IMPACT OF DIETARY MACRONUTRIENTS ON OBESITY IN DIFFERENT US ADULT ETHNIC SUBPOPULATIONS

By

Kwasi Yeboah-Afihene

A Dissertation Submitted in Partial Fulfillment of the Requirements for the Degree of Doctor of Philosophy in Biomedical Informatics

Department of Health Informatics

School of Health Professions

The State University of New Jersey, Rutgers

August 2016

Final Dissertation Approval Form

IMPACT OF DIETARY MACRONUTRIENTS ON OBESITY IN DIFFERENT US ADULT ETHNIC SUBPOPULATIONS

By

Kwasi Yeboah-Afihene

Dr. Shankar Srinivasan, Dissertation Committee Member/Chairman (Date)

Dr. Fredrick Coffman, Dissertation Committee Member (Date)

Dr. Syed Haque, Dissertation Committee Member (Date)

TABLE OF CONTENTS

ABSTRACT vi
ACKNOWLEDGEMENT viii
LIST OF TABLES ix
LIST OF FIGURES xi
CHAPTER 1 1
INTRODUCTION 1
1.1 Statement of Problem 1
1.2 Background of Problem 3
1.2.1 Trend in Obesity Incident rate 3
1.2.2 Obesity and Mortality Rate 7
1.2.3 Obesity and Growing Related Cost to the USA 8
1.2.4 Obesity and Poor Quality of Life 13
1.2.5 Obesity and what we eat 13
1.3 Goals and Objectives 14
1.4 Significance of Problem 15
1.4.1 Obesity's Impact on Families 15
1.4.2 Obesity's Impact on Healthcare Systems 16
1.5 Hypothesis and Related Questions 18
CHAPTER 2 20
LITERATURE REVIEW 20
2.1 Literature Source and Search Strings 20
2.2 General Overview of Previous Literature 20
2.3 Understanding Obesity's Impact 21
2.3.1 Non Communicable Diseases [NCD] 21
2.3.2 Body Mass Index 22
2.3.3 Common Obesity Comorbidities 23
2.3.4 Common Obesity Related Procedures 29
2.3.5 Obesity Prevention and Education 32
2.4 Obesity: Different Schools of Thought 35
2.4.1 Industrialization of Food Industry and Obesity 35
2.4.2 Behavioral Factors and Obesity 37
2.4.3 Environmental Contributions to Obesity 38
2.4.4 Socioeconomic Status, Culture and Obesity 39
2.5 Obesity and Dietary Macronutrients 39
2.5.1 Obesity and Sugar Consumption 39

2.5.2 Obesity and Carbohydrate Consumption....41
2.5.3 Obesity and Fiber Consumption....42
2.5.4 Obesity and Protein Consumption....43
2.5.5 Obesity and Fat Consumption....45

CHAPTER 3....46

RESEARCH METHODOLOGY....46

3.1 Research Overview....46
3.1.1 Thoretical Framework....47
3.1.2 Research Design....48
3.1.3 Research Questions and Associated Hypothesis....50

3.2 Data Sources....51
3.2.1 Study Period and Study Group Inclusion/Exclusion Criteria....53

3.3 NHANES Dataset and Elements....53
3.3.1 Data Cleaning and Data Processing....54
3.3.2 Demographic Data....55
3.3.3 Dietary Data....56
3.3.4 Examination Data....56
3.3.5 Questionnaire Data....57
3.3.6 Variables and Coding....57
3.3.7 Exposures and Outcomes....58

3.4 Data Analysis Methods....59
3.4.1 Data Analysis Plan....61

CHAPTER 4....64

RESULTS....64

4.1 Introduction....64

4.2 Results: Descriptive Statistics....64

4.3 Achievement of Objectives of the Study....77
4.3.1 Objectives of Hypothesis 1....77
4.3.2 Objectives of Hypothesis 2....80
4.3.3 Objectives of Hypothesis 3....84
4.3.4 Objectives of Hypothesis 4....87
4.3.5 Supplementary Categorical Analysis....90
4.3.6 Summary....97

CHAPTER 5....99

DISCUSSIONS, CONCLUSIONS AND STUDY LIMITATIONS....99

5.1 Discussions and Conclusions....99
5.1.1 Question 1....101
5.1.2 Question 2....102
5.1.3 Question 3....105
5.1.4 Question 4....106

5.2 Study Limitations 106
5.3 Suggested Future Research 107
5.4 Summary 108
REFERENCES 111

ABSTRACT

IMPACT OF DIETARY MACRONUTRIENTS ON OBESITY IN DIFFERENT US ADULT ETHNIC SUBPOPULATIONS

By

Kwasi Yeboah-Afihene

BACKGROUND: How we manage our energy intake and usage is very crucial to the obesity phenomenon. The body gets its energy supply from the macronutrients we consume, which are primarily, proteins, carbohydrates, fats, fibers and sugars. Their effect of on various US subpopulations so far as obesity and weight management is concerned has not been adequately explored in literature.

GOALS AND OBJECTIVES: Our main objective is to assist physicians in advising their patients on functional diets with relatively appropriate amounts of specific macronutrients for proper weight management.

METHOD: The study followed a retrospective, quantitative and correlational design which sought to examine the relationships between variables. Two NHANES dataset cycles were analyzed, NHANES 2009-2010 and 2011-2012. The youth and children below age 19 were excluded with pregnant women as well. The hypotheses were tested with hierarchical logistic regression models and Wald's test.

RESULTS: Different macronutrient compositions appeared to be associated with different levels of risk for obesity, Wald = 8.081, $p < 0.01$. Amongst the macronutrients fiber appeared to be associated with lowest obesity risk, with β = -0.012, *OR = 0.988, OR 95% CI (0.978, 0.998),* $p < 0.05$. Sugar also had an inverse or

negative relationship with β = -0.002, *OR=0.998, OR 95% CI (0.996, 0.999), p* < 0.05. Fat, carbohydrate and protein were positively associated to obesity however their level of significance or probabilities of occurrences were not within 95% confidence level, their beta values were respectively; β = 0.002, β = 0.001, β = 0.001. It was also observed that none of the macronutrients affected the various ethnic subpopulations differently. [Protein Wald = 2.32, p = 0.12, Carbohydrate Wald = 1.93, p = 0.17, Fiber Wald = 2.18, p = 0.13, Fat Wald =1.01, p = 0.45, and Sugar Wald = 2.86, p = 0.08].

CONCLUSION: High fiber diet has numerous health benefits, which include weight management benefits. Also sugar can be part of a functional diet, though has to be used in moderation due to its health risks other than obesity. The difference in the prevalence of obesity amongst various US ethnic subpopulations may be due to factors other than any anatomical or physiological differences.

ACKNOWLEDGEMENTS

Many thanks to my faculty adviser and dissertation committee chairman, Dr. Shankar Srinivasan. Dr. Shankar has been a friend, a mentor and an adviser to me, with his generosity of advice spanning beyond academics. It has been a long and winding road, and sometimes I get discouraged and frustrated. He always urges me on and encourages me to forge ahead and never give up. I do really like to express my sincerest gratitude to him for taking a personal and professional interest in my academic career.

I would also like to thank Dr. Syed Haque, Dr. Fredric Coffman and Dr. Masayuki Shabata for their numerous contributions, advice and continuous guidance. For your passionate dedication to your work which is so exemplary, and your personal efforts in getting me to this point in the program, I deeply appreciate and thank you.

To my deceased Dad, I would say thanks again, for your love and confidence in me. I deeply appreciate all the sacrifices you made to get me this far. Your most thoughtful words of wisdom have often been a compass in my journey of life. To my dear Mom, words cannot express my appreciation for your love and dedication toward my upbringing to this date. Thank you and God bless. To my Children Jeremiah and Racquelle, I would say thanks a million for your patience and understanding, and also your continuous prayers for me. Also, to all my siblings and Dr. and Mrs. Ata Bonna and Family, I would like to say a big thank you for all your love and support. Finally to all others who have been helpful in this journey, I would say thank you and many blessings.

LIST OF TABLES

Table 1: Top 10 causes of death by race in the USA 7

Table 2: Top 20 most expensive conditions treated in U.S. hospitals, all payers, 2011 9

Table 3: Variables and Recommended Coding..... 57

Table 4: Collinearity Test Results..... 60

Table 5: Summary Statistics NHANES 2009 – 2012 for Obese and Non Obese Adults 66

Table 6: Summary Statistics NHANES 2009 – 2012 Macronutrients Consumption by Race..... 72

Table 7: Summary Statistics NHANES 2009 – 2012 Frequency Distribution and Percent..... 74

Table 8: Summary Statistics NHANES 2009 – 2012 Row Percent [Obese-Non Obese] 77

Table 9: Hypothesis 1 Model Results 79

Table 10: Hypothesis 2 Model Results 81

Table 11: Hypothesis 2 Model Results 82

Table 12: Hypothesis 2 Model Results 83

Table 13: Hypothesis 3 Model Results 85

Table 14: Hypothesis 3 Model Results 86

Table 15: Hypothesis 4 Model Results 88

Table 16: Hypothesis 4 Model Results 89

Table 17: Percent Obese and Non Obese for Intake of Macronutrients by Quartile ... 91

Table 18: Total Protein Intake with Control Variables 93

Table 19: Total Carbohydrate Intake with Control Variables 94

Table 20: Total Fiber Intake with Control Variables 95

Table 21: Total Fat Intake with Control Variables 96

Table 22: Total Sugar Intake with Control Variables 97

LIST OF FIGURES

Figure 1: Obesity Trends amongst U.S Adults 4

Figure 2: Prevalence of Self-Reported Obesity among Non-Hispanic Black Adults by State 4

Figure 3: Prevalence of Self-Reported Obesity among Hispanic Adults, by State 5

Figure 4: Prevalence of Self-Reported Obesity amongst Hispanic Adults by State, BRFSS, 2011-1013 6

Figure 5: Prevalence of Self-Reported Obesity among Non-Hispanic White Adults, By State 6

Figure 6: Estimated Prevalence of Adults Diagnosed with Diabetes in the USA per State 25

Figure 7 Diagram of CDC Medical Examination Centers (MEC) Source: CDC 52

Figure 8: Collinearity Test Results 60

Figure 9: Logit Model 61

Figure 10: Daily Mean Energy Consumption (Kcal) 67

Figure 11: Daily Mean Protein Consumption (g) 68

Figure 12: Daily Mean Sugar Consumption (g) 68

Figure 13: Daily Mean Carbohydrate Consumption (g) 69

Figure 14: Daily Mean Fiber Consumption (g) 69

Figure 15: Daily Mean Fat Consumption (g) 70

Figure 16: Daily Mean Monounsaturated Fat Consumption (g) 70

Figure 17: Daily Mean Saturated Fat Consumption (g) 71

Figure 18: Daily Mean Cholesterol Consumption (g) 71

Figure 19: Obesity by Quartiles of Macronutrients Intake 92

CHAPTER 1

INTRODUCTION

1.1 Statement of Problem

The obesity phenomena started becoming an issue of critical importance in the last three decades [1]. The most notable change at its onset was the industrialization on our food systems [2]. This new paradigm of food processing and consumption patterns coupled with its effect on the body's physiological and other subsystems increased the rate of obesity systemically in the USA and globally [2]. It is believed that high dense energy foods including more refined carbohydrates and sugars were among the main culprits of the obesity phenomena [3].

There is also a myriad of socioeconomic and cultural elements which have in their own ways influenced life styles and others which have in turn exacerbated the incidence of obesity [4-7]. Some of these have influenced life style choices such as poor diet, sedentary life styles; lack of physical exercise and a lot more which influence our body's metabolism and other biological processes that eventually worsen the obesity and comorbidity outcomes [8-11].

Psychosocial factors or determinants of health such as poor self-esteem, stress and loneliness also create inertias that inherently influence people to indulge in counterproductive life styles which also affect obesity negatively [12]. These factors hamper the progress patients make in improving their health by reducing their weight.

Biological factors are also very prominent in the obesity phenomena. Factors like Genetics and Metabolic rate make some individuals naturally more susceptible to

the problem than others[13]. This increases the difficulty of successfully containing the problem by people with such propensities[13]. Although there are genetic predispositions that increase the risk unevenly, for many people, other factors when well controlled can prevent the onset of obesity, in spite of their natural predispositions[13].

Among the myriad of factors that affect obesity, the most prominent is how we balance our energy intake and usage [14]. There have been a lot of theories and a combination of food types that is believed to create the best mix of dietary combination that will help people lose weight and maintain the best body size with a normal body mass index [BMI] and good health [13]. The industry that sprang out of this need has grown to a multibillion dollar industry, with diverse products and services. In spite of all the effort and money being invested by businesses and spent by consumers, the response by far does not commensurate with all the efforts [13,15].

Products like low carbohydrate high protein diets, diets high in fiber low in saturated fats and so on has been some of the buzz words in the market place [15-17]. Often time, the combination keep shifting leaving consumers very confused and frustrated. Weight management and healthy eating standards keep shifting over the years [17]. The current USDA dietary guidelines 2015-2020, have advice providing some suggestions for diverse healthy eating patterns that can help reduce the risk of obesity and other popular chronic conditions and diseases that affect the general US population. The main theme in all of these diets is that they are higher in vegetables, fruits, whole grains, low-fat or nonfat dairy and or soy beverages and provide a variety of protein foods such as seafood, lean meats, and poultry. They are however limited in saturated fats, trans-fats, added sugars, and sodium. The right combination of

macronutrients in a functional diet, and their effect on various US subpopulations so far as obesity and weight management are concerned have not been adequately explored in the literature and we aim at making a contribution to shed more light on this area of dietary and nutritional needs for weight management and healthy living.

1.2 Background of Problem

1.2.1Trend in Obesity Incident rate

The prevalence of Obesity has risen since the early to mid-1980s [18]. This trend has been worsening since its onset, creating numerous health issues and burdens for rich and poor nations [18]. The following are maps generated by US Center for disease control [CDC], highlighting snap shots of OBESITY trends and prevalence per ethnic group among adults in the USA:

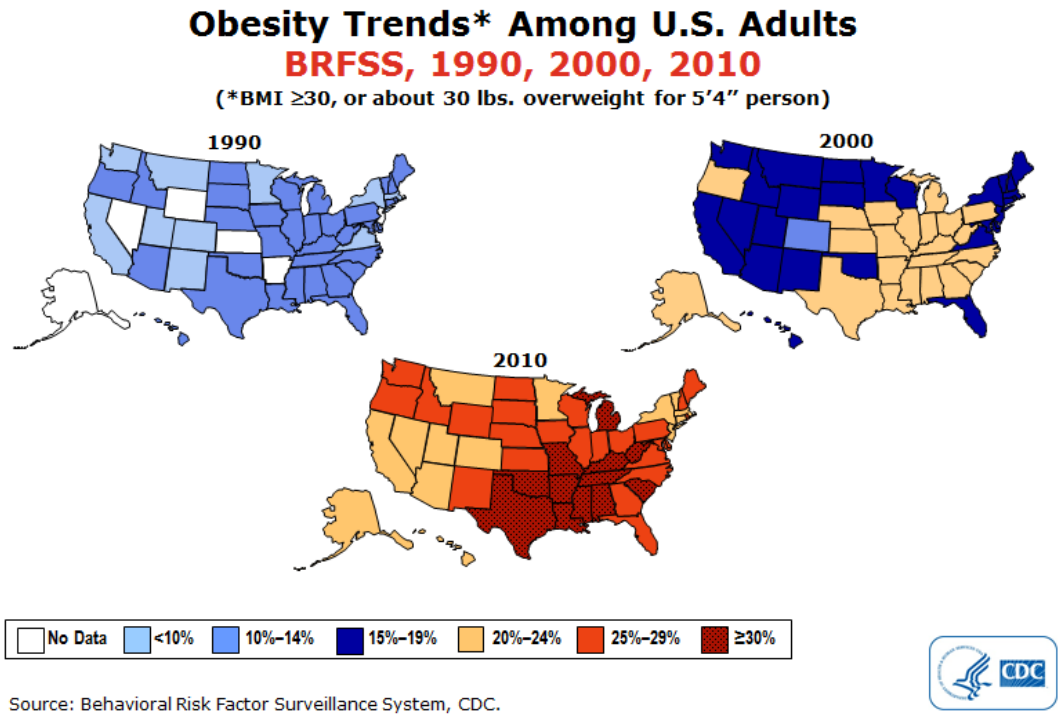


Figure 1: Obesity Trends amongst U.S Adults

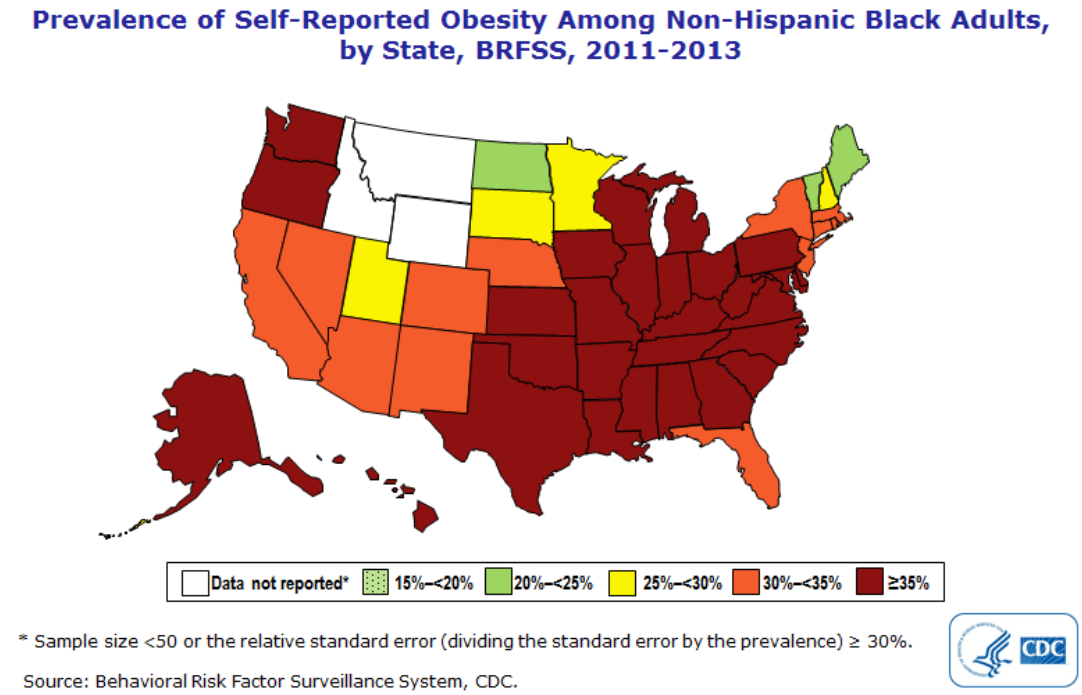


Figure 2: Prevalence of Self-Reported Obesity among Non-Hispanic Black Adults by State

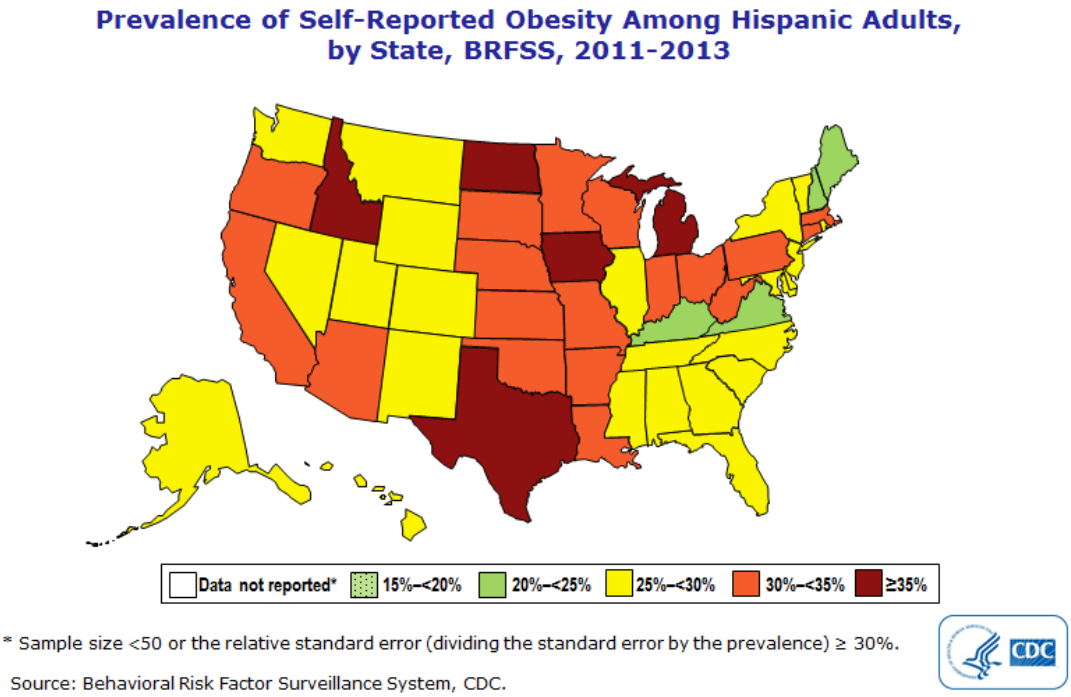


Figure 3: Prevalence of Self-Reported Obesity among Hispanic Adults, by State

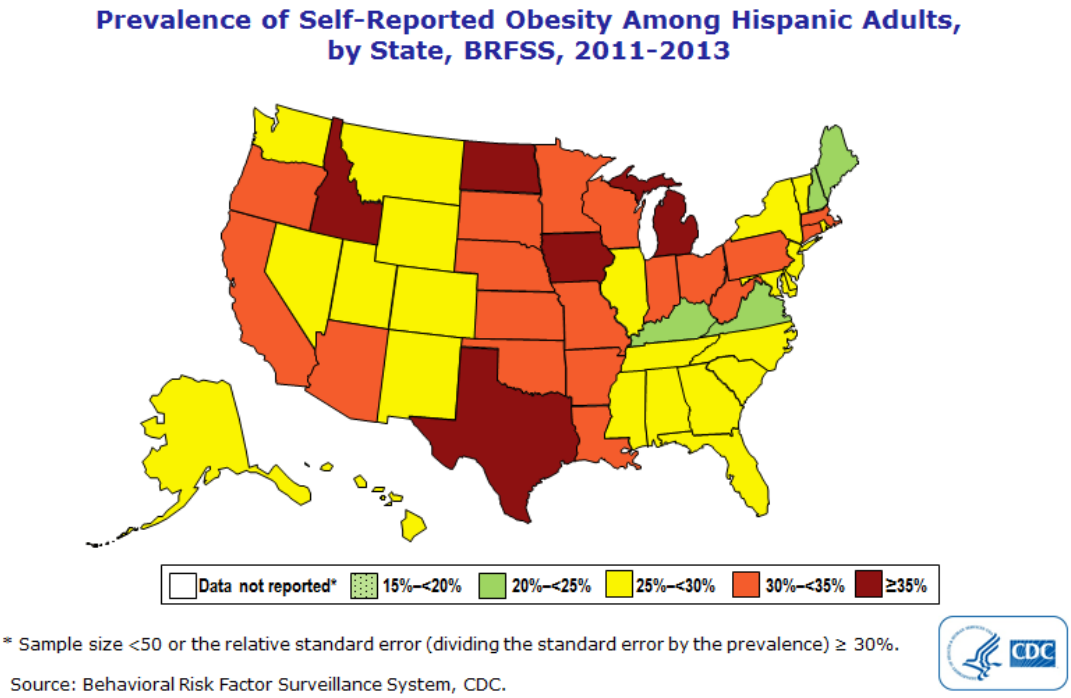


Figure 4: Prevalence of Self-Reported Obesity amongst Hispanic Adults by State, BRFSS, 2011-1013

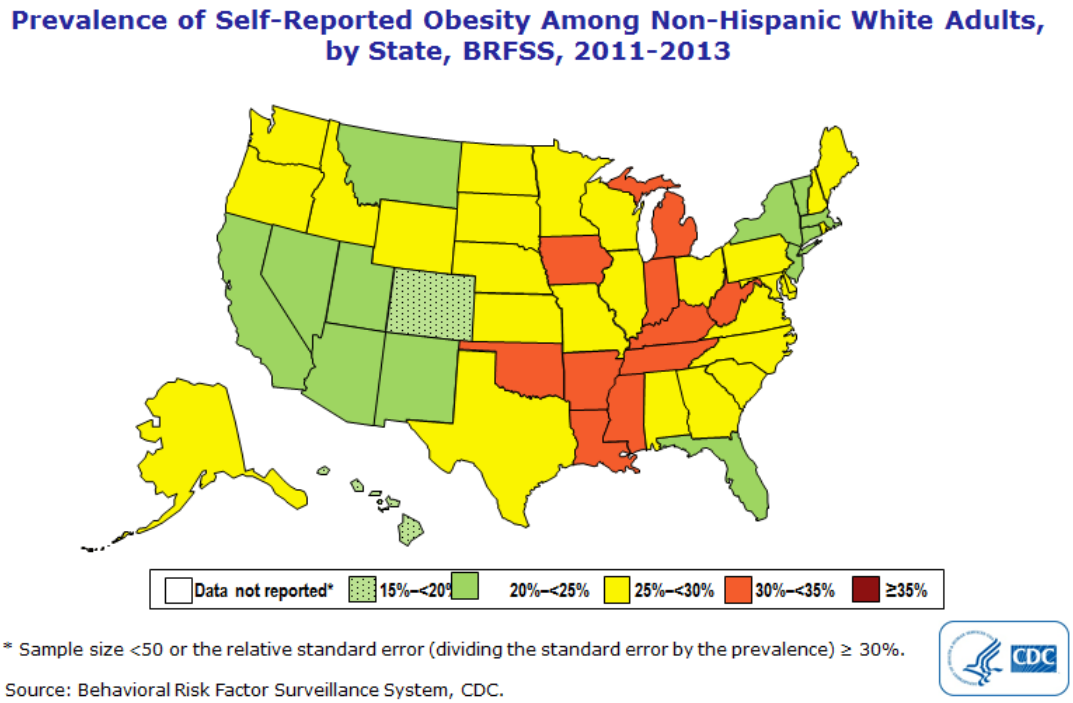


Figure 5: Prevalence of Self-Reported Obesity among Non-Hispanic White Adults, By State

Obesity's prevalence has been rapidly rising in the USA and other countries over the last thirty years with lots of health burdens that need to be studied and addressed [1,19]. There have been statements by some researchers indicating that over 60 percent of all US adults are overweight or obese. And also the rate is rapidly increasing for children and adolescence [20].

There are several diseases that are triggered by obesity. Some of these are heart diseases, diabetes Mellitus, sleep apnea, some types of cancer and osteoarthritis. It is by far the leading cause of preventable death in the US. It's viewed by healthcare professionals and authorities as a serious public health problem. Obesity was classified

as a disease by the American Medical Association in 2013 [21]. As has been observed by the above maps depicting obesity prevalence amongst various US ethnic groups, the rates are not uniform. Amongst the various ethnicities, the Non-Hispanic Blacks have the highest obesity prevalence rates. The next in ranking are the Hispanics and then the Non-Hispanic Whites.

1.2.2 Obesity and Mortality Rate

Table 1: Top 10 causes of death by race in the USA

TOP 10 CAUSES OF DEATH IN THE USA BY RACE 2010						
Rank	All Races	Hispanic	Black	White	American Indian/Alaska Native	Asian/Pacific Islander
1	Heart disease 24.9%	Cancer 20.7%	Heart disease 24.1%	Heart disease 25.1%	Heart disease 18.9%	Influenza & pneumonia 2.0%
2	Cancer 24.4%	Heart disease 20.6%	Cancer 23.3%	Cancer 24.6%	Cancer 18.6%	Heart disease 23.5%
3	Unintentional injuries 6.2%	Unintentional injuries 9.5%	Unintentional injuries 5.5%	Unintentional injuries 6.2%	Unintentional injuries 13.5%	Stroke 6.6%
4	Chronic lower respiratory diseases 5.3%	Stroke 4.2%	Stroke 4.8%	Chronic lower respiratory diseases 5.7%	Diabetes 5.1%	Unintentional injuries 5.0%
5	Stroke 4.2%	Diabetes 4.2%	Homicide 4.6%	Stroke 4.1%	Chronic liver disease 5.0%	Diabetes 3.5%
6	Diabetes 2.9%	Chronic liver disease 3.9%	Diabetes 3.9%	Diabetes 2.7%	Chronic lower respiratory diseases 4.1%	Chronic lower respiratory diseases 3.4%
7	Suicide 2.5%	Homicide 3.1%	Chronic lower respiratory	Suicide 2.6%	Suicide 4.0%	Influenza & pneumonia 3.1%

			diseases 3.1%			
8	Alzheimer's disease 2.1%	Chronic lower respiratory diseases 2.7%	Kidney disease 2.8%	Alzheimer's disease 2.2%	Stroke 3.0%	Suicide 2.8%
9	Kidney disease 2.0%	Suicide 2.7%	HIV disease 2.1%	Influenza & pneumonia 1.9%	Homicide 2.4%	Kidney disease 2.0%
10	Influenza & pneumonia 1.9%	Kidney disease 2.1%	Septicemia 1.8%	Kidney disease 1.9%	Influenza & pneumonia 2.0%	Alzheimer's disease 1.3%

**Percentages represent total deaths in the age group due to the cause indicated. Numbers in parentheses indicate tied rankings. Source: CDC*

Seven of the top ten causes of death as indicated by table 1 above are obesity comorbidities. Heart diseases top the chart as being the culprit responsible for 24.9% of all deaths among all races/ethnicities, and cancer comes next with 24.4% of all deaths among all races/ethnicities.

1.2.3 Obesity and Growing Related Cost to the USA

One of the obesity studies conducted by researchers indicates that the cost of obesity, which comprised of overweight, with a body mass index range [BMI] of [25 – 29.9] and the Obese, with a BMI range of [BMI > 30], the medical expenses was about 9.1 percent of all medical related expenses in the US in 1998 and may have reached as much as $78.5 billion[19,22].

According to CDC, a third of US adults [about 72 million or more] are obese. For children and adolescents aged between 2 – 19 years, about 17% of them are also obese. Non-Hispanic Blacks have 51% higher obesity prevalence than non-Hispanic

Whites, while Hispanics have a 21% higher obesity prevalence than non-Hispanic Whites. According to CDC's estimates for 2008, it cost the US about $147 billion annually on obesity related cost for US adults.

Table 2: Top 20 most expensive conditions treated in U.S. hospitals, all payers, 2011

Top 20 most expensive conditions treated in U.S. hospitals, all payers, 2011				
RANK	CCS principal diagnosis category and name	Aggregate hospital costs, U.S. $, in millions	National costs, %	Number of hospital discharges, in thousands
1	Septicemia (except in labor)	20,298	5.2	1,094
2	Osteoarthritis	14,810	3.8	964
3	Complication of device, implant or graft	12,881	3.3	699
4	Liveborn	12,390	3.2	3,818
5	Acute myocardial infarction	11,504	3	612

6	Spondylosis, intervertebral disc disorders, other back problems	11,218	2.9	667
7	Pneumonia (except that caused by tuberculosis and sexually transmitted diseases)	10,570	2.7	1,114
8	Congestive heart failure, non-hypertensive	10,535	2.7	970
9	Coronary atherosclerosis	10,400	2.7	605
10	Respiratory failure, insufficiency, arrest (adult)	8,749	2.3	404
11	Acute	8,361	2.2	597

	cerebrovascular disease			
12	Cardiac dysrhythmias	7,624	2	795
13	Complications of surgical procedures or medical care	6,850	1.8	529
14	Chronic obstructive pulmonary disease and bronchiectasis	5,700	1.5	729
15	Rehabilitation care, fitting of prostheses, and adjustment of devices	5,487	1.4	420
16	Diabetes mellitus with complications	5,380	1.4	561
17	Biliary tract disease	5,137	1.3	469

18	Fracture of neck of femur (hip)	4,866	1.3	316
19	Mood disorders	4,840	1.2	896
20	Acute and unspecified renal failure	4,668	1.2	498
	Total 20 top conditions	182,266	47.1	16,755
	Total all conditions	387,272	100	38,591

Source: Agency for Healthcare Research and Quality (AHRQ), Center for Delivery, Organization, and Markets, Healthcare Cost and Utilization Project (HCUP), Nationwide Inpatient Sample (NIS), 2011

Amongst the top 10 most expensive procedures performed in USA hospitals in 2011, six of them were directly associated with obesity comorbidities. Heart diseases topped the chart with the most costly related procedures. Extending the list to the top twenty most expensive procedures add more to the list of obesity comorbidities related expensive procedures. Controlling obesity therefore will reduce related illnesses which will possibly reduce the cost of maintenance of related comorbidities such as heart diseases and diabetes nationally [23].

1.2.4 Obesity and Poor Quality of Life

The quality of life for most obese people is poor [24]. This problem is due to poor physical health, poor mental well-being and psychosocial dysfunctions [24]. They face discrimination and prejudice and that is their biggest burden [24]. Psychosocial factors play an important role in predicting outcomes of physical as well as mental health [24].

Weight improvements of obese patients often improve obesity related illnesses [23]. For the morbidly obese patients, the effectiveness of surgery in improving health and quality of life has been common [23]. The most important outcome of bariatric surgery involves improved obesity related illnesses, quality of life and psychological wellbeing [23]. There is often dramatic improvement or resolution of serious medical comorbidities accompanied by weight loss following Laparoscopic adjustable gastric banding [23]. Metabolic syndrome also improves. Metabolic syndrome puts you at risk for cardiovascular diseases, dyslipidemia and impaired glucose tolerance [23]. Weight loss is associated with fertility and more favorable pregnancy outcomes. Other obesity aggravated diseases are also improved after LAP-BAND surgery. These health improvements influence quality of life via improvement in physical and mental health. Obesity therefore impedes on our quality of life in general [23].

1.2.5 Obesity and what we eat

Obesity is affected by many factors, amongst which are excess caloric intake and caloric expenditure[15]. The best way to balance that, and maintain a healthy weight is what the challenges revolve around [13]. It is so because of the complexity of the human organism and its sub systems that affects adiposity and also the complex

societies in which we live, that exerts indirect and direct influences in the way we consume these calories, and also the types of calories we consume [4,6,8,13,15].

The calories we consume from our daily meals came from various macronutrients [15]. The way the body process these macronutrients and their pathways to obesity are different and as such exert diverse influences respectively [13,15]. Some of these macronutrients are; Sugars, Carbohydrates, Proteins, Fiber, and Fats.

Our body needs these macronutrients in other to function normally [15]. All those macronutrients have their primary functions in the body when taken in the right proportions, however the excess of all of them in our regular meals turn into our energy repository, through the process of adiposity [15]. This ultimately increases our weight relative to our height, causing obesity.

All these dietary macronutrients therefore are culprits of what cause obesity when disproportionately taken [13,15], however some exerts more influence or are worse culprits than others and also different ethnic groups may react differently to these mix of macronutrients.

1.3 Goals and Objective

To assist physician advice their patients on functional diets with the relatively appropriate amount of specific macronutrients needed for proper weight management. Our goals are;

- To better understand the relative risk of macronutrients to obesity.
- To better understand the relative risk of macronutrients in different ethnic subpopulations.

- To better understand the relative risk of a combination of macronutrients for example fat and sugar, fiber and protein, as oppose to their individual component.

1.4 Significance of Problem

A good understanding of such benefits and risk factors as listed above will be of great benefits to medical practitioners in advising their patients on diverse dietary issue such as these. Physicians could advice patients who find it hard to cut back on steak and roast beef or diet high in say fat to incorporate more fiber or complex carbohydrates and fruits and vegetables. Also patients from different ethnic groups could be well advised of their risk in consuming disproportionate amount of sugar or saturated fat. Lastly patients could be advised on how food categories synergistically combined could increase their risk, so that they make adjustments accordingly.

1.4.1 Obesity's impact on Families

Obesity affects families in many ways, which make most obese families worse off. It often propagates itself via generations and creates a family line of obese members who are generally prone to related comorbidities. Male obesity is increasing globally, and about 70% of men are obese [25]. Awareness is increasing, that male obesity negatively impact fertility, subsequent pregnancy and the offspring's health burden [25]. Paternal overweight/obesity induces paternal programing of offspring phenotypes likely mediated through genetic epigenetic changes in spermatozoa [25]. For mothers who have obesity, this condition may have an adverse effect on their lives and family relationships [26]. Families with obesity face a lot of discrimination and public

rejection [23,24]. These kind of public attitudes not only affect their psychosocial state and mental health, it also affects their chances of being employed gainfully, perpetuating a cycle of poverty for some families [23,24]. There are some genetic risk factors that impacts obesity. They however requires interaction with environmental factors for their expression [27]. Home environment of most obese family have some emotional and financial stressors, and some people deal with stress through smoking cigarette and other addictive substances to ward out the stress, including comfort meals which are full of carbohydrates and sugars. These behaviors create environment that affect the entire family adversely, including the children [27].

Obesity also affects fertility, hence influencing the female's chances of conception and bearing children [23,24]. Obesity affects families in many ways, mostly adversely putting them at risk financially, and health wise, both physically and mentally [25].

1.4.2 Obesity's impact on Healthcare Systems

The US expenditure on healthcare is more than any other country in the industrialized world [28]. This spending pattern however, is not because of higher incomes or the fact that the population is growing old. It is also not because of greater supply or utilization of medical facilities and doctors [28]. The related research finding indicates that, the problem with higher spending in more likely because prices of medical care and procedures are high and technology is readily available. It is also because of the growing obesity phenomena [28].

Obesity has become an epidemic also amongst children. The growing prevalence of obesity amongst children has greatly influenced pediatric practice. It has

influenced the discipline in such a way that pediatrician and other childcare practitioners have become so used to obesity comorbidities such as diabetes, hypertension, and other metabolic syndrome related diseases [29]. This phenomenon has put a great economic burden on the US healthcare delivery systems. Healthcare utilization has increase due to the growth in obesity incident and related comorbidities such as cardiovascular diseases are also soaring. [29].

Obesity comorbidities present a great burden on health care utilization and cost [29,30]. Cardiovascular diseases and diabetes present a great challenge to the US health systems. Obesity and diabetes mellitus for example, have created crises for the healthcare systems and the health of the public, incurring cost and disease burden for adults and children. Increasing cost and prevalence are as well expected [30]. The current situation cannot be contained unless more coordinated efforts to address these conditions at the national level are implemented [30].

In 2001, the aggregate obesity related cost was more than $99 billion per year. This amount represented about 5.7% of US health care expenditure [31]. Loss of weight changes the onset of some comorbid conditions associated with obesity. In some situations a loss of weight of about 5% to 10% is significant enough to change the onset of such obesity comorbidities as coronary heart diseases, type two diabetes, stroke and osteoarthritis, resulting in significant health and economic benefit [31].

A 2008 estimated obesity related cost saw a jump to $147 billion. This amount accounted for more than 8% of the total Medicare expenditures and 11% of the Medicaid expenditures [32]. According to Wexler (2007), though obese people constituted about 37% of the United States' population, the cost of obesity related

diseases and health issues account for 61% of healthcare costs in the US annually [33]. The costs were in excess of $147 billion annually, causing a financial tension on the healthcare system. The last two paragraphs show the growing trend of obesity cost burden to the USA.

1.5 Hypothesis and Related Questions

The purpose of the study is to determine the relative obesity risks posed by disproportionate consumption of certain macronutrients. The outcome of our study will arm physicians with empirical evidence to help them better advice their patients on related dietary issues. It will also ultimately improve the awareness of patients of their apparent risks of consuming certain amount of food types as oppose to others, and encourage them to make the most needed adjustments in their lifestyles related to dietary habits. This will go a long way to help them manage their weights better. The study will be driven by the following research questions and associated statistical hypotheses:

Question 1:

Do different dietary macronutrients compositions lead to different risk of obesity?

H0:

Different dietary macronutrients compositions do not lead to different risk of obesity.

H1:

Different dietary macronutrients compositions lead to different risk of obesity.

Question 2:

Does diet in different compositions of macronutrients affect ethnic groups differently?

H0:

Diet in different compositions of macronutrients does not affect ethnic groups differently.

H1:

Diet in different compositions of macronutrients does affect ethnic groups differently.

Question 3:

Does a diet high in fat and sugar increases the risk of obesity relatively higher than either individual component?

H0:

Diet high in fat and sugar do not increase the risk of obesity relatively higher than either individual component.

H1:

Diet high in saturated fat and sugar increases the risk of obesity relatively higher than either individual component.

Question 4:

Does a diet with significant amount of fiber and protein reduce the risk of obesity relatively higher than either individual component?

H0:

Diet with significant amount of fiber and protein do not reduce the risk of obesity relatively higher than either individual component.

H1:

Diet with significant amount of fiber and protein reduce the risk of obesity relatively higher than either individual component.

CHAPTER 2

LITERATURE REVIEW

2.1 Literature Source and Search Strings

Literature search consisted of review of many peer reviewed articles published on macronutrients and obesity, obesity and socioeconomic status, obesity and related comorbidities and other synonyms and more. Reviewed were articles and information published on organization websites, relevant books and by other reputable publishers. Electronic search strategies were utilized to identify relevant peer-reviewed articles, reviews and meta-analysis. Search was done with MEDLINE and PUBMED (1980-2016) for abstracts, English articles, and titles. We also conducted hand and google searched.

2.2 General Overview of Previous Literature

There have been many research works resulting in various academic reports and research journal articles regarding Obesity, macronutrients and related co-morbidities internationally and in the United States of America. In the USA, a lot of these were done with the NHANES sample data, and a lot more were also longitudinal studies which generated their own sample data. There has also been lots of research done in modeling and simulation of various aspects of Obesity, using various stochastic models such as the artificial neural network and the Markov Chain. Lots more research has also been done on macronutrients such as sugar, carbohydrates, proteins, fiber and obesity separately.

However, there has not been a comprehensive research done on the differences in macronutrients on obesity risks and also how they affect various US ethnic subpopulations.

2.3 Understanding Obesity's Impact

It is estimated that a quarter or more of America's healthcare costs are associated to obesity [34,35]. About 20 to 30 percent of the rise in health care spending since 1979 is related to the rise in obesity prevalence. A more stable obesity rate would have kept the US healthcare spending nearly at 10% lower on the average per person [36,37]. Non communicable or chronic diseases and healthcare cost in the US can never be controlled unless the country finds a way to make its citizens healthier. About Two-thirds of US adults are overweight or obese [38]. Also childhood obesity epidemic is on the loose, putting today's youth in danger for poor health. They are more likely to be the first generation to live shorter years, with more health problems than their parents [39].

2.3.1 Non Communicable Diseases [NCD]

The World Health Organization [WHO] reported in 2010 that non communicable diseases or chronic diseases are the world's biggest cause of death. They stated that about 36 million people died from NCDs in 2008. Majority of the death were caused by cardiovascular diseases (48%), cancers (21%), chronic respiratory diseases (12%) and diabetes (3%) [18]. Over 9 million deaths were mainly preventable, and occurred before age 60. Most of these premature deaths were in low income countries and ranged from 22% among men and 35% among women. In high

income countries the rates were relatively low, 8% among men and 10% among women. [18].

Some of the major related behavioral risk factors are the use of tobacco, lack of physical activities, harmful consumption of alcohol and poor diet. These risk factors remains very high globally and more especially in low to middle income countries [18]. Some other risk factors include, raised blood pressure, overweight and obesity raised cholesterol levels and raised blood glucose. [18].

2.3.2 Body Mass Index [BMI]

The general definition of obesity is; "excess body fat that increases the risk of disease and premature death". The main measure of obesity is a function of weight and height which is called the Body Mass Index (BMI). Economists and healthcare workers use this measure to estimate the prevalence of obesity and other related healthcare statistics like disease burden created by obesity and so on. These help in the allocation of healthcare resources and also influences obesity related healthcare government policies [40]. Below is the BMI mathematical formula. This was established in 1997 and published in 2000 [41].

- A BMI less than18.5 is Underweight
- A BMI 18.5 – 24.9 is Normal weight
- A BMI 25.0 – 29.9 is Overweight
- A BMI 30 – 39.9 is Obese
- A BMI 40.0 or higher is severely or Morbidly Obese

BMI [Body Mass Index] Formula

$$BMI = \frac{\text{Weight (kg)}}{\text{Height}^2\text{ (m)}} \qquad BMI = 730\,\frac{\text{Weight (lb)}}{\text{Height}^2\text{ (in)}}$$

Obesity is multidimensional. This is so because excess of body fat can lead to different comorbidities, and to different extents, depending on it's location within the body and other genetic predispositions of the individual. Though BMI is one dimensional, it is the most favored measure by researchers and economist as well as others interested in related research, modeling and public policy issues. BMI is cheap to measure because related data is affordable and abundant [40].

2.3.3 Common Obesity Comorbidities

Obesity co-morbidities are many. These will include: Various forms of Cancer, Cardiovascular Diseases, Diabetes Mellitus, Osteoarthritis, Renal Diseases and more. This paper however will emphasize on Cardiovascular Diseases and Diabetes Mellitus as examples of obesity comorbidities. These will be described in more details in the next several sections.

2.3.3.1 Diabetes Mellitus

Diabetes is characterized by several disease states, which have its marker as high level of blood glucose. It usually happens as a result of defective insulin production, insulin action or both [42]. Insulin is the hormone needed to convert sugar and starches to energy for the body's needs [42]. Many serious diseases, such as heart disease, kidney failure, foot problems, blindness, dental disease, lower extremity amputations, diabetic ketoacidosis, pregnancy complications and death, can manifest

as diabetes complications. Though the main cause of diabetes is unknown, genetics and other risk factors such as lack of physical activities and poor diet appear to induce it [42].

Types/Classes of Diabetes

Type 1 Diabetes – Type 1 diabetes is also referred to as insulin-dependent or juvenile-onset diabetes. When the body's immune system destroys pancreatic beta cells, the cells that makes the hormone insulin, type 1 diabetes manifest as a result. Insulin is the hormone that regulates body's blood's sugar. Type 1 diabetes accounts for about 5% to 10% of all diagnosed cases of the disease. [42].

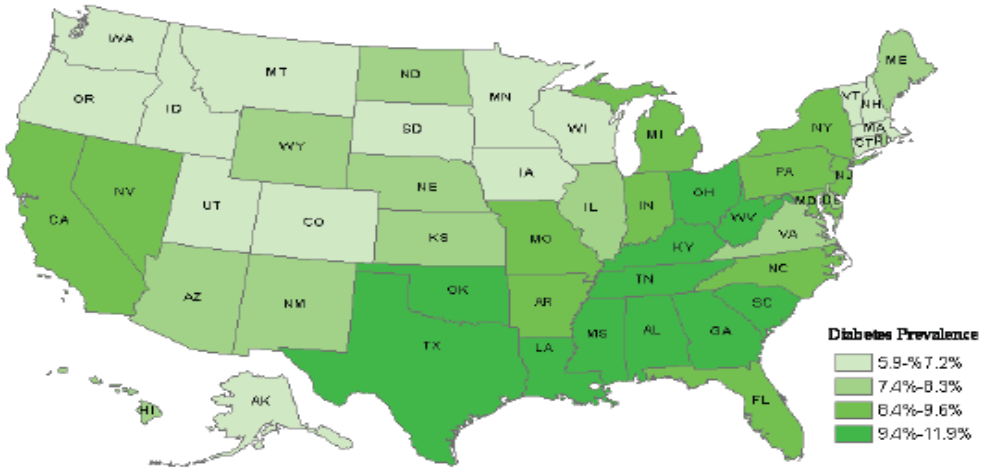
Type 2 Diabetes – Type 2 diabetes is also referred to as non-insulin dependent or adult-onset diabetes mellitus. It starts when the body's usage of insulin is not properly regulated. The phenomenon is referred to as insulin resistance. When it does happen the pancreas gradually loses the ability to produce insulin as the need arises. Type 2 diabetes accounts for about 90 to 95% of all diagnosed cases of the disease. [42].

Gestational Diabetes – When glucose intolerance occurs during pregnancy, the patient is diagnosed with gestational diabetes. It is signified by high levels of blood glucose during pregnancy, for women who had never been diagnosed with diabetes. The disease affects about 4% of all pregnant women.

Pre-diabetes – When ones blood sugar is high, yet not to the threshold of diabetes diagnosis, pre-diabetes is rather diagnosed [42]. Majority of patients diagnosed with pre-diabetes, often have an increased risk of developing type 2 diabetes as well as stroke and heart disease.

Estimated Prevalence of Adults Diagnosed with Diabetes*

UNITED STATES



*Question: Have you ever been told by a doctor that you have diabetes?
Source: CDC Behavioral Risk Factor Surveillance System, 2008.

Figure 6: Estimated Prevalence of Adults Diagnosed with Diabetes in the USA per State

***["An estimated** 23.6 million Americans, or 7.8% of the U.S. population, have diabetes.1 Approximately 75% of these individuals, or 17.9 million Americans, have been diagnosed with diabetes while the remaining 5.7 million are unaware that they have diabetes." (CDC)][43] NOTE: As observed, the Diabetes Prevalence Pattern on the above map closely matches that of Obesity Prevalence on the related maps shown earlier.*

From the onset of the use of insulin in 1921 diabetes has been a treatable chronic condition [44]. Childhood or juvenile diabetes or insulin dependent diabetes, also known as Type 1 diabetes, is mostly commonly diagnosed in children and adolescents [45]. Its incidence in adults is similar to that of children. Most adults with type 1 diabetes

are often diagnosed with type two, hence the misconception that it is a disease of children [45].

. Type 2 diabetes is incurable at the moment and is rapidly increasing in countries like the United State of America [46]. The rate of increase of type 2 diabetes is in epidemic proportions according to CDC [46].

Complex metabolic changes damage or impair many internal organs such as the cardiovascular systems [46]. Such defects lead to substantial increase in mortality and morbidity in sufferers of both types of diabetes [46].

2.3.3.2 Cardiovascular Diseases [CVDs]

As has been mentioned in this paper, Obesity has many co-morbid indications or comorbidities. These include diseases of the heart, which are well publicized disease states, mostly due to their adverse impact on society. Heart disease is a broad category of diseases which affects the heart [47]. Some common ones are, Coronary heart disease, ischemic heart disease, cardiovascular disease, pulmonary heart disease, hypertensive heart disease, inflammatory heart disease and valvular heart disease [47]. This document however will be focusing on cardiovascular diseases as mentioned earlier. It is a class of diseases that affects or involves the heart and or blood vessels, such as the arteries and veins [48]. Though cardiovascular disease may means any disease that affects the cardiovascular system, it is often referred to those with atherosclerosis or arterial disease [48].

Atherosclerosis formation is slow. It is a complex disease of which fatty substances, cholesterol, cellular waste products, calcium, and other substances, leaves

residues, that build up in the inner lining of an artery, which eventually clogs or narrow its path [49]. Its impact is different, depending upon the particular set of arteries in the body that narrow or become clogged. [49]. As an example, plaque buildup in the blood vessels that supply the heart with oxygen rich blood may lead to chest pain and cause heart attack. On the other hand plaque buildup in the arteries that supply blood to the brain could lead to a stroke [49]. As reported by CDC, heart disease is the leading cause of illness and death in the United States. In the US, about one million deaths are attributable to heart disease. This number of death is about twice the number of deaths from cancer.

Atherosclerosis is mostly a preventable disease. Its risk factors are well known and documented. As such preventive measures such as lowering blood pressure and LDL ("bad") cholesterol levels, losing weight, smoking cessation and increasing physical activity need to be followed to prevent the onset and fast development of atherosclerosis [49]. Its symptoms are normally hidden, until a significant percentage (40%) of a vessel become clogged with plaque and a complication follows [49]. The symptoms differ, depending upon which arteries in the body narrow and become clogged with plaque.

Both men and women are affected devastatingly by Cardiovascular diseases [50]. Since 1984 cardiovascular disease has claimed the lives of more females than males, in terms of total deaths [50]. It is however the No. 1 killer of women and men. Cardiovascular diseases causes about a death a minute amongst females, claiming about half a million female lives every year. At age 75 and on, the prevalence of cardiovascular disease among women is higher than among men [50].

For children under the age of 15 years, cardiovascular diseases ranks as the No. 3 cause of death. It falls behind certain conditions originating in the prenatal period and accidents [50]. In 2001, for children aged 15 and younger, about 197,000 cardiovascular procedures were performed [50]. There are thousands of babies who are born each year with congenital cardiovascular defects [50]. Congenital cardiovascular defects claim more lives than any other kind of congenital defects. It claims about 2,200 lives a year of children under age 15 [50]. In the USA, about 1 million people alive today have congenital cardiovascular defects, and nearly 25 percent are children [50].

Types/Classes of Cardiovascular Diseases:

Coronary Artery Disease (CAD)

When there is a clog or plaque buildup in the vessels that supply the heart with oxygen-rich blood, the associated cardiovascular disease is called coronary artery disease. Its related symptoms manifest itself when the tissues of the heart begin to become deprived of oxygen. Chest pain (angina) usually occurs as a result. In a situation when the artery becomes completely clogged or blocked, cells in the heart begin to die, and often results in a heart attack. Symptoms of coronary artery disease are often triggered by sexual activity, physical exercise, exposure to cold weather, stress or anger [50].

Cerebrovascular Disease

When the buildup of plaque occurs in the arteries that supply the brain with oxygen-rich blood, the related cardiovascular disease is called cerebrovascular disease. Its major symptoms are transient ischemic attack (a sudden loss of brain function with complete recovery within 24 hours) and stroke [50].

Peripheral Artery Disease

When the plaque buildup affects the arteries that supply the extremities of the body (such as the hands and feet) with oxygen-rich blood, the related cardiovascular disease is called peripheral artery disease [50]. Cardiovascular diseases are typically treated by the following medical practitioners; Cardiologists, thoracic surgeons, vascular surgeons, neurologist, and internal radiologist; depending on the organ system that is being treated [48].

Obesity is regarded as a very complex disorder and a major risk factor for many diseases. Often, it is linked to increased cardiovascular disease (CVD), stroke, cancer, hypertension, diabetes, and early death [51-53]. According to the Framingham Heart Study researchers, obese patients had about 104% increase in the risk of developing heart failure compared to non-overweight individuals [54]. Patients diagnosed with morbid or abdominal obesity are at particularly high risk for CVD, diabetes, and all-cause mortality [53,55]. A stroke patient with obesity typically has a longer hospital stays and a lower chance of being discharged home [53,56,57].

2.3.4 Common Obesity Related Procedures

Surgery has emerged as the choice of morbidly obese patients for assisting with weight loss. As a result of that, many studies have been done relating to gastric bypass and bariatric surgeries. One of such studies confirmed that bariatric surgery is the most effective ways of treating severe obesity or morbid obesity and its other conditions [57].

There has been an examination of gastric bypass trends from 1998-2003, focusing on the demographics and health profiles of the patients who took part in the procedure for outcomes such as complication rates, hospital stay and length of stay [58]. National Hospital Discharge Survey data, reported totals of about 288,000 discharges relating to gastric bypass surgery between the years of 1998 – 2003 [58]. There was an increase in the total number of gastric bypass surgeries from 14000 in 1998 to 108000 in 2003 [58]. The length of stay was decreased by 56% with 10% complication rate during the period [58].

Another study examined the increase of national open bariatric surgery, and its correlation with laparoscopic bariatric surgery [59]. There was a dramatic increase in both surgeries between 1998 and 2002 [59]. Open Bariatric and laparoscopic bariatric surgeries increased by 450% from 1998 to 2002. Whiles bariatric open surgery increased by 144% during the same period [59]. This pushed the growth of bariatric centers significantly during that period.

A study which was published in 2005 used the National Impatient Sample [NIS] data from 2000 to show the hospital and patient attributes for those who did bariatric surgery [60]. The following were discovered from the study:

- The length of stay, cost, morbidities and comorbidities were highest for men and those over 60 years old
- The length of stay, cost and comorbidities were greatest for African Americans as compared to Caucasians, and Hispanics.

- Lower income, Medicare and Medicaid insured patients has higher length of stay and cost as compared with those paying out of pocked and those that are privately insured.

Bariatric surgery operations performed worldwide in 2008 was 344221. They were performed by 4,680 bariatric surgeons. Out of that, 220,000 were performed in USA/Canada by 1,625 surgeons [61]. Laparoscopic adjustable gastric banding were the most commonly performed procedures during that period (AGB; 42.3%). For the laparoscopic adjustable gastric banding procedures, laparoscopic standard Roux-Y gastric bypass was the most performed (RYGB; 39.7%), and total sleeve gastrostomies 4.5%. Over 90% of procedures were performed laparoscopically [61]. Bariatric surgery is growing worldwide as the surgery of choice for the morbidly obese, but less so than in the past [61,62].

A systematic review and a meta-analysis on bariatric surgery were performed by some US researchers. The research was done because of the fact that morbid obesity was refectory to diet and drug therapy, however quite responsive to bariatric surgery [62]. Their main goal was to find out the impact of bariatric surgery on weight loss, operative mortality outcomes, and four obesity comorbidities. These were diabetes, hyperlipidemia, hypertension and obstructive sleep apnea [62]. Two levels of screening were used on 2738 citations from diverse sources including; Medline, Current Content, and Cochrane Library [62]. Their conclusion was that, effective weight loss was achieved in morbidly obese patients after undergoing the procedure [62]. Also a good majority of patients with hypertension, diabetes, hyperlipidemia and obstructive sleep apnea experienced improvement or complete cure [62]. Bariatric Surgery therefore

is the treatment of choice which has yielded good outcomes as per the evidence in the current section of this paper.

2.3.5 Obesity Prevention and Education

For the past twenty years and more, childhood overweight and obesity prevalence has rapidly grown worldwide [63-67]. The new trends in childhood obesity are due to socioeconomic changes and physical environment, with nutrition transition as well [17]. There has been a trend of decreased physical activity and more sedentary lifestyle amongst children. In addition, there is an increase in the consumption of energy dense foods that are low in fiber, but high in sugar, and more sugar sweetened drinks [68]. Globally, overweight and obesity have become a very serious public health concern [69,70]. There has been an overwhelming increase in healthcare cost that are associated with overweight and obesity. [68]. Children's physical appearance is impacted by childhood obesity and can result in additional psycho-social consequences. Most affected children have low self-esteem, experience social alienation, and lacks self-confidence [71,72]. They also experience discrimination [73] and, for girls, depression [74]. Most children with childhood overweight and obesity problems usually have additional long-term health risks into adulthood [68]. Long-term follow-up studies show that obese children typically grow up to become obese adults [75-77].

A systematic review on the issue was conducted in a study by C.M. Doak et al. The review was limited to school-based studies. It was a quantitative evaluation using anthropometric outcomes that intervene on diet or activity related behaviors [68].

Quantitative and qualitative approaches were used to identify factors related to successful interventions as well as adverse consequences [68].

Sixty eight per cent of the interventions, or 17 of the 25, were 'effective' based on a statistically significant reduction in body mass index (BMI) or skin-folds for the intervention group. Four of the interventions were effective by BMI as well as skin-fold measures [68]. Amongst the four, two were targeted toward reductions in television viewing [68]. The two remaining studies were targeted towards direct physical activity interventions, using the physical education program combined with nutrition education. Out of the former two interventions reported, one was effective in reducing childhood overweight but was also associated with an increase in underweight prevalence [68].

Most of the intervention or prevention programs for obesity and overweight included in the study were effective. Physical education in schools and reducing television viewing are two examples of interventions that have been successful [68]. However a few of the studies reported on underweight prevalence. As a result of that they recommended giving more attention to preventing adverse outcomes.

Mary Story, Karen Kaphingst, and Simone French argue that U.S. schools have and can provide many opportunities for developing obesity-prevention strategies. Some of their suggestions were providing more nutritious food, offering greater opportunities for physical activity and providing obesity-related health services [78]. Most schools offer a wide variety of food options. These include meals available both through the U.S. Department of Agriculture's school breakfast and lunch programs and through "competitive foods" sold à la carte in cafeterias, vending machines, and

snack bars [78]. Food provided by schools via the U.S Department of Agriculture's breakfasts and school lunches must meet federal nutrition standards, however the alternative competitive foods are exempt from such requirements [78]. Budget constraints force schools to sell the popular but nutritionally poor foods à la carte. This has made most parents express growing public discomfort with the school food environment [78].

In another research study on nutrition, obesity education and counselling, it was concluded that implementation of an 'Obesity Prevention in Pediatrics" curriculum appears to improve participants' knowledge base as well as their skills and level of personal comfort in the recognition, evaluation and management, including counseling, of both obese and at-risk pediatric patients and their families [79]. The variability of children's levels of physical activity is wide. It may be influenced by a multitude of factors including physiological, psychological, sociocultural and environmental determinants [80]. In spite of the fact that the relationship between physical activity and obesity is controversial and the protective mechanism unclear, it is hypothesized to protect individuals from the development of obesity. It does so by increasing energy expenditure and resting metabolic rate, leading to a favorable fuel utilization [80].

Controlled exercise intervention programs supports beneficial effects of physical activity in children. [80]. Schools, families and community interventions, are examples of where most public health interventions designed to increase children's levels of physical activity have been implemented. The results of these interventions suggest promising strategies for the prevention of childhood obesity [80]. Successful

prevention of childhood obesity, is most likely through physical activity promotion involving theory-based, culturally appropriate school, family and community interventions [80]. By the use of policy changes, environmental planning and educational efforts in schools and communities, enhanced opportunities for physical activity can be provided [80].

Due to the above literature and study reviews, it is quite apparent that obesity preventions and related programs can be more effective in the long run, if children and parents or guardians, who usually supervise their domestic activities in collaboration with the school systems, are targeted for their implementations. Since post and prenatal women activities also affect the weight of children, it is very important also that women's health and activities through conception and beyond is also closely monitored.

2.4 Obesity: Different Schools of Thought

2.4.1 Industrialization of Food Industry and Obesity

The U.S population's weight has been increasing throughout the 20th century [2]. The study increase in obesity since the 1980s however, is quite different [2]. Most part in the 20th century, the US population's weight was below recommended maximum life's pan levels (Fogel, 1984) [2]. During those times, an increase in weight represented an improvement in health [2]. Americans are now growing bigger and bigger, with weights higher than medical science recommends. In recent times, weights are still getting high [2]. The U.S is the most obese advanced nation.

People normally gain weight when their caloric intake exceeds their caloric expenditure [2]. David M. Cutler et. al. 2003, examined whether increase in obesity in the US is a result of decrease in exercise or increase in food intake or consumption. Their evidence suggested that increase in caloric intake is far more important than reduced caloric expenditure in explaining recent increase in obesity [2]. As they observed, our caloric expenditure has not changed significantly since 1980 [2], whiles our calories consumed have risen markedly.

Technological innovations in food production and transportation, appears to influence Food consumption [2]. These innovations has made it possible for companies to mass prepare food and ship to consumers for ready usage [2]. As a result there is a significant reduction in time cost of food, which has in turn led to increase food consumption, and ultimately increase weight [2].

Many facts are in agreement with this theory. First, there has been a significant increase in food variety in recent decades, and as a result, people eat many more times during the day [2]. This is a result of implications of declining price for mass produced food, due to economies of scale [2]. For a comparison between varieties of demographic groups, people who did not have many options in their food choices a few decades ago had the largest increase in obesity. Countries who have significant regulations, particularly on the food industry, the likes of the Scandinavian Countries, have had less of an increase in obesity [2].

Though it makes sense economically to suggest that this time cost savings and the apparent increase in consumption represents pure economic benefit, however lack of self-control tend to make the changes non beneficial, hence reduces welfare or

wellbeing [2]. Minimizing the time cost of food preparation disproportionately increased food consumption [2] because time delay is a particularly pertinent mechanism for discouraging those individuals from consuming [2].

2.4.2 Behavioral Factors and Obesity

Childhood weight gain in children is usually a results of a complex mix of factors associated to diet and activity as well as a background of genetic predispositions [81]. Some related risk factors include; high energy density diet, high consumption of sugar-sweetened beverages, large portion size of food, eating patterns such as meal skipping, high levels of sedentary behavior and low levels of physical activity [81].

Interactions between behavioral factors, especially diet and activity, is a growing concern [81]. There is therefore a need for more research to understand the complex interaction of behavior [81]. Parental obesity strongly predicts childhood obesity. The result reflects genetic predisposition to weight gain and environmental effects [81]. Hence the child's chances of positive energy balance may be affected by behavioral genetic factors in addition to environmental risk factors [81]. The effect of parental obesity on excess weight gain in children illustrates the complexity of the interaction between factors and the problem in determining which factors [81] are causal factors of obesity and which ones are amendable to interventions.

2.4.3 Environmental Contributions to Obesity

Our genes have not changed significantly during the past two decades. An environment which promotes behaviors that causes obesity, is one of the factors that is responsible for the recent obesity explosion [82]. To stop and eventually reverse the obesity epidemic, we must correct this obesogenic environment [82].

Simply, obesity is caused by excess energy intake beyond energy expenditure. The current obesogenic environment is characterized by lots of supply of convenient, relatively inexpensive, high palatable, energy dense food, coupled with a life style of low level of physical activities needed for subsistence, which promotes high energy intake and low expenditure [82]. Our body weight and composition are determined by interactions between diet, the environment and genetics [82]. The environment contributes to obesity by increasing the frequency of behaviors that increase the risk of positive energy balance. Hence the body mass index increases to restore energy balance [82].

Some of the environmental factors that promote overeating are:

- Food availability and portion size
- High fat diet

Some of the environmental factors that promote physical inactivity:

- Advances in technology and transportation
- Appeal to TV
- Appeal to electronic and computer games

Over all, energy balance under any condition is determined not by diet or activity alone, but by the interaction between the two [82].

2.4.4 Socioeconomic Status, Culture and Obesity

In the United States, obesity and hanger coexists and the phenomenon is called the hanger obesity paradox [11]. This paradox was first mentioned by William Dietz MD., Ph.D., in 1995 in his case study "Does Hunger cause Obesity" [11], published in Pediatrics. It was written because obesity connotes excessive energy intake and hunger reflects an inadequate food supply [11]. The increased prevalence of obesity and hunger in the same person hence seemed paradoxical [11].

Food insecurity leads to the purchase of high energy dense food which ultimately leads to obesity [11]. The poor with scarce financial resources often do not prioritize food choices favorably [11]. Government Programs like WIC and food stamps attempt to remedy the situation.

2.5 Obesity and Dietary Macronutrients

2.5.1 Obesity and Sugar Consumption

A significant amount of scientific research has been performed on Sugar consumption since the rising rates of diabetes, obesity, and hypertension became a major concern [83]. The questions of whether sugar causes type 2 diabetes and the effects on glycemic control and insulin resistance for those with the illness, has been examined by most researchers [83]. A lot of research works into the question of whether sugar is a major risk factor for obesity has also been done. Some of these researchers suggest that there is no evidence for sugar causing diabetes. Diets to control diabetes

must account for total carbohydrate, not just the sugar component [83]. Others however, suggest that sugar is a significant risk factor for type two diabetes mellitus [84-88].

Scientific evidence that sugar consumption leads to obesity is inconsistent, and diverse studies show both reduced and enhanced satiating effects [83]. In spite of the fact that there is evidence that sugar sweetened beverages [SSBs] may be less satiating than solid forms of carbohydrate, and increasing consumption of such beverages has generated scientific concern, most of the evidence is observational and well-designed intervention trials are lacking [83]. Therefore, the evidence for an effect of sugars on chronic diseases is generally not conclusive. [83].

There are other researchers that suggest that sugar is directly linked to obesity and its consumption has to be reduced [89-93]. The consumption of sugar sweetened beverages in America has increase about 300% since the 80s and so has the rate of obesity [94,95]. There have been several research investigations on this subject with conflicting views [96,97]. However several reviews on the subject indicate that the conflicting views were due to the methodology of the research work [96,97]. Most highly rated research methodologies on the subject links high sugar consumption to obesity, and sugar sweetened beverages [SSBs] being the most consumed added sugar in America turns out the worst culprit [96].

The fight to reduce obesity globally has been very challenging and some countries like India and England are even considering the imputation of taxes on sugar, to hopefully reduce sugar consumption and eventually curb obesity [85,98-103]. Others are saying that we should look beyond sugar and consider the entire glycemic index and well as the glycemic load [104]. Though sugar consumption stand out as one of

the factors that impacts obesity prevalence in America and globally, the entire eating habits and patterns of people needs to be critically examined, in lure of their relative obesity risks.

2.5.2 Obesity and Carbohydrates Consumption

With the epidemic increases in the prevalence of obesity in the U.S. and elsewhere, carbohydrate in diet has recently been under closer scrutiny [105,106]. This has resulted in the development of methods for analyzing the effects of dietary carbohydrate [106]. Among the derived methods of analyzing the effect of dietary carbohydrate, one of the primaries is the glycemic index [106]. It is a measure of food's effect on blood glucose levels [106]. It was initially designed purposely as a method for determining the right amount of carbohydrates for people with diabetes. The glycemic index however, does not address the other metabolic issues related to excess sugar consumption [106]. Some of these issues is the use of low glycemic index sweeteners, particularly fructose, which is increasingly present in processed food [106]. Associated with increased adiposity is fructose. This may be as a result of its effects on hormones associated with satiety [106]. There have been other methods of determining good carbohydrates. The main theme among them is increased non-starchy vegetables and higher-fiber legumes [106].

Carbohydrates in meals constitute a good percentage of our energy intake [13,15,107]. It metabolizes into glucose and also some fatty acids through adiposity [15,108]. More refined carbohydrates such as white rice have a very high glycemic load and metabolizes faster than less refined carbohydrates such as whole grains [13,15]. Whole

grains, fruits and vegetables with their higher inherent fiber present a healthier alternative to more refined carbohydrates [13,15]. Excess caloric consumption with a greater proportion of process carbohydrates is not good for weight management and health in general [13,15]. Excess refined carbohydrates are known to be a risk factor for type two diabetes mellitus and cardiovascular diseases [15,109].

2.5.3 Obesity and Fiber Consumption

The term dietary fiber, comprises of a heterogeneous group of natural food sources, processed grains, and commercial supplements [110]. Many different forms of dietary fiber have been used as alternative agents in the management of manifestations of the metabolic syndrome, including obesity [110]. The biological efficacy of dietary fiber in the metabolic syndrome and body weight control has varying levels. Many factors and mechanisms have been reported as mediators of the effects of dietary fiber on the metabolic syndrome and obesity[110]. Some of these are the modulation of gastric sensorimotor influences that appears to be crucial for the effects of dietary fiber [110].

There are a lot of evidence from Epidemiology which favors the hypothesis that obesity may result from the low fiber diet of industrialized societies, because hyperinsulinemia is a universal characteristic and perhaps causal factor of obesity [111]. The possibility is considered that dietary factors causing excess insulin secretion might lead to obesity [111]. A glucose diet causes a slightly greater insulin rise than cooked starch containing an equal amount of carbohydrate [111]. A high fiber starchy foods cause a lesser insulin response than glucose in solution [111]. Dietary fiber acts by displacing some of the carbohydrate that would have been absorbable in the small

intestine, or could translocate the carbohydrate to a point lower in the intestinal tract where less effect on insulin secretion would be observed [111].

Childhood obesity is one of the serious public health problems in the United States because of its assumed high prevalence and increasing secular trend [112]. The genetic contribution to obesity is at best estimated to ranges from 5% to 25% [112]. Environmental factors however play a major role in obesity development [112]. Low income and a low level of education are both high risks of obesity [112,113]. Several studies has been done, linking increased total fat intake, rather than caloric intake, with obesity [112]. Television viewing has also been linked to childhood obesity by several studies [112]. In developing countries where obesity is not too common among the populations, dietary fiber consumption is high [112]. An explanation for the role dietary fiber plays in obesity include a reduced caloric density of the foods, a slower rate of food ingestion, and possible effects on satiety [112,113]. Available data, primarily from adult studies shows, significantly lower risks for obesity, diabetes, and constipation could be expected with higher dietary fiber consumption [114].

2.5.4 Obesity and Protein Consumption

Proteins are large biomolecules or macromolecules, which consists of one or more long chains of amino acid residues. Proteins perform many different functions within living organisms, including catalyzing metabolic reactions, DNA replication, responding to stimuli, and transporting molecules from one location to another. Proteins are different from one another primarily in their sequence of amino acids, which is dictated by the nucleotide sequence of their genes, and which usually results

in protein folding into a specific three dimensional structure that determines its activity.

Diets high in protein are associated with greater satiety [115]. In a couple of studies, they are associated with greater weight loss compared with high carbohydrate diets [115]. High protein diets also lower plasma triglyceride and blood pressure [115]. There seems to be no harmful effects of high protein diets on bone density or renal function in weight loss studies [115]. According to Willi et. al., the high-protein, low-carbohydrate, low-fat ketogenic diet (K diet) can be used effectively for rapid weight loss in adolescents with morbid obesity [116]. Loss in lean body mass is blunted, blood chemistries remain normal, and sleep abnormalities significantly decrease with weight loss [116].

In recent years, the Atkins diet, a well-known low-carbohydrate and high-meat diet, has gotten a high interest from many researchers and the general public for weight loss and for the prevention of obese and type 2 diabetes [117]. There is a claim that the restriction of carbohydrates could assist with the body's metabolism from burning glucose to burning stored body fat [117]. There is however counter claim that the long term effectiveness beyond 6 months of Atkins diet is without evidence or prof [117]. Wang et. al. however concluded that there is an association with meat consumption and the risk of obesity [117]. The effectiveness of high protein diet as a solution for obesity is a bit controversial and researchers have different claims.

2.5.5 Obesity and Fat Consumption

Fats are a type of nutrient that is gotten from diet. It is important to eat some fats, however harmful when you eat too much of it [118]. Fats provide the body with energy that it needs to work properly[118]. When performing physical activity or exercise, the body uses calories from carbohydrates. However, after 20 minutes of exercise, it relies on the calories from fat to keep one going [118]. Fat is also needed for skin and hair health. [118]. It also helps in the absorption of vitamins A, D, E, and K, the so-called fat-soluble vitamins [118]. Fat also fills the fat cells, and helps in insulating the body to help keep it warm [118]. The fats the body gets from food give it essential fatty acids called linoleic and linolenic acid [118]. They are called "essential" because the body cannot make them itself, or work without them. They are needed by the body for brain development, controlling inflammation, and blood clotting [118]. Fat has 9 calories per gram, more than 2 times the number of calories in carbohydrates and protein, which each have 4 calories per gram [118]. Diet high in fat is well established as an obesity risk [119]. High fat diet induced obesity which however, comes with numerous health issues [119-126].

CHAPTER 3

RESEARCH METHODOLOGY

3.1 Research Overview

Obesity is the leading cause of preventable death in the United States and the most prevalent, fatal, chronic, disorder of the 21st century. African Americans prove to be disproportionally affected by obesity, followed by Hispanics. According to many experts, this prevalence of obesity is correlated with a staggering increase in diabetes and cardiovascular diseases. Socioeconomic and cultural obesity determinants also have a relationship with obesity. Balancing our caloric intake and expenditure is one of the most critical factors of obesity. This simple phenomenon is complicated by numerous other factors, such as socioeconomic and cultural factors, environmental and others. What we eat and its quantity in lure of our activity levels and the body's needs are very critical to the causation of obesity. As said before there are many factors that affect this seemingly simple phenomena.

In spite of the complex nature of obesity causal and confounding factors, it is very critical to note the most important dietary culprits of obesity, to enable physician and other medical practitioners to advice patients amongst others, how to manage their caloric intake and ultimately their weight. Calories come mainly from the major macronutrients, which are proteins, fiber, fat and carbohydrates. Their pathways to obesity are different and their influences also do differ by intensity. It is therefore very critical that their impact on various ethnic groups is well understood, in addition to their level of intensity as obesity risk factors. A good insight of these issues will help physicians to better advice their patients on related dietary matters. The main goal of

this study therefore is to contribute to the understanding such matters as mentioned above.

This would help medical practitioners personalize treatment for obese patients with a deeper understanding of the patient's related background, and also help influence national public health related issues. Patients will also understand their health state better in reference to their various health determinants and act accordingly to improve their health and wellbeing. The purpose of this retrospective, correlational, quantitative study is to examine obesity in lure of the effect of the major macronutrients on United States ethnic sub-population using data collected by CDC.

Chapter 3 will present an overview of the methodology used for this study. This overview will include the following: Research Overview, Theoretical Framework, Research Design, Research Questions and Hypothesis, Data Source, Data Sample Size, Study Period and Study Group Inclusion/Exclusion Criteria, NHANES DATASET and Elements, Data Cleaning and Data Processing, Variables and Coding Exposures and Outcomes, Data Analysis Method, and Data Analysis Plan.

3.1.1 Theoretical Framework

The theoretical framework of the study was the health belief model. The health belief model is one of the most frequently used health behavior theories in public health. The premise of the health belief model is that health-related action depends on the occurrence of three factors: (a) the individual must perceive there is an existence of a health concern, (b) the individual must believe that he or she is vulnerable to a serious health problem or threat, and (c) the individual must believe that engaging or

following a certain health recommendation will benefit him or her in reducing the perceived health threat (McKenzie, Neiger, & Thackeray, 2009) [127].

With the results generated from this study the health belief model can be used to further investigate why certain individuals are at higher risk based on ethnicity and portions of macronutrients consumption. In addition it is important for individuals to overcome their perceived barriers in following health recommendations. Some potential barriers an individual may face are financial and time barriers, whiles others may simply be the location of their residence. For example, some young adults may perceive that there is an existence of a health threat and that they are vulnerable to this threat, and they may also believe that following certain physical activity, and dietary guidelines would be beneficial, but they may think that they do not have the time or money to exercise, or access to proper diet due to food deserts within their metropolitan status codes or residential areas.

3.1.2 Research Design

This study will follow a retrospective, quantitative, and correlational design. A quantitative correlational design seeks to examine potential relationships between variables [128,129]. Further insight into why this design selection is appropriate for this study can be seen by examining the three parts of the design separately (retrospective, quantitative, and correlational).

The data will be retrospective due to the use of archival data for the analysis. In retrospective studies, the outcome of interest has already occurred at the time the study was initiated. For this study, the data is archival, and the outcome of interest

occurred from 2009 - 2012. Retrospective studies allow the researcher to estimate the effect of an exposure on an outcome and obtain measures of association, both of which are objectives of this study.

A quantitative research refers to the systematic empirical investigation of social phenomena via statistical, mathematical or numeric data, or computational techniques [130]. Using a quantitative design for this study will allow the researcher to explore the relationship between individual dietary macronutrients and covariates of interest and their effects on obesity.

Correlational studies should be used when independent variable variation has occurred without researcher control. In this study, the researcher is not able to control any of the independent variables; the variation within the independent variables occurred prior to data collection. All data is retrospective and; therefore, the researcher is unable to introduce any type of intervention, only examine the relationships between variables. The basic purpose of a correlational study is to determine the relationship between variables, but not the cause of this relationship. According to Triola (1998), coming to the conclusion that the results of a correlational study imply causality must be avoided [2,131].

3.1.3 Research Questions and Associated Hypothesis

Question 1:

Do different dietary macronutrients compositions lead to different risks of obesity?

H0:

Different dietary macronutrients compositions do not lead to different risks of obesity.

H1:

Different dietary macronutrients compositions lead to different risks of obesity.

Question 2:

Does diet in different compositions of macronutrients affect ethnic groups differently?

H0:

Diet in different compositions of macronutrients does not affect ethnic groups differently.

H1:

Diet in different compositions of macronutrients does affect ethnic groups differently.

Question 3:

Does a diet high in fat and sugar increases the risk of obesity relatively higher than either individual component?

H0:

Diet high in fat and sugar do not increase the risk of obesity relatively higher than either individual component.

H1:

Diet high in fat and sugar increases the risk of obesity relatively higher than either individual component.

Question 4:

Does a diet with significant amount of fiber and protein reduce the risk of obesity relatively higher than either individual component?

H0:

Diet with significant amount of fiber and protein do not reduce the risk of obesity relatively higher than either individual component.

H1:

Diet with significant amount of fiber and protein reduce the risk of obesity relatively higher than either individual component.

3.2 Data Sources

This research used an existing National Health and Nutrition Examination Survey (NHANES) data. The data was collected by the Centers for Disease Control (CDC), and is a cross – sectional sample of non-institutionalized U.S populations. The data is designed to access the health and nutrition status of adults and children in the

United States. The collection of the NHANES data started in the 1960s and contains information on the health and nutrition of the sample participants, which reflects the health and nutritional changes and trends amongst Americans. The data is collected in by annual cycles. Each cycle contains an average of [14000] children and adults with an average of about 5000 – 7000 adult respondents per cycle selected from all the counties in the United States. There are different subsets of data, which includes; demographic, examination, dietary, questionnaire, and laboratory data. The examination component consists of medical, dental and physiological measurements. The data is collected and administered by highly trained medical personnel. In addition to that it also contains measurements of blood pressure, information on sex, race, education and more. The NHANES dataset uses a stratified random sampling.

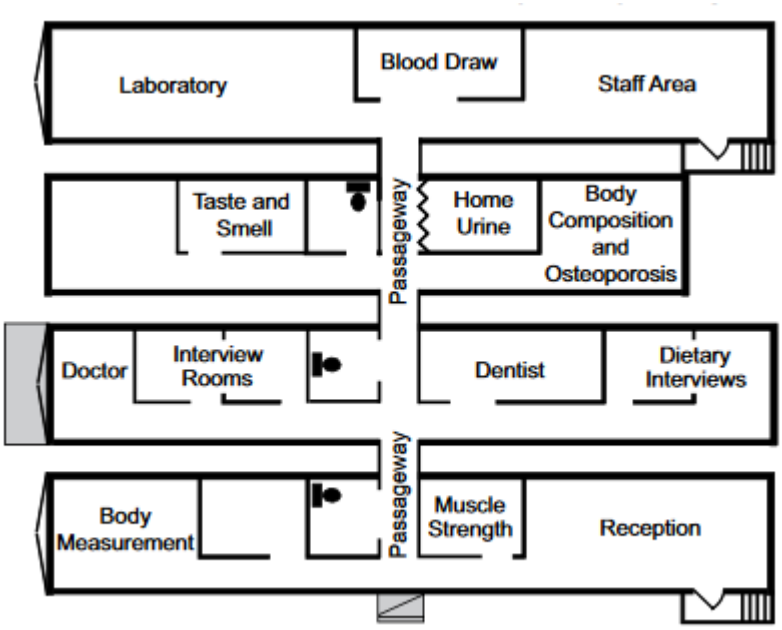


Figure 7 Diagram of CDC Medical Examination Centers (MEC) Source: CDC

3.2.1 Study Period and Study Group Inclusion/Exclusion Criteria

The research analyzed the NHANES data collected by CDC from 2009 – 2012. SAS version 9.3 and "R version 3.2.5" [An open source statistical package] were used for the statistical analysis and data cleaning. The NHANES data contains records of respondents aged a few months to over 80 years old. However children and adolescents 19 years and younger were excluded. Adults over 20 years old were the only respondents considered. Also all pregnant women were excluded from the sample used for the analysis. Weighting was used to ensure that descriptive statistics could be comparable to the national population.

3.3 NHANES Dataset and Elements

The NHANES data files downloaded are accessible to the public and they contain the follow variables:

Nutrition Variables:

1. SEQN – RESPONDENT SEQUENCE
2. DR1LINE – FOOD/INDIVIDUAL COMPONENT NUMBER
3. DR1TKCAL – ENERGY [KCAL]
4. DR1TPROT – PROTEIN [GM]
5. DR1TCARB – CARBOHYDRATE [GM]
6. DR1TSUGR – TOTAL SUGAR [GM]
7. DR1TFIBE – DIETARY FIBER [GM]
8. DR1TFAT – TOTAL FAT [GM]
9. DR1TSFAT – TOTAL SATURATED FATTY ACIDS [GM]
10. DR1TMFAT – TOTAL MONOSATURATED FATTY ACIDS [GM]
11. DR1TPFAT – TOTAL POLYSATURATED FATTY ACIDS [GM]
12. DR1_040Z – DID YOU EAT AT HOME [YES, NO]

Demographic Variables:

1. SEQN – RESPONDENT SEQUENCE
2. RIAGENDR – GENDER
3. RIDAGEYR – AGE IN YEARS AT SCREEINING
4. RIDRETH3 – RACE/HISPANIC ORIGIN W/NH ASIAN

5. DMDEDUC2 – EDUCATION LEVEL ADULT 20 +
6. RIDEXPRG – PREGNANCY STATUS AT EXAMINATION
7. WTINT2YR – FULL SAMPLE TWO YEAR INTERVIEW WEIGHT
8. WTMEC2YR – FULL SAMPLE TWO YEAR MCE EXAMINATION WEIGHT
9. SDMVSTRA – MARKED VARIANCE PSEUDO – STRATA
10. SDMVPSU – MARKED VARIANCE PSEUDO – PSU

Examination - Body Measures:

1. SEQN – RESPONDENT SEQUENCE
2. BMXBMI – BODY MASS INDEX [KG/M**2]

Questionnaire – Physical Activity:

1. BMXBMI – BODY MASS INDEX [KG/M**2]
2. PAQ650 – VIGOROUS RECREATIONAL ACTIVITIES
3. PAQ665 – MODERATE RECREATIONAL ACTIVITIES

The variable name SEQN was used to merge all the files for the analysis.

3.3.1 Data Cleaning and Data Processing

The NHANES data files were originally in SAS transport format with the extension XPT. The SAS datasets were extracted from the individual SAS transport files and saved in the normal SAS dataset format, with .sas7bdat extensions. The frequencies of the individual datasets were run and cross checked with NHANES reported numbers.

The SAS datasets were then merged into one dataset with all the variables from the individual component datasets to bring in all the variables needed for the analysis. After that process, all the needed variables were extracted from the merged file to create a smaller dataset with only the variables needed for the respective analysis. After that, the files were all condensed into one with the related variables extracted. The SAS (Proc Means, Freq) commands were run for all the variables to check for

their distributions, missing values and outliers. The content of the dataset for the cycles 2011 - 2012 was checked with the (Proc Content) command in SAS. The data cleaning process was repeated for the dataset cycle 2009 – 2010. All variable response codes were also checked referencing the code book as well and compared with the related dataset for the cycle 2009 – 2010 for consistency.

After the content and variables check were completed, the data set for the two cycles were appended to form a single dataset using the (Set) command in SAS. For all the categorical variables for which (Refused and Don't Know) were coded with 7, 9, or 77 and 99 were recoded as missing values. These included the following: DMDEDUC2, DMDMARTL, PAQ650 and PAQ655. In general the missing values were far less than 10% and were all eliminated from the analysis.

3.3.2 Demographic Data

As per CDC's claim, the demographic data was collected by interviewers who are trained to administer in English and Spanish. The Computer Assisted Personal Interview (CAPI) methodology was used by the interviewers in the process. Printed questionnaires were used in place of CAPI when its usage was not applicable in some circumstances. The demographic variables were checked with the respective code book referenced for details. Relevant variables were selected for the analysis and were also sorted and their frequencies checked to ensure consistency.

Details of the variables are all documented in an electronic code book. Also documents to enhance the analysis process are presented by CDC electronically on the NHANES related website. The code book and related documents sheds more light on

the variables, their functions and things to take into consideration whiles analyzing a particular cycle of data or merged multiple dataset for two or more cycles of data.

3.3.3 Dietary Data

As per CDC's claim, a 24-hour dietary recall interviews were conducted by trained Spanish and English data collectors on individuals with ages ranging from zero to eighty years and above. The interviews were done in Mobile Examination Centers (MEC) with standardized measures for all the respondents. The measuring tools for the standardized measures were intended to help respondents to quantify the amount of food they consume. A second dietary telephone recall is done within three to ten days after the first recall.

The first day interview data was used for the analysis. After carefully examining the data referencing the related code books, the variables with the totals of the macronutrients were selected. Frequencies and patterns were also checked for consistency. The first dietary recall interview is collected in person in the MEC, whiles the second interview is done by telephone three to ten days later.

3.3.4 Examination Data

Respondents are examined in MEC by medical and dental practitioners. Lots of medical records are taken including blood pressure readings, Body Mass Index and a lot more. There are multiple examination dataset depending of what is being considered. They were also examined in lure of the code book and the BMI (kg/m**2) was selected from the body measurement's file.

3.3.5 Questionnaire Data

Two variables were selected from the questionnaire data from the physical activity subset. They are:

PAQ650 – Vigorous recreational activities

PAQ665 – Moderate recreational activities

3.3.6 Variables and Coding

Table 3: Variables and Recommended Coding

VARIABLES	EXPLANATION	MEASURE	CODE
SEQN	Respondent sequence number	SCALE	
SDDSRVYR	Data release cycle	NOMINAL	7 -> NHANES 2011-2012 PUBLIC RELEASE 6 -> NHANES 2009-2010 PUBLIC RELEASE
RIAGENDR	Gender	NOMINAL	1 -> MALE 2 -> FEMALE . -> MISSING
RIDAGEYR	Age in years at screening	SCALE	
RIDRETH1	Race/Hispanic origin	NOMINAL	1 -> MEXICAN AMERICANS 2 -> OTHER HISPANICS 3 -> NH WHITES 4 -> NH BLACKS 5 -> OTHER RACE INCLUDING MULTI-RATIAL
DMDEDUC2	Education level - Adults 20+	ORDINAL	1 -> LESS THAN 9TH GRADE 2 -> 11-12 GRADE (INCLUDING 12TH GRADE WITH NO DIPLOMA) 3 ->HIGH SCHOOL GRADUATE OR GED EQUIVALENT 4 ->SOME COLLEGE OR AA DEGREE 5 -> COLLEGE GRADUATE OR ABOVE 7 -> REFUSED 9 -> DON'T KNOW . -> MISSING
DMDMARTL	Marital status	NOMINAL	1 -> MARRIED 2 -> WIDOWED 3 -> DIVORCED 4-> SEPARATED 5 -> NEVER MARRIED 6 -> LIVING WITH PARTNER 77 -> REFUSED 99 -> DON'T KNOW . -> MISSING
RIDEXPRG	Pregnancy status at exam	NOMINAL	1 -> YES, POSITIVE LAB TEST OR SELF REPORTED PREGNANCY AT EXAM 2 -> NOT PREGNANT AT EXAM 3 -> CAN'T ASCERTAIN IF PARTICIPANT IS PREGNANT AT EXAM 4 -> MISSING
WTINT2YR	Full sample 2 year interview weight	SCALE	
WTMEC2YR	Full sample 2 year MEC exam weight	SCALE	
SDMVPSU	Masked variance pseudo-PSU	NOMINAL	1 - 3 -> RANGE OF VALUES
SDMVSTRA	Masked variance pseudo-stratum	SCALE	
WTDRD1	Dietary day one sample weight	SCALE	
DR1TKCAL	Energy (kcal)	SCALE	
DR1TPROT	Protein (gm)	SCALE	
DR1TCARB	Carbohydrate (gm)	SCALE	
DR1TSUGR	Total sugars (gm)	SCALE	
DR1TFIBE	Dietary fiber (gm)	SCALE	
DR1TTFAT	Total fat (gm)	SCALE	
DR1TSFAT	Total saturated fatty acids (gm)	SCALE	
DR1TMFAT	Total monounsaturated fatty acids (gm)	SCALE	
DR1TPFAT	Total polyunsaturated fatty acids (gm)	SCALE	
DR1TCHOL	Cholesterol (mg)	SCALE	
BMXBMI	Body Mass Index (kg/m**2)	SCALE	
PAQ650	Vigorous recreational activities	NOMINAL	1 -> YES 2 -> NO 7 -> REFUSED 8 -> DON'T KNOW . -> MISSING
PAQ665	Moderate recreational activities	NOMINAL	1 -> YES 2 -> NO 7 -> REFUSED 8 -> DON'T KNOW . -> MISSING

3.3.7 Exposures and Outcomes

The main outcome variable was a transformation of the Body Mass Index variable BMXBMI into a categorical variable called “obesity” with responses Yes =1 and No = 0. This was done to enable the use of hierarchical logistic regression models for the inferential statistics. The major exposures variables used in the models were

the major macronutrients. They were proteins, carbohydrates, fiber, sugar and total fat, and were represented respectively as: DRITPROT, DRITCARB, DRITSUGR, DRITFIBE AND DRITTFAT. The confounding factors or variables were education, marital status, race and gender, moderate recreational activities, and vigorous recreational activities. These were also represented respectively as: DMDEDUC2, DMDMARTL, RIDETH1, RIAGENDR, PAQ655 and PAQ650.

3.4 Data Analysis Methods

Two cycles of NHANES [2009-2010 and 2011-2012] data were appended and used for the analysis. The combined data was cleaned as stated above. The missing values were less than 10% of the responses and were therefore ignored. The multi-level sample design was used as recommended in the NHANES related document. The complex sampling technique used SDMVSTRA, SDMVPSU, and WTMEC2YR respectively as the Strata, Cluster and Weight variables as recommended by CDC's NHANES. All the categorical variables were appropriately coded with CDC's related code book's recommendations. The complex sampling plan for the various datasets was finally used to run, respective related tests.

Collinearity test was performed and collinear variables were eliminated from the analysis. The following are the results of the collinearity test:

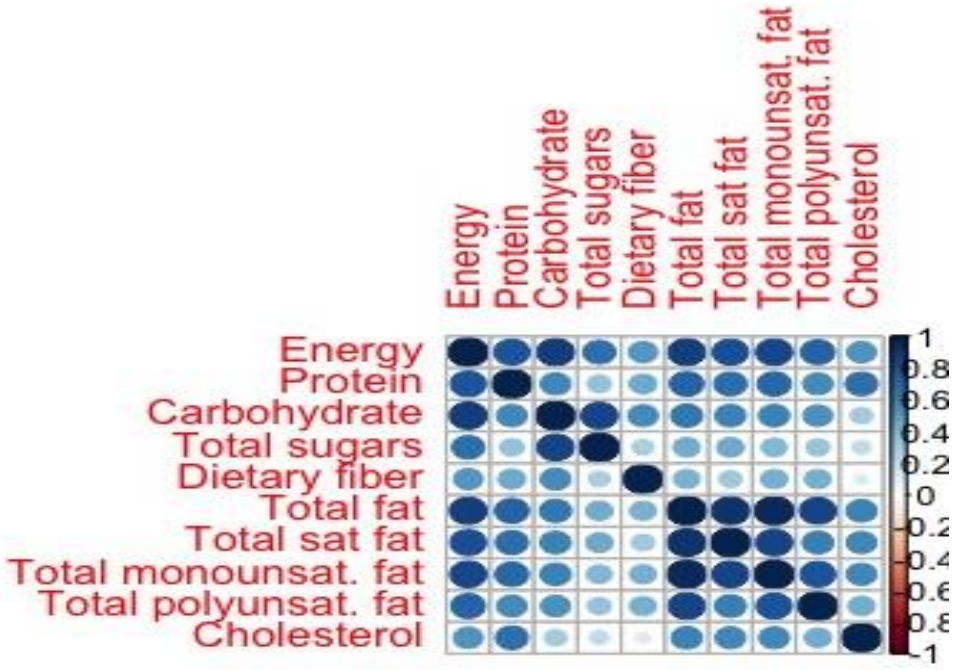


Figure 8: Collinearity Test Results

Table 4: Collinearity Test Results

	Energy	Protein	Carbohydrate	Total sugars	Dietary fiber	Total fat	Total sat fat	Total monounsat. fat	Total polyunsat. fat	Cholesterol
Energy	1	0.786	0.875	0.652	0.512	0.868	0.802	0.831	0.719	0.527
Protein	0.786	1	0.567	0.345	0.448	0.717	0.667	0.696	0.551	0.653
Carbohydrate	0.875	0.567	1	0.844	0.555	0.626	0.581	0.583	0.53	0.299
Total sugars	0.652	0.345	0.844	1	0.273	0.434	0.441	0.39	0.332	0.212
Dietary fiber	0.512	0.448	0.555	0.273	1	0.418	0.314	0.42	0.422	0.115
Total fat	0.868	0.717	0.626	0.434	0.418	1	0.914	0.968	0.841	0.582
Total sat fat	0.802	0.667	0.581	0.441	0.314	0.914	1	0.841	0.599	0.56
Total monounsat. fat	0.831	0.696	0.583	0.39	0.42	0.968	0.841	1	0.789	0.562
Total polyunsat. fat	0.719	0.551	0.53	0.332	0.422	0.841	0.599	0.789	1	0.434
Cholesterol	0.527	0.653	0.299	0.212	0.115	0.582	0.56	0.562	0.434	1

Polyunsaturated and monounsaturated fats and cholesterol were all collinear with total fat. They were hence all eliminated from the analysis and total fat was used instead.

The descriptive analysis included population count or frequencies, and table percent. The mean and standard deviations were calculated for all the respective scale

or numerical variables. For the inferential statistics, the hierarchical logistic regression models and analysis were used for the various hypothesis testing. The odds ratios of the various resulted estimates or beta values were also calculated. The Wald's test was then finally used to test the validity of the various hypotheses. The logit model is as follows:

Logit Model

$$\text{logit}\left(\Pr\left(Y=1\right)\right)=\log\left(\text{odds}\left(Y=1\right)\right)=\log\left[\frac{\Pr\left(Y=1\right)}{1-\Pr\left(Y=1\right)}\right]=\beta_0+\beta_1X_1+\beta_1X_2+\ldots+\beta_1X_j$$

Figure 9: Logit Model

The analyses were run with respect to the various hypotheses, and in light of the research questions being asked. The descriptive analyses for all the variables were calculated with all results recorded in excel tables. The results of the respective hierarchical logistic regressions and related odds ratio and other statistics were also imported and formatted in excel tables.

3.4.1 Data Analysis Plan

R version 3.2.5 was used to conduct the data analysis. All the analyses were done within 95% confidence interval (***α = .05)***. Data analysis was performed in several steps. Descriptive statistics were calculated for study variables including frequencies and percentages for categorical variables and means and standard deviations for continuous variables.

Hypothesis 1 was tested using hierarchical logistic regression analysis. Hypothesis 1 stated that consumption of different compositions of dietary macronutrients led to different risks of obesity. Hierarchical regression was performed in two steps. First, the relationships between demographic variables and likelihood of obesity were analyzed. Then, macronutrient intake was entered into the model. Odds ratios calculated for each macronutrient and Wald's test was used to compare the two models. The results of the regression models were then used to assess the validity of Hypothesis 1.

Hypothesis 2 was also tested using hierarchical logistic regression analysis. Hypothesis 2 stated that diet with different compositions of macronutrients affected the likelihood of obesity in different ethnic groups differently. A separate hierarchical regression model was performed for each nutrient, including, proteins, sugar, fat, fiber and carbohydrates. First the demographic variables and the consumption of the nutrient was entered into each model. Then, the interaction between the nutrient and race/ethnicity was entered into the model. The interactions were then tested for statistical significance using Wald's test in order to determine whether different compositions of macronutrients affected the likelihood of obesity in different ethnic groups differently. The results of the regression models were then used to assess the validity of Hypothesis 2.

Hypothesis 3 was also tested using hierarchical logistic regression analysis. Hypothesis 3 stated that diet high both in fat and sugar increases the risk of obesity to a greater degree than a diet high in either fat or sugar individually. The hierarchical logistic model was performed in three steps. First, demographic variables were entered

into the model. Second, fat consumption and sugar consumption were entered into the model. Finally, the interaction between sugar consumption and fat consumption was entered into the model. The models were compared using Wald's test and the interaction between fat consumption and sugar consumption was tested for statistical significance. The results of the regression models were then used to assess the validity of Hypothesis 3.

Hypothesis 4 was also tested using hierarchical logistic regression analysis. Hypothesis 4 stated that a diet high in both fiber and protein reduces the risk of obesity to a greater degree than a diet high in fiber or protein individually. The hierarchical logistic model was performed in three steps. First, demographic variables were entered into the model. Second, fiber consumption and protein consumption were entered into the model. Finally, the interaction between fiber consumption and protein consumption was entered into the model. The models were compared using Wald's test and the interaction between protein consumption and fiber consumption was tested for statistical significance. The results of the regression models were then used to assess the validity of Hypothesis 4.

CHAPTER 4

RESULTS

4.1 Introduction

In this chapter of the study, the data was analyzed to assess the validity of the hypotheses. Data from two NHANES data cycles 2009-2010 and 2011-2012 were used for the analysis, with children nineteen years or less and pregnant women excluded. Data analysis was performed in several steps. First, descriptive statistics were calculated. Second, data was analyzed using hierarchical regression analysis. Finally, the hypotheses of the current study were assessed for validity.

4.2 Results: Descriptive Statistics

Descriptive statistics were presented for both continuous variables and categorical variables. There were ten continuous variables used in this study including age, energy consumption (kcal/day), protein consumption (g/day), carbohydrate consumption (g/day), sugar consumption (g/day), fiber consumption (g/day), fat consumption (g/day), saturated fat consumption (g/day), monounsaturated fat consumption (g/day) and cholesterol consumption (g/day). However, saturated fat consumption (g/day), monounsaturated fat consumption (g/day), cholesterol consumption (g/day), and energy consumption (Kcal/day) were not used in the regression models due to collinearity; saturated fat consumption (g/day), monounsaturated fat consumption (g/day), and cholesterol consumption (g/day) were strongly correlated with fat consumption (g/day), and energy consumption (kcal/day)

was strongly correlated with protein consumption (g/day), carbohydrate consumption (g/day), and fat consumption (g/day).

Table 5 displays the means and standard deviations for the continuous variables of the study for the obese and non-obese participants. The average age for non-obese participants was 48.56 years, and 50.18 years for obese participants. The average energy consumption of the non-obese participants was slightly higher than the obese participants. They were 2138.83 Kcal and 2070.96 Kcal respectively. Amongst the macronutrients, the fat related nutrients were the only ones that the obese group is slightly higher than the non-obese group. The total fat, saturated fat, monounsaturated fat and cholesterol average consumption for the obese group was respectively; 78.31g, 25.36g, 28.34g and 290.71gm. On the other had the total fat, saturated fat, monounsaturated fat, and cholesterol average consumption for the non-obese group was respectively; 77.71g, 24.98g, 27.99g and 282.03g. The average consumption patterns for the rest of the macronutrients were different in direction. The non-obese group had a slightly higher averages that the obese group. The average consumption of protein, carbohydrate, sugar and fiber for the non-obese group were respectively, 81.9g, 263.88g, 117.1g, 17.34 g whiles those for the obese group were; 80.31g, 252.51g, 112.83g, and 16.23g.

Table 5: Summary Statistics NHANES 2009 – 2012 for Obese and Non Obese Adults

	Non Obese		Obese	
	Mean	SD	Mean	SD
Age	48.56	18.36	50.18	16.51
Energy (kcal)	2138.83	993.47	2070.96	972.31
Protien (g)	81.9	41.78	80.31	40.84
Carbohydrate (g)	263.88	127.68	252.51	124.41
Total Sugar (g)	117.1	80.39	112.83	77.55
Total Fiber (g)	17.34	10.7	16.23	10.17
Total Fat (g)	77.71	45.09	78.31	45.2
Total saturated fat (g)	24.98	16.41	25.36	15.88
Total monounsaturatesaturated fat (g)	27.99	17.19	28.34	17.47
Total cholesterol (g)	282.03	233.77	290.71	225.56

Table 6 displays average composition of macronutrient consumption by race/ethnicity. On average, non-Hispanic whites consumed the most calories (2160.12 Kcal/day), followed by Mexican Americans (2156.06 Kcal/day). Participants from other races including multiracial groups consumed the least calories on the average (1983.04 Kcal/day). Mexican Americans also had the highest level of protein consumption (85.72 g/day), followed by non-Hispanic whites (81.19 g/day). Non-Hispanic blacks had the lowest protein consumption (79.47 g/day).

On average, Mexican Americans consumed the largest amount of carbohydrates (272.38 g/day), followed by the non-Hispanic whites (261.07 g/day). Hispanics, other than Mexican Americans, on average, consumed the least amount of carbohydrates (249.91 g/day).

Non-Hispanic whites also had the highest level of sugar consumption (120.7 g/day). Non-Hispanic blacks had the second highest level of sugar consumption (118.91 g/day), followed by Mexican Americans (113.53 g/day). The lowest level of

sugar consumption was in participants from other races and multiracial participants (95.13 g/day).

Mexican Americans ranked highest in level of fiber intake (20.12 g/day), followed by participants of other races and multiracial participants (18.13 g/day). Non-Hispanic whites had an average daily fiber consumption of 16.96 g/day. Additionally, the lowest level of fiber consumption was in Non-Hispanic Blacks (14.53 g/day). On average, Non-Hispanic whites consumed more fat than other racial groups (81.94 g/day), followed by Non-Hispanic blacks (79.18 g/day).

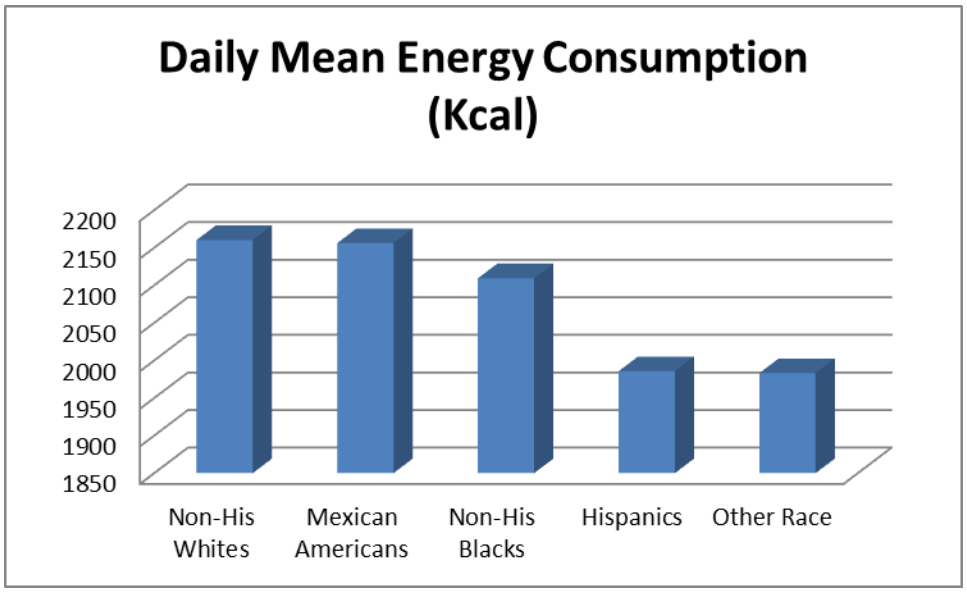


Figure 10: Daily Mean Energy Consumption (Kcal)

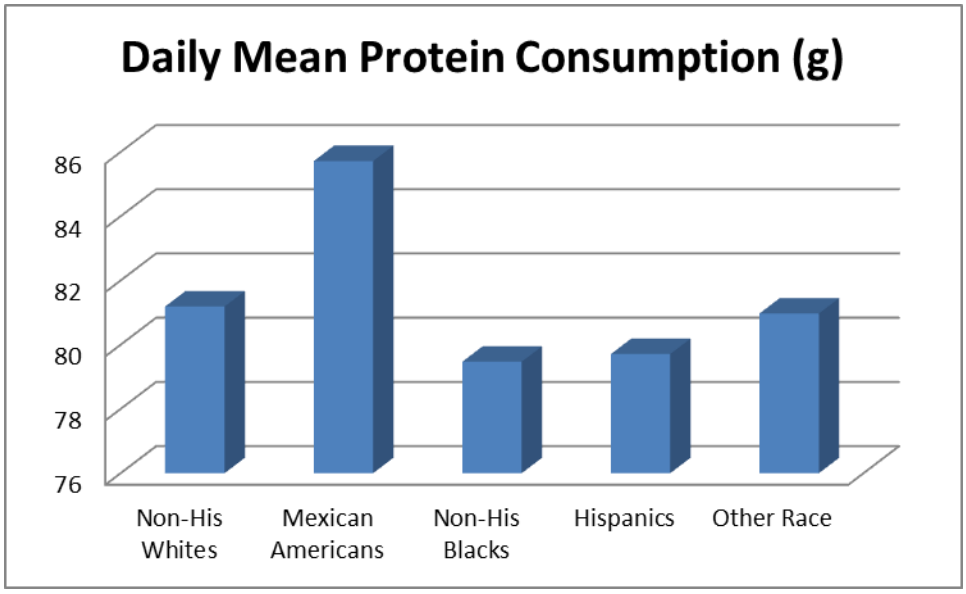


Figure 11: Daily Mean Protein Consumption (g)

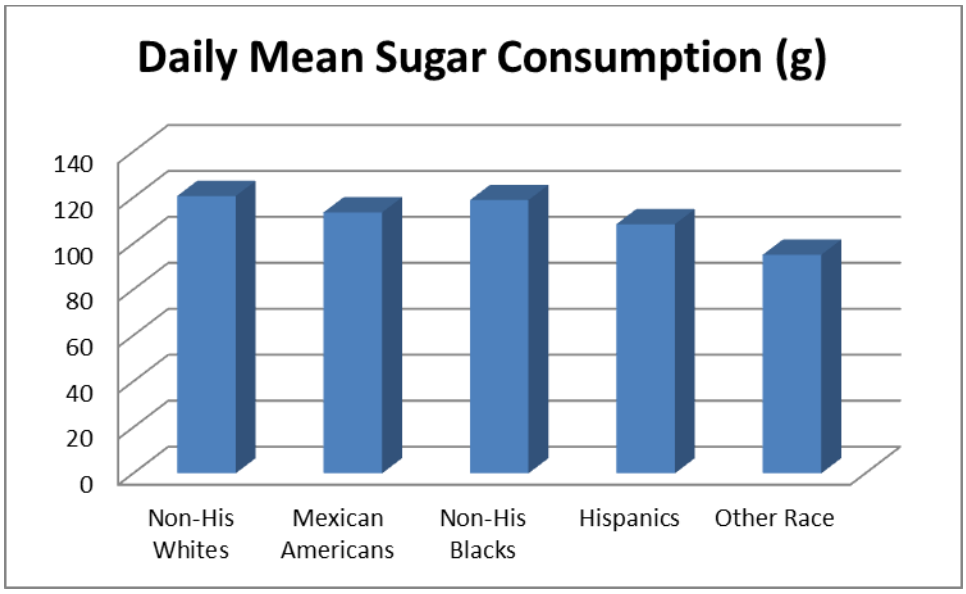


Figure 12: Daily Mean Sugar Consumption (g)

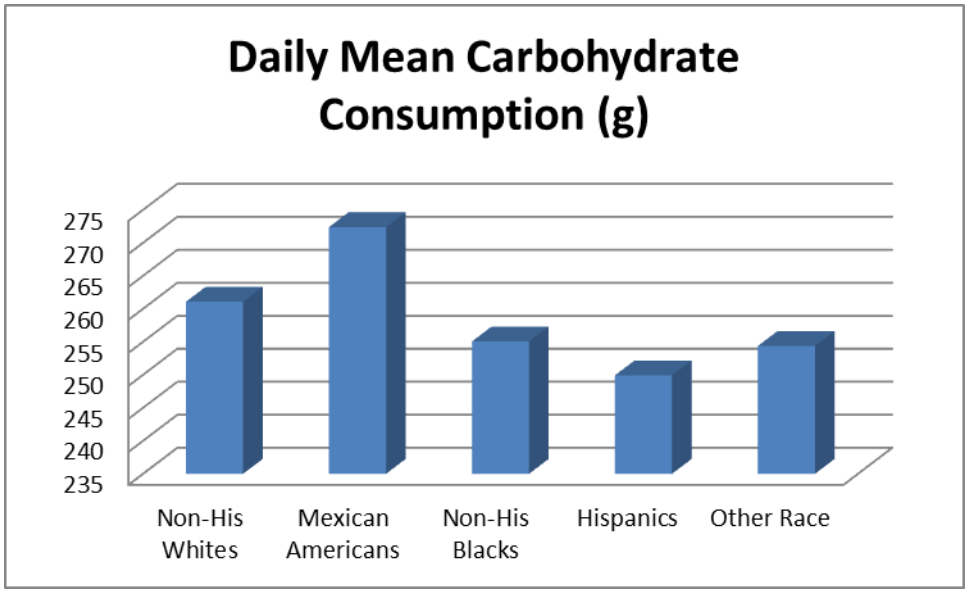


Figure 13: Daily Mean Carbohydrate Consumption (g)

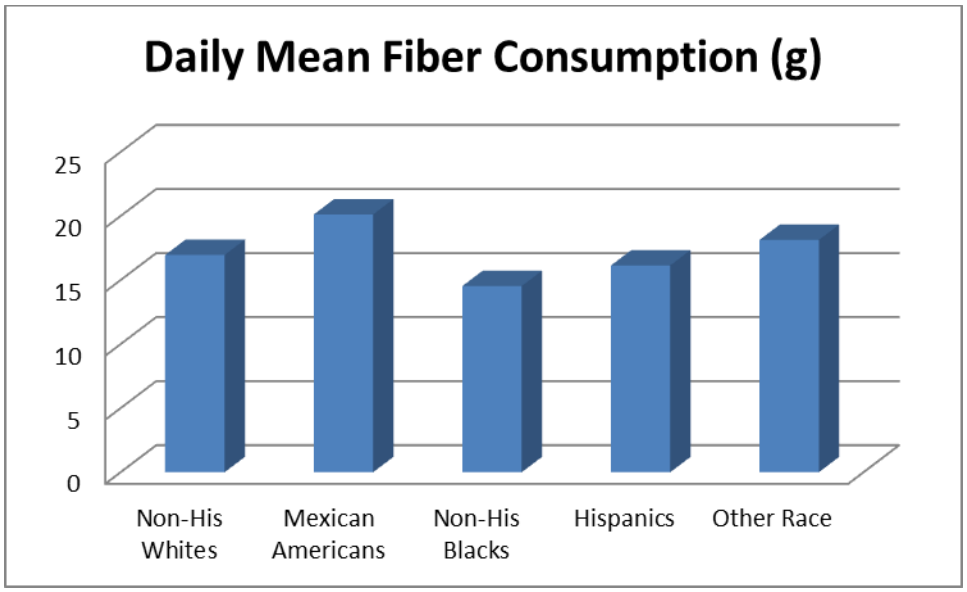


Figure 14: Daily Mean Fiber Consumption (g)

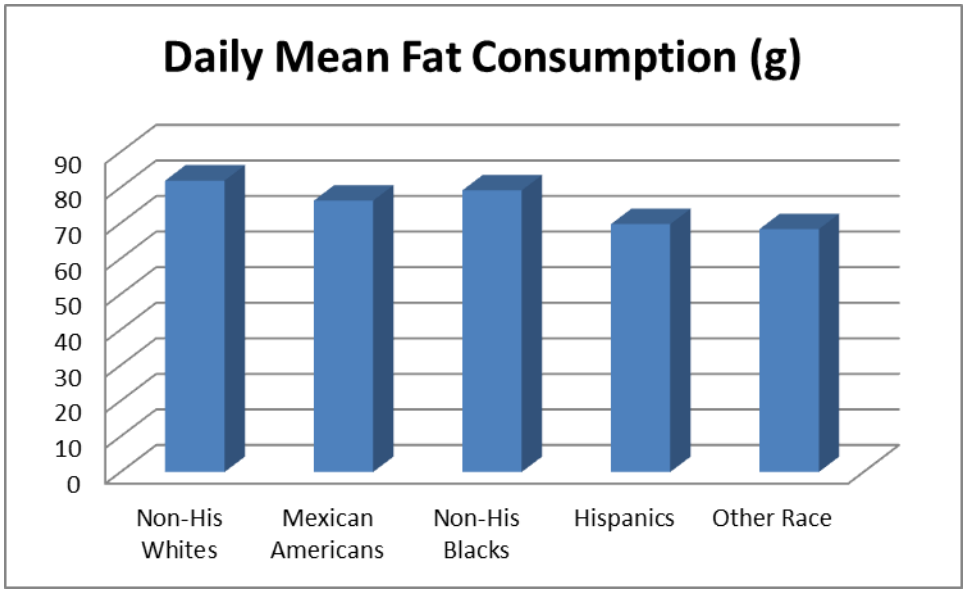


Figure 15: Daily Mean Fat Consumption (g)

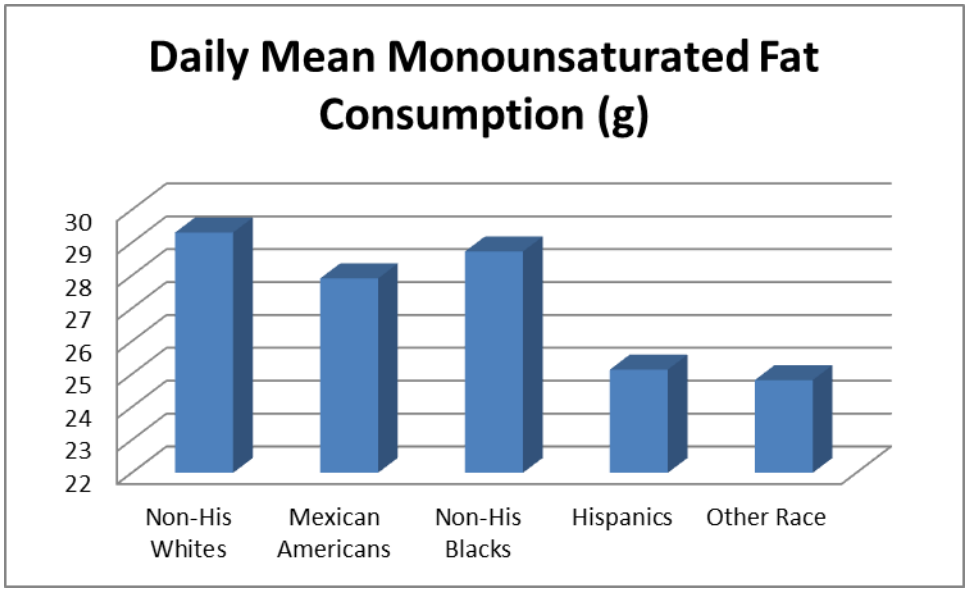


Figure 16: Daily Mean Monounsaturated Fat Consumption (g)

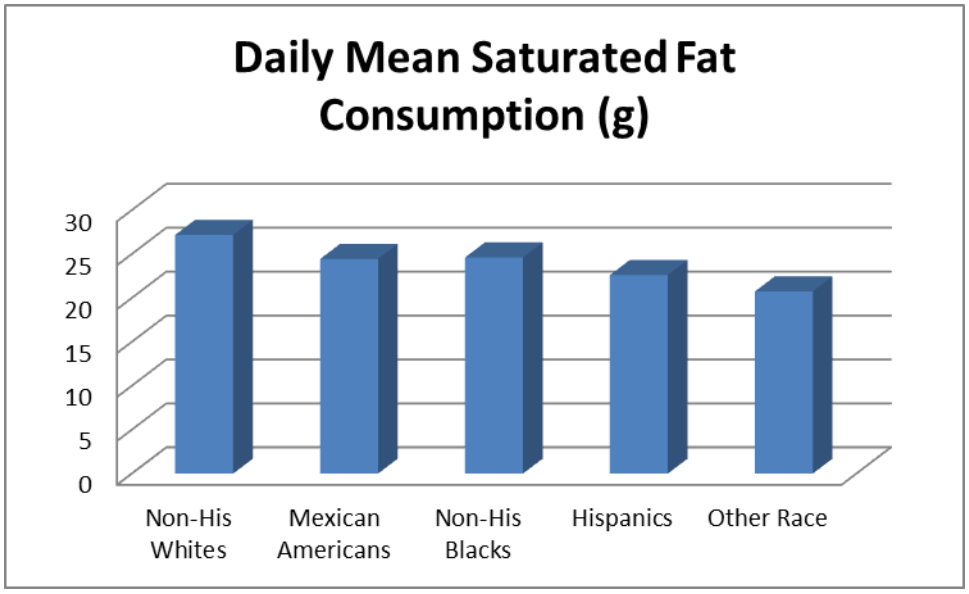


Figure 17: Daily Mean Saturated Fat Consumption (g)

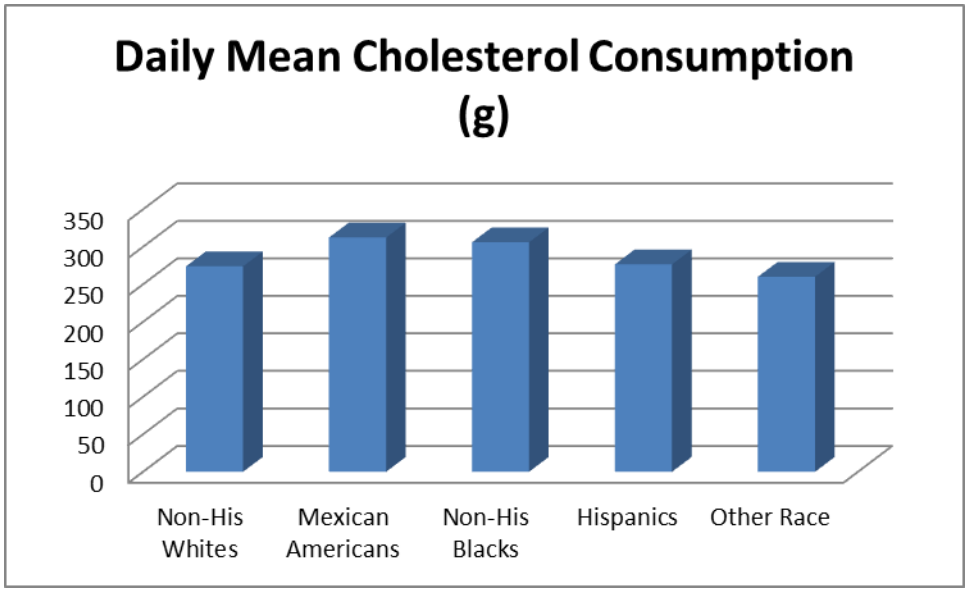


Figure 18: Daily Mean Cholesterol Consumption (g)

Table 6: Summary Statistics NHANES 2009 – 2012 Macronutrients Consumption by Race

Race/Ethnicity	Macronutrient	Mean	SD
Non-Hispanic Whites	Energy (kcal)	2160.12	977.18
	Protien (gm)	81.19	40.33
	Carbohydrate (gm)	261.07	127.71
	Total Sugar (gm)	120.7	84.83
	Total Fiber (gm)	16.96	10.27
	Total Fat (gm)	81.94	45.57
	Total saturated fat (gm)	27.18	16.88
	Total monounsaturatesaturated fat (gm)	29.31	17.38
	Total cholesterol (gm)	273.91	219.53
Mexican Americans	Energy (kcal)	2156.06	973.3
	Protien (gm)	85.72	43.82
	Carbohydrate (gm)	272.38	125.97
	Total Sugar (gm)	113.53	72.69
	Total Fiber (gm)	20.12	11.97
	Total Fat (gm)	76.3	43.84
	Total saturated fat (gm)	24.44	15.33
	Total monounsaturatesaturated fat (gm)	27.91	17.2
	Total cholesterol (gm)	312.32	253.85
Non-Hispanic Blacks	Energy (kcal)	2109.28	1050.48
	Protien (gm)	79.47	42.03
	Carbohydrate (gm)	255.05	132.2
	Total Sugar (gm)	118.91	80.21
	Total Fiber (gm)	14.53	9.43
	Total Fat (gm)	79.18	47
	Total saturated fat (gm)	24.61	16.23
	Total monounsaturatesaturated fat (gm)	28.73	17.76
	Total cholesterol (gm)	305.95	244.6
Other Hispanics	Energy (kcal)	1985.64	976.01
	Protien (gm)	79.71	40.43
	Carbohydrate (gm)	249.91	121.92
	Total Sugar (gm)	108.42	71.09
	Total Fiber (gm)	16.14	9.39
	Total Fat (gm)	69.69	41.75
	Total saturated fat (gm)	22.61	14.84
	Total monounsaturatesaturated fat (gm)	25.14	15.91
	Total cholesterol (gm)	276.51	216.92
Other Races Including Multiracial	Energy (kcal)	1983.04	881.75
	Protien (gm)	80.97	41.96
	Carbohydrate (gm)	254.37	112.29
	Total Sugar (gm)	95.13	64.64
	Total Fiber (gm)	18.13	11.29
	Total Fat (gm)	68.28	41.44
	Total saturated fat (gm)	20.74	14.25
	Total monounsaturatesaturated fat (gm)	24.82	16.6
	Total cholesterol (gm)	260	219.14

Descriptive statistics for categorical variables were also calculated. Table 7 displays descriptive statistics for both obese and non-obese participants. From table 7 below, amongst the obese, 55.00% were women and 45.00% are men. With the perspective of race, 41.11% of Non-Hispanic Whites were obese as compared to the

total obese population. The next in rank was Non-Hispanic Black with 27.59% of the total obese population. Mexican Americans came next with 16.43%, and other Hispanics and other races including multiracial groups followed with 10.18% and 4.69% respectively.

From the overall adult obese population, again 23.88% were high school graduates or GED equivalent in level of education, and 30.92% had some college exposure or AA degree. In this category, 17.82% were having college degrees or above whiles 16.06% had some high school exposure. Those with less than 9th grade exposure in education constituted 11.32% of the population. The distribution for marital status was as followed; 50.12% of the obese adults were married, whiles 17.84% had never married. In this population, 12.09% were divorced and 8.82% were widowed. Those who were living with partners constituted 7.30% of the population, whiles 3.84% were separated from their spouses.

Table 7: Summary Statistics NHANES 2009 – 2012 Frequency Distribution and Percent

		Not Obese		Obese	
		N	%	N	%
Gender	Female	3078	47.68	2135	55.00
	Male	3377	52.32	1747	45.00
Race	Non-Hispanic Whites	2935	45.47	1596	41.11
	Mexican Americans	856	13.26	638	16.43
	Non-Hispanic Blacks	1174	18.19	1071	27.59
	Other Hispanics	642	9.95	395	10.18
	Other Races Including Multiracial	848	13.14	182	4.69
Education	High School Graduate or GED Equivalent	1355	21.03	926	23.88
	11th to 12th Grade	936	14.53	623	16.06
	College Graduate or Above	1660	25.76	691	17.82
	Less than 9th Grade	677	10.51	439	11.32
	Some College or AA Degree	1816	28.18	1199	30.92
Marital Status	Married	3214	49.81	1944	50.12
	Divorced	651	10.09	469	12.09
	Living with Partner	547	8.48	283	7.30
	Never Married	1315	20.38	692	17.84
	Seperated	214	3.32	149	3.84
	Widowed	512	7.93	342	8.82
Vigerous Recreational	No	4901	75.93	3329	85.75
Activity	Yes	1554	24.07	553	14.25
Moderate	No	3684	57.08	2483	63.96
Recreational Activity	Yes	2770	42.92	1399	36.04

Table 8 displays the percent of obese and non-obese participants for the value of each categorical variable. Fifty-nine percent of females were non-obese, whiles 41.0% were obese. There were 65.9% of men that were non-obese whiles 34.1% of them were obese. These percentages indicated that, there were relatively more women who were obese within their category than men.

Ethnicity followed gender as the next category on the table. Within the Non-Hispanic Whites, 64.8% were non-obese whiles, 35.2% were obese. Relatively, they had the least percentage of obese within their group, besides the other races including multiracial. This group had about 82.3% of them as non-obese, whiles 17.7% of them were obese. They included Asians, and all other races besides, the major categories such as Non-Hispanic Whites, Non-Hispanic Blacks, Mexican Americans, and Other

Hispanics. Amongst the major groups, other Hispanics came next to non-Hispanic Whites with 61.9% of them as non-obese and 38.1% obese. Mexican Americans followed with 57.3% non-obese and 42.7% obese. The group with the highest relative rates of obese within the group was the non-Hispanic Blacks. In this group there were 52.3% obese and 47.7% non-obese.

College graduates and above had the least obesity rate within their group, with 70.6% of them non-obese and 29.4% obese. For the rest of the education level categories the numbers were very close. Those with less than 9^{th} grade of education had 60.7% of them as non-obese, whiles 39.3% of them were obese. The next were those with some college or AA degree. Amongst them, 60.2% of them were non-obese, whiles 39.8% of them were obese. For those with 11^{th} to 12^{th} grade of education, 60% were non-obese whiles 40% were obese. High School Graduates of GED equivalent had 59.4% of them as non-obese and 40.6% of them as obese.

For the marital status category, those living with partners, turned out the least obese within group, with 65.9% of them non-obese and 34.1% obese. They were followed by those who had never married. Amongst them, 65.5% were non-obese and 34.5% obese. After these groups, married couple appeared to be the next in rank, with 62.3% of them obese and 37.7% non-obese. Those who were separated and those who were widowed come close with one percentage point difference in the obese and non-obese numbers. The widows had 60.0% of them as non-obese and 40.0% of them obese, whiles those who were separated had, 59.0% as non-obese and 41.0% obese. The divorced group had the most obese within group. 58.1% of them were non-obese and 41.9% of them were obese.

The physical activity category had a very striking difference between those who performed some level of physical activity and those who did not. For the rigorous physical activity sub category, amongst those who said no to rigorous physical activities, 59.6% were non-obese and 40.4% were obese. On the other hand, for those who said yes to rigorous physical activities, 73.8% were non-obese and 26.2% were obese. Within the moderate physical activities subcategory, 59.7% of those who said no were non obese and 40.3% were obese. For all those who said yes to moderate physical activity however, 66.4% we non-obese whiles 33.6% were obese.

From the above, it appears that the following categories and their related subcategories have some level of association with obesity; Gender, Race/Ethnicity, Education, Marital Status, Rigorous Physical Activity and Moderate Physical activity.

Table 8: Summary Statistics NHANES 2009 – 2012 Row Percent [Obese-Non Obese]

		Not Obese	Obese
Gender	Female	59.0%	41.0%
	Male	65.9%	34.1%
Race/Ethnicity	Non-Hispanic Whites	64.8%	35.2%
	Mexican Americans	57.3%	42.7%
	Non-Hispanic Blacks	52.3%	47.7%
	Other Hispanics	61.9%	38.1%
	Other Races Including Multiracial	82.3%	17.7%
Education	High School Graduate or GED Equivalent	59.4%	40.6%
	11th to 12th Grade	60.0%	40.0%
	College Graduate or Above	70.6%	29.4%
	Less than 9th Grade	60.7%	39.3%
	Some College or AA Degree	60.2%	39.8%
Marital Status	Married	62.3%	37.7%
	Divorced	58.1%	41.9%
	Living with Partner	65.9%	34.1%
	Never Married	65.5%	34.5%
	Seperated	59.0%	41.0%
	Widowed	60.0%	40.0%
Rigorous Physical Activity	No	59.6%	40.4%
	Yes	73.8%	26.2%
Moderate Physical Activity	No	59.7%	40.3%
	Yes	66.4%	33.6%

4.3 Achievement of Objectives of the Study

4.3.1 Objectives of Hypothesis 1

Hypothesis 1 was analyzed using hierarchical logistic regression analysis. Hypothesis 1 stated that consumption of different compositions of dietary macronutrients compositions led to different risks of obesity.

Analysis was conducted in two steps. First, the demographic variables were entered into the model and their respective odds ratios and *p*-values calculated.

Demographic variables included level physical activity, gender, age, race/ethnicity highest completed level of education, and marital status. Then, the macronutrients were introduced to the model including total fiber consumption (g/day), total carbohydrate consumption (g/day), total protein consumption (g/day), total sugar consumption (g/day), and total fat consumption (g/day). In order to test the effect of the macronutrients on obesity for statistical significance, Wald's test was performed.

According to the analysis, consumption of different compositions of dietary macronutrients compositions led to different risks of obesity, Wald = 8.081, $p < 0.01$. Higher levels of fiber consumption were associated with a lower risk of obesity, β = -.012, OR = 0.988, OR 95% CI (0.978, 0.998), $p < 0.05$. Fat consumption was not significantly associated with obesity risk. However higher levels of fat consumption was associated with higher risk of obesity, β = .002, OR = 1.002, OR 95% CI (1, 1.004), however $p > 0.05$. Higher levels of sugar consumption were associated with a lower risk of obesity, β = -.002, OR = 0.998, OR 95% CI (0.996, 0.999), $p < 0.05$. There was no statistically significant relationship between protein consumption, carbohydrate consumption, and obesity risk.

Table 9: Hypothesis 1 Model Results

	Model 1				Model 2			
	β	*OR*	*OR* 95% CI	*p* -value	*β*	*OR*	*OR* 95% CI	*p* -value
(Intercept)	-0.355	0.701	(0.561, 0.877)	0.007	-0.371	0.690	(0.531, 0.898)	0.018
Moderate Physical Activity	-0.660	0.517	(0.443, 0.602)	<.001	-0.649	0.522	(0.451, 0.605)	<.001
Vigerous Physical Activity	-0.138	0.871	(0.788, 0.963)	0.016	-0.127	0.881	(0.793, 0.978)	0.037
Gender: Female								
Gender: Male	0.011	1.011	(0.907, 1.126)	0.850	-0.030	0.970	(0.849, 1.109)	0.667
Age	0.001	1.001	(0.997, 1.005)	0.555	0.002	1.002	(0.998, 1.006)	0.409
Race/Ethnicity: Non-Hispanic White								
Race/Ethnicity: Mexican Americans	0.379	1.461	(1.236, 1.726)	<.001	0.406	1.502	(1.264, 1.783)	0.001
Race/Ethnicity: Non-Hispanic Blacks	0.622	1.862	(1.608, 2.157)	<.001	0.609	1.838	(1.589, 2.127)	<.001
Race/Ethnicity: Other Hispanics	0.141	1.152	(0.958, 1.384)	0.151	0.147	1.159	(0.964, 1.393)	0.145
Race/Ethnicity: Other Races Including Multiracial	-0.599	0.549	(0.458, 0.659)	<.001	-0.595	0.552	(0.455, 0.668)	<.001
Education: Graduated High School or GED								
Education: 11th to 12th Grade	-0.197	0.821	(0.702, 0.96)	0.025	-0.189	0.828	(0.708, 0.968)	0.038
Education: College Graduate or Above	-0.303	0.739	(0.623, 0.876)	0.003	-0.288	0.749	(0.627, 0.896)	0.009
Education: Less than 9th Grade	-0.240	0.787	(0.658, 0.941)	0.018	-0.219	0.804	(0.673, 0.96)	0.035
Education: Some College or AA Degree	0.049	1.051	(0.9, 1.227)	0.540	0.053	1.054	(0.91, 1.222)	0.497
Marital Status: Married								
Marital Status: Divorced	0.143	1.154	(0.971, 1.371)	0.124	0.145	1.156	(0.965, 1.384)	0.144
Marital Status: Living with Partner	-0.310	0.733	(0.618, 0.87)	0.003	-0.309	0.734	(0.617, 0.873)	0.005
Marital Status: Never Married	-0.196	0.822	(0.736, 0.918)	0.003	-0.193	0.824	(0.737, 0.923)	0.006
Marital Status: Seperated	-0.189	0.828	(0.628, 1.091)	0.198	-0.196	0.822	(0.62, 1.089)	0.200
Marital Status: Widowed	-0.069	0.933	(0.708, 1.231)	0.630	-0.067	0.936	(0.712, 1.229)	0.641
Total Protein					0.001	1.001	(0.999, 1.004)	0.274
Total Carbohydrate					0.001	1.001	(0.999, 1.002)	0.413
Total Fiber					-0.012	0.988	(0.979, 0.998)	0.031
Total Fat					0.002	1.002	(1, 1.004)	0.073
Total Sugar					-0.002	0.998	(0.996, 0.999)	0.025
Wald's Test							8.081	
p-value							0.002	

4.3.2 Objectives of Hypothesis 2

Hypothesis 2 was analyzed using hierarchical logistic regression analysis. Hypothesis 2 stated that consumption of different compositions of macronutrients affects different ethnic groups differently.

Analysis was conducted for each macronutrient, including protein, fat, carbohydrates, fat, and sugar. Analysis was performed in several steps. First, the demographic variables and the macronutrient were entered into the model and their respective odds ratios and *p*-values calculated. Demographic variables included level physical activity, gender, age, race/ethnicity highest completed level of education, and marital status. Then, the interactions between macronutrients and race/ethnicity entered into the model. In order to test the effect of the interaction between macronutrients and race/ethnicity on obesity for statistical significance, Wald's test was performed.

According to the results, diet consisting of different compositions of macronutrients did not affect ethnic groups differently. There was no statistically significant relationship between the interaction between protein and race/ethnicity, carb and race/ethnicity, fiber and race/ethnicity, total fat and race/ethnicity, and sugar and race/ethnicity, and likelihood of obesity, $Wald = 2.32$, $p = 0.12$; $Wald = 1.93$, $p = 0.17$; $Wald = 2.18$, $p = 0.13$; $Wald = 1.01$, $p = 0.45$; $Wald = 2.86$, $p = 0.08$.

Table 10: Hypothesis 2 Model Results

	Model 1(Protein)				Model 2 (Carb)			
	β	*OR*	*OR* 95% CI	*p* -value	*β*	*OR*	*OR* 95% CI	*p* -value
(Intercept)	-0.561	0.571	(0.427, 0.764)	0.003	-0.239	0.788	(0.612, 1.013)	0.090
Moderate Physical Activity	-0.666	0.514	(0.442, 0.598)	<.001	-0.660	0.517	(0.444, 0.602)	<.001
Vigerous Physical Activity	-0.140	0.869	(0.785, 0.963)	0.021	-0.138	0.871	(0.789, 0.963)	0.020
Gender: Female								
Gender: Male	-0.036	0.965	(0.853, 1.092)	0.583	0.044	1.045	(0.928, 1.177)	0.483
Age	0.002	1.002	(0.998, 1.006)	0.432	0.001	1.001	(0.997, 1.005)	0.775
Race/Ethnicity: Non-Hispanic White								
Race/Ethnicity: Mexican Americans	0.636	1.889	(1.252, 2.852)	0.011	0.459	1.582	(1.153, 2.172)	0.016
Race/Ethnicity: Non-Hispanic Blacks	0.917	2.501	(1.873, 3.339)	<.001	0.661	1.937	(1.482, 2.531)	0.001
Race/Ethnicity: Other Hispanics	0.323	1.381	(0.955, 1.999)	0.114	0.489	1.631	(1.174, 2.265)	0.014
Race/Ethnicity: Other Races Including Multiracial	-0.334	0.716	(0.439, 1.167)	0.207	-0.944	0.389	(0.231, 0.656)	0.005
Education: Graduated High School or GED								
Education: 11th to 12th Grade	-0.195	0.823	(0.704, 0.961)	0.032	-0.197	0.821	(0.701, 0.961)	0.032
Education: College Graduate or Above	-0.310	0.733	(0.618, 0.87)	0.005	-0.310	0.733	(0.617, 0.871)	0.005
Education: Less than 9th Grade	-0.244	0.783	(0.655, 0.936)	0.021	-0.256	0.774	(0.65, 0.921)	0.015
Education: Some College or AA Degree	0.045	1.046	(0.896, 1.221)	0.582	0.045	1.046	(0.897, 1.219)	0.578
Marital Status: Married								
Marital Status: Divorced	0.149	1.160	(0.978, 1.377)	0.116	0.143	1.153	(0.972, 1.369)	0.131
Marital Status: Living with Partner	-0.309	0.734	(0.619, 0.87)	0.004	-0.305	0.737	(0.621, 0.875)	0.005
Marital Status: Never Married	-0.189	0.828	(0.74, 0.926)	0.007	-0.194	0.824	(0.736, 0.922)	0.006
Marital Status: Seperated	-0.192	0.826	(0.626, 1.089)	0.202	-0.186	0.831	(0.629, 1.096)	0.217
Marital Status: Widowed	-0.066	0.936	(0.709, 1.236)	0.649	-0.066	0.936	(0.71, 1.234)	0.649
Nutrient	0.002	1.002	(1.001, 1.004)	0.019	0.000	1.000	(0.999, 1)	0.104
Nutrient x Race (Non- Hispanic White)								
Nutrient x Race (Mexican American)	-0.003	0.997	(0.993, 1.001)	0.149	0.000	1.000	(0.999, 1.001)	0.568
Nutrient x Race (Non-Hispanic Hispanic Black)	-0.004	0.996	(0.994, 0.999)	0.027	0.000	1.000	(0.999, 1.001)	0.724
Nutrient x Race (Other Hispanic)	-0.002	0.998	(0.994, 1.002)	0.327	-0.001	0.999	(0.997, 1)	0.043
Nutrient x Race (Other Races Including Multiracial)	-0.003	0.997	(0.991, 1.003)	0.340	0.001	1.001	(0.999, 1.003)	0.200
Wald's Test(to test significance of nutrient race interactions)			2.35				1.93	
p-value			0.12				0.17	

Table 11: Hypothesis 2 Model Results

	Model 3 (Fiber)				Model 4 (Total Fat)			
	β	*OR*	*OR* 95% CI	*p* -value	*β*	*OR*	*OR* 95% CI	*p* -value
(Intercept)	-0.266	0.766	(0.593, 0.991)	0.068	-0.521	0.594	(0.444, 0.795)	0.005
Moderate Physical Activity	-0.647	0.523	(0.449, 0.611)	<.001	-0.653	0.520	(0.447, 0.605)	<.001
Vigerous Physical Activity	-0.129	0.879	(0.793, 0.973)	0.031	-0.139	0.870	(0.786, 0.963)	0.021
Gender: Female								
Gender: Male	0.032	1.033	(0.927, 1.151)	0.572	-0.027	0.974	(0.869, 1.091)	0.657
Age	0.001	1.001	(0.997, 1.005)	0.505	0.002	1.002	(0.998, 1.006)	0.415
Race/Ethnicity: Non-Hispanic White								
Race/Ethnicity: Mexican Americans	0.214	1.238	(0.94, 1.632)	0.157	0.511	1.668	(1.189, 2.338)	0.013
Race/Ethnicity: Non-Hispanic Blacks	0.558	1.747	(1.304, 2.34)	0.003	0.794	2.213	(1.689, 2.9)	<.001
Race/Ethnicity: Other Hispanics	0.413	1.511	(1.098, 2.079)	0.028	0.416	1.516	(1.009, 2.279)	0.070
Race/Ethnicity: Other Races Including Multiracial	-0.572	0.565	(0.375, 0.851)	0.020	-0.608	0.545	(0.388, 0.764)	0.005
Education: Graduated High School or GED								
Education: 11th to 12th Grade	-0.198	0.820	(0.701, 0.96)	0.031	-0.196	0.822	(0.704, 0.959)	0.013
Education: College Graduate or Above	-0.280	0.756	(0.637, 0.897)	0.008	-0.306	0.736	(0.621, 0.873)	<.001
Education: Less than 9th Grade	-0.249	0.780	(0.653, 0.931)	0.019	-0.245	0.783	(0.654, 0.938)	0.070
Education: Some College or AA Degree	0.057	1.058	(0.911, 1.23)	0.473	0.045	1.046	(0.897, 1.219)	0.005
Marital Status: Married								
Marital Status: Divorced	0.139	1.149	(0.962, 1.372)	0.155	0.138	1.148	(0.967, 1.363)	0.144
Marital Status: Living with Partner	-0.312	0.732	(0.615, 0.871)	0.005	-0.311	0.733	(0.618, 0.87)	0.005
Marital Status: Never Married	-0.199	0.819	(0.732, 0.917)	0.005	-0.191	0.826	(0.739, 0.922)	0.006
Marital Status: Seperated	-0.194	0.824	(0.624, 1.087)	0.197	-0.190	0.827	(0.626, 1.092)	0.207
Marital Status: Widowed	-0.074	0.929	(0.704, 1.225)	0.610	-0.071	0.932	(0.706, 1.229)	0.626
Nutrient	-0.007	0.993	(0.985, 1)	0.092	0.002	1.002	(1, 1.004)	0.054
Nutrient x Race (Non- Hispanic White)								
Nutrient x Race (Mexican American)	0.010	1.010	(0.999, 1.021)	0.110	-0.001	0.999	(0.996, 1.001)	0.350
Nutrient x Race (Non-Hispanic Hispanic Black)	0.003	1.003	(0.99, 1.017)	0.619	-0.002	0.998	(0.995, 1.001)	0.146
Nutrient x Race (Other Hispanic)	-0.017	0.983	(0.963, 1.004)	0.138	-0.003	0.997	(0.992, 1.001)	0.148
Nutrient x Race (Other Races Including Multiracial)	-0.001	0.999	(0.978, 1.02)	0.905	0.001	1.001	(0.996, 1.006)	0.832
Wald's Test(to test significance of nutrient race interactions)			2.18				1.01	
p-value			0.13				0.45	

Table 12: Hypothesis 2 Model Results

	Model 5 (Sugar)			
	β	*OR*	*OR* 95% CI	*p* -value
(Intercept)	-0.157	0.855	(0.658, 1.11)	0.263
Moderate Physical Activity	-0.668	0.513	(0.44, 0.598)	<.001
Vigerous Physical Activity	-0.138	0.871	(0.788, 0.962)	0.020
Gender: Female				
Gender: Male	0.047	1.049	(0.938, 1.172)	0.422
Age	0.000	1.000	(0.996, 1.004)	0.917
Race/Ethnicity: Non-Hispanic White				
Race/Ethnicity: Mexican Americans	0.345	1.412	(1.084, 1.839)	0.027
Race/Ethnicity: Non-Hispanic Blacks	0.520	1.682	(1.368, 2.067)	<.001
Race/Ethnicity: Other Hispanics	0.309	1.362	(1.078, 1.722)	0.025
Race/Ethnicity: Other Races Including Multiracial	-0.952	0.386	(0.267, 0.557)	<.001
Education: Graduated High School or GED				
Education: 11th to 12th Grade	-0.193	0.824	(0.704, 0.966)	0.036
Education: College Graduate or Above	-0.321	0.725	(0.609, 0.864)	0.004
Education: Less than 9th Grade	-0.257	0.774	(0.648, 0.924)	0.016
Education: Some College or AA Degree	0.041	1.042	(0.894, 1.214)	0.609
Marital Status: Married				
Marital Status: Divorced	0.149	1.161	(0.978, 1.379)	0.117
Marital Status: Living with Partner	-0.305	0.737	(0.621, 0.876)	0.005
Marital Status: Never Married	-0.195	0.823	(0.736, 0.92)	0.006
Marital Status: Seperated	-0.182	0.833	(0.629, 1.103)	0.229
Marital Status: Widowed	-0.059	0.943	(0.716, 1.241)	0.683
Nutrient	-0.001	0.999	(0.998, 0.999)	0.009
Nutrient x Race (Non- Hispanic White)				
Nutrient x Race (Mexican American)	0.000	1.000	(0.998, 1.002)	0.832
Nutrient x Race (Non-Hispanic Hispanic Black)	0.001	1.001	(0.999, 1.002)	0.349
Nutrient x Race (Other Hispanic)	-0.002	0.998	(0.997, 1)	0.064
Nutrient x Race (Other Races Including Multiracial)	0.003	1.003	(1, 1.006)	0.052
Wald's Test(to test significance of nutrient race interactions)			2.86	
p-value			0.08	

4.3.3 Objectives of Hypothesis 3

Hypothesis 3 was analyzed using hierarchical logistic regression analysis. Hypothesis 3 stated that diet high in both fat and sugar increases the risk of obesity relatively to a greater degree than a diet high in either fat or sugar individually,

Analysis was performed in several steps. First, the demographic variables were entered into the model and their respective odds ratios and *p*-values calculated. Demographic variables included level physical activity, gender, age, race/ethnicity highest completed level of education, and marital status. Then, total fat consumption (g/day) and total sugar consumption (g/day) were entered into the model. Finally, the interaction between fat and sugar consumption was entered into the model. In order to test the effect of the interaction between fat consumption and sugar consumption on obesity for statistical significance, Wald's test was performed.

Individually, greater levels of fat consumption were associated with greater likelihood of obesity and greater levels of sugar consumption were associated with lower likelihood of obesity, $Wald = 13.14$, $p < 0.001$. However, according to the results, there was no statistically significant evidence that diet high in both fat and sugar increases the risk of obesity relatively to a greater degree than a diet high in either fat or sugar individually, $Wald = 0.62$, $p = 0.44$.

Table 13: Hypothesis 3 Model Results

	Model 1				Model 2			
	β	*OR*	*OR* 95% CI	*p* -value	*β*	*OR*	*OR* 95% CI	*p* -value
(Intercept)	-0.355	0.701	(0.561, 0.877)	0.007	-0.321	0.725	(0.555, 0.947)	0.033
Moderate Physical Activity	-0.660	0.517	(0.443, 0.602)	<.001	-0.664	0.515	(0.443, 0.598)	<.001
Vigerous Physical Activity	-0.138	0.871	(0.788, 0.963)	0.016	-0.139	0.870	(0.786, 0.963)	0.018
Gender: Female								
Gender: Male	0.011	1.011	(0.907, 1.126)	0.850	-0.006	0.994	(0.886, 1.115)	0.919
Age	0.001	1.001	(0.997, 1.005)	0.555	0.001	1.001	(0.997, 1.005)	0.729
Race/Ethnicity: Non-Hispanic White								
Race/Ethnicity: Mexican Americans	0.379	1.461	(1.236, 1.726)	<.001	0.374	1.454	(1.231, 1.717)	0.001
Race/Ethnicity: Non-Hispanic Blacks	0.622	1.862	(1.608, 2.157)	<.001	0.628	1.873	(1.619, 2.168)	<.001
Race/Ethnicity: Other Hispanics	0.141	1.152	(0.958, 1.384)	0.151	0.150	1.162	(0.969, 1.394)	0.127
Race/Ethnicity: Other Races Including Mu	-0.599	0.549	(0.458, 0.659)	<.001	-0.596	0.551	(0.457, 0.664)	<.001
Education: Graduated High School or GED								
Education: 11th to 12th Grade	-0.197	0.821	(0.702, 0.96)	0.025	-0.189	0.828	(0.709, 0.967)	0.032
Education: College Graduate or Above	-0.303	0.739	(0.623, 0.876)	0.003	-0.327	0.721	(0.606, 0.859)	0.003
Education: Less than 9th Grade	-0.240	0.787	(0.658, 0.941)	0.018	-0.232	0.793	(0.662, 0.95)	0.024
Education: Some College or AA Degree	0.049	1.051	(0.9, 1.227)	0.540	0.041	1.041	(0.894, 1.213)	0.611
Marital Status: Married								
Marital Status: Divorced	0.143	1.154	(0.971, 1.371)	0.124	0.151	1.164	(0.98, 1.382)	0.106
Marital Status: Living with Partner	-0.310	0.733	(0.618, 0.87)	0.003	-0.307	0.735	(0.62, 0.873)	0.003
Marital Status: Never Married	-0.196	0.822	(0.736, 0.918)	0.003	-0.192	0.825	(0.739, 0.922)	0.004
Marital Status: Seperated	-0.189	0.828	(0.628, 1.091)	0.198	-0.178	0.837	(0.632, 1.108)	0.234
Marital Status: Widowed	-0.069	0.933	(0.708, 1.231)	0.630	-0.058	0.943	(0.717, 1.241)	0.683
Total Sugar Consumption					-0.002	0.998	(0.998, 0.999)	<.001
Total Fat Consumption					0.002	1.002	(1.001, 1.004)	0.006
Sugar and Fat Interaction								
Wald's Test(to test significance of nutrient sugar and fat consumption)							13.140	
p-value							<.001	

Table 14: Hypothesis 3 Model Results

	Model 3			
	β	*OR*	*OR* 95% CI	p-value
(Intercept)	-0.386	0.680	(0.508, 0.911)	0.023
Moderate Physical Activity	-0.665	0.514	(0.443, 0.597)	<.001
Vigerous Physical Activity	-0.140	0.870	(0.786, 0.963)	0.018
Gender: Female				
Gender: Male	-0.009	0.991	(0.882, 1.114)	0.883
Age	0.001	1.001	(0.997, 1.005)	0.713
Race/Ethnicity: Non-Hispanic White				
Race/Ethnicity: Mexican Americans	0.373	1.452	(1.231, 1.712)	0.001
Race/Ethnicity: Non-Hispanic Blacks	0.629	1.876	(1.622, 2.17)	<.001
Race/Ethnicity: Other Hispanics	0.153	1.165	(0.969, 1.401)	0.128
Race/Ethnicity: Other Races Including Mu	-0.592	0.553	(0.46, 0.666)	<.001
Education: Graduated High School or GED				
Education: 11th to 12th Grade	-0.189	0.828	(0.709, 0.968)	0.034
Education: College Graduate or Above	-0.328	0.720	(0.605, 0.858)	0.003
Education: Less than 9th Grade	-0.228	0.796	(0.665, 0.952)	0.027
Education: Some College or AA Degree	0.040	1.041	(0.893, 1.212)	0.618
Marital Status: Married				
Marital Status: Divorced	0.154	1.166	(0.982, 1.386)	0.104
Marital Status: Living with Partner	-0.306	0.736	(0.62, 0.874)	0.004
Marital Status: Never Married	-0.190	0.827	(0.74, 0.923)	0.005
Marital Status: Seperated	-0.178	0.837	(0.632, 1.108)	0.236
Marital Status: Widowed	-0.057	0.945	(0.718, 1.244)	0.693
Total Sugar Consumption	-0.001	0.999	(0.998, 1)	0.079
Total Fat Consumption	0.003	1.003	(1.001, 1.005)	0.023
Sugar and Fat Interaction	0.000	1.000	(1, 1)	0.446
Wald's Test(to test significance of nutrient sugar and fat interactions)			0.620	
p-value			0.44	

4.3.4 Objectives of Hypothesis 4

Hypothesis 4 was analyzed using hierarchical logistic regression analysis. Hypothesis 4 stated that diet high in both fiber and protein decreases the risk of obesity relatively to a greater degree than a diet high in either fiber or protein individually.

Analysis was performed in several steps. First, the demographic variables were entered into the model and their respective odds ratios and *p*-values calculated. Demographic variables included level physical activity, gender, age, race/ethnicity highest completed level of education, and marital status. Then, protein consumption (g/day) and total fiber consumption (g/day) were entered into the model. Finally, the interaction between protein and fiber consumption was entered into the model. In order to test the effect of the interaction between protein consumption and fiber consumption on obesity for statistical significance, Wald's test was performed.

Individually, greater levels of fiber consumption were associated with greater likelihood of obesity and greater levels of protein consumption were associated with lower likelihood of obesity, $Wald = 6.04$, $p < 0.01$. However, according to the results, there was no statistically significant evidence that diet high in both protein and fiber increases the risk of obesity relatively to a greater degree than a diet high in either fiber or protein individually, $Wald = 0.81$, $p = 0.38$.

Table 15: Hypothesis 4 Model Results

	Model 1				Model 2			
	β	*OR*	*OR* 95% CI	*p* -value	*β*	*OR*	*OR* 95% CI	*p* -value
(Intercept)	-0.355	0.701	(0.561, 0.877)	0.007	-0.436	0.647	(0.492, 0.85)	0.007
Moderate Physical Activity	-0.660	0.517	(0.443, 0.602)	<.001	-0.650	0.522	(0.448, 0.607)	0.000
Vigerous Physical Activity	-0.138	0.871	(0.788, 0.963)	0.016	-0.128	0.880	(0.793, 0.975)	0.029
Gender: Female								
Gender: Male	0.011	1.011	(0.907, 1.126)	0.850	-0.030	0.971	(0.856, 1.101)	0.651
Age	0.001	1.001	(0.997, 1.005)	0.555	0.002	1.002	(0.998, 1.007)	0.309
Race/Ethnicity: Non-Hispanic White								
Race/Ethnicity: Mexican Americans	0.379	1.461	(1.236, 1.726)	<.001	0.408	1.504	(1.273, 1.777)	0.000
Race/Ethnicity: Non-Hispanic Blacks	0.622	1.862	(1.608, 2.157)	<.001	0.605	1.832	(1.585, 2.117)	0.000
Race/Ethnicity: Other Hispanics	0.141	1.152	(0.958, 1.384)	0.151	0.141	1.151	(0.959, 1.382)	0.154
Race/Ethnicity: Other Races Including Mu	-0.599	0.549	(0.458, 0.659)	<.001	-0.583	0.558	(0.464, 0.672)	0.000
Education: Graduated High School or GED								
Education: 11th to 12th Grade	-0.197	0.821	(0.702, 0.96)	0.025	-0.194	0.824	(0.705, 0.963)	0.029
Education: College Graduate or Above	-0.303	0.739	(0.623, 0.876)	0.003	-0.271	0.763	(0.641, 0.907)	0.009
Education: Less than 9th Grade	-0.240	0.787	(0.658, 0.941)	0.018	-0.219	0.803	(0.674, 0.957)	0.028
Education: Some College or AA Degree	0.049	1.051	(0.9, 1.227)	0.540	0.059	1.061	(0.914, 1.233)	0.450
Marital Status: Married								
Marital Status: Divorced	0.143	1.154	(0.971, 1.371)	0.124	0.138	1.149	(0.961, 1.373)	0.151
Marital Status: Living with Partner	-0.310	0.733	(0.618, 0.87)	0.003	-0.313	0.731	(0.615, 0.869)	0.003
Marital Status: Never Married	-0.196	0.822	(0.736, 0.918)	0.003	-0.192	0.825	(0.736, 0.925)	0.005
Marital Status: Seperated	-0.189	0.828	(0.628, 1.091)	0.198	-0.205	0.815	(0.62, 1.072)	0.165
Marital Status: Widowed	-0.069	0.933	(0.708, 1.231)	0.630	-0.075	0.927	(0.704, 1.222)	0.600
Total Protein Consumption					0.003	1.003	(1.001, 1.004)	0.012
Total Fiber Consumption					-0.011	0.990	(0.983, 0.996)	0.007
Protein Fiber Interaction								
Wald's Test(to test significance of nutrient fiber and protein consumption)							6.040	
p-value							0.01	

Table 16: Hypothesis 4 Model Results

	Model 3			
	β	*OR*	*OR* 95% CI	p -value
(Intercept)	-0.364	0.695	(0.514, 0.938)	0.034
Moderate Physical Activity	-0.650	0.522	(0.448, 0.608)	0.000
Vigerous Physical Activity	-0.128	0.880	(0.794, 0.976)	0.031
Gender: Female				
Gender: Male	-0.028	0.972	(0.856, 1.104)	0.672
Age	0.002	1.002	(0.998, 1.007)	0.300
Race/Ethnicity: Non-Hispanic White				
Race/Ethnicity: Mexican Americans	0.410	1.507	(1.275, 1.78)	0.000
Race/Ethnicity: Non-Hispanic Blacks	0.603	1.827	(1.582, 2.11)	0.000
Race/Ethnicity: Other Hispanics	0.140	1.151	(0.959, 1.381)	0.155
Race/Ethnicity: Other Races Including Mu	-0.581	0.559	(0.465, 0.673)	0.000
Education: Graduated High School or GED				
Education: 11th to 12th Grade	-0.195	0.823	(0.705, 0.961)	0.028
Education: College Graduate or Above	-0.267	0.766	(0.644, 0.911)	0.010
Education: Less than 9th Grade	-0.222	0.801	(0.673, 0.955)	0.028
Education: Some College or AA Degree	0.060	1.062	(0.914, 1.234)	0.444
Marital Status: Married				
Marital Status: Divorced	0.137	1.147	(0.958, 1.373)	0.160
Marital Status: Living with Partner	-0.315	0.730	(0.614, 0.868)	0.003
Marital Status: Never Married	-0.193	0.824	(0.735, 0.925)	0.006
Marital Status: Seperated	-0.207	0.813	(0.619, 1.068)	0.161
Marital Status: Widowed	-0.078	0.925	(0.701, 1.22)	0.589
Total Protein Consumption	0.002	1.002	(0.999, 1.004)	0.215
Total Fiber Consumption	-0.015	0.985	(0.973, 0.998)	0.041
Protein Fiber Interaction	0.000	1.000	(1, 1)	0.383
Wald's Test(to test significance of nutrient fiber and protein interactions)			0.810	
p-value			0.383	

4.3.5 Supplementary Categorical Analysis

The supplementary categorical analysis was performed to further understand the directions of the beta values in the previous analysis used to validate the hypothesis. As has been displayed in the subsequent tables, they all go to support the validity of the directions of the beta values. For example, from table 13, the percent of obesity decreased with higher consumption of fiber and sugar which is in line with the previous numbers. Similar trends were true for the remaining macronutrients.

Table 17: Percent Obese and Non Obese for Intake of Macronutrients by Quartile

Percent Obese and Non Obese for Intake of Macronutrients by Quartile				
Total Fat				
	Quartile 1	Quartile 2	Quartile 3	Quartile 4
Non Obese	62.4	64	62.1	61.3
Obese	37.6	36	37.9	38.7
Total Protein				
	Quartile 1	Quartile 2	Quartile 3	Quartile 4
Non Obese	61.2	63	61.9	63.8
Obese	38.8	37	38.1	36.2
Total Fiber				
	Quartile 1	Quartile 2	Quartile 3	Quartile 4
Non Obese	59.1	60.6	64.1	66.1
Obese	40.9	39.4	35.9	33.9
Total Sugar				
	Quartile 1	Quartile 2	Quartile 3	Quartile 4
Non Obese	59.6	64.2	62.4	63.5
Obese	40.4	35.8	37.6	36.5
Total Carb				
	Quartile 1	Quartile 2	Quartile 3	Quartile 4
Non Obese	59.6	62	63.2	65
Obese	40.4	38	36.8	35

Figure 19 is a graphic display of the trends observed in table 18. It is the distribution of obesity in different quartile of macronutrient intakes or consumption. As an example, for fiber, those in the first quartile, who consume the least amount of fiber has the longest bar, representing the percent of obese in that group. The next quartile or the second quartile, followed with a slightly less obese percent as represented by the respective bar. It goes on to the forth quartile, which represents the

group that consumes the most fiber. In this group, the bar is the shortest, representing the least percent of obese in that group.

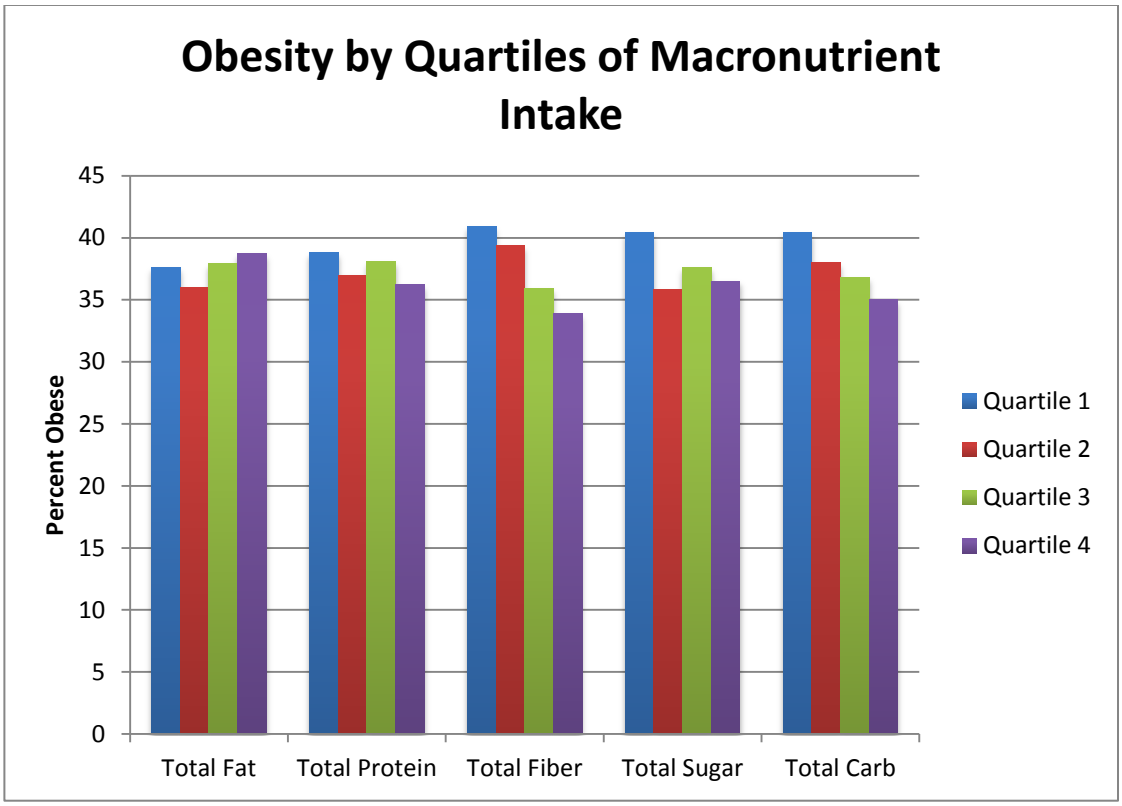


Figure 19: Obesity by Quartiles of Macronutrients Intake

The subsequent tables show results of logistic regression models for the various macronutrients. As observed from table 23, sugar consumption still shows a very significant negative association with obesity.

Table 18: Total Protein Intake with Control Variables

Total Protein Intake with Control Variables						
	Estimate	OR	Std. Error	t value	Pr(>|t|)	
(Intercept)	-0.452	0.637	0.125	-3.624	0.003	*
Moderate Physical Activity	-0.665	0.514	0.078	-8.535	0.000	***
Vigerous Physical Activity	-0.140	0.869	0.051	-2.727	0.017	
Gender: Female						
Gender: Male	-0.042	0.959	0.068	-0.623	0.544	
Age	0.002	1.002	0.002	0.826	0.424	
Race/Ethnicity: Non-Hispanic White						
Race/Ethnicity: Mexican Americans	0.367	1.444	0.084	4.380	0.001	**
Race/Ethnicity: Non-Hispanic Blacks	0.625	1.869	0.075	8.389	0.000	***
Race/Ethnicity: Other Hispanics	0.138	1.148	0.094	1.469	0.166	
Race/Ethnicity: Other Races Including Multiracial	-0.592	0.553	0.095	-6.241	0.000	***
Education: Graduated High School or GED						
Education: 11th to 12th Grade	-0.194	0.824	0.079	-2.451	0.029	*
Education: College Graduate or Above	-0.315	0.730	0.088	-3.558	0.004	**
Education: Less than 9th Grade	-0.226	0.798	0.091	-2.478	0.028	*
Education: Some College or AA Degree	0.047	1.048	0.078	0.602	0.557	
Marital Status: Married						
Marital Status: Divorced	0.149	1.161	0.089	1.677	0.117	
Marital Status: Living with Partner	-0.304	0.738	0.087	-3.486	0.004	**
Marital Status: Never Married	-0.186	0.830	0.058	-3.233	0.007	**
Marital Status: Seperated	-0.194	0.823	0.140	-1.387	0.189	
Marital Status: Widowed	-0.064	0.938	0.142	-0.451	0.660	
Total Protein Quartile 1						
Total Protein Quartile 2	0.025	1.025	0.087	0.283	0.781	
Total Protein Quartile 3	0.167	1.182	0.079	2.121	0.054	
Total Protein Quartile 4	0.187	1.205	0.101	1.854	0.087	

*** $p < 0.001$, ** $p < 0.1$, * $p < 0.05$

Table 19: Total Carbohydrate Intake with Control Variables

Total Carbohydrate Intake with Control Variables						
	Estimate	OR	Std. Error	t value	Pr(>|t|)	
(Intercept)	-0.322	0.725	0.111	-2.904	0.012	*
Moderate Physical Activity	-0.660	0.517	0.078	-8.488	0.000	***
Vigerous Physical Activity	-0.137	0.872	0.052	-2.664	0.019	*
Gender: Female						
Gender: Male	0.027	1.028	0.061	0.451	0.660	
Age	0.001	1.001	0.002	0.407	0.690	
Race/Ethnicity: Non-Hispanic White						
Race/Ethnicity: Mexican Americans	0.381	1.463	0.087	4.397	0.001	**
Race/Ethnicity: Non-Hispanic Blacks	0.621	1.861	0.074	8.359	0.000	***
Race/Ethnicity: Other Hispanics	0.141	1.151	0.093	1.505	0.156	
Race/Ethnicity: Other Races Including Multiracial	-0.603	0.547	0.093	-6.459	0.000	***
Education: Graduated High School or GED						
Education: 11th to 12th Grade	-0.194	0.824	0.080	-2.423	0.031	*
Education: College Graduate or Above	-0.308	0.735	0.087	-3.524	0.004	**
Education: Less than 9th Grade	-0.243	0.784	0.090	-2.705	0.018	*
Education: Some College or AA Degree	0.048	1.049	0.079	0.607	0.554	
Marital Status: Married						
Marital Status: Divorced	0.145	1.156	0.089	1.621	0.129	
Marital Status: Living with Partner	-0.307	0.736	0.087	-3.532	0.004	**
Marital Status: Never Married	-0.197	0.821	0.056	-3.493	0.004	**
Marital Status: Seperated	-0.184	0.832	0.142	-1.296	0.217	
Marital Status: Widowed	-0.066	0.936	0.143	-0.460	0.653	
Total Carb Quartile 1						
Total Carb Quartile 2	-0.039	0.962	0.066	-0.593	0.563	
Total Carb Quartile 3	0.048	1.049	0.072	0.657	0.522	
Total Carb Quartile 4	-0.093	0.911	0.062	-1.497	0.158	

*** $p < 0.001$, ** $p < 0.1$, * $p < 0.05$

Table 20: Total Fiber Intake with Control Variables

Total Fiber Intake with Control Variables						
	Estimate	OR	Std. Error	t value	Pr(>|t|)	
(Intercept)	-0.337	0.714	0.111	-3.034	0.010	*
Moderate Physical Activity	-0.647	0.524	0.080	-8.123	0.000	***
Vigerous Physical Activity	-0.131	0.877	0.053	-2.486	0.027	*
Gender: Female						
Gender: Male	0.033	1.033	0.057	0.575	0.575	
Age	0.001	1.001	0.002	0.709	0.491	
Race/Ethnicity: Non-Hispanic White						
Race/Ethnicity: Mexican Americans	0.413	1.512	0.085	4.891	0.000	***
Race/Ethnicity: Non-Hispanic Blacks	0.608	1.837	0.072	8.424	0.000	***
Race/Ethnicity: Other Hispanics	0.145	1.156	0.095	1.526	0.151	
Race/Ethnicity: Other Races Including Multiracial	-0.614	0.541	0.103	-5.968	0.000	***
Education: Graduated High School or GED						
Education: 11th to 12th Grade	-0.191	0.826	0.080	-2.384	0.033	*
Education: College Graduate or Above	-0.272	0.762	0.087	-3.112	0.008	**
Education: Less than 9th Grade	-0.228	0.796	0.091	-2.490	0.027	*
Education: Some College or AA Degree	0.053	1.055	0.077	0.690	0.502	
Marital Status: Married						
Marital Status: Divorced	0.131	1.140	0.094	1.392	0.187	
Marital Status: Living with Partner	-0.314	0.731	0.089	-3.509	0.004	**
Marital Status: Never Married	-0.197	0.821	0.059	-3.324	0.005	**
Marital Status: Seperated	-0.199	0.820	0.141	-1.411	0.182	
Marital Status: Widowed	-0.075	0.928	0.140	-0.531	0.605	
Total Fiber Quartile 1						
Total Fiber Quartile 2	0.051	1.052	0.073	0.700	0.496	
Total Fiber Quartile 3	-0.069	0.934	0.077	-0.898	0.385	
Total Fiber Quartile 4	-0.204	0.815	0.090	-2.269	0.041	

*** $p < 0.001$, ** $p < 0.1$, * $p < 0.05$

Table 21: Total Fat Intake with Control Variables

Total Fat Intake with Control Variables						
	Estimate	OR	Std. Error	t value	Pr(>|t|)	
(Intercept)	-0.408	0.665	0.122	-3.340	0.005	**
PAQ650Yes	-0.659	0.517	0.077	-8.536	0.000	***
PAQ665Yes	-0.137	0.872	0.051	-2.678	0.019	*
RIAGENDRMale	-0.033	0.968	0.059	-0.552	0.590	
RIDAGEYR	0.002	1.002	0.002	0.869	0.401	
RIDRETH1Mexican Americans	0.382	1.465	0.085	4.492	0.001	**
RIDRETH1Non-Hispanic Blacks	0.626	1.870	0.074	8.422	0.000	***
RIDRETH1Other Hispanics	0.163	1.177	0.094	1.733	0.107	
RIDRETH1Other Races Including Multiracial	-0.570	0.565	0.094	-6.092	0.000	***
DMDEDUC211th to 12th Grade	-0.194	0.824	0.079	-2.467	0.028	
DMDEDUC2College Graduate or Above	-0.307	0.736	0.087	-3.537	0.004	**
DMDEDUC2Less than 9th Grade	-0.227	0.797	0.094	-2.421	0.031	*
DMDEDUC2Some College or AA Degree	0.047	1.048	0.079	0.595	0.562	
DMDMARTLDivorced	0.138	1.148	0.088	1.558	0.143	
DMDMARTLLiving with Partner	-0.314	0.730	0.087	-3.593	0.003	**
DMDMARTLNever Married	-0.195	0.823	0.056	-3.511	0.004	**
DMDMARTLSeperated	-0.186	0.831	0.143	-1.303	0.215	
DMDMARTLWidowed	-0.067	0.935	0.140	-0.475	0.643	
Total Fat Quartile 1						
Total Fat Quartile 2	-0.083	0.920	0.085	-0.979	0.345	
Total Fat Quartile 3	0.063	1.065	0.071	0.894	0.388	
Total Fat Quartile 4	0.160	1.174	0.086	1.860	0.086	

*** $p < 0.001$, ** $p < 0.1$, * $p < 0.05$

Table 22: Total Sugar Intake with Control Variables

Total Sugar Intake with Control Variables						
	Estimate	OR	Std. Error	t value	Pr(>|t|)	
(Intercept)	-0.159	0.853	0.129	-1.234	0.239	
Moderate Physical Activity	-0.659	0.517	0.079	-8.341	0.000	***
Vigerous Physical Activity	-0.135	0.874	0.051	-2.667	0.019	*
Gender: Female						
Gender: Male	0.025	1.025	0.058	0.429	0.675	
Age	0.001	1.001	0.002	0.364	0.722	
Race/Ethnicity: Non-Hispanic White						
Race/Ethnicity: Mexican Americans	0.383	1.466	0.084	4.550	0.001	**
Race/Ethnicity: Non-Hispanic Blacks	0.623	1.864	0.076	8.213	0.000	***
Race/Ethnicity: Other Hispanics	0.140	1.150	0.092	1.516	0.153	
Race/Ethnicity: Other Races Including Multiracial	-0.622	0.537	0.095	-6.558	0.000	***
Education: Graduated High School or GED						
Education: 11th to 12th Grade	-0.198	0.821	0.080	-2.477	0.028	
Education: College Graduate or Above	-0.307	0.735	0.088	-3.497	0.004	**
Education: Less than 9th Grade	-0.255	0.775	0.091	-2.795	0.015	
Education: Some College or AA Degree	0.052	1.054	0.079	0.661	0.520	
Marital Status: Married						
Marital Status: Divorced	0.144	1.154	0.087	1.645	0.124	
Marital Status: Living with Partner	-0.322	0.725	0.090	-3.572	0.003	**
Marital Status: Never Married	-0.200	0.818	0.057	-3.531	0.004	**
Marital Status: Seperated	-0.193	0.825	0.142	-1.362	0.196	
Marital Status: Widowed	-0.073	0.930	0.141	-0.517	0.614	
Total Sugar Quartile 1						
Total Sugar Quartile 2	-0.294	0.745	0.099	-2.957	0.011	
Total Sugar Quartile 3	-0.182	0.834	0.075	-2.437	0.030	*
Total Sugar Quartile 4	-0.232	0.793	0.071	-3.263	0.006	**

*** $p < 0.001$, ** $p < 0.1$, * $p < 0.05$

4.3.6 Summary

In this study, we identified strong evidence that fiber and sugar are negatively associated to obesity. However for proteins, fat and carbohydrates, there was not a strong enough association with obesity. Their association with obesity was not significant at $p < 0.05$. Fiber and sugar has a relatively liner relationship from the supplementary categorical analysis tables with a negative association to obesity. It is therefore evident that the consumption of fiber has a lower risk to obesity while that of

fat has a relatively higher risk of obesity. Though the consumption of sugar seemed to also pose a lower risk to obesity, the trend is not consistent within the different quartiles.

There appeared to be no significant difference in obesity risk exposure for macronutrients to different ethnic groups. For example fiber did not appear to have any significantly more benefit for Non-Hispanic Whites, than either Non-Hispanic Black or Hispanics. In other words their risk and benefit exposures by these macronutrients had no significant difference. Also the risk and benefit exposures by fat and sugar, and protein and fiber together, appeared to be not different from their respective individual components.

In this chapter we detailed the data and results for specific analysis conducted in the research. Chapter 5 contains interpretation of the findings of the conducted research, recommendations for further action, information concerning the limitations and implications of the research and further recommendations about the practical significance and possibility for broader impact of the study.

CHAPTER 5

DISCUSSIONS, CONCLUSIONS AND STUDY LIMITATIONS

5.1 Discussion and Conclusions

This study was conducted to examine the relationship between macronutrients and obesity and also how they affect different ethnic subpopulations. The evaluations included a determination of how different macronutrients differ in terms of their obesity risk. Also, how different macronutrients affect different ethnic subpopulations. I.e. whether there is a difference in let's say how proteins affect Non-Hispanic White as opposed to non-Hispanic Blacks. Or total fats on say Mexican Americans and Hispanics. We also evaluated the obesity risk of synergistic and antagonistic combinations of some macronutrients as compared to their respective individual components.

A retrospective study with a correlational and quantitative research design was used. The entire related hypotheses were tested with hierarchical logistic regression models and Wald's test for their validity. The targeted population was US adults, ages 20 years and above, excluding pregnant women. This chapter contains discussions pertaining to the interpretation and conclusions of the findings, structured per research question, the limitations of the study, and the study implications.

From table 5 it was observed that on the average, the obese are slightly older than the non-obese, with mean ages of 50.18 years and 48.56 years respectively. With the non-obese being slightly younger than the obese, their obesity risk may have been impacted by high activity levels of the relatively younger group. Middle age and older folks are relatively less active than the younger group.

The total calories consumed by the non-obese are again slightly higher than the obese group. These suggest that total caloric intake alone may not be the sole determinant of obesity. However the types of calories in combination with other factors do also matters. Again from table 5 it is observed that the mean consumption of most of the macronutrients is slightly higher for the non-obese group than the obese, with the exception of the fat group. I.e. total fat, saturated fat, monounsaturated fat and cholesterol. Amongst these the mean consumption of the obese group are slightly higher than the non-obese group. This may suggest that total fat may pose and higher obesity risk. Therefore diet high in fat may be associated with high obesity risk, and hence a reduction in fat intake may help reduce one's obesity risk, though there wasn't significant evidence to suggest that within 95% confidence level. Gender also appeared to have association with obesity risk. As observed from table 8, amongst the female 41.0% are obese whiles for men, only 34.1% are obese. Women are therefore relatively more likely to be obese than men.

Again from table 8, it appears that there was a wide disparity of obesity prevalence amongst the various ethnic groups. The group with the least obesity prevalence was the Other Race including Multiracial. This group was predominantly Asians and amongst them, only17.7% was obese. The group with the highest obesity prevalence was the Non-Hispanic Blacks. Amongst them 47.7% were obese. However from table 6, the average macronutrient consumption patterns did not vary very widely, though there were slight differences. These observations made one wonder if various macronutrients exert different obesity risks and also whether macronutrients affects different ethnic groups differently. It is important however to also note that,

some folks may have high BMI, but strong muscle tone, and may not have as much health risk as those whose weight is predominantly due to fat.

5.1.1 Question 1

Do different dietary macronutrients compositions lead to different risks of obesity?

The findings based on the results from our modeling suggest that different macronutrient compositions lead to different risks of obesity, $Wald = 8.081, p < 0.01$. The null hypothesis was therefore dropped for the alternative hypothesis. Fiber had a significantly lower risk association to obesity, $p < 0.05$. Therefore the more fiber you east the lesser your risk to obesity would be, whereas the more fat in a diet, the higher your risk to obesity would be, though the evidence for fat was not significant within 95% confidence level. Sugar also appeared to have a significantly lower risk association to obesity, $p < 05$. This finding appears to be contrary to the general believe, that high sugar consumption leads to obesity. As for protein and carbohydrate, the risk associations were not very significant at 95% confidence level.

From table 21, which shows fiber intake in quartiles, after controlling for physical activity, gender, age, race, education and marital status, the highest quartile, still showed a significant benefit for fiber consumption over the first, $(OR = 0.815, p < 0.05)$. This also goes to concur with the indication that the more fiber you consume the less likely you are prone to obesity. High fiber content in a diet may tend to prevent overeating and excessive weight gain as suggested by Van Itallie et. al, 1978 [132]. Liu Sinin et. al, 2003 [133] also suggested that high fiber whole grain food is

inversely associated with weight gain. There are several other researchers who also suggest similar benefits and more of diet high in dietary fiber [110,111,133-136].

As has already been mentioned, sugar consumption in observed to be inversely associated with obesity risk. In other words eating more sugar is not associated with high obesity risk. Excessive consumption of sugar however is associated with health risks, like type 2 diabetes mellitus, and oral health [84,86]. A low to moderate use of sugar can therefore be part of a healthy meal. High or excessive use of fat however creates a high obesity and other health risks such as cardiovascular diseases and cancer [120,121,124,137,138]. Gibson et. al. 1996, concluded from their research that, there is little evidence that either fatty foods, or diets high in sugar, are associated with obesity [139]. They further emphasized that sugar appears to have a weak negative association with BMI that is not totally explained by confounders such as dieting, under-reporting or the inverse correlation between energy from sugar and fat [139].

5.1.2 Question 2

Does diet in different compositions of macronutrients affect ethnic groups differently?

Our research and analysis findings suggested that, diet in different compositions of macronutrients do not affect ethnic groups differently. The results for the respective macronutrients are as follows. Protein, *Wald = 2.32, p > 0.05; Carbohydrate, Wald = 1.93, p > 0.05; Fiber, Wald = 2.18, p > 0.05; Fat, Wald = 0.01, p > 0.05; and Sugar, Wald = 2.86, p > 0.05.* The macro nutrients interactions with race in all cases were not significant. In other word each of the macronutrients affect each of the races or ethnicities relatively the same, with not significant

difference. Therefore the effect of say, proteins on Non-Hispanic Whites, Non-Hispanic Blacks, Hispanics, Mexicans and Other races has no significant different.

Numerous obesity epidemiological researches however suggest significant disparities in obesity prevalence amongst various ethnicities. Our findings however suggest that the difference has not much to do with the anatomical or physiological makeup of the various ethnicities. As an example, figures one through five in this paper shows clearly the ethnic differences in obesity prevalence.

Socioeconomic status, sociocultural, environmental and life styles amongst others play a very important role in explaining the differences amongst inter racial disparities in obesity prevalence. Kelley et. al. 2016 also observed racial/ethnic differences within geographical regions [140]. Black men they said had greater odds of obesity in the South, West and Midwest than white men [140]. Whiles Hispanic men have greater odds in the South and West [140]. Asian men however have lower odds of obesity than white men in all regions [140]. Most poor people lives in neighborhoods with high crime rate and less opportunities of physical activity, since indoors presents the safest abode for them. With some working two minimum wage jobs, time to go to the gym is most often unaffordable luxury. With lots of food deserts in such neighborhoods, food choices become narrow and often leave them with fast food and non-nutritious obesogenic diets.

Cultural norm and perceptions, practices and nutritional patterns also play a very important role in inter racial disparities in obesity prevalence. Some cultures perceive being heavy attractive whiles other observes being thin attractive. As an example, for most African indigenous cultures, being heavy is more attractive than

being on the thin side. On the other hand in most western cultures, thinness is more attractive than being heavy. Eating patterns amongst various cultural and ethnic groups also make a lot of difference in their size. For example most Asian cultures eat more fiber and other less obesogenic meals than most Western cultures hence their observed edge over them in size, in terms of being less likely to be obese as observed by Kelley et. al. As an example, most Asians, especially Indians, a lot of them are vegetarians and do not eat meat, but more vegetables and fruits, which has more fiber.

Educational levels and their interracial disparities also influence the differences in obesity amongst the various races. Generally the higher your education the more income you are most likely going to earn. The highly educated therefore are also more likely to have the advantages of those in the higher socioeconomic class. This alone gives them an edge over those in the lower socioeconomic class. The highly educated also tend to have a better understanding of their health status and as a result of that consciousness leads a healthy lifestyle. They most likely live in less obesogenic environments with less crime and local parks and safe pavements to either walk or run on routine base. Also with a bigger budget for groceries and more supper markets within reach, they have better choices for meals which will most likely lead to a superior nutrition pattern, and subsequent reduced risk of obesity.

Interracial disparities in these factors and others are what most likely explain the racial differences of obesity prevalence in the USA. Our findings above suggests that, the physiological make up and the anatomy of various races do not have any significant influence on the obesity risk imposed by the various macronutrients they consume.

5.1.3 Question 3

Does a diet high in fat and sugar increase the risk of obesity relatively higher than either individual component?

Our research and analysis finding suggest that, a diet high in fat and sugar does not increase the risk of obesity relatively higher than either individual component, $Wald = 0.620, p > 0.05$.

La Fleur et. al. performed a study to examine the effect of different free choice high caloric, obesity inducing diet on glucose metabolism on rat models. They observed that both high fat high sugar (HFHS) and high fat (HF) diets resulted in obesity with comparable plasma concentration of free fatty acids [141]. The HF diets, they said did not affect glucose metabolism, whereas the HFHS diet resulted in hyperglycemia, hyperinsulinemia and as well as glucose intolerance because of diminution glucose response [141]. They concluded that their results suggested that diet content is crucial for glucose intolerance, not only obesity or total caloric intake [141].

This research work goes to support the fact that the combination of high fat and high sugar diet does not pose higher obesity risk than their individual component. The issue however is beyond obesity for the high sugar in the diet. High sugar diet as has already been mentioned in this paper causes more health risks such as type two diabetes mellitus, triggered by glucose intolerance. It is therefore important that one moderates their sugar intake to avoid its associated health hazards, beyond obesity concerns.

5.1.4 Question 4

Does a diet with significant amount of fiber and protein reduce the risk of obesity relatively higher than either individual component?

The findings of our research and analysis for this question suggest that a diet with significant amount of fiber and protein do not reduce the risk of obesity relatively higher than either individual components, $Wald = 0.810, p > 0.05$.

There are numerous benefits for consuming fiber rich or high fiber diets. It typically slows digestion and delays hunger. In so doing it helps in controlling frequent consumption of food and hence helps control weight. There is not much evidence of protein rich food, being good for weight control, especially diet high in animal protein or processed animal protein. Other sources like lean poultry and plant protein sources may have a bit of an advantage in weight loss.

Combinations of fiber and protein diets have several known health advantages. It is known to reduce high blood pressure in hypertensives and also good for the prevention of some cardiovascular issues [142,143]. They are also good in controlling chronic kidney problems and other reproductive functions [144,145]. In spite of the numerous health benefits of the combination of fiber and protein, there is no evidence of their interaction reducing obesity risks better than their component individuals.

5.2 Study Limitations

The current findings for the dissertation were subject to little limitations; though two cycles of NHANES data representing 4 years data analysis of NHANES provided large sample size that calls for precision in outcome measurement. Dietary

recall can be over-estimated or under-estimated when giving account of caloric and macronutrients intake. And self-reported macronutrient intake can result in under estimation which can create reporting bias because one cannot estimate for macronutrients that was added while cooking. NHANES being a cross-sectional study does not allow casual inference in the study. NHANES data has previously been used for estimations of prevalence in several studies: Meanwhile, primarily NHANES data are restricted to non-institutionalized participants. This means institutionalized populations such as those in prisons, assisted living and other institutions are not represented. Moreover, research findings on non-institutionalized population do not represent hundred percent of the population. Besides, people eat differently on different days; people turn to eat more restaurant foods on weekends and depending on the day that the interview was done can also affect the portion sizes reported. Finally, in addition to the aforementioned limitations, the multi-stage probability nature of NHANES does not address time. Sampling parameters can vary over time.

5.3 Suggested Future Research

We recommend an investigation within and across ethnic groups to find reasons for the wide inter racial variabilities amongst them, so far as obesity prevalence within the various groups are concerned. Asian Americans have very excellent results, why African Americans and American Indians appear to have very unfavorable results. A good understanding of the intra racial socioeconomic and cultural dynamics as well as life styles will go a long way improving our understanding of the inter racial variabilities and health inequities and ultimately help

us bridge the gap, by learning from the successful groups, and replicate the success in the least successful groups.

Another area that needs further investigation is communities within different Metropolitan Status Codes [MSCs]. We also recommends further investigation in this area, accessing the obesity risk factors and others that will help explain why certain Metropolitan Status Codes appears to be more obesogenic than others, hence the disparities in their obesity rates. A good understanding in this area will help in designing more healthy communities that are less obesogenic.

5.4 Summary

This study investigated the impact of dietary macronutrients on obesity in different US adult ethnic subpopulations, using NHANES data set collected by CDC, in addition to reviewing several related pear reviewed articles and other published materials. The hierarchical logistic regression analysis was use with Wald's test to test our various hypotheses at 95% confidence level. The hypotheses were tested while controlling for physical activity, gender, age, race/ethnicity, education and marital status, all of which were confounding obesity risk factors.

Our first hypothesis tested for varying obesity risk levels for the various macronutrients. It turned out that the various macronutrients have different obesity risk level. The most significant amongst them were fiber, sugar and fat. Fiber turned out with the least risk association to obesity, with an inverse relationship. Hence the more fiber you take in your diet, the less likely you are to be obese. Sugar also had some evidence of negative association. Fat however turned out with some evidence of

positive association to obesity. Hence the more fat you consume, the most likely you are to be obese.

Contrary to our hypothesis two which investigated the effect of macronutrients on the various US sub populations; there was no significant difference or association in the effect of various macronutrients on the various race or ethnic groups. For example there was no significant difference in the way fiber affected Non-Hispanic Whites and the others such as Non-Hispanic Blacks, Hispanics, and Mexican American. In spite of this observation, there is a wide disparity in the obesity prevalence amongst, the various ethnic groups in the literature. Our findings suggest that, these can be explained by factors either than the anatomy and physiology of the various ethnic groups. Some of these factors such as lifestyle adjustment, can "somewhat" be controlled by the individual, yet there are many others far beyond the control of the respective victims. Such factors as socioeconomic, cultural and environmental issues at the societal level, which's effect on obesity are beyond the control of some of its victims. These issues impact our lifestyle and dietary choices, which ultimately affects our chances of being obese, via our bodies' biological mechanism and systems. As a result, leaving the responsibility for solutions to victims alone may not only be unfair but an ineffective way of dealing with the obesity issue.

To have more sustainable results, responsibilities has to be rightly apportioned to individuals and society at large. All related stakeholders, including the food industry, the government; NGOs, Educational institutions, the healthcare industry and the likes need to collaborate to find a more harmonized solution via a systemic integrative thinking and approach to problem solving. This we believe will engender

more practical and sustainable solutions. Over leveraging respective industry, entity or stakeholders conveniences undermines the others interests and effectiveness in dealing with the issue, for instance the food industries in their quest to maximize their profits alone, without any consideration for other societal health needs, generally reacts in a way to undermines it. Also town planners acting without such tenets, also work to undermine health need for their residents and so on. There is therefore a need for a general societal health consciousness at the government planning and regulatory level which trickles down to entities and individuals.

Also for medical practitioners, a good understanding of socioeconomic and cultural as well and environmental profile of their obese patients will help them prescribe a more personal and practical solution, that could effectively intervene and help them deal with their obesity problem. A practical advice will be to increase dietary fiber intake of patients or in combination with proteins, since either have good health benefits and also the tendency to reduce weight. Sugar can be part of a functional diet, but has to be used in moderation. It is however important to note that, consuming all dietary macronutrients in meals, in appropriate proportions, will provide the needed balanced diet the body needs to function properly. Excessive consumption of these however will expose victims to obesity and numerous other health risks, such as diabetes mellitus, cancer, cardiovascular diseases, and more.

REFERENCES

1. Rubenstein AH. Obesity: A modern epidemic. . 2005.

2. Cutler DM, Glaeser EL, Shapiro JM. WHY HAVE AMERICANS BECOME MORE OBESE? Cambridge, MA: NATIONAL BUREAU OF ECONOMIC RESEARCH; 2003.

3. Drewnowski A, Specter SE. Poverty and obesity: the role of energy density and energy costs. The American Journal of Clinical Nutrition 2004;79:6-16.

4. Ball K, Crawford D. Socioeconomic status and weight change in adults: a review. Soc Sci Med 2005;60:1987-2010.

5. Baum CL, 2nd, Ruhm CJ. Age, socioeconomic status and obesity growth. J Health Econ 2009;28:635-48.

6. Cynthia L. Ogden PD, Molly M. Lamb PD, Margaret D. Carroll MSPH, Katherine M. Flegal PD. Obesity and Socioeconomic Status in Adults: United States, 2005-2008. N C H S D a t a B r i e f - CDC 2010;50.

7. Zhang Q, Wang Y. Socioeconomic inequality of obesity in the United States: do gender, age, and ethnicity matter? Soc Sci Med 2004;58:1171-80.

8. Brennan SL, Henry MJ, Nicholson GC, Kotowicz MA, Pasco JA. Socioeconomic status and risk factors for obesity and metabolic disorders in a population-based sample of adult females. Prev Med 2009;49:165-71.

9. Fitzgerald N, Himmelgreen D, Damio G, Segura-Pérez S, Peng Y-K, Pérez-Escamilla R. Acculturation, socioeconomic status, obesity and lifestyle factors among low-income Puerto Rican women in Connecticut, U.S., 1998–1999. Rev Panam Salud Publica 2006;19(5):306-13.

10. McLaren L. Socioeconomic status and obesity. Epidemiol Rev 2007;29:29-48.

11. Scheier LM. What is the hunger-obesity paradox? J Am Diet Assoc 2005;105:883-4, 6.

12. Cawley, Rosaleen M. The Psycosocial Aspects of Obesity: A Quantitative & Qualitative Study; PhD Thesis: Dept of General Practice & Primary Care (Division of Community Based Sciences); October 2004.

13. Tubes G. Why we get fat and what to do about it.

14. Brun JF. Exercise Makes More than an Energy Deficit: Toward Improved Protocols for the Management of Obesity? EBioMedicine 2015;2:1862-3.

15. Tubes G. Good Calories Bad Calories.

16. Organization WH. World Health Organization; Health Impact Assessment. World Health Organization; 2015.

17. Popkin BM. The shift in stages of the nutrition transition in the developing world differs from past experience!.. Pub Health Nutr 2002;5:205-14.

18. Organization WH. Non Communicable Disease Country Profile: WHO; March 2011.

19. Prevention CfDCa. Overweight and Obesity: Obesity Trends: US Obesity Trends 1985-2004. CDC; 2004.

20. Wyatt SB, Winters KP, Dabbert PM. Overweight and Obesity; Prevalence. Consequences, and cause of a growing public health problem. April, 2006.

21. AMA. AMA News Room [American Medical Association]. 2013.

22. Finkelstein EA, Fiebelken IC, Wang G. National Medical Spending Attributable to Overweight and Obesity; How much and who's paying. 2003.

23. Dixon JB, O'Brien PE. Changes in Comorbidities and Improvement in Quality of Life after LAP-BAND Placement. The American Journal of Surgery 2002;184:551-4.

24. Kral JG, Sjostrom LV, Sullivan MBE. Assesment of quality of life before and after surjury for sever obesity. The American Journal of Clinical Nutrition 1992;55:6115-45.

25. McPherson MD, Fulson T, Aitken RT, Lane M. Paternal Obesity, Intervention and Mechanistic Pathways to Impaired Health in Offspring. Ann Nutr Metab 2014;64:231-8.

26. Akay AP, Ozturk Y, Avcil SN, Kavurma C, Tufan E. Relationships between pediatric obesity and maternal emotional states and attitude. Int J Psychiatry 2015;50:178 - 90.

27. Hunts, Hart CL, Haig L, Upton MN, Watt GL, Lean ME. Contributions of maternal and paternal adiposity and smoking to adult offsprings adiposity and cardiovascular risk: The Midspan Family Study. BMT Open 2015;5:76-82.

28. International Program in Health Policy an Innovation, The Commonwealth Fund. Explaining high heatlh care spending in the Unites States; an international comparison of supply, utilization. prices and quality: Issue Brief: Commonwealth Fund; 2012.

29. Trasande L, Elbel B. The economic burden placed on health care systems by childhood obesity. Expert Rev Pharmacoecon 2012;12:39-45.

30. Ryan VG. Cost and policy implications from the increasing prevalence of obesity and diabetes mellitus. Gend Med 2009;1:86-108.

31. Wolf AM. The economic impact of obesity.Building bridges with managed care. Postgrad Med 2001;109(6 suppl):34-9.

32. Finkelstein EA, Cohen JW, Dielta W. Annual spending attributable to obesity: Payer and specific estimates. Health Aff (Millwood) 2008;28(5):822-31.

33. Wexler B. The economic overview of obesity. " Weight in America, Obesity, Eating Disorders, and other health Risks. Information Plus Reference Series 2007.

34. Prevention CfDCa. Preventing Obesity and Chronic Diseases Through Good Nutrition and Physical Activities. 2008.

35. Anderson LH, al. e. Healthcare Charges Associated with Physical Inactivity, Overweight and Obesity. Preventing Chronic Disease 2 no 4 2005 October:1-12.

36. Office USC. Technology Change and Growth of Healthcare Spending. Whashington DC: US Gov. Printing Office; 2008.

37. Disease PtFC. The Unhealthy Truth: Rising Rate of Chronic Diseases and the Future of Health in America. 2007.

38. Prevention CfDCa. Press Release: Obesity Still a Major Problem CDC; 2006.

39. Olshansky SJ, Passaro DJ, Hershow RC, al. e. A Potential Decline in Life Expectancy in the United States in the 21st CenturyMarch, 2005.

40. Pellas JC, al. e. Qualifying Obesity in Economic Research: How Misleading is the Body Mass Index (Draft)2011.

41. Wikipedia. Body Mass Index. 2015.

42. Biddle J, al. e. The Burden of Obesity in Arkansas: Diabetes Prevention and Control, Arkansas Department of Health; 2008.

43. The Burden of Obesity in Arkansas: Diabetes Prevention and Control, Arkansas Department of Health; 2008.

44. Wikipedia. Diabetes Mellitus. Wikipedia; 2015.

45. Wikipedia. Diabetes Mellitus Type 1. Wikipedia; 2015.

46. Wikipedia. Diabetes Mellitus Type 2. Wikipedia; 2015.

47. Wikipedia. Heart Disease. Wikipedia; 2015.

48. Wikipedia. Cardiovascular Disease. Wikipedia; 2015.

49. Institute HW. Cardiaovascular Disease Fact Sheet. ADAM Inc 2004.

50. Association AH. Heart Disease and Stroke Statistics 2004: American Heart Association; 2004.

51. Flegal KM, al. e. Association of All-Cause Mortality with Overweight and Obesity Using Standard Body Mass Index. JAMA 2013:71-82.

52. Lavie CJ, Milani RV, Ventura HO. Obesity and Cardiovascular Disease - Risk Factors, Paradox, and Impact of Weight Loss. J Am Coll Cardiol 2009;May 26.

53. Association AH. Obesity and Cardiovascular Diseases (CVDs). Washington DC: American Heart Association; American Strock Association; 2013.

54. Satish Kenchaiah MD, Jane C. Evans DS, Daniel Levy MD, et al. Obesity and the Risk of Heart failure. 2002.

55. Katzmarzyk OT, Mire E, Bouchard C. Abdominal Obesity and Mortality; The Pennington Center Longitudianal Study. Nutr Diabetes 2012;August

56. Razimia T, al. e. BMI and Stroke Discharge Outcome after Ischemic Stroke. Arch Neurol 2007.

57. Talbot ML, Jorgensen JO, Loi KW. Difficulty in Provision of Bariatric Surgical Services to the Morbidly Obese. Med J 2005;182(7):344-7.

58. Shinogle JA, Owings MF, Kozak LI. Gastric bypass as Treatement for Obesity: Trends Characteristics and Complications. Obes Res 2005;13(12):2202-9.

59. Nguyen MT, Root J, Zainabadi K, Sabio A, Chalifous S, al. e. Accelerated growth of bariatric surgery with Introduction of Minimally Invasive Surgery. Arch Serg 2005;140(12):1198-202.

60. Carbonell AM, Lincourt AE, Matthews BD, Kercher KW, Sing RF, Heniford BT. National Study of the effect of Patient and Hospital Characteristics on Bariatric Surgery outcomes. Am Surg 2005;7(194):308-14.

61. Buchwarld H, O'brien DM. Metabolic/Bariatric Surgery Worldwide: Springer Science + Business Media LLC; 2008.

62. Buchwarld H, Avidor Y, Brauwarld E. Bariatric Surgery: A systemic Review and Meta Analysis. JAMA 2004;292:1724 - 37.

63. Lissau I, Overpeck MD, Ruan WJ. Body Mass Index and overweight in adolescents in 13 European countries, Iseael and the United States. Arch Pediatr Adolesc Med 2004;158:27 - 33.

64. Spurgeon D. Childhood Obesity in Canada has tripled in the past 20 years. BMJ 2002;324:1416.

65. Wang Y, Monteiro C, Popkin BM. Trends of obesity and underwight in older children and adoloscents in the United States, Brazil, China, and Russia. Am J Clin Nutr 2002;75:971 - 7.

66. Kelie H, Lenz J, Kries Rv. Prevalence of overweight and obesity and trends in Body Mass Index in Germany Pre-School Children Int J Obes Rlat Metab Disord 2002;26:1211 - 7.

67. Rocchini AP. Childhood obesity and a diabetes epidemic. N Engl J Med 2002;855:854.

68. Doak, al. e. The prevention of childhood overweight and obesity. Obesity review. Arch Pediatr Adolesc Med 2006;154:931 - 5.

69. Onis Md, Blossner M. Prevalence and trends of overweight among preschool children in developing countries. Am J Clin Nutr 2002;72:1032 - 9.

70. JC S. Obesity: a growing problem. Acta Paediatr Suppl 1999;88:46 - 50.

71. Wabitsch M. Overweight and obesity in European Children: Definition and diagnostic procedures, risk factors and consequences for later health outcome. Eur J Pediatr 2000;159:S8 - S13.

72. Must A, Strauss RS. Risk and consequences of childhood and adolescent obesity. Int J Obes Relat Metab Disord 1999;23:S2 - S11.

73. Dietz WH. Health consequenses of obesity in youth: Childhood predictors of adult disease. Pediatrics 1998;101:518 - 25.

74. S.J. E, T.N. R, K.F. H, J.D.. K. Are overweight children unhappy?: Body mass index, depressive symptoms, and overweight concerns in elementary school children. Arch Pediatr Adolesc Med 2000;130.

75. Witakar RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. N Engl J Med 1997;337:869 - 73.

76. Freedman DS, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. Inter-relationships among childhood BMI, childhood heght, and adult obesity: the Bogalusa Heart Study. Int J Obes Relat Metab Disord 2004;28:10 - 6.

77. Guo SS, Chumblea WC. Tracking of Body Mass Index in Children in relation to overweight in adulthood. Am J Clin Nutr 1999;70:145S - 8S.

78. Story M, Kaphingst KM, French S. The role of schools in Obesity Prevention2006.

79. Gonzalez JL, Gilmer L, al. e. Obesity Prevention in Pediatrics: A Pilot Perdiatric Resident Carriculum Intervention on Nutrition and Obesity Education and Counseling. J Natl Med Assoc 2006;98:1483 - 8.

80. Goran MI, Reynold KD, Lindquist CH. Role of Physical activity in prevention of obesity in children. Int J Obes 1999;23:S18 - S33.

81. Rennie KL, Johnson L, Jebb SA. Behavioural determinants of obesity. Best Pract Res Clin Endocrinol Metab 2005;19:343-58.

82. Hill JO, Peters JC. Environmental Contributions to the Obesity Epidemic. Science 1998;280.

83. Schorin MD, Sollid K, Edge MS, Bouchoux A. The Science of Sugars, Part 3. Nutr Today 2012;47:252-61.

84. Apovian CM. Sugar-sweetened soft drinks, obesity, and type 2 diabetes. JAMA 2004;292:978-9.

85. Basu S, Vellakkal S, Agrawal S, Stuckler D, Popkin B, Ebrahim S. Averting obesity and type 2 diabetes in India through sugar-sweetened beverage taxation: an economic-epidemiologic modeling study. PLoS Med 2014;11:e1001582.

86. Beaglehole R. Sugar sweetened beverages, obesity, diabetes and oral health: a preventable crisis. Pac Health Dialog 2014;20:39-42.

87. Gulati S, Misra A. Sugar intake, obesity, and diabetes in India. Nutrients 2014;6:5955-74.

88. Hu FB, Malik VS. Sugar-sweetened beverages and risk of obesity and type 2 diabetes: epidemiologic evidence. Physiol Behav 2010;100:47-54.

89. Malavazos AE, Briganti S, Morricone L. Sugar-sweetened beverages, genetic risk, and obesity. N Engl J Med 2013;368:286.

90. Malik VS, Hu FB. Sweeteners and Risk of Obesity and Type 2 Diabetes: The Role of Sugar-Sweetened Beverages. Curr Diab Rep 2012.

91. Malik VS, Popkin BM, Bray GA, Despres JP, Hu FB. Sugar-sweetened beverages, obesity, type 2 diabetes mellitus, and cardiovascular disease risk. Circulation 2010;121:1356-64.

92. Martin-Calvo N, Martinez-Gonzalez MA, Bes-Rastrollo M, et al. Sugar-sweetened carbonated beverage consumption and childhood/adolescent obesity: a case-control study. Public Health Nutr 2014;17:2185-93.

93. McGill AT. The sugar debate and nutrition: obesity and 'empty calories'. N Z Med J 2014;127:6-11.

94. Bray GA, Popkin BM. Sugar consumption by Americans and obesity are both too high--are they connected? Response to letter by John White, PhD. Pediatr Obes 2014;9:e78-9.

95. Harrington S. The role of sugar-sweetened beverage consumption in adolescent obesity: a review of the literature. J Sch Nurs 2008;24:3-12.

96. Bucher Della Torre S, Keller A, Laure Depeyre J, Kruseman M. Sugar-Sweetened Beverages and Obesity Risk in Children and Adolescents: A Systematic Analysis on How Methodological Quality May Influence Conclusions. J Acad Nutr Diet 2016;116:638-59.

97. Gibson S. Sugar-sweetened soft drinks and obesity: a systematic review of the evidence from observational studies and interventions. Nutr Res Rev 2008;21:134-47.

98. Briggs AD, Mytton OT, Madden D, O'Shea D, Rayner M, Scarborough P. The potential impact on obesity of a 10% tax on sugar-sweetened beverages in Ireland, an effect assessment modelling study. BMC Public Health 2013;13:860.

99. Buhler S, Raine KD, Arango M, Pellerin S, Neary NE. Building a strategy for obesity prevention one piece at a time: the case of sugar-sweetened beverage taxation. Can J Diabetes 2013;37:97-102.

100. Cabrera Escobar MA, Veerman JL, Tollman SM, Bertram MY, Hofman KJ. Evidence that a tax on sugar sweetened beverages reduces the obesity rate: a meta-analysis. BMC Public Health 2013;13:1072.

101. Conkle J, Carter M. Taxing sugar-sweetened beverages: the fight against obesity. Nurse Pract 2013;38:1-4.

102. Manyema M, Veerman LJ, Chola L, et al. The potential impact of a 20% tax on sugar-sweetened beverages on obesity in South African adults: a mathematical model. PLoS One 2014;9:e105287.

103. Novak NL, Brownell KD. Taxation as prevention and as a treatment for obesity: the case of sugar-sweetened beverages. Curr Pharm Des 2011;17:1218-22.

104. Boswell R. Sugar: there's more to the obesity crisis. Nature 2012;482:470.

105. Ortega RM, Andres P. [Carbohydrates and obesity]. Med Clin (Barc) 1998;110:797-801.

106. Wylie-Rosett J, Segal-Isaacson CJ, Segal-Isaacson A. Carbohydrates and increases in obesity: does the type of carbohydrate make a difference? Obes Res 2004;12 Suppl 2:124S-9S.

107. Jebb SA. Carbohydrates and obesity: from evidence to policy in the UK. Proc Nutr Soc 2015;74:215-20.

108. Howard BV, Abbott WG, Swinburn BA. Evaluation of metabolic effects of substitution of complex carbohydrates for saturated fat in individuals with obesity and NIDDM. Diabetes Care 1991;14:786-95.

109. Lopez-Alarcon M, Perichart-Perera O, Flores-Huerta S, et al. Excessive refined carbohydrates and scarce micronutrients intakes increase inflammatory mediators and insulin resistance in prepubertal and pubertal obese children independently of obesity. Mediators Inflamm 2014;2014:849031.

110. Papathanasopoulos A, Camilleri M. Dietary fiber supplements: effects in obesity and metabolic syndrome and relationship to gastrointestinal functions. Gastroenterology 2010;138:65-72 e1-2.

111. Ullrich IH, Albrink MJ. The effect of dietary fiber and other factors on insulin response: role in obesity. J Environ Pathol Toxicol Oncol 1985;5:137-55.

112. Kimm SY. The role of dietary fiber in the development and treatment of childhood obesity. Pediatrics 1995;96:1010-4.

113. Kobayakawa A, Suzuki T, Ikami T, Saito M, Yabe D, Seino Y. Improvement of fasting plasma glucose level after ingesting moderate amount of dietary fiber in

Japanese men with mild hyperglycemia and visceral fat obesity. J Diet Suppl 2013;10:129-41.

114. Kranz S, Brauchla M, Slavin JL, Miller KB. What do we know about dietary fiber intake in children and health? The effects of fiber intake on constipation, obesity, and diabetes in children. Adv Nutr 2012;3:47-53.

115. Clifton P. Effects of a high protein diet on body weight and comorbidities associated with obesity. Br J Nutr 2012;108 Suppl 2:S122-9.

116. Willi SM, Oexmann MJ, Wright NM, Collop NA, Key LL, Jr. The effects of a high-protein, low-fat, ketogenic diet on adolescents with morbid obesity: body composition, blood chemistries, and sleep abnormalities. Pediatrics 1998;101:61-7.

117. Wang Y, Beydoun MA. Meat consumption is associated with obesity and central obesity among US adults. Int J Obes (Lond) 2009;33:621-8.

118. Dietary fats explained. Medline Plus 2016. (Accessed May 10, 2016, 2016, at https://www.nlm.nih.gov/medlineplus/ency/patientinstructions/000104.htm.)

119. Molinero LL, Yin D, Lei YM, et al. High-Fat Diet-Induced Obesity Enhances Allograft Rejection. Transplantation 2016;100:1015-21.

120. Fujita Y, Maki K. High-fat diet-induced obesity triggers alveolar bone loss and spontaneous periodontal disease in growing mice. BMC Obes 2015;3:1.

121. Kentish SJ, Vincent AD, Kennaway DJ, Wittert GA, Page AJ. High-Fat Diet-Induced Obesity Ablates Gastric Vagal Afferent Circadian Rhythms. J Neurosci 2016;36:3199-207.

122. Lima VV, Giachini FR, Matsumoto T, et al. High-fat diet increases O-GlcNAc levels in cerebral arteries: a link to vascular dysfunction associated with hyperlipidaemia/obesity? Clin Sci (Lond) 2016;130:871-80.

123. Marques C, Meireles M, Norberto S, et al. High-fat diet-induced obesity Rat model: a comparison between Wistar and Sprague-Dawley Rat. Adipocyte 2016;5:11-21.

124. Skaznik-Wikiel ME, Swindle DC, Allshouse AA, Polotsky AJ, McManaman JL. High-Fat Diet Causes Subfertility and Compromised Ovarian Function Independent of Obesity in Mice. Biol Reprod 2016.

125. Takase K, Tsuneoka Y, Oda S, Kuroda M, Funato H. High-fat diet feeding alters olfactory-, social-, and reward-related behaviors of mice independent of obesity. Obesity (Silver Spring) 2016;24:886-94.

126. Yamasaki M, Hasegawa S, Imai M, Takahashi N, Fukui T. High-fat diet-induced obesity stimulates ketone body utilization in osteoclasts of the mouse bone. Biochem Biophys Res Commun 2016;473:654-61.

127. McKenzie JF, Neiger BL, Thackeray R. Planning, implementing, & Evaluating Health Promotion Programs (5th Ed). San Francisco, CA: Pearson Benjamin Cumming; 2009.

128. Johnson RB. Educational Research; Quantitative, Qualitative and Mixed Method Approaces 3rd ed. Thousand Oaks, CA: Sage Publications; 2007.

129. Creswell JW. Educational Research; planning, Conducting and Evaluating Qualitative and Quantitative Research 2nd ed. Upper Saddle River, NJ: Prentice Hall; 2005.

130. Givens LM. The Sage Encyclopedia of Quality Research Methods. Los Angeles, Calif: Sage Publication; ISBN: 1-4129-4163-6; 2008.

131. Triola M. Elementary Statistics 7th ed. Reading, MA: Addison Wesley Longman; 1998.

132. Van Itallie TB. Dietary fiber and obesity. Am J Clin Nutr 1978;31:S43-52.

133. Liu S, Willett WC, Manson JE, Hu FB, Rosner B, Colditz G. Relation between changes in intakes of dietary fiber and grain products and changes in weight and development of obesity among middle-aged women. Am J Clin Nutr 2003;78:920-7.

134. Maki KC, Beiseigel JM, Jonnalagadda SS, et al. Whole-grain ready-to-eat oat cereal, as part of a dietary program for weight loss, reduces low-density lipoprotein cholesterol in adults with overweight and obesity more than a dietary program including low-fiber control foods. J Am Diet Assoc 2010;110:205-14.

135. Melanson KJ, Angelopoulos TJ, Nguyen VT, et al. Consumption of whole-grain cereals during weight loss: effects on dietary quality, dietary fiber, magnesium, vitamin B-6, and obesity. J Am Diet Assoc 2006;106:1380-8; quiz 9-90.

136. Owens TJ, Larsen JA, Farcas AK, Nelson RW, Kass PH, Fascetti AJ. Total dietary fiber composition of diets used for management of obesity and diabetes mellitus in cats. J Am Vet Med Assoc 2014;245:99-105.

137. Allerton TD, Primeaux SD. High-fat diet differentially regulates metabolic parameters in obesity-resistant S5B/Pl rats and obesity-prone Osborne-Mendel rats. Can J Physiol Pharmacol 2015:1-10.

138. Dudek M, Kolodziejski PA, Pruszynska-Oszmalek E, et al. Effects of high-fat diet-induced obesity and diabetes on Kiss1 and GPR54 expression in the

hypothalamic-pituitary-gonadal (HPG) axis and peripheral organs (fat, pancreas and liver) in male rats. Neuropeptides 2016;56:41-9.

139. Gibson SA. Are high-fat, high-sugar foods and diets conducive to obesity? Int J Food Sci Nutr 1996;47:405-15.

140. Kelley EA, Bowie JV, Griffith DM, Bruce M, Hill S, Thorpe RJ, Jr. Geography, Race/Ethnicity, and Obesity Among Men in the United States. Am J Mens Health 2016;10:228-36.

141. la Fleur SE, Luijendijk MC, van Rozen AJ, Kalsbeek A, Adan RA. A free-choice high-fat high-sugar diet induces glucose intolerance and insulin unresponsiveness to a glucose load not explained by obesity. Int J Obes (Lond) 2011;35:595-604.

142. Burke V, Hodgson JM, Beilin LJ, Giangiulioi N, Rogers P, Puddey IB. Dietary protein and soluble fiber reduce ambulatory blood pressure in treated hypertensives. Hypertension 2001;38:821-6.

143. He J, Whelton PK. Effect of dietary fiber and protein intake on blood pressure: a review of epidemiologic evidence. Clin Exp Hypertens 1999;21:785-96.

144. Evenepoel P, Meijers BK. Dietary fiber and protein: nutritional therapy in chronic kidney disease and beyond. Kidney Int 2012;81:227-9.

145. Remer T. Dietary protein and fiber intake and sex hormone-binding globulin. J Clin Endocrinol Metab 2001;86:950.