar los factores de la dieta, estilo de vida, del medio ambiente, y biológicos que afectan el riesgo de desarrollar diabetes y enfermedades del corazón en adultos Latinos.

#### DESCRIPCION DEL ESTUDIO DE INVESTIGACION

Si decide tomar parte en este estudio, usted va a ser participante en un estudio de investigación que va a enfocarse a estudiar distintos factores de la dieta, estilo de vida, del medio ambiente, y biológicos que afectan si la gente desarrolla diabetes o enfermedades del corazón. Todos los procedimientos del estudio descritos abajo se llevarán a cabo únicamente con propósito de investigación.

Le vamos a hacer preguntas de un cuestionario breve por teléfono para saber si usted cumple con los requisitos para participar en el estudio. De ser así, y si usted decide participar, vamos a programar una cita para que usted venga a uno de nuestros tres sitios del estudio en Arizona State University: el Centro de Ciencias de la Salud en el campus Politécnico, la Unidad de Investigación Clínica en el campus de Tempe, o el Centro de Salud de ASU en el campus del centro (Downtown). Usted puede elegir el sitio que más le convenga. Esta cita se va a programar temprano en la mañana para que usted pueda venir antes de desayunar.

Antes de su visita al estudio, le vamos a enviar por correo un cuestionario de frecuencia de alimentos. Este es un cuestionario con preguntas acerca de la frecuencia con la que usted come varios alimentos, y toma alrededor de 45 minutos para completarlo. Usted puede completar este cuestionario antes de venir a su visita del estudio, o puede llenarlo durante su visita, especialmente si prefiere que alguien le ayude a llenarlo. Si usted llena el cuestionario antes de tiempo, le pedimos que lo traiga a la visita del estudio.

Usted tendrá que ayunar por 12 horas antes de su visita del estudio. Esto significa que usted no debe comer o beber nada, excepto agua, desde 12 horas antes de su cita.

Al inicio de su visita le vamos a dar la oportunidad de preguntar cualquier otra pregunta que pueda tener acerca del estudio. Le vamos a pedir que use el baño para vaciar su vejiga, y posteriormente vamos a medir su estatura, peso y la medida alrededor de su cintura y cadera. Después le vamos a pedir que se siente por varios minutos y posteriormente vamos a medir su presión tres veces.

10/27/09

Version 1

Page 1 of 4

Factors related to obesity in Latinos living in the Phoenix area PI: Vega-López

Spanish

Vamos a sacarle una muestra de sangre para medir su colesterol y triglicéridos, azúcar y otros indicadores de cómo se transportan y procesan el colesterol y el azúcar en sus venas, y como se remueven de la sangre. También vamos a medir indicadores del tipo de grasa, vitaminas y otros compuestos que normalmente come en su dieta, así como indicadores de cómo responde su sistema antioxidante. La cantidad de sangre que le vamos a sacar es de 40 ml (alrededor de 3 cucharadas). Vamos a guardar algo de la sangre que le saquemos (15 ml o alrededor de 1 cucharada) para medir en el futuro indicadores adicionales del riesgo de diabetes y enfermedades del corazón, de la calidad de la dieta y de la respuesta a la oxidación. Le vamos a dar la opción de decidir si quiere que guardemos su sangre para usarla en el futuro. Si accede a que guardemos su sangre para usarla en el futuro, usted nos da permiso de compartir esta sangre con otros investigadores sin notificarle.

Después de sacar su sangre, le vamos a dar algo ligero de comer (snack) y le vamos a pedir que llene una encuesta. Esta encuesta incluye preguntas acerca de usted, sus hábitos de la dieta y actividad física, su vecindario, y acerca de cuánto sabe de la diabetes y enfermedades del corazón. Usted puede saltarse preguntas de esta encuesta si no se siente a gusto contestando preguntas específicas. Completar esta encuesta le tomará alrededor de 1 hora, o hasta 1 hora con 45 minutos si decide completar el cuestionario de frecuencia de alimentos el día de la visita.

Si usted toma parte en el estudio, la cantidad de tiempo que tendrá que estar en el Centro de Ciencias de la Salud en el campus Politécnico, la Unidad de Investigación Clínica en el campus de Tempe, o el Centro de Salud de ASU en el campus del centro (Downtown) es de hasta 2.5 horas. Cerca de 100 personas del área de Phoenix van a participar en este estudio.

#### RIESGOS

Si decide participar en este estudio, existe el riesgo de que al sacar su sangre se formen moretones, sienta molestias, se sienta mareado, o que se desmaye. Sin embargo, este riesgo es bajo. El equipo de investigación hará lo posible para minimizar estos riesgos: (1) teniendo personal entrenado para sacar su sangre, y (2) dándole algo ligero de comer después de sacar su sangre. Usted puede sentir una sensación incómoda cuando le midamos su presión mientras se infla el aparato para medir la presión. Este riesgo es bajo y la incomodidad pasará cuando el aparato se desinfle. También hay un pequeño riesgo de que no se sienta a gusto contestando algunas de las preguntas de la encuesta, las cuales no tiene que contestar. Como que todavía no se han identificado. No existen alternativas viables para este estudio. Usted puede pedirle a su doctor que le haga un examen médico.

#### BENEFICIOS

Aunque puede que no haya beneficios directos para usted, con su participación podemos ganar información que pueda beneficiar a la salud de otras personas.

#### NUEVA INFORMACION

Si los investigadores encuentran nueva información durante el estudio que pudiera cambiar razonablemente su decisión acerca de participar, ellos le harán llegar esta información.

#### CONFIDENCIALIDAD

Toda la información que se obtenga en este estudio es estrictamente confidencial a menos que su divulgación se requiera por ley. Puede que los resultados de este estudio de investigación se usen en reportes, presentaciones y publicaciones, en los que los investigadores no revelarán la identidad de quienes participen. Para mantener la confidencialidad de su expediente, la Dra. Vega-López va a asignar un código a todos los datos y muestras de sangre para que no contengan ninguna información que pueda identificarlo a

10/27/09

Version 1

Page 2 of 4

usted. Toda la información confidencial se mantendrá en un archivero bajo llave en la oficina de la Dra. Vega-López o en una computadora protegida con contraseña, y sólo será disponible a los miembros del equipo de investigación. Todas las muestras y materiales del estudio se destruirán 10 años después de que se haya completado el estudio, o si usted decide salirse del estudio. Las muestras de sangre serán desechadas y todos los documentos relacionados con el estudio se triturarán.

#### PRIVILEGIO DE DARSE DE BAJA

Tomar parte en este estudio de investigación es absolutamente su decisión. Está bien decir no si usted no quiere participar. Incluso si ahora decide participar, usted tiene la libertad de decidir después que prefiere no participar. Usted puede decidir dejar de participar en este estudio de investigación en cualquier momento y por cualquier razón.

#### COSTOS Y COMPENSACION

Los investigadores quieren que su decisión acerca de participar en este estudio sea absolutamente voluntaria. Sin embargo, también reconocen que su participación pueda causarle inconvenientes debido al tiempo que se necesita para completar las actividades de investigación, y debido a que le vamos a sacar una muestra de sangre. Para compensarle por su tiempo y molestias, usted puede recibir una tarjeta de regalo para una tienda local con valor de \$20 al final de la visita del estudio, incluso si los procedimientos del estudio no se completaron.

Su participación en este estudio de investigación no le va a costar. Nosotros vamos a pagar su estacionamiento usando estampillas de validación o pases de estacionamiento temporales.

#### COMPENSACION POR ENFERMEDAD Y LESIONES

Si usted decide participar en el estudio, su consentimiento no le quita ninguno de sus derechos legales. Sin embargo, no existen fondos para compensarlo en caso de alguna lesión.

#### CONSENTIMIENTO VOLUNTARIO

La Dra. Vega-López puede contestarle cualquier pregunta que tenga con respecto al estudio de investigación o a su participación en este estudio, antes o después de dar su consentimiento a participar. Puede contactar a la Dra. Vega-López en 6950 E. Williams Field Road, Mesa, Arizona, 85212, <u>sonia.vega.lopez@asu.edu</u>, o al 480-727-5016.

Si tiene preguntas acerca de sus derechos como sujeto/participante en este estudio de investigación, o si siente que se le ha puesto en riesgo, usted puede contactar al Jefe del Comité Institucional de sujetos Humanos, por medio de la Oficina de Integridad y Aseguramiento de Investigaciones de ASU al 480-965-6788.

Esta forma explica la naturaleza, requisitos, beneficios y riesgos del proyecto. Al firmar esta forma usted está de acuerdo y con el conocimiento de que asume cualquier riesgo relacionado con su participación. Recuerde, su participación es voluntaria. Usted puede elegir no participar o retirar su consentimiento y dejar de participar en cualquier momento sin ninguna penalidad o pérdida de beneficios. Al firmar esta forma de consentimiento usted está renunciando a ningún derecho, reclamo o remedio legal. Se le dará (ofrecerá) una copia de esta forma de consentimiento.

10/27/09

Version 1

Page 3 of 4

#### Spanish

Factors related to obesity in Latinos living in the Phoenix area PI: Vega-López

Su firma indica que usted da su consentimiento para participar en el estudio descrito anteriormente.

Firma del Participante	Nombre Impreso	Fecha
Otra Firma (si es adecuado)	Nombre Impreso	Fecha

Sus iniciales a continuación indican si usted nos da permiso de guardar 15 ml (1 cucharada) de su sangre para uso en el futuro.

Yo 🗌 ESTOY de acuerdo en que se guarde mi sangre para hacer análisis en el futuro.

Yo 🗌 NO ESTOY de acuerdo en que se guarde mi sangre para hacer análisis en el futuro.

Iniciales del Participante

#### DECLARACION DEL INVESTIGADOR

Spanish

"Certifico que he explicado a la persona cuyo nombre aparece arriba la naturaleza, el propósito, los beneficios potenciales y posibles riesgos asociados con su participación en este estudio de investigación, he contestado a las preguntas que surgieron y he sido testigo de la firma de arriba. Estos elementos del Consentimiento Informado se conforman con el Aseguramiento que Arizona State University le da a la Oficina de Protección de Sujetos Humanos para proteger los derechos de sujetos humanos. He dado (ofrecido) al participante una copia firmada de esta forma de consentimiento."

Firma del Investigador\_\_\_\_\_ Fecha\_\_\_\_ Fecha\_\_\_

10/27/09

Version 1

Page 4 of 4

APPENDIX VI

MAIN SURVEY

INTERVIEW INFORMATION INFORMACION DE LA ENTREVISTA
Study ID Date
1) Interviewer name / Nombre de Entrevistador.
2) Language of the interview / Idioma en el que se lleva a cabo la entrevista
□₁ English / <i>Inglés</i> □₂ Spanish / <i>Español</i> □₃ Both
SOCIODEMOGRAPHIC CHARACTERISTICS/ PERSONAL INFORMATION CARACTERISTICAS SOCIODEMOGRAFICAS/ INFORMACION PERSONAL
3) Gender/ Sexo: 1 Male / Hombre 2 Female / Mujer
4) How old are you?/ Edad
5) Do you identify yourself as any of the following? / ¿Cómo se identifica usted a sí mismo?
(interviewer, piease read the options)
<ul> <li>I Mexican / Mexicano</li> <li>American / Americano</li> <li>Mexican-American / Mexicano-Americano</li> <li>Hispanic or Latino/ Hispano o Latino</li> <li>Hispanic or Latino/ Hispano o Latino</li> <li>Tro Other (please specify)/ Otro, por favor especifique:</li> <li>Refuse / Rehúsa</li> </ul>
6) Do you speak? / Usted habla (Interviewer, please read options)
<ul> <li>☐1 English only / Inglés sólamente</li> <li>2 English and Spanish / Inglés y español</li> <li>3 Spanish only / Español sólamente</li> <li>77 Other (please specify)/ Otro, por favor especifique:</li> <li>99 Refused / Rehúsa</li> </ul>
7) What language do you speak in your home most of the time? ¿Que idioma habla en su casa la mayoría del tiempo?
<ul> <li>☐1 English only / Inglés sólamente</li> <li>2 English and Spanish / Inglés y español</li> <li>3 Spanish only / Español sólamente</li> <li>77 Other (please specify)/ Otro, por favor especifique:</li> <li>99 Refused / Rehúsa</li> </ul>
12) Are you currently working? / ¿Está trabajando actualmente?
$\square_1$ Yes / Sí $\square_2$ No, <b>go to question # 14</b>

13) Which of the following best describes your current employment status? ¿Cuál de los siguientes describe mejor su estado actual de empleo?

Working full-time, 35 hours per week or more / Trabaja de tiempo completo, 35 horas o más por semana 2 Working part-time, less than 35 hours per week / Trabaja de medio tiempo, menos de 35 horas por semana

### Skip to question 15

14) Which of the following best describes your current employment status? ¿Cuál de los siguientes describe mejor su estado actual de empleo?

### (Interviewer please read all options and check only one box.)

- $\Box_3$  Unemployed or laid off <u>and</u> looking for work Desempleado o suspendido temporalmente por falta de trabajo y buscando trabaio
- , Unemployed and not looking for work / Desempleado y no esta buscando trabajo 5 Homemaker / Ama(o) de Casa

6 In school / En la escuela

- 17 Retired / Retirado
- Disabled, not able to work / Incapacitado, no puede trabajar

□<sub>9</sub> Other? (Please specify) /¿Algún otro? (Por favor especifique):\_

15) Including money from all salaries/work, government assistance and (if applicable) unemployment, what is the total amount of money your household receives PER MONTH?

¿Cuánto es el ingreso total de dinero que su CASA recibe POR MES, incluyendo el dinero de todos los salarios de trabajo, asistencia del gobierno y el desempleo (si aplica)?

(Interviewer, if participant is not sure read all the options; make sure answer includes food stamps, alimony, and foster care)

□₁ \$0-1000  $\Box_2$  \$1001-2000 3 \$2001-3000 **\_**₄ \$3001-4000 □<sub>5</sub> >\$4000 Other amount, (specify) / Otra cantidad, (especifique) 388 DK / No sabe 99 Refused / Rehúsa

16) Last grade you completed in school / Grado final que se ha cumplido en la escuela:

 $\square_1$  Less than 6<sup>th</sup> grade / *Menos de 6<sup>o</sup> grado*  $\Box_2$  Completed elementary school (6<sup>th</sup> grade) / *Escuela primaria completa (6º grado)*  $\boxed{\Box}_{3}^{1}$  Completed middle school (9<sup>th</sup> grade) / *Escuela secundaria completa (9º grado)*  $\boxed{12^{\circ}}_{4}$  Completed high school (12<sup>th</sup> grade) / Escuela preparatoria completa (12° grado)  $\Box_5$  Some college / Algo de universidad 6 College graduate or higher / Graduado de la universidad o posgrado Other, (specify) / Otra, (especifique) \_\_\_\_\_ □<sub>88</sub> DK / No sabe ge Refused / Rehúsa

### MIGRATION PATTERNS AND ACCULTURATION PATRONES DE IMIGRACION Y ACULTURACION

25) Where were you born? City:	Country:	99
¿En dónde nació? (Ciudad, estad	do, país)	
26) For how long have you been living in ¿Cuánto tiempo lleva viviendo er	the United States?months los Estados Unidos?	years
27) Which of the following best describes ¿Cuál de los siguientes lo describe mo (Mark only one answer)	you? əjor?	
□ <sub>1</sub> 1st generation = You were born in $1^a$ generación = Nació en México □ <sub>2</sub> 2nd generation = You were born country. $2^a$ generación = Nació en los Est	n Mexico or other country outside th o u otro país fuera de los Estados U in USA; either parents born in Mexi	ne U.S. Inidos. ico or other
México u otro país.	n USA, both parents born in USA a	
grandparents born in Mexico or o	ther country.	nu an
3ª generación = Nació en los Est Estados Unidos y todos sus abue	ados Unidos; sus dos padres nacie elos nacieron en México u otro país	ron en los
4 4th generation = You and your page grandparents born in Mexico or of 4 <sup>a</sup> generación = Usted y sus pada menos uno de sus abuelos nación nacieron en los Estados Unidos.	arents born in USA; at least one of y ther country with reminder born in t res nacieron en los Estados Unidos en México u otro país. Sus otros a	your the USA. s; por lo abuelos
☐ <sub>5</sub> 5th generation = You and your pa born in the USA	arents born in the USA and all grand	dparents
5ª generación = Usted y sus pada abuelos nacieron en los Estados	res nacieron en los Estados Unidos Unidos.	s y todos sus
□ <sub>88</sub> DK / <i>No sabe</i> □ log Refused / <i>Rehúsa</i>		

The following questions are about how much you identify with the Hispanic culture and the American culture. Please indicate how often you do each of the following. *Las siguientes preguntas son acerca de cuánto se identifica con la cultura hispana y la cultura americana. Por favor indique con qué frecuencia hace cada una de las siguientes.* 

	Not at all <i>Nunca</i>	Very little or not very often Casi nunca o pocas veces	Moderately <i>Moderadamente</i>	Much or very often Muchas veces o frecuentemente	Extremely often or almost always Muchas veces o casi siempre
28) I speak Spanish Hablo español	1	2	3	4	5
29) I speak English Hablo inglés	1	2	3	4	5
30) I enjoy speaking Spanish	1	2	3	4	5
31) I associate with Anglos	1	2	3	4	5
32) I associate with Mexicans					
and/or Mexican Americans Me asocio con Mexicanos o con Mexicano Americanos	1	2	3	4	5
33) I enjoy listening to Spanish language music Me gusta la música Mexicana (música en idioma español)	1	2	3	4	5
34) I enjoy listening to English language music Me gusta la música en idioma inglés	1	2	3	4	5
35) I enjoy Spanish language TV					
Me gusta ver programas en la televisión que sean en español	1	2	3	4	5
36) I enjoy English language TV					_
Me gusta ver programas en la televisión que sean en inglés	1	2	3	4	5
37) I enjoy English language movies	1	2	3	4	5
Me gusta ver películas en inglés			-		-
38) I enjoy Spanish language movies Me gusta ver películas en español	1	2	3	4	5
39) I enjoy reading (e.g. books) in Spanish Me gusta leer (libros) en	1	2	3	4	5
español 40) I enjoy reading (e.g.					
books) in English Me gusta leer (libros) en inglés	1	2	3	4	5
41) I write (e.g. letters) in Spanish <i>Escribo (cartas) en español</i>	1	2	3	4	5
42) I write (e.g. letters) in English <i>Escribo (cartas) en inglés</i>	1	2	3	4	5
43) My thinking is done in the English language Mis pensamientos ocurren en el idioma inglés	1	2	3	4	5
44) My thinking is done in the Spanish language Mis pensamientos ocurren en el idioma español	1	2	3	4	5
45) My contact with Mexico has been <i>Mi contacto con México ha</i> <i>sido</i>	1	2	3	4	5
46) My contact with USA has been Mi contacto con los Estados Unidos ha sido	1	2	3	4	5
47) My father identifies/identified himself as 'Mexicano' <i>Mi padre se identifica (o se identificaba) como Mexicano</i>	1	2	3	4	5

	Not at all <i>Nunca</i>	Very little or not very often Casi nunca o pocas veces	Moderately Moderadamente	Much or very often Muchas veces o frecuentemente	Extremely often or almost always Muchas veces o casi siempre
48) My mother identifies/identified herself as 'Mexicana' <i>Mi madre se identifica (o se identificaba) como Mexicana</i>	1	2	3	4	5
49) My friends, while I was growing up, were of Mexican origin Mis amigos(as) de mi niñez eran de origen mexicano	1	2	3	4	5
50) My friends, while I was growing up, were of Anglo origin Mis amigos(as) de mi niñez eran de origen Anglo Americano	1	2	3	4	5
51) My family cooks Mexican foods <i>Mi familia cocina comida</i> <i>mexicana</i>	1	2	3	4	5
52) My friends now are of Anglo origin Mis amigos recientes son Anglo Americanos	1	2	3	4	5
53) My friends now are of Mexican origin Mis amigos recientes son Mexicanos	1	2	3	4	5
54) I like to identify myself as an Anglo American <i>Me gusta identificarme como</i> <i>Anglo Americano(a)</i>	1	2	3	4	5
55) I like to identify myself as Mexican American Me gusta identificarme como Mexicano(a) Americano(a) (Estadounidense de origen mexicano)	1	2	3	4	5
56) I like to identify myself as Mexican Me gusta identificarme como Mexicano(a)	1	2	3	4	5
57) I like to identify myself as an American Me gusta identificarme como Americano(a)	1	2	3	4	5

### END OF INTERVIEW

Interviewer: Read...

Thank you so much for your time. / Muchas gracias por su tiempo

## APPENDIX VII

## ANTHROPOMETRICS DOCUMENTATION FORM

### Anthropometrics Documentation Form

Study ID		Date
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	Measurement 1	Measurement 2	Measurement 3
HEIGHT			
(cm)			
WEIGHT			
(kg)			
WAIST			
CIRCUMFERENCE			
(cm)			
HIP			
CIRCUMFERENCE			
(cm)			
BLOOD			
PRESSURE			
(mm Hg)			
	Calavilated		
	- Calculated		

## Initials of person taking measurements \_\_\_\_\_

If procedures were NOT completed, indicate rescheduling date and time: \_\_\_\_\_

## APPENDIX VIII

BLOOD SAMPLE DOCUMENTATION FORM

### **Blood Draw Documentation Form**

Study ID:	Date:	Time:
Olddy ID	Dute	

Phlebotomist:

Before you draw the blood, please ask the patient the following questions to assess how well the participant complied with the guidelines to prepare him/her for the blood draw. If the participant did not comply with the guidelines, reschedule the blood draw. Remind them of each of the guidelines and encourage him/her to follow the guidelines.

1. At what time did you last eat or drink something (excluding water)? \_\_\_\_\_ ¿A qué hora consumió el último alimento o bebida (excepto agua)?

# If he/she ate or drank something less than 8 hours ago, reschedule the blood draw.

2.	Have you had coffee in the past 12 hours? ¿Tomó café en las últimas 12 horas?	🗌 Yes	🗌 No
3.	Have you exercised in the past 12 hours? ¿Hizo ejercicio en las últimas 12 horas?	Yes	🗌 No

Please confirm that the following tubes of blood were collected:

<ul> <li>One 2 ml gray top</li> <li>One 6 ml lavender top</li> <li>One 10 ml lavender top</li> <li>Two 10 ml red top</li> </ul>			
Blood draw successfully completed?	🗌 Yes	🗌 No	
Comments:			

If blood was NOT collected, indicate rescheduling date and time:

Proceed to Survey Completion.

## APPENDIX IX

## ACCULTURATION RATING SCALE

The following is the Acculturation scoring instructions as explained by Cuellar et al. [182].

ARSMA-II employs a bilingual format with both language versions (English and Spanish) on the same page. Scale I of ARSMA-II is a 30-item self-rating scale composed of an Anglo Orientation Subscale (AOS) of 13 items (Items 2, 4, 7, 9, 10, 13, 15, 16, 19, 23, 25, 27, and 30) and a Mexican Orientation Subscale (MOS) composed of 17 items (Items 1, 3, 5, 6, 8, 11, 12, 14, 17, 18, 20, 21, 22, 24, 26, 28, and 29). The sum of the AOS scale is divided by 13 to obtain a mean score for that subscale. The sum of the MOS is divided by 17 to obtain a mean for that subscale. The MOS mean is subtracted from the AOS mean to obtain a linear acculturation score that represents an individual's score along a continuum from very Mexican oriented to very Anglo oriented. The acculturation score can be used to obtain an acculturation level for the subject by employing the suggested cutting scores shown in Table 1 below. These cutting scores were selected based on standard deviation unites from the mean of the combined sample of 379 subjects representing five generations.

Individuals scoring greater than -1/2 standard deviation below the mean on both the AOS and the MOS scales are classified as high integrated biculturals. Similarly, using fractional deviations from the AOS and MOS means, cutting scores were set for defining low integrated biculturals as those individuals scoring between -1.5 standard deviations below the mean to -.5 standard deviations below the mean on both the MOS and AOS scales.

Acculturation Levels	Description	ARSMA-II Acculturation Score*
Level I	Very Mexican oriented	< -1.33
Level II	Mexican oriented to approximately balanced bicultural	$\geq$ -1.33 and $\leq$ 07
Level III	Slightly Anglo oriented bicultural	>07 and < 1.19
Level IV	Strongly Anglo oriented	$\geq$ 1.19 and < 2.45
Level V	Very assimilated; Anglicized	> 2.45

 Table 16. Cutting Scores for Determining Acculturation Level Using

 ARSMA-II

\*Raw score means were used to calculate the Acculturation Score. The choices selected for each item are added and divided by the number of items on the MOS and AOS scales separately to obtain the raw score mean for each scale. These means were used in the formula: Acculturation Score = AOS (mean) – MOS (mean).