

2018

Relationship Between Exposure to Phthalate and Obesity in the United States

Gladys Chidiebere Ezem
Walden University

Follow this and additional works at: <https://scholarworks.waldenu.edu/dissertations>

 Part of the [American Studies Commons](#), [Environmental Health and Protection Commons](#), and the [Epidemiology Commons](#)

This Dissertation is brought to you for free and open access by the Walden Dissertations and Doctoral Studies Collection at ScholarWorks. It has been accepted for inclusion in Walden Dissertations and Doctoral Studies by an authorized administrator of ScholarWorks. For more information, please contact ScholarWorks@waldenu.edu.

Walden University

College of Health Sciences

This is to certify that the doctoral dissertation by

Gladys Ezem

has been found to be complete and satisfactory in all respects,
and that any and all revisions required by
the review committee have been made.

Review Committee

Dr. Nicoletta Alexander, Committee Chairperson, Public Health Faculty
Dr. Scott McDoniel, Committee Member, Public Health Faculty
Dr. Diana Naser, University Reviewer, Public Health Faculty

Chief Academic Officer

Eric Riedel, Ph.D.

Walden University

November 2018

Abstract

Relationship Between Exposure to Phthalate and Obesity in the United States

by

Gladys Ezem

M. S. N., University of Phoenix, 2011

B.S.N., Imo State University, 2002

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Public Health

Walden University

November 2018

Abstract

Obesity is a public health problem. The global obesity problem has been attributed to unhealthy diet, inactivity, and hereditary factors. However, phthalates may also contribute to the obesity epidemic. The purpose of this cross-sectional study was to examine whether there was a relationship between exposure to phthalates and obesity among adults 18 years and older in the United States. The theoretical framework was based on the socioecological model. The quantitative, cross-sectional design, and data analysis were based on the National Health and Nutrition Examination Survey, 2011-2012 database. Descriptive statistics and multiple regression analysis were used to analyze the association between phthalates, obesity, and other independent variables (phthalates exposure, socioeconomic status, gender, age, race/ethnicity, physical activity and place of residence) According to study results, exposure to phthalates does not have a statistically significant effect on the odds of obesity. In terms of the influence of the sociodemographic variables such as race, age, and gender on the relationship between total phthalate exposure and obesity, only race showed statistical significance at $p < 0.05$. In addition, physical activity did not have influence on relationship between total phthalate exposure and obesity among adults 18 years and older in the United States. Despite the lack of significant relationships, the results of this study are useful in addressing the concerns about the exacerbation of obesity. The results of this study could enhance academic research by showing the effect of phthalate chemical exposure, age, gender, race/ethnicity, physical activity, income, and educational level on obesity.

Relationship Between Exposure to Phthalate and Obesity in the United States

by

Gladys Ezem

M.S. N., University of phoenix, 2011

B.S.N., Imo state University, 2002

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Public Health

Walden University

November 2018

Dedication

I dedicate this dissertation to my God, my family, and my friends for their constant support and unconditional love. I love you all dearly.

Acknowledgments

My first gratitude goes to my God who granted me his grace to complete this Course. Despite him, I am nothing. I would like to express my sincere gratitude to my Chair, Dr. Nicoletta, Alexander, for her continuous support, for her patience, motivation, and immense knowledge. Her guidance helped me in all the time of research and writing of this dissertation. I could not have imagined having a better chair for my Ph.D. study. Besides my chair, I would like to thank my dissertation committee members: Dr. Scott, McDoniel and Dr. Diana Naser for their insightful comments and encouragements.

I also thank my friends and family members (too many to list here but you know who you are!) for providing support and friendship that I needed. I would like to thank Dr. Emmanuel Salawu, Dr. Edward Irobi, and Dr. Adaku Madubuko for being supportive throughout my time here and for helping me with proofreading my papers.

I especially thank my mom, dad, sisters, and brothers. My hard-working parents have sacrificed their lives for my survival and provided unconditional love and care. I love them so much, and I would not have made it this far without them. I know I always have my family to count on when times are rough. I thank my beloved sons and daughters, Juliet Ezem, Justice Ezem, Augusta Ezem, and Victor Ezem, who stood by me during the rainy days. God reward you for all your efforts.

Table of Contents

Background of the Study	2
Obesity Level Increase Tendency.....	3
Overview of Obesity	3
The Gap in Knowledge	4
Problem Statement	5
Purpose of the Study	7
Research Questions and Hypotheses	7
Definitions	11
Assumptions.....	12
Biases	14
Significance.....	15
Summary.....	15
Chapter 2: Literature Review	17
Literature Search Strategy.....	18
Synopsis of the Current Literature on the Relevance of the Problem.....	18
Theoretical Foundation	21
Individual	23
Association.....	23
District.....	23
Community	24

Literature Review Related to Key Variables and Concepts.....	24
Obesity	24
Chapter 3: Research Method.....	45
Research Design and Rationale	45
Study Variables.....	45
Research Design.....	46
Methodology.....	48
Population and Setting	48
Sampling and Sampling Procedure.....	49
Sampling Frame	49
Sample Size and Power Analysis.....	50
Justification for the Effect Size.....	51
Research Design.....	51
Study Participants and Informed Consents.....	52
Chapter 4: Results.....	70
Data Collection	70
Secondary Dataset: NHANES 2011-2012	70
Sampling and Sampling Procedure.....	71
Dataset Download and Data Cleaning	71
Results.....	73
Descriptive Statistics: Study Variables.....	73

Descriptive Statistics.....	73
Table 3	75
Numerical Descriptive Statistics of the Categorical Variables in the Study	75
Table 4	78
Numerical Descriptive Statistics for the Continuous Variables	78
Table 7	80
Table 8	80
Graphical Descriptive Statistics.....	82
Table 9	84
Logistic regression assessing Total Phthalate Exposure on Obesity (RQ1).....	84
Table 10	87
Logistic regression results for Total Phthalate Exposure by Race, Age, & Gender*.....	87
Table 11	90
Logistic regression for Total Phthalate Exposure excluding the effect of age, race, and gender*	90
Table 12	93
Logistic regression results Physical Activity Score and Total Phthalate Exposure*.....	93
Chapter 5: Discussion, Conclusion, and Recommendations	98
Interpretation of Findings	99
Research Question 1	99
Research Question 2	100

Appendix A: Title of Appendix	126
Figure A1. Frequency distribution of BMI Category	126
Figure A2. Frequency distribution of marital status	126
Figure A3. Frequency distribution of annual household income.....	127
Figure A4. Frequency distribution of annual family income.....	128
Figure A5. Frequency distribution of gender.....	128
Figure A6. Frequency distribution of race	129
Figure A7. Distribution of total phthalate exposure (ng/mL).....	129
Figure A8. Distribution of age in years at screening	130
Figure A9. Distribution of the number of days of vigorous work	130
Figure A10. Distribution of the number of days of moderate work	131
Figure A11. Distribution of the number of days of walk or bicycle.....	132
Figure A12. Distribution of the number of days of vigorous recreational activities	132
Figure A13. Distribution of the number of days of moderate recreational activities	133
Figure A14. Distribution of the physical activity score	134

Chapter 1: Introduction to the Study

Within the last 30 years, obesity prevalence has risen steadily in the United States (Obesity Statistics, 2014). From 2009 to 2010, the obesity rate in the United States increased to 35.7% in adults and 17% in children (Obesity Statistics, 2014). Obesity has reached epidemic level worldwide (Dahlberg & Krug, 2015). Over 600 million adults are clinically obese; while about 78% of this number is overweight (Obesity Statistics, 2014). Obesity kills more people than hunger or malnutrition (Masters, Reither, Powers, Yang, Burger, & Link, 2013). This health problem leads to roughly 3 million deaths annually (Trasanda et al., 2015). Obesity impacts the mortality rate of cardiovascular diseases, cancers, and Type 2 diabetes (Biro, 2010).

The treatment of obesity affects the financial status of the United States and other countries. For instance, the medical cost of treating obesity in the United States was estimated to be \$147 billion in 2009 (Trasanda et al., 2015). Some endocrine disrupting chemicals like phthalates have been hypothesized to be associated with the increasing incidence of obesity (Trasande, Attina, Sathyanarayana, Spanier, & Blustein, 2013). The huge financial expenditure on the prevention of obesity and the related diseases has formed part of the medical programs all over the world (Trasanda et al., 2015).

In this chapter, I introduced the study topic and presented an overview of the research. The first segment consists of the background, problem statement, study purpose, the research questions, and the study's associated hypothesis. The next part presented the socioecological model (SEM) and its application to the issue of obesity.

The section that follows contains the nature of the study, variables, assumptions, the study scope, and delimitations and limitations. Last, the significance of the study in the advancement of public health knowledge and contribution to positive social change were described.

Background of the Study

Obesity

Obesity diagnosis is based on the body mass index (BMI) expressed as weight/height (Kg/m^2). The growth chart is used to calculate the percentile that is compared with the percentile of people between the same age and sex (Bleich, Pickett-Blakely, & Cooper, 2011). A person with a BMI of 25 to 29.9 is considered to be overweight, while someone with a BMI over 30 is considered to be obese (Bleich et al., 2011).

The treatment of obesity impacts the financial status of the United States and other countries. For instance, the medical cost of treating obesity in the United States was estimated to be \$147 billion in 2009 (Trasanda et al., 2015). Chemicals like phthalates have been hypothesized to be associated with the increasing incidence of obesity (Chang & Nayga, 2009). Phthalates are also known chemically as phthalates esters; they are chemical additives added to plastics, especially polyvinyl chloride (PVC) to improve its properties (Chang & Nayga, 2009).

Obesity Level Increase Tendency

The prevalence of obesity has increased over the last several decades in the United States, Europe, and in developing countries (Hatch et al., 2008). Obesity prevalence rises steadily and almost doubled up universally between 1980 and 2008 (Hatch et al., 2008). Although dietary patterns and physical activity are causal factors in the obesity epidemic, endocrine disrupting chemicals like phthalates may impact obesity-related pathways by altering hormone action or gene expression (Hatch et al., 2008). Obesity can cause chronic health concerns, emotional, metabolic, and physical health problems (Chang & Nayga, 2009)

Overview of Obesity

Scholars who analyzed samples obtained from Human provided useful information that shows the link between exposures to phthalate chemicals and obesity (Plotnikoff, Lightfoot, Spinola, Predy, & Barrett, 2011). Thayer, Heindel, Bucher, and Gallo (2012) used National Health and Nutrition Examination Survey (NHANES), a representative sample of people living in the United States, to examine if phthalate chemicals alter the differentiation of adiposities and found that 30% of obesity is associated with phthalates exposure.

The compounds of the food consumed represent one of the most significant aspects of obesity (Kelishadi, Parinaz, & Fahmeh (2013). Kelishadi et al. (2013) observed that exposure to phthalate chemicals could increase the risk of excess body fat. Baillie-Hamilton (2002) examined the relationship of plastic chemicals to obesity and postulated

that people exposed to higher levels of phthalates are more likely to be obese than those who are exposed to lower levels. Wang, Zhou, Tang, He, Wu, Chen et al., 2013 stated that phthalates impede the metabolism of fat, thereby causing obesity despite the consumption of healthy food and daily exercise.

The disorders related to obesity represent a problem not only for patients but also for the health system of any community or country (Robinson, 2008). When treating this disease, people should engage in activities aimed at the normalization of metabolic disorders and weight losses. The effectiveness of the treatment of obese patients is low, as the majority of patients lose weight slowly (Robinson, 2008). The slow weight loss is caused mainly by people taking a passive position on the stage of decreased body weight stabilization. Some doctors express pessimism towards the patients' effort to reduce weight. In controlled clinical trials, there is a small average weight loss and a high degree of recurrence (Robinson, 2008).

The Gap in Knowledge

Knowledge of obesity risk factors is paramount to the prevention of obesity, especially in a community at risk for obesity. Although the CDC (2016) recommended that phthalates chemicals and their effect on human health be studied further, “only very few studies have examined the health effects of phthalates” on humans (Casas, 2011, p. 858). In addition, there is a gap in the study of the mechanisms and factors of disturbance of mental adaptation to obesity (Russell-Mayhew, McVey, Bardick, & Ireland, 2012). Solving these problems is in alignment with the biopsychosocial approach and adaptation as a

common methodological basis for medical psychodiagnostic in its understanding of the system.

There are little data known about the features of coping with stress and protective behavior and their influence on the process of adaptation to overweight in the United States. However, scholars have not focused on members' self-esteem in the structure of self-evaluation and prospects in life, psychological factors of resistance to therapy, and related to comorbid disorders. This study will contribute to filling that knowledge gap.

Problem Statement

Obesity is a prevailing public health problem that impacts both developed and developing countries (Janesick, & Blumberg, 2011). The prevalence of obesity and its related health problems has increased over several years and is a risk factor for chronic health diseases like diabetes, cardiovascular illness, and cancer (Sameera & Amar, 2012). These results entail the need for a more detailed study on obesity

Earlier studies were centered on the risk factors of obesity like diet pattern, increased caloric consumption, and physical inactivity; however, information on the obesogen hypotheses that endocrine-disrupting substances like phthalates contribute to obesity remains comparatively under- examined (Merrill & Birnbaum. 2011). Merrill and Birnbaum (2011) showed the impact of substance groups called phthalates on obesity. Moisse (2012) demonstrated the association between endocrine-disrupting substances and weight gain. Baillie-Hamilton (2002) noted the role of endocrine disruptors in the etiology of obesity and emphasized that the obesity epidemic amplified as a result of the

increase of industrial chemicals over the past 4 decades. Scholars should investigate the impact of endocrine disrupting substances like phthalates on obesity to illuminate one of the possible causes of obesity. In this study, I investigated if a relationship existed between obesity and exposure to phthalates in the United States.

To understand better the essence of obesity, it is critical to denote more data about phthalates in the context of the current study. Phthalates (directory o-phthalic acid) are used as plasticizers in the manufacture of polymeric materials. They are mainly based on polyvinyl chloride and polystyrene, as well as synthetic and natural rubber products for industrial, consumer, food, and medical use (Kim & Park, 2014). Phthalates do not form chemical bonds with the polymer. Hence, they can gradually migrate from the finished products into the environment. In the application and use of polymer products, phthalates can enter the water, soil, air, food, and the human body. Due to the high thermal and photostability, resistance to hydrolysis can occur in neutral environments; these compounds can accumulate in the environment. Phthalates are toxic substances that can accumulate in the fatty tissues of living organisms and can result in mutagenic, carcinogenic, and teratogenic effects (Kim & Park, 2014).

Phthalates also are known chemically as phthalates esters, which are primarily chemical additives used in plastics to improve its properties (Kim & Park, 2014). They are used in the production of a variety of common household and health products from vinyl flooring, adhesives, detergents, plastic care products, urinary catheter, blood-storage containers, and medical tubing (Kim & Park, 2014). Their health effects are

significant. Environmental exposure to phthalates has been implicated in some public health issues ranging from fertility, cancers, metabolic disorders, and obesity (Kim & Park, 2014). The mechanism by which environmental exposure to phthalates leads to obesity is through the antiandrogenic effects, reactivation of peroxisome proliferators' activated receptors (PPARs), and deregulation of lipid metabolism (Kim & Park, 2014).

Purpose of the Study

The purpose of this study was to examine if there was a relationship between exposure to phthalates and obesity among adults 18 years and older in the United States. I also investigated if education level, place of residence (zip code), socioeconomic status (defined by income and educational level), and race/ethnicity influenced the relationship between exposure to phthalates and obesity. Lastly, I evaluated whether or not the gender, age, and /or the behavioral variable (physical activity) influenced the relationship between exposure to phthalates and obesity in this group.

The dependent variable was identified as obesity status. Obesity was calculated by using the participant's height and weight. The independent variable was phthalate exposure. Covariates considered were gender, education level, place of residence (zip code), age (sociodemographic variables), and physical activity (behavioral).

Research Questions and Hypotheses

I explored the relationship between phthalate chemicals exposure and obesity among adults 18 years and older in the United States. I also investigated whether or not

there was a relationship between gender, age, physical activity, socioeconomic status, place of residence, race/ethnicity, and exposure to phthalates and obesity in this group.

RQ1: Is there an association between exposure to phthalates and obesity among adults 18 years and older in the United States?

H_01 : There is no association between phthalates exposure and obesity among adults 18 years and older in United States.

H_11 : There is association between phthalates exposure and obesity among adults 18 years and older in the United States.

RQ2: Do the sociodemographic variables (SES, race/ethnicity, gender, age, place of residence) influence the relationship between exposure to phthalates and obesity among adults 18 years and older in the United States?

H_02 : The sociodemographic variables have no statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older the United States.

H_12 : The sociodemographic variables have statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older in the United States.

RQ3: Does physical activity influence the relationship between exposure to phthalates and obesity among adults 18 years and older in the United States?

H₀₃: Physical activity has no statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older in the United States.

H₁₃: Physical activity have statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older the United States.

Theoretical/Conception Framework

The theoretical framework is used to establish the structure that guides the research by providing the background that supports the investigation and by offering the justification for the study of a research problem (Cassel, 2010). An efficient control of obesity could be achieved by placing an emphasis on the correction of behaviors, culture, and the belief systems, all of which are contributing factors to obesity (Wang et al., 2013). Obesity is an escalating “public health problem that is best tackled by an integrated approach, which is enabled by integrated public health policies” (Wang et al., 2013, p.86). A comprehensive framework that can help to identify options for improvement and to systematically develop solutions will assist in handling the obesity crisis (Su, 2015).

I employed the socioecological model as the theoretical framework. This model was used to highlight the problem of overweight and obesity by conceptualizing several levels of pressure, supportive, and preventive programs that would handle the various levels (Plotnikoff et al., 2011). This model provides a framework for examining behavior

and relationships within the environment by focusing on the social, physical, societal, and governmental influences on behavior (Plotnikoff et al., 2011). This theory centers on individual, relationship, community, and society and explains that the complicated relationships that exist between the individual, relationship, community, and the society need to be examined for a successful understanding of the problem of obesity. I used the constructs of this theory to understand the impact of phthalates on obesity.

Nature of the Study

This study is a quantitative, cross-sectional study using secondary data. In the cross-sectional study design, the prevalence of the outcome of interest, for the population or subgroups within the population at a given time-point, are investigated (Levin, 2006). This design was chosen because it can provide empirical data on the extent of the lack of knowledge of obesity risk factors as the driving force behind the increasing obesity risk. Also, the study's results emphasize the need for enhanced preventive measures in quality obesity awareness strategies.

The data were analyzed using IBM SPSS statistical software, version 21. The association between phthalate chemical exposure and obesity was assessed using the odds ratios (ORs). The assessment was done using logistic regression models (Field, 2009). The dependent (outcome variable) variable for this study was obesity status (obese or not obese), while the independent variables were the phthalate chemical constituents in food and knowledge of the chemical composition. Sociodemographic variables such as gender, educational level, and zip code were analyzed as covariates, among others. Logistic

regression is a statistical approach for analyzing a dataset in which there are one or more independent variables that determine an outcome. The outcome is measured with a dichotomous variable (in which there are only two possible outcomes; MedCalc, 2016). The regression models were designed to make sure they were not violated. Logistic regression can include all sorts of relationships, because it applies a nonlinear log transformation to the predicted ORs. Further, the independent variables do not need to be multivariate normal—although multivariate normality yields a more stable solution. Behavioral variables data, such as smoking and alcohol consumption, were also collected. Pertinent medical history such as hypertension, diabetes, family history of obesity, prior overweight or obesity, and high cholesterol were retrieved. These assisted in defining the at-risk population.

Definitions

The operational terms and definitions below will clarify the terms and concepts used in this study.

Body mass index (BMI): BMI is often used for obesity diagnosis (Bray, 1985).

Obesity: Obesity results from a caloric imbalance in which an individual burns few calories when compared to the amount consumed (Hu, 2008). In this study, a person with a BMI greater than or equal to 30 was considered overweight (Bray, 1985).

Overweight: Excess body weight for a height confirms a person as overweight (Hu, 2008). In this study, a person with a BMI between 25 and 30 was considered obese (Bray, 1985).

Phthalates: Chemically known as phthalates esters. They are chemical additives used in the manufacture of plastics to improve its properties. As an additive to plastics products, phthalates make them flexible and makes the fragrance of personal care products last longer. Phthalates are used in the production of a variety of common household and health products; this makes their health effects significant (Kwack, 2010). The link between phthalates and obesity has become a subject of interest among researchers, although animal models have also been implored (Kwack, 2010).

The independent variables for this study were knowledge of risk factors for obesity (phthalates chemicals). The study covariates included age, weight, height, family history of obesity, prior overweight or diabetes, high cholesterol, hypertension, number of days of vigorous work, and moderate intensity work. Vigorous work is a vigorous-intensity activity that causes large increases in breathing or heart rate and is done for at least 10 minutes continuously (NHANES, 2011, 2012). Moderate is a moderate-intensity activity that causes small increases in breathing or heart rate and is done for at least 10 minutes continuously (NHANES, 2011, 2012)

Assumptions

I made some assumptions in this study. I assumed that the participants answered the questions honestly and that the scale used was a valid measure of the construct of interest. Also, I assumed that participants who participated in similar surveys in the past would not introduce stereotyped responses. I also assumed that the tools used for data collection adequately measured the designated variables. These assumptions are of

particular importance for the study because they direct the investigation and serve as a basis for the achieving the results expected.

Scope and Delimitations

The study covered the problem of the obesity rates in the United States. The study provides new information about the role of individual personality and emotional and behavioral factors in the disturbances of mental adaptation with nutritional obesity and associated pathology. The study also covers the complex psycho-social relations, determining eating disorders, psychological adjustment disorders, and overweight. I defined the importance of self-corporeality (appearance) of these subjects in the hierarchy of their actual values.

Limitations

Study limitations are the recognized weaknesses that affect the rigor of a study (Salazar, Crosby, & DiClemente, 2006). This study was limited to the people of the United States, which may not be used for generalization on other counties. The use of secondary data that was originally meant for another study also acted as a limitation. The inflexibility of the questions designed to allow the extraction of honest answers to enhance the reliability and validity of the study also added to the limitations (Creswell, 2009). Another limitation involved the literacy standard of the respondents, which may be below the expected standard. To reduce the impact of the limitations, increase the diversity of the population, and to avoid the use of one-sided participants, a random sample size was used.

Biases

Any systematic error that results in a wrong estimate of the association between exposure and risk of disease is known as a bias (biases that may impact this study include,

Information bias results from differences in the way of obtaining data on exposure or outcome from the participants. Secondary data gathered for another purpose and issues of lag between events and reporting may introduce bias to this study (Creswell, 2009). To prevent this bias, the NHANES with standard questionnaires was used, and protocol was developed for the collection, measurement, and interpretation of information.

Observer bias may occur as a result of the investigator's prior knowledge of the hypothesis under investigation or knowledge of an individual's exposure status (Creswell, 2009). This may lead to differences in the way information is collected, measured, or interpreted by the investigator for each of the study groups. Members were blinded to the hypotheses to prevent this bias.

Selection bias is introduced when the two groups being compared differ systematically. There are differences in the characteristics between those who are selected for a study and those who are not selected (Creswell, 2009). Randomization used in this study may help to minimize this bias.

Reporting bias is selective revelation or suppression of relevant information about the study by either the participants or the researcher may introduce bias to the study result (Creswell, 2009). This bias was controlled by using reliable data from the NHANES.

Significance

I explored the impact of phthalates chemicals on overweight and obesity among adults 18 years and older in the United States. I also investigated whether or not there is a relationship between gender, age, physical activity, SES, race/ethnicity, and exposure to phthalates and obesity in this group. The information from this study helps in the establishment of effective programs that will reduce the prevalence of overweight and obesity. The identification of the factors that drive the increased prevalence of obesity help in primary prevention and reduction of the incidence of obesity. Primary obesity prevention is important because it is not easy to lose weight once a person gets to obesity status. The findings lead to a greater awareness and implementation of obesity risk factor preventive measures in the communities, health care providers, and policy change. According to Bienkowski (2012), endocrine-disrupting chemicals like phthalates destabilize the metabolism of fat by impacting the action of weight-controlling hormones in the body and decrease their sensitivity to neurotransmitters. There is a need in a metabolic plan and other associated with obesity disorders.

Summary

Many scholars have established that a relationship exists between the exposure to phthalates and obesity (Fulcher & Gibb, 2014; Sargis, Choudhurg, & Brady, 2010). There is a need for primary obesity prevention in the United States. Although obesity has been discussed in the scholarly research, the current investigation plays a different role because it covers the United States as a whole.

Knowledge of the relationship between exposure to phthalates and obesity is of importance in the prevention of obesity. Obesity is one of the leading cause of long-term disability (Newbold, 2010), and the need for primary obesity prevention in the United States is critical. Obesity prevention can be achieved by addressing the issues from a socioecological perspective, because, although the individual may be directly involved, the after effect of obesity constitutes an individual as well as the national problem both in direct and indirect costs (Newbold, 2010)).

In Chapter 2, I presented a review of existing literature on the relationship between phthalates exposure and obesity. This section includes an overview of the socioecological framework. The chapter ended with the implications of past research and its relationship to this study. In Chapter 3, I focused on the design and methodology used in conducting the research. I discussed the tools used in data collection and result analysis: sample population, sample size, the procedure for data collection, the questionnaire, ethical considerations, measures, and data analysis. In Chapter 4, I presented the results. I also addressed the research questions and the relationship between the variables. In Chapter 5, I discussed the findings and the implications of the results of the study. Recommendations for future studies were made based on results and the social change implications of the study.

Chapter 2: Literature Review

Environmental exposure to phthalates has been implicated in a number of public health issues including obesity, infertility, cancers, and metabolic disorders (Fulcher & Gibb, 2014; Newbold, 2010; Sargis et al., 2010). As a result of the use of phthalate-related products, human can be exposed to this chemical through many ways, such as ingestion, inhalation, dermal contact, and placenta transmission (Hao, Cheng, Xia, & Ma, 2012; Heindel, Newbold, & Schug, 2015). Ingestion of food is the major exposure pathway for phthalate exposure, especially for high molecular weight phthalates, like di (2-ethylhexyl phthalate (DEHP) and Butyl benzyl phthalates ((Thayer et al., 2012). Obesity has been linked to environmental exposure to phthalates (Moisse, 2012).

The relationship between obesity and exposure to phthalate chemicals in the United States and the individual risk factors for obesity has not been fully explored. The purpose of this study, therefore, was to examine whether or not there was an association between phthalates chemicals exposure and obesity.

In this chapter, I present current, peer-reviewed articles on the impact of phthalates chemicals on obesity as it relates to the United States. The approach and methodology for the study lends support from current literatures. The theoretical framework was based on the constructs of the socioecological angle, which states that multiple levels of influence exist around the individual to impact the outcome of behavior. I review the socioecological theory by stressing the significance of the various levels of influence in addressing the increased obesity prevalence in this population. The

influence of health literacy and obesity literacy on obesity among the people in United States were discussed, while addressing the influence of past research on the study.

Literature Search Strategy

A methodical literature exploration was conducted using Walden Library, Pub med, Cochrane library, Google scholar, Google, and Internet web of science. In Walden library, I used medical subject headings and free text terms like *endocrine disruption and endocrine suppression chemical, obese or overweight, endocrine-disrupting chemicals like phthalates, phthalate, endocrine disruptors, obesity, and obesogens*. The same text word searches were used in Pub med, Google scholar, Google, Cochrane library, and Internet web of science. Studies were included that defined the phthalates chemical groups and its association to obesity. Current literature published between year 2000 and 2006, and articles that discussed current theories and methods were searched. The studies excluded were those without abstract and full texts, letters, case reports, and reviews.

Synopsis of the Current Literature on the Relevance of the Problem

The consumption of high-calorie fast foods, soft drinks, and sedentary activities has been responsible for the increased incidence of obesity (Brown, DeBanate, & Rother, 2010). However, these factors are insufficient to explain the increase in obesity during the 20th century (Mayo Clinic, 2010). Baillei-Hamilton (2002) projected that the global obesity epidemic was as a result of exposure to endocrine disrupting chemicals (EDCs) and established that increased manufacture of industrial chemicals coincided with exacerbation of obesity in the Unites States. Catecholamines, like adrenaline and

dopamine, that help weight loss are reduced by phthalate chemicals, which affects both metabolism and the desire to go out for a walk (Baillie-Hamilton, 2010). Thayer et al. (2012) showed a link between some EDCs and obesity. According to Thayer et al. (2012) “chemicals like phthalates alter differentiation of adiposities or the development of neural circuits that regulate feeding behavior,” (p. 783) thereby leading to increased BMI. Tang-Peronard, Andersen, Jensen, and Heitmann (2011) showed that some obesogens-like phthalates may play a role in the development of obesity, thereby highlighting the relationship between EDCs and obesity.

Obesogen is the name given to a set of EDCs that support weight gain and obesity. These chemicals may cause obesity in a number of ways including disruption of critical lipid metabolism pathways to promote adiposeness and fat storage, the alteration of the metabolic set point to induce positive energy balance, or increasing appetite (Kim & Park, 2014). There are positive associations between obesogen levels, including phthalates and body weight or BMI (Kim & Park, 2014). Thayer et al. (2012) used NHANES to examine if phthalates chemical alter differentiation of adiposities, and they found that 30% of obesity is associated with phthalates exposure.

Phthalates were originally designed to soften plastic products to enhance their flexibility. They are also used as perfumes in cosmetics. They are mostly seen in a range of domestic products or private care products, ranging from building materials, shower curtains, food packaging, and medical devices (Kim & Park, 2014). The exposure of human beings to phthalates can occur through ingestion of contaminated food and water,

dermal contact, inhalation of polluted air, and parental exposure from medical devices (Kim & Park, 2014)). Phthalates may support obesity through antiandrogenic effects, antithyroid hormone activities, and/or activation of PPARs (Chamorro-Garcia et al., 2012). explored the association between endocrine hormones and obesity and found that some endocrine disruptors like phthalates may play a role in the development of obesity. Wang et al. (2013) investigated the association between phthalate exposures, BMI and waist circumference (WC) in Chinese people. Wang et al. showed that some phthalate exposures were associated with BMI or WC in Chinese people. Moisse (2012) postulated that individuals get obese when exposed to high levels of bisphenol A (BPA). BPA is the chemical banned from baby bottles that are still being used in food containers. This chemical belongs to the endocrine-disrupting group. Moisse observed that, although over 92% of the study participants had detectable levels of BPA in their urine, those with the highest levels were 2.6 times more likely to be obese than those with the lowest levels.

Kelishadi et al. (2013) conducted a study on ecological chemicals and their mode of action and concluded that exposure to environmental factors could increase the risk of excess body fat. According to the Kelishadi et al., (2013), “the devastating effects of these chemicals are mostly observed during the developmental stage of life, when reorganization of adipose tissues occurs” (p. 116). Trasande, Attina, Sathyanarayana, Spanier, and Blustein (2015) stated that “less than 20% causation of obesity, and type 2 diabetes are associated with high molecular weight endocrine disruptors” (p. 216).

Theoretical Foundation

The socioecological model served as the theoretical framework for this study. This model was used to highlight the problem of overweight and obesity by conceptualizing several levels of pressure and supportive preventive programs that would handle the various levels of the pressure (Plotnikoff et al., 2011). The socioecological theory was developed by Bronfenbrenner and Condry (1970) to scrutinize human development, the developing person, the environment, and the interaction between the two (Cassel, 2010). Bronfenbrenner and Condry believed that interrelated levels of association exist between the individual and the environment and that the relationship influences the life of the individual (Bronfenbrenner & Condry, 1970). I chose this model is because it provides a framework for examining behavior and the relationship within the environment by focusing on the social, physical, societal, and governmental influences on behavior (Plotnikoff et al., 2011). This theory is related to the research questions by building on the association between the individual, relationship, community, and society that may impact obesity. I will use this theory to examine the complex relationship that exists between the individual, relationship, community, and the society to understand the problem of obesity. The constructs of this theory were used to understand the impact of endocrine-disruptors on obesity. The socioecological model has been previously used as a study and intervention outline to help in the comprehension and alleviation of obesogenic factors in the Samoan community in the state-level tobacco prevention (Cassel, 2010).

This figure shows the relationship between the individual, community, and the society.

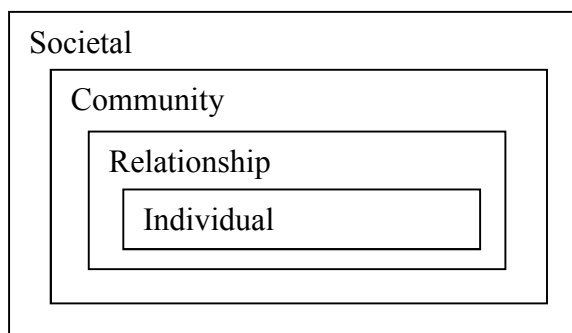


Figure 1. Ecological framework view of health.

The objective of this theory is to prevent an action before its occurrence. The preventive mechanism will include consideration of the factors that impact the problem (Dahlberg & Krug, 2015). The CDC (2016) advocated the use of a four-level social-ecological model to better understand a problem and the effect of potential prevention strategies. This model includes the intricate interplay between individual, relationship, community, and societal factors. It allows people to recognize the range of factors that put them at risk for a problem or protect them from developing health problems (Cassel, 2010). The overlapping rings in the model illustrate how factors at one level influence factors at another level. In order to prevent a health problem, it is essential to act across several levels of the model at the same time. This will help to maintain preventive efforts over time than any single intervention (Dahlberg & Krug, 2015; Morley & Pratte, 2013).

Individual

The first level identifies biological and personal history factors that increase the likelihood of becoming obese. Some of these factors are inactivity, sedentary life style, consumption pattern, and behavior. Prevention strategies at this level are often designed to promote attitudes, beliefs, and behaviors that prevent obesity. Definite strategies may involve education and life skills training (Cassel, 2010; Dahlberg & Krug, 2015).

Association

The second level includes relationships that may increase the risk of becoming obese. Usually, an individual's closest social circle like, peers, partners, and family members influence the behavior and contribute to the range of experiences. Prevention strategies at this level may include parenting or family-focused prevention programs and mentoring and peer programs designed to reduce obesity and promote healthy eating patterns (Dahlberg & Krug, 2015; Morley & Pratte, 2013).

District

The third level includes the settings, like schools, workplaces, and neighborhoods in which social relationships occur and seeks to identify the uniqueness of these settings that are associated with obesity (Marley & Pratte, 2013). Prevention strategies at this level are typically designed to impact the social and physical environment by reducing the serving of sodas in schools; improving the availability of parks and recreational activities in neighborhoods; and improving the climate, processes, and policies within schools and places of work (Dahlberg & Krug, 2015).

Community

The fourth level includes the societal factors that help create an environment in which obesity is encouraged or inhibited. These factors comprise social and cultural norms that sustain obesity as an acceptable way of life. Other large communal factors include the health, economic, educational, and social policies that help maintain economic or social inequalities between groups in society (Cassel, 2010).

Literature Review Related to Key Variables and Concepts

Obesity

Obesity affects one in six individuals in the United States (Ogden et al., 2014). Obesity diagnosis is based on the BMI expressed as weight/height (Kg/m²). Growth chart is used to calculate the percentile that is compared with the percentile of people between the same age and sex (CDC, 2016). Between 85th and 94th percentile or above is regarded as overweight, while 95th percentile or above indicates obesity and more than 1.2 times the 95th percentile depicts extreme obesity (CDC, 2016). The treatment of obesity impacts the financial status of the United States and other countries (Obesity: Implications for Prevention and Treatment, 2012). The medical cost of treating obesity in the United States was \$147 billion in 2010 (Trasande et al., 2015) Obesity resulting from environmental exposure to phthalates can begin from the time a baby is still in the uterus or continue from birth ((Holtcamp, 2012). The clinical manifestation of obesity is still dependent on factors such as type of phthalate ester exposed to race, sex, and gender (Plotnikoff et al., 2011). Scholars have shown the link between exposures to phthalate

chemicals and obesity (Lind, Roos, Ronn, & Johansson, 2012; Plotnikoff et al., 2011). Understanding this relationship is critical to solving this endemic public health issue (Song et al., 2014). Most epidemiologic studies examining the association between phthalate exposure and obesity have been based on the data from the NHANES. Marijke, Boer, Lamoree, Legler, and Margot (2013) explored the association between prenatal exposure to the EDCs and child growth early in life. Marijke et al. showed a positive relationship between exposure to phthalates and BMI. However, the small sample size limits the study findings. The findings added more insight to this research by showing that being overweight in early life may be prognostic of obesity later in later life.

La Merrill and Birnbaum (2011) postulated that phthalates exposure alone might not lead to obesity. Risk factors like diet components, inactivity, heredity, and the built environment are areas for further investigation because exposure to chemicals before and during pregnancy can contribute to the risk of obesity. La Merrill and Birnbaum concluded that organ chlorine chemicals and other related chemicals are possible risk factors for obesity. The findings from this study may help to view obesity not only in terms of heredity and human existence, but also in terms of how early life exposure to the obesogenic chemicals could set the pace for increase in body size later in life. Sargis et al. (2010) added that the latent contributions of environmental pollutants that disrupt endocrine metabolism should be considered in the pathogenesis of obesity because glucocorticoids' signaling is vital to adiposity differentiation. Sargis et al. assessed the ability of the glucocorticoid compounds to promote the deposition of adipose tissues by

using quantitative oil red O staining and immunoblotting for adiposity-specific protein in ten specimens. Sargis et al. observed that steroids like glucocorticoids promote adiposity differentiation by synergizing with agents present in the differentiation cocktail. Therefore, these hormones are able to promote adipogenesis through the stimulation of glucocorticoid receptor (Sargis et al., 2010).

Gittner, Ludington-Hoe, and Haller (2013) examined whether growth pattern in healthy infants in the beginning of life can predict obesity in later life. Gittner et al. concluded that children who were obese at age 5 had a BMI pattern that is different from children who exhibited normal weight at age 5. Gittner et al. illuminated that the identification of obesity development early in life may help in the prevention of obesity. Gittner et al. also showed that life BMI growth pattern in the beginning is clinically important as it allows discrimination of those who do not fit a normal weight pattern, which includes individualized treatment in early and in later years while precursors of later on health are still forming.

Behl et al. (2015) examined the relationship between maternal smoking in pregnancy and obesity. Behl et al. showed that early exposure to nicotine chemical through maternal smoking habit leads to obesity. Nevertheless, Behl et al. agreed that meta-analytical method may not be suitable for the study because “its results can only be as good as the original data is valid” (Behl et al., 2015, p. 121). Thayer et al. (2012) postulated that exposure to EDCs can impact subsequent generations, even though the current generation may not have been exposed to the chemicals. Thayer et al. confirmed

this transgenerational effect of exposure to phthalates chemicals after the analysis of literature from humans and experimental animals exposed to certain environmental chemicals. Chemical exposures may increase the risk of obesity by altering the differentiation of adiposities or the development of neural circuits that regulate feeding behavior. Avoidance of chemical exposure should start from previous generations. Thayer et al. did not consider the differing social and cultural contexts that will affect the study result. Also, research on environmental chemical exposures and type 1 diabetes was limited. I stopped reviewing here. Please go through the rest of your chapter and look for the patterns I pointed out to you. I will now look at your Chapter 3.

Endocrine System/ Endocrine Disruption

The endocrine system is made up of the group of glands that manufacture hormones that control metabolism, growth and development, tissue function, sexual function, reproduction, sleep, and mood, (Newbold, 2010). These group of glands are controlled directly by stimulation from the nervous system and by chemical receptors in the blood and hormones formed by other glands (Newbold, 2010). These glands maintain homeostasis by regulating the functions of organs in the body. Endocrine disrupting chemicals like phthalates imitate or partly mimic naturally occurring hormones in the body there by producing overstimulation (Newbold, 2010). Endocrine disrupting chemicals can also bind to a receptor within a cell and block the endogenous hormone from binding. The normal signal then fails to occur and the body fails to respond properly. Also, this chemical can interfere or block the way natural hormones or their

receptors are made or controlled, by altering their metabolism in the liver (Newbold, 2010).

When absorbed in the body, an endocrine disruptor can decrease or increase normal hormone levels (left), mimic the body's natural hormones (middle), or alter the natural production of hormones (right).

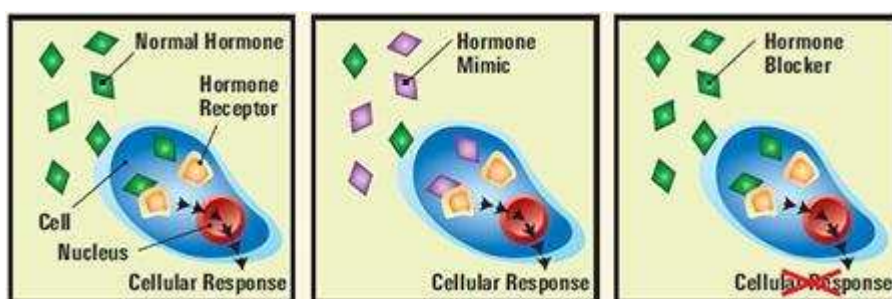


Figure. 2. How endocrine disruptors affect the body. (Left) Absorbed endocrine disruptors cause decrease or increase in hormone levels. (Middle) Absorbed endocrine disruptors mimic the natural hormones of the body. (Right) Absorbed endocrine disruptors can alter hormones' natural production. Source: National Institute of Environmental Health Sciences (2016).

Obesogen is the name given to a set of EDCs that support weight gain and obesity. These chemicals may cause obesity in a number of ways including; disruption of critical lipid metabolism pathways to promote adiposeness and fat storage; the alteration of the metabolic set point to induce positive energy balance, or increasing appetite (Kim & Park, 2014). Evidence show positive associations between obesogen levels, including phthalates, and body weight or body mass index (BMI) (Kim & Park, 2014). In a related

prospective study, Thayer et al. (2012) used NHANES, a generally representative sample of people living in the United States to examine if phthalates chemical alter differentiation of adiposities. They found that 30% of obesity is associated with phthalates exposure.

Endocrine disrupting chemicals

BPA. BPA stands for biphenol A. This is an industrial chemical that has been used to make certain plastics and resins since the 1960s. Bisphenol A is an organic artificial compound with the chemical formula $(\text{CH}_3)_2\text{C}(\text{C}_6\text{H}_4\text{OH})_2$ (Singh, & Li, 2011). It is an EDC and belong to the group of diphenyl methane derivatives and bisphenols, with two hydroxyphenyl groups (Mayo Clinic, 2012). Bisphenol A may interfere with the endocrine system in the body. Human beings get exposed to Bisphenol A via ingestion of food, dust and water, inhalation of gases and particles in the air, and through the skin (Mayo Clinic, 2012). EDCs can also be transferred from the pregnant woman to the fetus or child through the placenta and breast milk (Moisse, 2012). These chemicals are suspected to be associated with altered reproductive function in males and females; increased incidence of breast cancer, abnormal growth patterns and obesity (Singh, & Li, 2011; Tang-Peronard et al., 2011; Thayer, Heindel, Bucher & Gallo, 2012).

Phthalates. Phthalates are also known chemically as phthalates esters, they are essentially chemical additives added to plastics, especially polyvinyl chloride (PVC) to improve its properties (Shin & Park, 2014). Phthalates are used in the manufacture of plastics, solvents, and personal care products. As an additive to PVC products phthalates

make them flexible and makes the fragrance of personal care products last longer (Kwack, 2010). They are colorless, odorless, oily liquids that do not evaporate easily and do not chemically bind to the material they are added to (Shin & Park, 2014). Phthalates are readily absorbed into the human body and are converted quickly to their respective metabolites (Hatch et al., 2013). Unlike some chemicals, they tend to pass out of the body quickly in urine and feces. The exposure effect can be increased as a result of synergetic action (Shin & Park, 2014).

Phthalates are used in the production of a wide variety of common household and health products from vinyl flooring, adhesives, detergents, plastic care products, urinary catheter, blood storage containers, medical tubing (Kwack, 2010). This makes their health effects significant. As esters they are derived from the chemical reaction between phthalic anhydride and alcohol. The number of carbon atoms in the alcohol chain of a phthalate is the criteria used in grouping the various phthalates into two major groups (Shin & Park, 2014). There is the lower molecular weight phthalate which contains between 3- 6 carbon atoms in its alcohol chain and the higher molecular weight phthalates with more than 6 carbon atoms in their alcohol chain (Hatch, 2013). The linkages between phthalates and obesity in humans have continuously become a subject of interest among researches, although animal models have also been implored (Kwack, 2010).

Potential Source of Exposure to Phthalates

Since 1930 manufacturing industries have been using phthalates as plasticizers, and this chemical is currently used as additives in various consumer products. It has been estimated that, the global use of phthalates should be about several million tons per year (Kim & Park, 2014). The high molecular weight (HMW) phthalates, are mostly used in the manufacture of polyvinyl chloride (PVC) plastics for food packaging, building materials, and medical devices, while Low molecular weight (LMW) phthalates, are usually used in the manufacture of personal care products like perfumes, lotions, cosmetics, shampoo, paints, and adhesives (Kim & Park, 2014). Phthalates particles are always emitted from PVC and plastic materials, resulting in contamination of indoor air, house dust, or food. As a result, the primary methods of HMW phthalate exposure are ingestion of the particles. Phthalates are weak endocrine disruptors and androgen blocking chemicals. The chemicals can be released from a product by heat, agitation, and prolonged storage (Kim & Park, 2014). When absorbed into the blood stream, phthalates can either mimic or block hormones or suppress the hormonal activity (Kim & Park, 2014). Table 1 presents phthalates, source of exposure and metabolite.

Table 1

Sources of Phthalates Exposure

Phthalates	Source of Exposure	Metabolite
Low molecular weight Diethyl phthalates (Low molecular weight)	Personal care products like deodorant, fragrance, shampoos, after shave	Monomethyl phthalate (MMP)

Diethyl phthalate (DEP)	Skin care, nail care, makeup	Monoethyl phthalates (MEP).
Di-n-butyl phthalates (DBP)	Paints, adhesives, perfumes	Mono-n-butyl phthalates (MBP).
Di-iso-butyl phthalates (DiBP)	Paints, adhesives	Mono-iso-butyl phthalates (MiBP)
HIGH MOLECULAR WEIGHT BBzP	Toys, food packaging, synthetic leather, paint	Monobenzyl phthalates (MBzP)
DEHP	Furniture, gloves, dust, medical devices, floor tiles, wall coverings	Mono(2-ethylhexyl) MEHP Mono (2-Carboxy-hexyl) MCHP
Di-iso-nonyl phthalate DNP	Clothes, footwear, car interior	Mono-iso-nonyl phthalate (MnNP) Mono (carboxy-iso-octyl) phthalate (MCiOP)
Di-n-octyl phthalate (DnOP)	Package sealants, bottle cap liners, garden hoses, wire and cable insulators, vinyl gloves	Mono- (3-carboxypropyl) phthalate (MCP)

Di-isodecyl phthalate (DiDP)	Lamination film, foot wear, school supplies, scented erasers, pencil case	Mono-isodecyl phthalate (MiDP) Mono (Carboxynonyl phthalate (MCNP).
------------------------------	---	---

Metabolism of Phthalates

After exposure to Phthalates, the chemicals are rapidly metabolized and excreted in urine and feces. The initial hydrolyses involve the breakdown of diester phthalates by esterases and lipases present in the intestine and parenchyma to their respective monoester phthalates (Janesick & Blumberg, 2011). This low molecular weight phthalate (LMW) are excreted in urine and feces as a monoester, without more metabolisms. Conversely, high molecular weight phthalates (HMW) are additionally broken down from monoesters through hydroxylation or oxidation, to produce a number of oxidative metabolites (Song et al., 2014). These oxidative metabolites of phthalates are excreted in urine within 24 hours of exposure. On the other hand, oxidative metabolites can undergo another phase of conjugation to form hydrophilic glucuronide conjugates, which are excreted rapidly in urine.

Mechanism Through Which Phthalates Cause Obesity

Phthalates may promote obesity through the interruption of thyroid function, which plays a major role in the regulation of energy balance and metabolism. Thyroid function has been seen to impact the regulation of BMI. A slight change in thyroid-stimulating hormone (TSH) or thyroxin levels can cause considerable differences in resting energy expenditure (Schug, Janisick, & Blumberg, 2011). For example, slight increase of the blood TSH levels is linked to both weight gain over 5 years and obesity in a population study (Schug, Janisick, & Blumberg, 2011).

The "frugal phenotype" that results from contact to malnourished setting and endocrine-disrupting chemicals could be one of likely mechanisms through which phthalates promote obesity (Kim & Park, 2014) It has been shown that changes, resulting from an undesirable environment, may result in augmented uptake and preservation of nutrients, and prompt individuals to obesity and other metabolic disorders (Hatch et al., 2013).) Epidemiological studies provide evidence that maternal malnutrition during pregnancy and subsequent low birth weight is associated with obesity later in life (Hatch et al., 2013).

Risk Factors for Obesity

Multiple risk factors are associated with obesity and to date; research has not been able to isolate the effects of a single factor related to increased risk of obesity (Herrera, Keildson & Lindgren, 2011). Some common factors include inactivity (Mitchell, & Byun, 2014), unhealthy eating patterns (Montoya et al., 2013), genetics, socioeconomic

status, race/ethnicity, interaction factors, media, marketing, and the physical environment (Herrera, Keildson, & Lindgren, 2011).

Access and Opportunity

It is likely that ease of access to foods with high sugar, fat, and sodium content contribute to the increase in the prevalence of over- weight and obesity (Jennings et al., 2011). To reduce the prevalence of obesity, some restrictions on access to high fat foods are appropriate, to establish a foundation for subsequent public health interventions. This is because, there is no restriction whatsoever on the retail and commercial availability of these snack and fast food products which are readily available in vending machines, gas stations, convenience stores, and many other places (Jennings et al., 2011). Almost every retail and commercial outlets sell candies, cookies, soft drinks and gums. Restriction of access to these products is unlikely as long as they can be legally sold and even if it is possible, there is another concern whether restriction would have a public health effect. The changing pattern of consumption and its effect on intervention need to be considered. Over the past few years, there have been increases in eating outside the home especially at fast food restaurants (Jennings et al., 2011).

Role of the Environment

Environment plays a vital role in the exacerbation of obesity. These include the home, church, school, and community (Dunton, Kaplan, Wolch, Jarrett & Reynolds, 2010). People develop norms, behaviors and attitudes on food within the home, school, and community environments, and these factors impact a one's physical activity level and

diet pattern (Carter, 2012; Saelens et al., 2012). School environment impact obesity because almost 95% of Americans are enrolled in schools (CDC, 2016). Schools can help students adopt and maintain healthy eating and physical activity behaviors.

Unfortunately, most schools do not have physical education in their curriculum (Sharma, 2011). Also, the school environment plays important role in the promotion of healthy behaviors, like the prevention of obesity (Karnik, & Kanekar, 2015). The CDC (2016) has issued guidelines for nutrition and physical activity programs in schools. Proper management of the school cafeteria and encouragement of physical activity environment can assist in reducing body mass index; although the declining requirement for physical education in schools shows that the school environment may need enhancement (Sherma, 2011). Both the American Public Health Association and the American Academy of Pediatrics has called for the development of school policies for the promotion of healthful eating environments and the prohibition of soft drinks and other low-nutrition foods during the school day. Fortunately, this has been achieved by state legislation or local school board policy.

Economic Factors

Government's taxing and spending power can help in fostering healthy behavior to the public; because, levying taxes can make people engage in healthy behaviors while acting as disincentive to engage in risky behaviors (Nederkoorn, Havermans, Giesen, & Jansen, 2011). For instance, excise tax policy on products like tobacco, and alcohol will discourage some people from buying them. If the same strategy is applied for reducing

the consumption of calorie-dense and low-nutritional-quality foods, it would likely yield good outcome. Also, incentives or subsidies to make more fruits and vegetables easily available and affordable should be considered (Han & Powell, 2011). Considering the impact of price on consumption, it has been noted that, lower prices are associated with higher consumption (Han & Powell, 2011). In their pragmatic study, these authors confirmed the existence of this same pattern of behavior in individuals and found it to be strong across diverse age groups and food types.

Laws

Law helps public health authorities to preserve the health of the public. Laws have helped in the prevention of chronic diseases, for example the bans or restrictions on public smoking, laws on blood alcohol concentration, and food fortification (Burriss, Ashe, Levin Penn & Larkin, 2016). In the area of obesity prevention, little related federal laws exist, other than efforts to provide liability protection to food and soft drink manufacturers. So, most of the legislative initiatives occurred only at the state level.

Regulations

Legislation ensures that administrative action is carried out to regulate products that might have an adverse effect on the public's health (Burriss et al., 2016). Food products are regulated in terms of certain aspects of health and safety, including nutritional labeling and health claims (Andreyeva, Kelly, & Harris, 2011). Currently, the FDA does not regulate the nutritional content of food products, portion size, or marketing strategies as needed (Andreyeva et al., 2011). Nevertheless, if food products were to

make an unjustified health claim, the FDA could act. Also, if food advertising were false, misleading, or deceptive, the FTC could take action (Burriss, Ashe, Levin, Penn, & Larkin, 2016). Concerns about food product advertising are not centered mainly on health claims or dishonesty, but on making calorie-dense and low-nutritional-quality food attractive to people. So, it is doubtful that the food and drug administration (FDA) or the Federal Trade commission (FTC) would assist in the area of maximum concern concerning promotion of unhealthful food products to people (Andreyeva et al., 2011). However, if governmental regulation is not possible, compulsory industry standards could be used to guide minimum nutrient content, portion size, and marketing of products targeted to individuals (Burrisset al., 2016). Also, local authorities could regulate food products, mainly in the areas of licensing, sampling, zoning restrictions, land use (Burrisset al., 2016) and conditional use permits. Recently, litigation has become an influential tool in preventing product-related injuries and ensuring the public health in areas like tobacco, gun violence, and lead paint (Burrisset et al., 2016). Although litigation is not an ideal tool, it is helpful in making some products safe. For example, there were some initial attempts to sue fast food restaurants based on the claim that they were partially responsible for the epidemic of obesity, and for other reasons, such as consumer safety (Burrisset al., 2016).

Health Literacy

According to the World Health Organization, many Americans have poor health literacy, and people with low levels of literacy are associated with poorer overall health

status, and reduced ability to manage chronic diseases (Boodman, 2011). Low literacy in individuals leads to low knowledge of their medical conditions. Individuals with the greatest healthcare needs in the United States are least able to understand the information they need to successfully navigate and function in our complex healthcare system (Cha et al., 2014). Research has shown that health literacy may be a stronger forecaster of health than age, employment status, education level, race, and income (Kuczmarski, 2016).

Obesity Literacy

Obesity literacy is the knowledge of obesity risk factors and warning signs or symptoms (Cha et al., 2014). Inadequate health literacy has been tied to poorer health outcomes, lower utilization of preventive health services, higher risk of hospitalization, and increased mortality rates (Lam & Yang, 2014). Although the knowledge of the warning signs of obesity is important, the most effective treatment of obesity is prevention and a major step in obesity prevention is knowledge of the risk factors and learning how to reduce those factors (Frerichs, 2016). Awareness therefore is the first step or strategy in obesity prevention (Kuczmarski, 2016), and is a strategy to stall the incidence of obesity in the United States (Lam & Yang, 2014). Prevention requires knowledge of the risk factors and a willingness to implement prevention strategies (Cha et al., 2014). Lack of understanding of the risk factors for obesity impedes any high technology prevention strategies. There is strong evidence that modifying the risk factors for obesity will greatly reduce the incidence of obesity by as much as 80% (Frerichs, 2016).

Diversity Variables and Obesity

Obesity impacts people from all social and economic cadres. The impact of socioeconomic status extends to adulthood (Selassie & Sinha, 2011). Poor community environment impede access to physical activity opportunities and affordable healthy foods. This is why people from low -income communities tend to have the highest obesity rate (Bradley & Corwyn, 2002). Availability of healthy food will help individuals in making healthy food choices, while availability of sidewalks, safe bike paths, and parks in the community will encourage people to participate in physical activity (Carson, Spence, Cutumisu & Cargill, 2010).

Screen time has been linked to socioeconomic factors (SEF). People living in low socio-economic communities have been found to be more video game users and spend more screen time compared to people living in high SES neighborhoods (Carson et al., 2010). Also, literature suggests that obesity does disproportionately affect certain minority groups based on race, gender, SES, and ethnicity. According to Wang (2011) African American and Hispanic- American youths are more likely to be overweight than non-Hispanic white youths.

A number of studies reveal that amongst non-Hispanic white children, SES is negatively correlated with obesity (Vander Wall & Mitchell, 2011). In most cases, the resources needed to make healthy eating choices, and to engage in outdoor activities, are often scarce in low income populations and amongst minority groups (Wan, 2011).

Implications of Obesity

Obesity breeds some emotional and social problems including; decreased self-esteem. An obese individual may exhibit low self-image and reduced self-worth based on socio-cultural influences (Mond, Van, Boutelle, Hannan, & Neumark-Sztainer, 2011). The stigma of being fat lowers one's self-esteem and prevent them from having close interactions within the family, community and society (Mond et al., 2011).

The negative impact of obesity on an obese person's self-esteem may affect the long-term happiness and success in life. The anxiety and stress from being obese may impact learning and social wellbeing (CDC, 2016). An obese individual can be socially withdrawn from others, and obesity can result to a vicious cycle for depression, and abnormal eating patterns (Mond et al., 2011)

Co-morbidities in health like hypertension, type 2 diabetes, some cancers and cardiovascular diseases are associated with obesity (Juonala et al., 2011). Obese people are at an increased risk for asthma, sleep apnea and other breathing problems (Mayo Clinic, 2010). The cost of treating obesity poses serious problems to the community (Mayo clinic, 2010).

Gaps in the Literature

Environmental exposure to phthalates has been implicated in a number of public health issues including obesity. Knowledge of this risk factor is paramount to the prevention of obesity especially in a community at risk. While CDC (2016), recommended that phthalates chemicals and their effect on human health be studied further, only very few studies have examined the health effects of phthalates on humans

(Casas, 2011). A small number of studies have examined the impact of phthalates on obesity in the United States (Kim & Park, 2014). Also, few research studies have been conducted on the impact of phthalate chemicals on the endocrine system, and on how the chemicals disrupt the endocrine system to cause obesity. Considering the importance of obesity to public health, the number of published studies on the impact of phthalates chemicals to obesity is limited. Phthalates is a huge class of chemicals comprising of many low molecular weight and high molecular weight chemicals which need to be studied comprehensively because, many of these chemicals have been shown to have negative health impacts (Singh & Li, 2011). There are limited data regarding the risk factors for the exposure to phthalates and association with obesity (Kim & Park, 2014; Casas, 2011). The previous studies conducted by Kim & Park (2011) and Casas (2011) did not investigate the relationship between exposure to phthalates and knowledge of the constituents and obesity. Therefore, this study furthered examined what we already know in literature by specifically examining if there is a relationship between exposure to phthalates and the perception of the risk for obesity. This is of particular importance to determine which of the obesity risk factors drive the increased incidence of obesity in the United States.

Conclusion

Many studies show that phthalates are likely obesogens that promote obesity through numerous mechanisms, including activation of PPARs, antithyroid effects, and epigenetic modulation (Casas, et al, 2011; Hatch et al., 2013; Merrill & Birnbaum, 2011).

The relationship between phthalate chemical exposure and obesity and risk factors for obesity tends to be limited (Janesick & Blumberg, 2016). If the burden of obesity must be reduced the risk factors reduction should be considered as the strategy for primary prevention and positive social change. Enough distinct phthalates have been studied to indicate that companies should proceed with caution when using any chemical in the phthalate class (Chamorro-Garcia, & Blumberg, 2014). The eradication of phthalates should start by addressing areas of significant exposure to phthalates (Cha, et al., 2014). Positive social change starts with health literacy and obesity education to the community at risk. Racial disparities in obesity risk factors awareness should be emphasized in populations with increased obesity risk. Research has consistently shown that the prevalence of obesity with the associated high morbidity and mortality rate is more in the United States (Wang, 2011). The lack of awareness on the impact of phthalate exposure on obesity is modifiable (Wang, 2011). According to the WHO (2013), “literacy is a stronger predictor of an individual’s health status than income, employment status, education level and racial or ethnic group”.

When properly educated, consumers can avoid products packaged in “recycling-code-3” plastic, products that include the vague ingredient “fragrance” on their label, and purchasing organic products packaged in glass as much as possible (Chiellini, Ferri, Morelli, Dipaola & Latini, 2013). Also, consumers can remove any food packaged in plastic from its packaging and place them in glass because, DEHP continues to leech over time, so one can actually reduce exposure by changing the storage container (Chielliniet

al., 2013). A lot of food exposure to phthalate chemicals could be taken care of through the food and drug Administrative regulation and toys through the Consumer Product Safety Commission (Fulcher, & Gibb, 2014). Retailers like Walmart and Target could also play a significant role, as they have with other chemicals of concern (Chiellini et al., 2013). Their suppliers should manufacture both alternatives to phthalates and adopt ways to remove the chemicals from their products altogether. Manufacturing companies could be using flexible polymers that don't require a plasticizer because they probably do not leech the way phthalates do (Chiellini et al., 2013).

Current human studies have examined the possible effects of phthalate exposure on the development of obesity, even though most of them are cross-sectional and short-term prospective studies. While casual concentrations of phthalate metabolites have good reproducibility, large-scaled longitudinal studies including measures at different life ages are needed to establish the impact of phthalate exposure on obesity.

Chapter 3: Research Method

I examined whether or not there was an association between exposure to phthalates and obesity in the United States. I also investigated if SES (education level and income) and place of residence (zip code) influenced the relationship between exposure to phthalates and obesity. Further, I examined whether or not physical activity influenced the relationship between exposure to phthalates and obesity. Race/ethnicity, gender, and age were included as each research question was examined. I explored the single and collective impact of the independent variables on the prevalence of obesity. This is important to determine which of the obesity risk factors drive the increased incidence of obesity in the United States. In this chapter, the study design, sample, instrumentation, data analysis, and ethical concerns are described. Also, the rationale for the study design, the sample characteristics and size, and data collection process are explained.

Research Design and Rationale

Study Variables

Dependent variable for this study was obesity status (obese or not obese). This was determined using height and weight to calculate BMI. The independent variable for this study was phthalate exposure. The independent covariates are listed under the following categories. The covariates are the sociodemographic variables.

Sociodemographic variables for this study included the participant's gender, SES (education level and income), place of residence (zip code), race/ethnicity, and age. The

behavioral variable was physical activity. Collectively, these assisted in defining the at-risk population.

Research Design

A cross-sectional analytic study design was used for this research. This design was used to ascertain the relationship between exposure to phthalate chemicals and obesity. In a cross-sectional, analytic study design, the independent and the dependent variables are measured at the same point in time (Lee, 1994; Schmidt & Kohlmann, 2008). In a cross-sectional, analytic study design, a representative sample is identified and selected from the population of interest, and the needed information regarding the dependent variable (i.e., the outcome under study, which was obesity in the current case) and the independent variables (i.e., the factors/exposures under study) were collected from the sample at one point in time.

Quantitative researchers use statistical techniques and investigate the relationships between events and factors that influence or cause them. Therefore, quantitative research is a systematic, objective, and formal process for obtaining quantifiable information. A quantitative study design was selected for this study because it has the potential to provide information and statistics on the relationship between exposure to phthalate chemicals and obesity in the United states. The findings from this study provide additional support on the need for preventive measures against obesity and help in improving obesity awareness strategies.

A cross-sectional design has many advantages. It is affordable and relieves resource constraints, it consumes less time, and it gives a picture of the population to be studied (Frankfort-Nachmias & Nachmias 2008, p.116-118). I chose this design because it evaluates the relationship among variables for a given population, and it also enables the evaluator to make inferences about the population from its subset at a defined point in time (Crosby et al. 2006). Additionally, a cross-sectional design reveals trend patterns and identifies areas for further studies within a short time with minimal resources (Crosby et al., 2006). According to Crosby et al. (2006), “the cross-sectional design allows for collection of data on multiple variables, maximizes completeness of key data, large number of subjects, dispersed subjects, and answers who, what, how and where questions” (p. 186). This design has been proven to be beneficial to other researchers, because it can be used to record the incidence of health problems and does not require follow up (Crosby et al., 2006).

I used this design to explore the relationship between the dependent and the independent variables. Although previous research has been done on some of the variables, no scholar has examined the association between exposures to phthalate chemicals and obesity in the United States. The variable (s) of interest that may have association with the increasing prevalence of obesity need to be exposed to advance knowledge in the discipline and enable public health professionals, health care providers, and consumers to render suitable preventive services.

Methodology

Population and Setting

I used secondary dataset from the NHANES 2011-2012. The NHANES sample represents the total noninstitutionalized civilian U.S. population residing in the 50 states and District of Columbia, and the U.S. population was the target population. The survey examines a nationally representative sample of about 5,000 persons each year from all races/ethnicities. These people are located across the country. Each year data is collected in a minimum of 15 states.

As with former NHANES samples, a four-stage sample design was used in the NHANES 2011–2012. The first stage consisted of selecting primary sampling units from a frame of all U.S. counties. In some cases, adjacent counties were combined to keep PSUs above a certain lowest size. NHANES PSUs were selected with probabilities proportionate to a measure of size (PPS). The second stage of selection for the NHANES 2011–2012 sample included a sample of area segments, comprising census blocks, or combinations of blocks. The third stage of sample selection consisted of dwelling units (DUs), including noninstitutionalized group quarters such as dormitories. The fourth stage of sample selection consisted of persons within occupied households. All eligible members within a household were listed, and a subsample of individuals was selected based on sex, age, race and Hispanic origin, and income.

Sampling and Sampling Procedure

The main objective of a sampling premise is to get the theoretical base that will help a researcher get the correct estimate of the unknown values of the parameters based on premeditated sample data (Frankfort-Nachmias & Nachmias, 2008). The survey was designed to produce statistics for four broad geographic regions of the United States and for the total population by sex, age, race, and income classification.

The NHANES used a stratified, multistage, probability cluster sample of households throughout the United States. Sampling domains were defined by race and Hispanic origin, sex, age, and low-income status to obtain the sample. The sampling rate for a domain depended on the target examination sample size, the expected examination response rate, and the estimated population size. The sample selection process involved a number of factors, including the selection of primary sampling units, household clusters, and households.

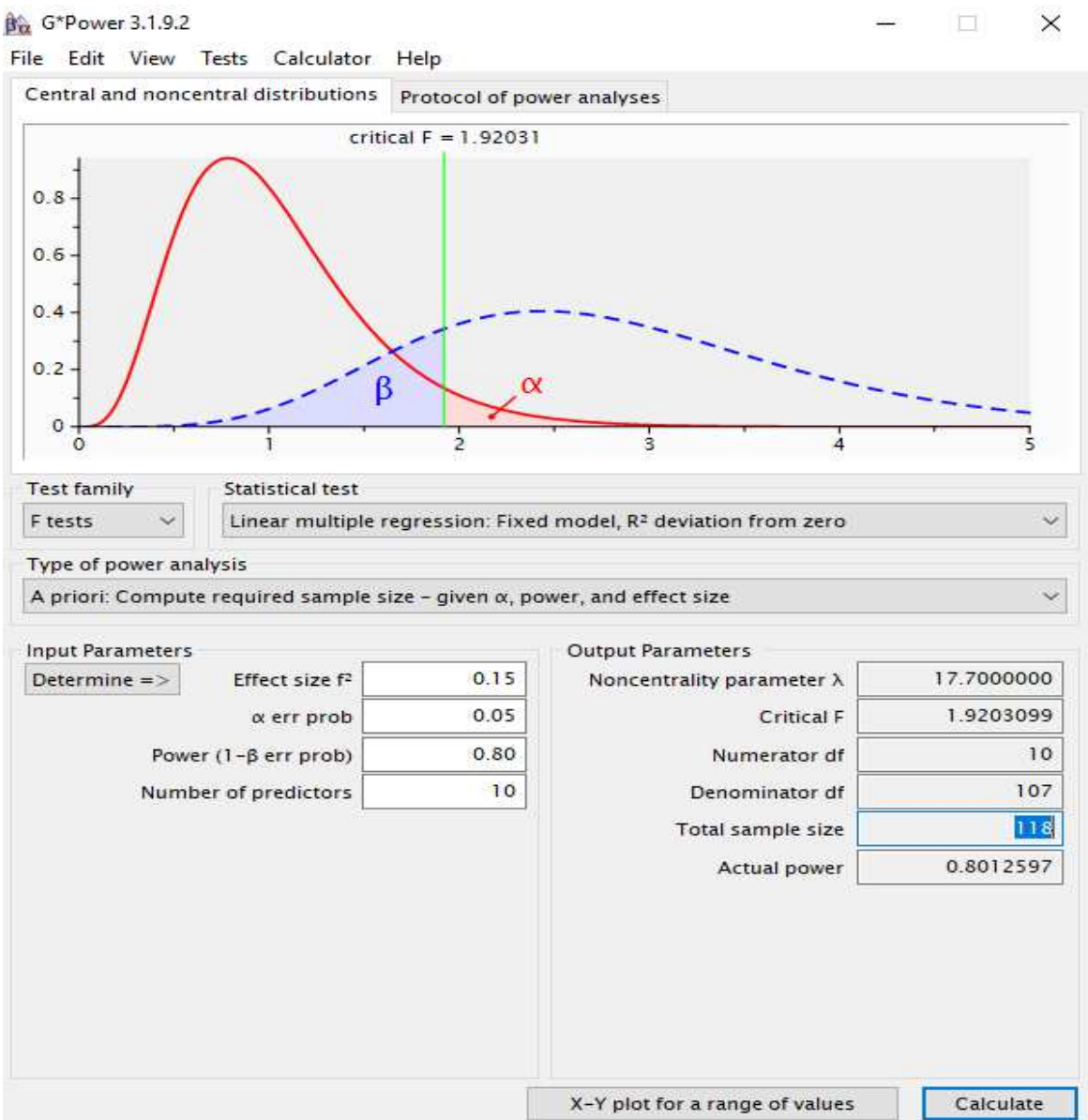
Sampling Frame

Inclusion criteria. The inclusion criteria for selection included resident of the United States; adult men and women aged 18 and older; and noninstitutionalized, civilian residents.

Exclusion criteria. Individuals who did not reside in the United States, individuals who were institutionalized (prison, mental hospitalization, etc.), children, pregnant women, and military personnel were excluded.

Sample Size and Power Analysis

Using GPower (Faul, Erdfelder, Lang, & Buchner, 2007), an alpha of 0.05, a beta of 80%, and a moderate effect size of 0.15, the minimum required sample size for a multiple linear regression model (with 10 independent variables) was 118. A screenshot of the GPower software used for calculating the sample size is shown in Figure 3.



*Figure. 3. G*power calculation.*

Justification for the Effect Size

The sample size from GPower (Faul et al., 2007) was based on an effect size of 0.15, alpha of 0.05, and a power of 80%. The possibility of missing data/values during data collection was accounted for, such that in the worst case of 10% missing data, the remaining valid data would be up to the required minimum sample size. Attempts were made to ensure that the sample was balanced between the groups. This was beneficial for the subsequent analysis and helped in ensuring that the sample was a good representative of the population. If a representative sample is used, the results of the study will be similar to what would be obtained if the study is done on the entire population (Frankfort-Nachmias & Nachmias, 2008).

According to Faul et al. (2009), in order to effectively calculate the necessary sample size for a study, it is necessary to know the values for statistical power, alpha, and effect size. A researcher may estimate the values for his or her parameters from existing literature or from a knowledge of possible values of the parameters (Faul et al., 2009). Information from existing literatures often guide researchers in deciding what statistical power, alpha, and effect size would be most appropriate.

Research Design

A cross sectional, analytic study design was used for this research. In cross sectional analytic study design, the independent and the dependent variables are

measured at the same point in time (Schmidt & Kohlmann, 2008). In this type of study, either the entire population or a subset of the population is selected, and from these individuals, data are collected to answer research questions of interest. It is called cross-sectional because the information about X and Y that is gathered represents what is going on at only one point in time (Schmidt & Kohlmann, 2008). The NHANES was designed to produce statistics for four broad geographic regions of the United States and for the total population by sex, age, race, and income classification. NHANES was a probability sample of the civilian, noninstitutionalized population of the United States aged 6 months through 74 years.

I used the cross sectional research design to find answers to research questions that will help to advance the knowledge of obesity prevention in public health. This design was chosen because it can provide empirical data on the exposure to phthalates as the driving force behind the increasing obesity risk. Also, the study's results will give further support on the need for enhanced preventive measures in quality obesity awareness strategies.

Study Participants and Informed Consents

The NHANES is a cross-sectional, complex, probability sample of the U.S. noninstitutionalized population, with both interview and examination components. The NHANES is a survey conducted by the National Center for Health Statistics (NCHS). The NCHS is a part of the CDC, U.S. Department of Health and Human Services. Health information collected in the NHANES is kept in strictest confidence. During the

informed consent process, survey participants were assured that data collected will be used only for the stated purposes and will not be disclosed or released to others without the consent of the individual or the establishment in accordance with section 308(d) of the Public Health Service Act (42 U.S.C. 242m). The consent form was signed by participants in the survey, and the participants agreed to participate in the study (NHANES, 2011, 2012)

The procedure for recruitment involved dividing all of the counties in the United States into 15 groups based on their characteristics. One county was selected from each large group, and together they formed the 15 counties in the NHANES surveys for each year. Within each county, smaller groups (with a large number of households in each group) were formed, and between 20 and 24 of these small groups were selected. All of the houses or apartments within those selected small groups were identified, and a sample of about 30 households were selected within each group. The NHANES interviewers went to each selected household and asked for information (age, race, and gender) on all persons in the household. A computer algorithm randomly selected some, all, or none of the household members.

The participation involved finding if the participants were eligible. An NHANES interviewer went people's homes and asked a few questions about the participant and the other members of the household. The NHANES interviewers had identification with them that they showed the subjects immediately they enter their house for survey. Once the

participant answered a few questions, the interviewer let him/her know if they were needed to participate further.

There was no permission needed to gain access to the NHANES datasets, which was meant for public use. The NCHS offers downloadable public-use data files through the CDC's FTP file server. Users have access to data sets, documentation, and questionnaires from the NCHS surveys and data collection systems. Public-use data files are prepared and disseminated to provide access to the full scope of the data. This allows researchers to manipulate the data in a format appropriate for their analyses (NHANES, 2011, 2012). Nevertheless, users of NCHS public-use data files must comply with data use restrictions to ensure that the information will be used solely for statistical analysis or reporting purposes.

The NCHS releases public-use data sets from the continuous NHANES in 2-year groupings (cycles). This release does not contain all of the data collected on persons who participated in the survey during 2011-2012. As more data becomes available, there will be additional releases posted on the What's New page. The NHANES data in this release were in SAS transport file format. Export engine was used to access these data in the SAS version. The transported files were copied to a permanent SAS library, and then they were transported to SPSS format.

Analyses were conducted with SAS version 9.3 and SUDAAN version 11.0. All analyses used NHANES examination sample weights that adjust for nonresponse, noncoverage, and unequal probabilities of selection. Standard errors were estimated with

Taylor series linearization to take into account the complex sample design. Pregnant females were excluded from all analyses. Obesity estimates for total adults aged 18 years and older were standardized to the projected estimates of the 2000 U.S. Census by the direct method, using the age groups 18 to 39 years, 40 to 59 years, and 60 years and older. Crude estimates of obesity among all adults were also presented. I stopped reviewing here due to time constraints.

Operational Definitions

The operational terms and definitions below will clarify the important terms and concepts used in this study.

Obesity: Body mass index (BMI) is a simple index of weight-for-height that is commonly used to classify overweight and obesity in adults. It is defined as a person's weight in kilograms divided by the square of his height in meters (kg/m^2) (Karnik, & Kanekar, 2015).

Height: The measurement from base to top or (of a standing person) from head to foot (Karnik, & Kanekar, 2015). Height will be used to calculate BMI for the obesity variable.

Weight: A body's relative mass or the quantity of matter contained by it, giving rise to a downward force; the heaviness of a person or thing (Bray, 1985). Weight will be used to calculate BMI for the obesity variable.

Level of Education: Education was used for the SES variable. For the purposes of statistical comparability, the United States has defined lower secondary education as

grades 7 through 9 and upper secondary as grades 10 through 12. For the attainment indicators, a person is classified in the highest level for which they completed the last grade or degree for the level (NHANES, 2011-2012). In this study, the following levels of education were considered: never attended school or only kindergarten, attended elementary school, attended some high school, graduated from high school, attended college or technical school, and graduated from college.

Income: Used for the SES variable, income is money that an individual or business receives in exchange for providing a good or service or through investing capital. Income is consumed to fuel day-to-day expenditures. Most people age 65 and under receive the majority of their income from a salary or wages earned from a job (CDC, 2016). In this study, the following levels of income were considered: less than \$10,000; \$10,000 to less than \$15,000; \$15,000 to less than \$20,000; \$20,000 to less than \$25,000; \$25,000 to less than \$35,000; \$35,000 to less than \$50,000; \$50,000 to less than \$75,000; \$75,000 and above.

Residence: A place of abode with some permanence; residence requires bodily presence and is distinct from domicile, which requires both bodily presence and intent to make it one's home (NHANES, 2011-2012). Zip codes was used for the residence variable. *Zip code* are a system of postal *codes* used by the United States Postal Service since 1963. The term *ZIP*, an acronym for *Zone Improvement Plan*, was chosen to suggest that the mail travels more efficiently, and therefore more quickly (zipping along), when senders use the *code* in the postal address (NHANES, 2011-2012).

Age: The length of time that a person has lived or a thing has existed, a distinct period of history (Zhang et al, 2014). Age was categorized in this study using appropriate age groups.

Gender: This is a nominal variable with binomial measure as male and female which can be coded as 1 for female and 2 for male. The impact of gender can be assessed for any significance in overweight and obesity. This state of being male or female is (typically used with reference to social and cultural differences rather than biological ones (Zhang et al, 2014).

Race and Ethnicity. The term race refers to groups of people who have differences and similarities in biological traits deemed by society to be socially significant, meaning that people treat other people differently because of them. *Ethnicity is defined* as someone's cultural background or where they came from. Hu, 2008).

Physical activity is any bodily exercise that enhances or maintains physical fitness and overall health and wellness. Frequent and regular physical exercise boosts the immune system and helps prevent "diseases of affluence" such as cardiovascular disease, type 2 diabetes, and obesity (Bray, 1985).

Study Variables

The independent variables and covariates that was used in this study were presented in Table 1

Table 1

Study Variables

Variable Name	Research Question(s)	Role	Potential Responses	Level of Measurement
Obesity status (BMI)	1, 2, 3	Dependent	Yes/No	Nominal
Weight	1, 2, 3	Used for calculating the BMI	Body weight in kilograms	Interval
Height	1, 2, 3	Used for calculating the BMI	Height in meters	Interval
Phthalates chemicals exposure	1, 2, 3	Independent	Urine level of phthalate chemicals	Ratio
Age	1, 2, 3	Covariate	Age in Years	Continuous
Gender	1, 2, 3	Covariate	Male/Female	Nominal
Race/ethnicity	1, 2, 3	Covariate	Caucasian, African American, Hispanics	<i>Nominal</i>
Zip code	2	Covariate	Places of residence	Nominal
Physical activity	3	Covariate	High, moderate, low	Ordinal
Income	2	Covariate	\$10,000; \$10,000 to less than \$15,000; \$15,000 to less than \$20,000; ... \$75,000 and above	Ordinal
Education Level	2	Covariate	Never attended school or only kindergarten, attended elementary school, attended some high school, graduated	Ordinal

from high school,
attended college or
technical school, and
graduated from college

Statistical Analysis

The nature of the variables being analyzed is an important component to addressing the research questions in this study. This study did not seek causal effect, but rather an association or relationship between variables that can refute or validate the study hypothesis. As a result, the following sets of statistical methods were used to analyze the data.

Descriptive statistics. Descriptive statistics for each of the study variables in Table 2 were calculated. Frequencies was used to describe the dichotomous and nominal variable, zip code, while mean and standard deviation were used for the continuous variable, age, interval variable, weight, and ordinal variable, number of days of physical activities.

Correlation analysis. Correlation analysis was performed to see whether there is a relationship between phthalate chemicals exposure and obesity prevalence. Correlation analysis includes the computation of Pearson's correlation coefficient and its significance level. The Pearson's correlation coefficient revealed how much of the change or variation in obesity incidence and obesity prevalence in United States can be attributed to phthalate chemicals exposure.

Logistic Regression analysis. Logistic regression analysis was used to predict the impact of phthalate chemicals on obesity in the United states. Logistic regression was

appropriate because, it can handle all sorts of relationships, through the application of a non-linear log transformation to odds ratios. Secondly, the independent variables do not need to be multivariate normal – although multivariate normality yields a more stable solution. The associated assumptions of logistic regression include; the model should be fitted correctly, each observation needs to be independent, that is, the model should have little or no multicollinearity, logistic regression assumes linearity of independent variables and log odds. Logistic regression also requires large sample size (Statistics solutions, 2016). The fitted regression models were carefully diagnosed to make sure they were free of various possible model violations (Field, 2009). SPSS version 21 (Statistical Package for the Social Sciences) is the statistical software that was used for data analysis (Faul et al., 2009).

Research Questions and Hypotheses

As a result of several opinions on the cause and methods of preventing obesity, many questions call for further investigations (CDC, 2016). This study explored the relationship between phthalates exposure and obesity among adults 18 years and older in the United States. The study also investigated whether or not there is a relationship between gender, age, physical activity, socioeconomic status, race/ethnicity and exposure to phthalates and obesity among adults 18 years and older in the United States.

The subsequent research questions (RQs) and hypotheses were obtained from the literature reviewed on the association between phthalates chemical exposure and obesity.

RQ1: Is there an association between exposure to phthalates and obesity among adults 18 years and older in the U.S?

H_{I0} : There is no association between phthalates exposure and obesity among adults 18 years and older in U.S.

H_{IA} : There is association between phthalates exposure and obesity among adults 18 years and older in the U.S.

RQ2: Do the socio-demographic variables (SES, race/ethnicity, gender, age, place of residence) influence the relationship between exposure to phthalates and obesity among adults 18 years and older in the US?

H_{20} : The socio-demographic variables have no statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older the U.S.

H_{2A} : The socio-demographic variables have statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older in the U.S.

RQ3: Does physical activity influence the relationship between exposure to phthalates and obesity among adults 18 years and older in the U.S.?

H_{30} : Physical activity has no statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older in the U.S.

H3_A: Physical activity have statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older the U.S.

Table 4

Null and Alternative Hypotheses for Research Question

Research Questions	Null Hypotheses	Alternative Hypotheses
RQ 1	There is no association between exposure to phthalates and obesity among adults 18 years and older in the U. S.	There is an association between exposure to phthalates and obesity among adults 18 years and older in the U. S.
RQ 2	The socio-demographic variables have no statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older the U.S.	The socio-demographic variables have statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older in the U.S.
RQ3	Physical activity has no statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older in the U.S.	Physical activity has statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older the U.S.

Threats to Validity

Validity is the ability of instrument to measure what it is intended to measure (Frankfort-Nachmias & Nachmias). The number of days of physical activities as shown

by the construct validity involves the use of nominal scale. The BMI scores are used to estimate the prevalence of overweight and obesity and the potential odds associated with social, physical, and environmental determinants.

Reliability is the ability of an instrument to reproduce results under similar characteristics and circumstances (Frankfort-Nachmias & Nachmias, 2008). Unambiguous questions help preserve the reliability of an instrument and subject anonymity guarantees honest reporting of weight status (Frankfort-Nachmias & Nachmias, 2008). Using weight and height provides a measureable scale appropriate for the study population. NHANES uses several methods to monitor the quality of the analyses performed by the laboratories. These methods include performing blind split samples collected on “dry run” sessions (NHANES 2011-2011). In addition, contract laboratories randomly perform repeat testing on 2.0% of all specimens (NHANES 2011-2012).

Internal Threats to Validity

Internal validity assesses whether the independent variable produced the differences observed. Some internal threats are particularly pertinent to the present study and the conclusions that it can meaningfully support. The digital scale currently used to obtain the body weight was linked to the study database, as is the audiometer used to measure height (CDC, 2016)

History. This threat type arises due to events occurring during the study period that may unduly influence outcomes (Creswell, 2009). History is a threat to conclusions

drawn from longitudinal studies because, the greater the period of time that elapses between measurements, the more the risk of a history effect. History is not a threat to cross-sectional studies which are conducted at one-time point.

Biases in sample selection are threats seen in both the cross-sectional and longitudinal approaches. However, because of the cost and time commitment, they are only devastating to the conclusions drawn from the longitudinal study and not for cross-sectional studies.

Regression. This occurs where repeated measures are used. This is mostly observed when participants selected for the study are on the classification variable of interest, e.g. intelligence. When this happens, the test-retest scores tend to steadily drift to the mean rather than remain stable (Fayter, McDavid, & Eastwood, 2007). The bivariate/multivariate data screening process of NHANES data sets help to minimize this problem (NHANES, 2011-2012).

Selection. This threat type arises when participants who have peculiar characteristics that would predispose them to certain outcomes are included (Fayter et al., 2007). Behavioral differences may not have any relationship with age. However, it may rather be the result of differing educational, cultural and nutritional/health habits which characterized living conditions differently for each generation (Fayter et al., 2007). When a classification variable like age is being examined within a cross-sectional approach, many developmental researchers argue that "age" as a variable is irrelevant in any case since the real interest lies in experiential variables as well as neuro-maturational factors

and their interplay (Fayter et al., 2007). To minimize the potential of distortion of this type of threat, this survey sampled only adults with the age of 18 and above in the United States.

Mortality or attrition. This is a major threat to a prolonged longitudinal study, not for a cross-sectional study. This is because, in a longitudinal study, the sample remaining at the end of the study is not likely to be comparable to the initial sample (Fayter et al, 2007).

Instrumentation. These problems are not peculiar to cross-sectional studies; they are mostly seen in longitudinal studies where the researchers leave the study and testing instruments for a long time period which make them invalid due to cultural change (Fayter et al, 2007).

Testing. Testing consist of either reactivity as a result of testing or practice/learning from exposure to repeated testing (Vogt, 2007). Is not routinely seen in cross-sectional studies. To lessen the testing threat, the researcher from previous study administered questionnaire once and the subjects completed the questionnaire just once to avoid replication and excessive scores (Vogt, 2007).

External Threats to Validity

External validity determines whether the results of the study can be generalized to an entire population from which the samples were drawn in the study (Ihantola & Kihn, 2011). External validity is secondary to and dependent on adequate attention given to the threats to internal validity.

The main sources of external threats to validity are; characteristics of the individuals selected for the sampling, the exclusiveness of the setting, and the timing of the data collection (Henderson, Kimmelman, Fergusson, Grimshaw, & Hackam, 2013). These types of validity threats arise when the researcher's findings generalize further than the target group and into other racial and social groups, or comprise other settings, a past or future situation (Henderson et al., 2013). Restricting of the primary comparative references to recent studies of similar settings and racial groups, and undertaking age group analysis of participants help to minimize this type of validity threat. Though not specific to the study of environmental obesogens, the time-varying nature of phthalates exposure, confounders, and outcomes can increase analytic complexity.

Quantitative research studies deal with numerical numbers which do not accommodate non-numerical explanations. This is a potential methodological weakness because; this may not accommodate explanations which cannot be numerically matched for a better understanding of the problem (Creswell, 2009 p. 145). This weakness can be overcome by conducting a mixed methods research combining the quantitative and qualitative research methods for a more robust understanding of the variable relationships and experiences in the practice setting.

Strengths and Limitations

The basic strengths of this quantitative study comprises the ability of comparing and generalizing from a small data to larger population. The study attempts to examine

the participants and their relationship to the variables such as physical activities, dietary changes, and sedentary lifestyles.

The utilization of survey method enables a wider coverage and increase in sample size that enhances generalization. A key strength of NHANES for assessing obesity prevalence is that heights and weights are measured by staff trained “to follow standardized examination protocols, to calibrate equipment according to a prescribed schedule and method, and to measure and record the survey data with precision” (CDC, 2016). The limitation of the study involves the rigidity of the process with questions fixed to maintain validity and reliability (Creswell, 2009). Another limitation involves the educational standard of the respondents and use of secondary data which was initially meant for another study.

Ethical Considerations

Ethical concerns were involved in this study because of the social stigma attached to obesity.

This stigma attached to obesity can result to the denial of obesity existence. Obesity is associated with psychological problems, so the privacy of the obese were considered. Since this study will be using secondary data set from the National Health and Nutrition survey, the necessary steps to protect the study participants and to obtain informed consents were already taken by the Centers for Disease Control and Prevention. Some of the steps used by CDC include:

1. Use of study codes on data documents (e.g., completed questionnaire) instead of recording identifying information and keep a separate document that links the study code to subjects' identifying information locked in a separate location and restrict access to this document (e.g., only allowing primary investigators access);
2. Encrypting of identifiable data
3. Removal of face sheets containing identifiers (e.g., names and addresses) from survey instruments containing data after receiving from study participants;
4. Properly disposing, destroying, or deleting of study data / documents
5. Limiting access to identifiable information;
6. Securing store data documents within locked locations; and/or
7. Assigning security codes to computerized records (CDC, 2013).

The subjects were informed about the research objectives, data collection, purpose, procedure, and potential risks and benefits of participating in the study before asking them about their willingness to participate in the study and their willingness to sign the informed consent form/agreement. They were given the opportunity to accept or reject the offer of participating in the study (CDC, 2016). Walden University Institutional Review Board (IRB) was contacted and approval obtained before using the National Health and Nutrition Examination Survey. NHANES which is operated by the Centers for Disease Control and Prevention Representative, said that the 2011-2012 dataset was

open to public use. This study employed secondary data collected from the NHANES participants.

Summary

This study is aimed at investigating the possibility of a relationship between exposure to phthalates and obesity. The cross sectional study design, using secondary data was used for this research so that the dependent and the independent variables could be measured at the same point in time. The needed data was from the National Health and Examination Nutrition Survey 2011-2012, which suit the needs of the current study. Being a quantitative research, it would be possible to have a highly systematic, objective, and formal process for obtaining quantifiable information on the relationship between phthalate chemicals and obesity. The findings, which was presented in Chapter 4, of this study provided new scientific information and drew attention to important players that lead to obesity and shed lights on the contributions of phthalates to obesity.

Chapter 4: Results

Obesity has been a public health problem for many years. Obesity can lead to chronic health diseases like cardiovascular disease, diabetes, and emotional diseases (Biro, 1010). The global obesity problem has been attributed to unhealthy diet, inactivity, and hereditary factors. However, exposure to phthalates may also contribute to the obesity epidemic (Heindel, Newbold & Schug, 2015). Phthalates are endocrine disrupting chemicals used to make plastics more flexible and harder to break. In this cross-sectional study, I examined whether there was a relationship between exposure to phthalates and obesity in the United States. The research design, methods, and data analysis were based on the NHANES survey, 2011-2012 dataset. Descriptive statistics and multiple regression analysis were used to analyze the association between each independent variable and the dependent variable.

Data Collection

Secondary Dataset: NHANES 2011-2012

I used a secondary dataset from a national survey (i.e., NHANES 2011-2012 dataset). The NHANES sample represents the total noninstitutionalized civilians in the U.S. population residing in the 50 states and District of Columbia. The NHANES used the U.S. population as the target population. The survey examines a nationally representative sample of about 5,000 persons each year from all races/ethnicities. These people are located across the country. Each year data is collected in a minimum of 15 states.

Sampling and Sampling Procedure

The sample selection process involved a number of factors, including the selection of primary sampling units, household clusters, and households. A four-stage sample design was used in the NHANES 2011–2012, just as in the previous NHANES. The first stage consisted of selecting primary sampling units from a frame of all U.S. counties. NHANES PSUs were selected with probabilities proportionate to a measure of size PPS. The second stage of selection for the NHANES 2011–2012 sample included a sample of area segments, comprising census blocks or combinations of blocks. The third stage of sample selection consisted of DUs, including noninstitutionalized group quarters such as dormitories. The fourth stage of sample selection consisted of persons within occupied households. All eligible members within a household were listed, and a subsample of individuals were selected based on sex, age, race and Hispanic origin, and income. The inclusion criteria for selection are; residents of the United States; adult men and women aged 18 and older; and noninstitutionalized civilian residents. Individuals who did not reside in the United States, individuals who were institutionalized (prison, mental hospitalization, etc.), children, pregnant women, and military personnel were excluded.

Dataset Download and Data Cleaning

The NCHS offers downloadable public-use data files through the CDC's FTP file server. There is no special permission needed to gain access to the NHANES datasets, which are meant for public use. Users have access to data sets, documentation, and

questionnaires from the NCHS surveys and data collection systems. Public-use data files are prepared and disseminated to provide access to the full scope of the data. This allows researchers to work on the data in a format appropriate for their analyses (NHANES, 2011, 2012).

The downloaded datasets are in SAS transport file format. Export engine was used to export the contained data into nonbinary, comma-separated value (CSV) file. The CSV file was then loaded into SPSS. In the data view section of SPSS, the needed variables listed in Table 2 of the Methods Chapter were identified and moved to the top in the list of the variables in the dataset. The variables that were not needed were moved to the bottom. Using the data dictionary obtained from NHANES website, the most appropriate names for each of the needed variables were entered into the space provided for variable labels in the data view of SPSS. Thereafter, the level of measurement (nominal, ordinal, or scale) was entered for each of the variables in the SPSS variable view window. In SPSS, the scale level of measurement is used to represent both interval and ratio levels of measurement. The improved/cleaned dataset was successively saved during the data cleaning to avoid data loss and to allow for possible reverting to an older version of the cleaned data if necessary.

For the categorical variables (i.e., nominal and ordinal), I added the text value of each number in the space provided for it in the variable view of SPSS. For example, under gender, 1 was linked to the value male, while 2 was linked to the value female. Using the compute variable command under the transform menu in SPSS, new variables

were calculated. For example, the new variable phthalate exposure was calculated by adding together all of the levels of exposure to each of the recorded phthalate compounds. In a similar way, the new variable physical activity core was calculated by summing together the number of minutes spent doing each of the listed physical activity.

Results

Descriptive Statistics: Study Variables

The NHANES, from which the dataset for this study was obtained, is a cross-sectional, four-stage, probability sample of the U.S. noninstitutionalized population, with both interview and examination components (NHANES, 2011, 2012). Obesity status was the dependent variable of interest in this study. Obesity was calculated by using the participants' height and weight to determine their BMI. The main independent variables of interest were phthalate exposure, gender, education level, race, age (sociodemographic variables), and physical activity (behavioral).

Descriptive Statistics

Frequency distribution for the categorical variables. The numerical descriptive statistics for the categorical variables (BMI category, marital status, annual household income, annual family income, gender, and race) are presented in a frequency table (Table 3). For the annual household income and annual family income, a family consisted of two or more people related by birth, marriage, or adoption residing in the same housing unit. A household consisted of all people who occupied a housing unit regardless of relationship. A household consisted of a person living alone or multiple

unrelated individuals or families living together. Numerical values showing the frequencies for each of the categories and the percentage that each of the categories constitute are shown in the frequency tables (Table 3). For example, I found that the sample was roughly the same number of males (50.6 %) and females (49.4 %). Non-Hispanic Whites constituted 35.9% of the study participants, while Non-Hispanic Black constituted 26.6% (Table 3). The proportion constituted by the other races, and distribution of the other categorical variables are shown in Table 3.

Table 3

Numerical Descriptive Statistics of the Categorical Variables in the Study

	Frequency	Percent
<i>Gender</i>		
Male	946	50.6
Female	925	49.4
Total	1871	100.0
<i>Race</i>		
Mexican American	174	9.3
Other Hispanic	188	10.0
Non-Hispanic White	672	35.9
Non-Hispanic Black	497	26.6
Non-Hispanic Asian	285	15.2
Other Race - Including Multi-Racial	55	2.9
Total	1871	100.0
<i>BMI Category</i>		
Underweight	46	2.5
Overweight	578	31.5
Obesity	601	32.8
Normal Weight	608	33.2
Total	1833	100.0
<i>Marital status</i>		
Married	864	48.7
Widowed	153	8.6
Divorced	171	9.6
Separated	53	3.0
Never married	394	22.2
Living with partner	138	7.8
Total	1773	100.0
<i>Annual household income</i>		
\$ 0 to \$ 4,999	48	2.7
\$ 5,000 to \$ 9,999	90	5.1
\$10,000 to \$14,999	143	8.1
\$15,000 to \$19,999	129	7.3
\$20,000 to \$24,999	128	7.3
\$25,000 to \$34,999	203	11.5
\$35,000 to \$44,999	162	9.2

\$45,000 to \$54,999	127	7.2
\$55,000 to \$64,999	76	4.3
\$65,000 to \$74,999	83	4.7
NOT sure but knows it is \$20,000 and Over	83	4.7
NOT sure but knows it is Under \$20,000	26	1.5
\$75,000 to \$99,999	165	9.3
\$100,000 and Over	302	17.1
Total	1765	100.0
<i>Annual family income</i>		
\$ 0 to \$ 4,999	83	4.7
\$ 5,000 to \$ 9,999	107	6.0
\$10,000 to \$14,999	157	8.9
\$15,000 to \$19,999	130	7.3
\$20,000 to \$24,999	129	7.3
\$25,000 to \$34,999	212	12.0
\$35,000 to \$44,999	156	8.8
\$45,000 to \$54,999	125	7.0
\$55,000 to \$64,999	76	4.3
\$65,000 to \$74,999	86	4.8
NOT sure but knows it is \$20,000 and Over	63	3.6
NOT sure but knows it is Under \$20,000	27	1.5
\$75,000 to \$99,999	155	8.7
\$100,000 and Over	268	15.1
Total	1774	100.0

Descriptive statistics for the continuous variables. The numerical descriptive statistics for the continuous variables (total phthalate exposure, age in years at screening, physical activity score, number of days of vigorous work, and number of days of moderate work) are presented in Table 4, which shows minimum, maximum, mean, standard error, standard deviation, skewness, and kurtosis values for each of the continuous variables considered in this study. Vigorous work is a vigorous-intensity activity that causes large increases in breathing or heart rate and is done for at least 10

minutes continuously (NHANES, 2011, 2012). Moderate work is a moderate-intensity activity that causes small increase in breathing or heart rate and is done for at least 10 minutes continuously (NHANES, 2011, 2012).

Table 4
Numerical Descriptive Statistics for the Continuous Variables

	Minimum	Maximum	Mean	Std. Error	Std. Deviation	Skewness	Kurtosis
Total Phthalate Exposure (ng/mL)	.00	31156.50	365.9927	21.75541	941.03169	20.609	625.487
Age in years at screening	18.0	80.0	47.114	.4327	18.7179	.140	-1.150
Number of days of vigorous work	.0	7.0	.632	.0376	1.6278	2.536	5.190
Number of days of moderate work	.0	7.0	1.296	.0506	2.1867	1.397	.446
Number of days of walk or bicycle	.0	7.0	1.670	.0605	2.6159	1.146	-.387
Number of days of vigorous recreational activities	.0	7.0	.764	.0365	1.5805	2.094	3.512
Number of days of moderate recreational activities	.0	7.0	1.497	.0508	2.1965	1.283	.366
Physical Activity Score	.00	35.00	5.8589	.12694	5.49101	1.084	1.445

I found that the total phthalate exposure (ng/mL) varied widely among the study participants (Table 4). Its mean value was 365.99 while its standard deviation was 941.03 (which was very large and indicates large variation across the study participants). Furthermore, the skewness value for total phthalate exposure was 20.61. The distribution of phthalate exposure was highly skewed to the right (positive skewness) among the study participants (Table 4). The number of days of vigorous recreational activities was

another variable that varied widely across the study participant. It had a mean of 0.76, a standard deviation of 1.58, and a skewness of 2.09.

Cross-tabulation of the study variable by BMI categories. The distribution and nature of each of the continuous and the categorical variables under each level of BMI category (underweight, normal weight, overweight, and obesity) are presented in Table 5. I found that the summary statistics of most of the study variables differed across the categories of BMI (Table 5). For example, the mean phthalate exposure (ng/mL) appeared to be lowest in the underweight people (191.85) and highest in the people with obesity (424.26). The mean phthalate exposure in the normal (310.56) and overweight (388.43) people fell in between these two. On the other hand, the mean number of days of vigorous recreational activities was highest in the people with normal body weight (1.1) and lowest in the people who are obese (0.4). The distributions of the various categories of marital status, income, gender, race, and education level across the across the BMI categories are also shown in Table 5.

Table 7
Summarized Numerical Descriptive Statistics of the Continuous Variables for Each Level of BMI Category

	BMI Category*			
	Underweight	Normal Weight	Overweight	Obese
Total Phthalate Exposure (ng/mL)	191.85 (201.77)	310.56 (616.72)	388.43 (725.84)	424.26 (1359.48)
Physical Activity Score	5.91 (4.72)	6.63 (5.82)	6.03 (5.41)	5.05 (5.07)
Number of days of vigorous work	.5 (1.5)	.6 (1.6)	.7 (1.7)	.7 (1.6)
Number of days of moderate work	1.0 (2.0)	1.2 (2.2)	1.3 (2.2)	1.4 (2.2)
Number of days of walk or bicycle	2.1 (2.9)	2.1 (2.8)	1.7 (2.6)	1.3 (2.4)
Number of days of vigorous recreational activities	.8 (1.5)	1.1 (1.8)	.8 (1.6)	.4 (1.2)
Number of days of moderate recreational activities	1.5 (2.2)	1.7 (2.3)	1.6 (2.2)	1.3 (2.0)

*The standard deviations are shown in parenthesis for each of the variables listed on the left.

Table 8
Summarized Numerical Descriptive Statistics of the Categorical Variables for Each Level of BMI Category

	BMI Category			
	Underweight	Normal Weight	Overweight	Obese
Marital status				
Married	13	264	299	277
Widowed	1	41	38	59
Divorced	3	41	61	62
Separated	1	22	15	14
Never married	15	146	99	128
Living with partner	6	46	43	43
Annual household income				
\$ 0 to \$ 4,999	2	12	14	18
\$ 5,000 to \$ 9,999	5	29	28	26
\$10,000 to \$14,999	2	39	38	58
\$15,000 to \$19,999	3	47	32	44

\$20,000 to \$24,999	4	32	39	50
\$25,000 to \$34,999	4	64	61	71
\$35,000 to \$44,999	2	52	52	54
\$45,000 to \$54,999	4	40	45	38
\$55,000 to \$64,999	1	25	23	25
\$65,000 to \$74,999	2	23	34	23
NOT sure but knows it is \$20,000 and Over	5	21	24	27
NOT sure but knows it is Under \$20,000	1	9	6	9
\$75,000 to \$99,999	1	51	57	56
\$100,000 and Over	9	121	86	82
Annual family income				
\$ 0 to \$ 4,999	4	33	19	25
\$ 5,000 to \$ 9,999	6	34	34	31
\$10,000 to \$14,999	2	41	49	59
\$15,000 to \$19,999	3	45	35	44
\$20,000 to \$24,999	5	32	40	49
\$25,000 to \$34,999	4	69	65	71
\$35,000 to \$44,999	2	50	49	52
\$45,000 to \$54,999	3	42	41	39
\$55,000 to \$64,999	1	25	20	29
\$65,000 to \$74,999	2	23	34	26
NOT sure but knows it is \$20,000 and Over	5	14	15	23
NOT sure but knows it is Under \$20,000	1	9	7	9
\$75,000 to \$99,999	1	46	55	53
\$100,000 and Over	6	106	80	72
Gender				
Male	15	312	323	281
Female	31	296	255	320
Race				
Mexican American	2	47	52	67
Other Hispanic	1	46	75	65
Non-Hispanic White	19	224	212	201
Non-Hispanic Black	10	111	137	230
Non-Hispanic Asian	13	155	85	26
Other Race - Including Multi-Racial	1	25	17	12
Education Level				
Less than 9th grade	3	53	64	54
9-11th grade (Includes 12th grade with no diploma)	15	107	92	119

High school graduate/GED or equivalent	6	104	105	129
Some college or AA degree	11	156	169	188
College graduate or above	11	186	148	111

Graphical Descriptive Statistics

The graphical descriptive statistics for the categorical variables (namely, BMI category, marital status, annual household income, annual family income, gender, and race) are presented in bar charts (Figure 4 to 9 in the Appendix). The vertical axis of each of the bar charts shows the frequencies for each of the categories in the respective categorical variable (Figures 4 to 9). The graphical descriptive statistics for the continuous variables (namely, total phthalate exposure, age in years at screening, and physical activity score, etc.) are presented in histograms shown in Figures 10 to 17 in the Appendix .

Results of Hypotheses Testing by Research Question

To answer the research questions posed, I fitted binary logistic regression models and tested the null hypotheses in the process.

Research Question 1

RQ1: Is there an association between exposure to phthalates and obesity among adults 18 years and older in the U.S.?

H_0 : There is no association between phthalates exposure and obesity among adults 18 years and older in U.S.

H_A : There is association between phthalates exposure and obesity among adults 18 years and older in the U.S.

The results of the fitted binary logistic regression model show that Total Phthalate Exposure is not statistically significant in the model ($p = 0.393$, Table 9). Therefore, I cannot reject the null hypothesis There is no statistically significant association between exposure to phthalates and obesity among adults 18 years and older in the U.S. after controlling for the effects of potential confounders.

Table 9

Logistic regression assessing Total Phthalate Exposure on Obesity (RQ1)

	B	S.E.	Wald	df	Sig.	Exp(B)	95% CI for EXP(B)	
							Lower	Upper
Total Phthalate Exposure (ng/mL)	.000	.000	.729	1	.393	1.000	1.000	1.000
Marital status (Married is the reference category)			14.186	5	.014			
Marital status (Widowed)	-.064	.275	.055	1	.815	.938	.547	1.607
Marital status (Divorced)	-.051	.255	.039	1	.843	.951	.576	1.568
Marital status (Separated)	-	.407	7.313	1	.007	.333	.150	.739
Marital status (Never married)	-.558	.198	7.959	1	.005	.572	.388	.843
Marital status (Living with partner)	-.523	.273	3.658	1	.056	.593	.347	1.013
Annual family income, AFI (\$ 0 to \$ 4,999 is the reference category)			12.605	13	.479			
AFI (\$ 5,000 to \$ 9,999)	-.116	.406	.082	1	.774	.890	.402	1.973
AFI (\$10,000 to \$14,999)	.586	.390	2.264	1	.132	1.797	.837	3.857
AFI (\$15,000 to \$19,999)	.237	.396	.357	1	.550	1.267	.583	2.754
AFI (\$20,000 to \$24,999)	.535	.401	1.777	1	.182	1.708	.778	3.751
AFI (\$25,000 to \$34,999)	-.078	.360	.047	1	.828	.925	.457	1.873
AFI (\$35,000 to \$44,999)	.032	.377	.007	1	.932	1.033	.493	2.163
AFI (\$45,000 to \$54,999)	.171	.399	.183	1	.669	1.186	.542	2.596
AFI (\$55,000 to \$64,999)	.199	.445	.201	1	.654	1.221	.510	2.922
AFI (\$65,000 to \$74,999)	.078	.446	.031	1	.861	1.081	.451	2.590
AFI (NOT sure but knows it is \$20,000 and Over)	.444	.507	.768	1	.381	1.559	.577	4.209
AFI (NOT sure but knows it is Under \$20,000)	.028	.641	.002	1	.965	1.028	.293	3.611
AFI (\$75,000 to \$99,999)	.376	.402	.875	1	.350	1.457	.662	3.205
AFI (\$100,000 and Over)	-.150	.373	.162	1	.687	.860	.414	1.788

Gender (Male is the Reference Category)	.134	.138	.936	1	.333	1.143	.872	1.499
Age in years at screening	-.007	.005	2.006	1	.157	.993	.983	1.003
Race (Mexican American Is the reference category)			104.396	5	.000			
Race (Other Hispanic)	-.264	.312	.716	1	.397	.768	.416	1.416
Race (Non-Hispanic White)	-.683	.257	7.032	1	.008	.505	.305	.837
Race (Non-Hispanic Black)	.180	.267	.454	1	.500	1.197	.709	2.022
Race (Non-Hispanic Asian)	-	.330	55.116	1	.000	.086	.045	.165
	2.448							
Race (Other Race – Including Multi-Racial)	-	.453	5.443	1	.020	.347	.143	.844
	1.057							
Education Level, EL (Less than 9th grade is the reference category)			9.414	4	.052			
EL (9-12th grade with no diploma)	.608	.293	4.309	1	.038	1.837	1.035	3.260
EL (High school graduate/ GED or equivalent)	.378	.279	1.840	1	.175	1.460	.845	2.522
EL (Some college or AA degree)	.414	.277	2.235	1	.135	1.513	.879	2.602
EL (College graduate or above)	.017	.295	.003	1	.955	1.017	.570	1.812
Physical Activity Score	-.039	.013	8.582	1	.003	.962	.937	.987
Constant	.882	.518	2.897	1	.089	2.415		

* Variable(s) entered on step 1: Total Phthalate Exposure (ng/mL), Marital status, Annual

family income, Gender, Age in years at screening, Race, Education Level, Physical Activity Score.

Cox & Snell R Square = 0.169; Nagelkerke R Square = 0.226.

The variables that are statistically significant ($p < 0.05$) in the model are shown in boldface.

Research Question 2

RQ2: Do the socio-demographic variables (race/ethnicity, gender, age, education level, and income) influence the relationship between exposure to phthalates and obesity among adults 18 years and older in the US?

H₂₀: The socio-demographic variables have no statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older the U.S.

H_{2A}: The socio-demographic variables have statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older in the U.S.

The null hypothesis was tested by fitting two regression models. The fitted binary logistic regression models are shown in Tables 15 and 16. The first model shown in Table 10 is the logistic regression model that includes the interactions between Total Phthalate Exposure and each of Race, Age, and Gender; while the second model shown in Table 11 is the Logistic regression model that does not include the effects of Race, Age, and Gender and their interactions with Total Phthalate Exposure.

The results of the fitted binary logistic regression model show that while the main effects of Race and Education Level are statistically significant ($p < 0.05$) in the model, the main effect of Gender ($p=0.450$) is not statistically significant in the model (Table 10). On the other hand, only the interaction effect of Race with Total Phthalate Exposure is statistically significant ($p < 0.05$, Table 10) in the model. The removal of Race, Age,

and Gender from the second model (shown in Table 11) makes the effect of Phthalate Exposure on the odds of obesity to approach statistical significance ($p = 0.077$, Table 11). The statistical significance ($p < 0.05$, Table 10) of the interaction effect of Race with Total Phthalate Exposure on obesity and the change in the statistical significance ($p = 0.077$, Table 11) of the effect of Phthalate Exposure upon the removal of the socio-demographic variables from the model help in answering the second research question. Based on the results, I can reject the null hypothesis and conclude that (although the interactions are weak) socio-demographic variables (such that race, age, gender, education level and income) have a weak influence on the relationship between Total Phthalate Exposure and obesity, with only the interaction effect of race been statistically significant ($p < 0.05$, Table 10).

Table 10

*Logistic regression results for Total Phthalate Exposure by Race, Age, and Gender**

	B	S.E.	Wald	df	Sig.	Exp(B)	95% CI for EXP(B)	
							Lower	Upper
Total Phthalate Exposure (ng/mL)	.001	.001	2.501	1	.114	1.001	1.000	1.003
Marital status (Married is the reference category)			15.760	5	.008			
Marital status (Widowed)	-.069	.278	.062	1	.803	.933	.541	1.609
Marital status (Divorced)	-.044	.258	.029	1	.865	.957	.577	1.587
Marital status (Separated)	-1.127	.409	7.578	1	.006	.324	.145	.723
Marital status (Never married)	-.618	.201	9.476	1	.002	.539	.363	.799
Marital status (Living with partner)	-.550	.276	3.965	1	.046	.577	.336	.991

Annual family income, AFI (\$ 0 to \$ 4,999 is the reference category)		11.834	13	.541			
AFI (\$ 5,000 to \$ 9,999)	-.047	.412	.013	1	.908	.954	.425 2.140
AFI (\$10,000 to \$14,999)	.591	.397	2.223	1	.136	1.806	.830 3.930
AFI (\$15,000 to \$19,999)	.280	.400	.491	1	.483	1.323	.605 2.896
AFI (\$20,000 to \$24,999)	.534	.406	1.728	1	.189	1.706	.769 3.785
AFI (\$25,000 to \$34,999)	-.084	.364	.053	1	.819	.920	.450 1.879
AFI (\$35,000 to \$44,999)	.036	.382	.009	1	.924	1.037	.491 2.192
AFI (\$45,000 to \$54,999)	.157	.404	.151	1	.697	1.170	.530 2.582
AFI (\$55,000 to \$64,999)	.230	.450	.262	1	.609	1.259	.522 3.039
AFI (\$65,000 to \$74,999)	.117	.452	.067	1	.795	1.125	.464 2.727
AFI (NOT sure but knows it is \$20,000 and Over)	.422	.512	.680	1	.409	1.526	.559 4.163
AFI (NOT sure but knows it is Under \$20,000)	.098	.662	.022	1	.883	1.102	.301 4.035
AFI (\$75,000 to \$99,999)	.401	.406	.975	1	.323	1.493	.674 3.307
AFI (\$100,000 and Over)	-.135	.378	.128	1	.721	.874	.416 1.833
Gender (Male is the Reference Category)	.127	.168	.571	1	.450	1.135	.817 1.576
Age in years at screening	-.004	.006	.466	1	.495	.996	.985 1.007
Race (Mexican American Is the reference category)			102.506	5	.000		
Race (Other Hispanic)	.111	.405	.075	1	.784	1.117	.505 2.474
Race (Non-Hispanic White)	-.685	.336	4.146	1	.042	.504	.261 .975
Race (Non-Hispanic Black)	.533	.341	2.443	1	.118	1.705	.873 3.327
Race (Non-Hispanic Asian)	-2.576	.420	37.621	1	.000	.076	.033 .173
Race (Other Race – Including Multi-Racial)	-.903	.580	2.423	1	.120	.405	.130 1.264
Education Level, EL (Less than 9th grade is the reference category)			10.358	4	.035		
EL (9-12th grade with no diploma)	.677	.299	5.130	1	.024	1.967	1.095 3.533
EL (High school graduate/ GED or equivalent)	.439	.284	2.387	1	.122	1.551	.889 2.706
EL (Some college or AA degree)	.459	.282	2.652	1	.103	1.582	.911 2.747
EL (College graduate or above)	.057	.301	.036	1	.849	1.059	.587 1.908
Physical Activity Score	-.040	.014	8.836	1	.003	.960	.935 .986

Race * Total Phthalate Exposure (ng/mL)	16.760	5	.005					
Race (Other Hispanic) by Total Phthalate Exposure (ng/mL)	-.001	.001	1.793	1	.181	.999	.997	1.000
Race (Non-Hispanic White) by Total Phthalate Exposure (ng/mL)	.000	.001	.035	1	.852	1.000	.999	1.002
Race (Non-Hispanic Black) by Total Phthalate Exposure (ng/mL)	-.001	.001	1.888	1	.169	.999	.998	1.000
Race (Non-Hispanic Asian) by Total Phthalate Exposure (ng/mL)	.001	.001	.337	1	.562	1.001	.999	1.002
Race (Other Race) by Total Phthalate Exposure (ng/mL)	-.001	.001	.280	1	.597	.999	.997	1.002
Age in years at screening by Total Phthalate Exposure (ng/mL)	.000	.000	2.371	1	.124	1.000	1.000	1.000
Gender (Female) by Total Phthalate Exposure (ng/mL)	.000	.000	.076	1	.782	1.000	1.000	1.001
Constant	.471	.574	.673	1	.412	1.602		

* Variable(s) entered on step 1: Total Phthalate Exposure (ng/mL), Marital status, Annual

family income, Gender, Age in years at screening, Race, Education Level, Physical

Activity Score, Race * Total Phthalate Exposure (ng/mL), Age in years at screening *

Total Phthalate Exposure (ng/mL), Gender * Total Phthalate Exposure (ng/mL).

Cox & Snell R Square = 0.187; Nagelkerke R Square = 0.249.

The variables that are statistically significant ($p < 0.05$) in the model are shown in boldface.

Table 11

*Logistic Regression for Total Phthalate Exposure excluding the effect of age, race, and gender**

	B	S.E.	Wald	df	Sig.	Exp(B)	95% CI for EXP(B)	
							Lower	Upper
Total Phthalate Exposure (ng/mL)	.000	.000	3.128	1	.077	1.000	1.000	1.000
Marital status (Married is the reference category)			9.262	5	.099			
Marital status (Widowed)	.061	.242	.064	1	.800	1.063	.662	1.708
Marital status (Divorced)	.207	.237	.762	1	.383	1.230	.773	1.958
Marital status (Separated)	-	.382	4.535	1	.033	.443	.210	.937
	.813							
Marital status (Never married)	-	.168	2.766	1	.096	.757	.545	1.051
	.279							
Marital status (Living with partner)	-	.251	.770	1	.380	.802	.490	1.313
	.221							
Annual family income, AFI (\$ 0 to \$ 4,999 is the reference category)			9.188	13	.759			
AFI (\$ 5,000 to \$ 9,999)	-	.388	.349	1	.555	.795	.372	1.700
	.229							
AFI (\$10,000 to \$14,999)	.319	.367	.754	1	.385	1.376	.670	2.827
AFI (\$15,000 to \$19,999)	-	.373	.052	1	.820	.919	.442	1.910
	.085							
AFI (\$20,000 to \$24,999)	.305	.378	.654	1	.419	1.357	.647	2.845
AFI (\$25,000 to \$34,999)	-	.345	.007	1	.934	.972	.495	1.910
	.029							
AFI (\$35,000 to \$44,999)	-	.358	.005	1	.942	.974	.483	1.965
	.026							
AFI (\$45,000 to \$54,999)	-	.378	.049	1	.824	.919	.438	1.929
	.084							
AFI (\$55,000 to \$64,999)	.139	.417	.111	1	.739	1.149	.507	2.602
AFI (\$65,000 to \$74,999)	.088	.423	.044	1	.835	1.092	.477	2.503

AFI (NOT sure but knows it is \$20,000 and Over)	.421	.473	.791	1	.374	1.524	.602	3.853
AFI (NOT sure but knows it is Under \$20,000)	.189	.623	.092	1	.762	1.208	.356	4.097
AFI (\$75,000 to \$99,999)	.116	.376	.096	1	.757	1.123	.537	2.349
AFI (\$100,000 and Over)	-	.354	.563	1	.453	.767	.383	1.534
	.265							
Education Level, EL (Less than 9th grade is the reference category)					19.790	4	.001	
EL (9-12th grade with no diploma)	.615	.271	5.151	1	.023	1.850	1.088	3.148
EL (High school graduate/GED or equivalent)	.395	.256	2.386	1	.122	1.484	.899	2.449
EL (Some college or AA degree)	.364	.248	2.153	1	.142	1.439	.885	2.340
EL (College graduate or above)	-	.265	.744	1	.388	.796	.474	1.337
	.228							
Physical Activity Score	-	.012	8.088	1	.004	.966	.943	.989
	.035							
Constant	.051	.388	.017	1	.895	1.052		

* Variable(s) entered on step 1: Total Phthalate Exposure (ng/mL), Marital status, Annual family income, Education Level, Physical Activity Score.

Cox & Snell R Square = 0.061; Nagelkerke R Square = 0.082.

The variables that are statistically significant ($p < 0.05$) in the model are shown in boldface.

Research Question 3

RQ3: Does physical activity influence the relationship between exposure to phthalates and obesity among adults 18 years and older in the U.S?

H3₀: Physical activity has no statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older in the U.S.

H3_A: Physical activity have statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older the U.S.

The null hypothesis was tested by fitting two logistic regression models shown in Table 12 and 13. The first logistic regression (Table 12) includes the interactions between Total Phthalate Exposure and Physical Activity; while the second logistic regression model (Table 13) does not include the effects of Physical Activity and its interactions with Total Phthalate Exposure.

The results of the fitted binary logistic regression models show that while the main effects of Physical Activity Score is statistically significant ($p < 0.05$) in the models (Table 12 and 13), the effect of the interaction between Physical Activity Score and Total Phthalate Exposure on obesity is not statistically significant ($p = 0.268$, Table 12) in the model. Furthermore, the removal of Physical Activity Score from the second model (Table 13) does not affect the level of significance of Phthalate Exposure in the models (Table 12 and 13). These help in answering the third research question such that, based on these results, I cannot reject the null hypothesis and state that Physical Activity does

not have influence on any possible relationship between Total Phthalate Exposure and obesity (Table 12 and 13) among adults 18 years and older in the U.S.

Table 12

*Logistic regression results Physical Activity Score and Total Phthalate Exposure**

	B	S.E.	Wald	df	Sig.	Exp(B)	95% CI for EXP(B)	
							Lower	Upper
Total Phthalate Exposure (ng/mL)	.000	.000	.076	1	.783	1.000	1.000	1.000
Marital status (Married is the reference category)			14.423	5	.013			
Marital status (Widowed)	-.066	.275	.057	1	.812	.937	.546	1.606
Marital status (Divorced)	-.048	.255	.035	1	.851	.953	.578	1.572
Marital status (Separated)	-1.110	.407	7.462	1	.006	.329	.148	.731
Marital status (Never married)	-.562	.198	8.057	1	.005	.570	.387	.840
Marital status (Living with partner)	-.527	.273	3.712	1	.054	.591	.346	1.009
Annual family income, AFI (\$ 0 to \$ 4,999 is the reference category)			12.876	13	.457			
AFI (\$ 5,000 to \$ 9,999)	-.116	.406	.082	1	.775	.890	.401	1.974
AFI (\$10,000 to \$14,999)	.593	.390	2.313	1	.128	1.810	.843	3.889
AFI (\$15,000 to \$19,999)	.240	.396	.368	1	.544	1.272	.585	2.764
AFI (\$20,000 to \$24,999)	.541	.402	1.814	1	.178	1.718	.782	3.774
AFI (\$25,000 to \$34,999)	-.080	.360	.049	1	.824	.923	.456	1.870
AFI (\$35,000 to \$44,999)	.030	.378	.006	1	.936	1.031	.492	2.161
AFI (\$45,000 to \$54,999)	.178	.400	.199	1	.656	1.195	.546	2.616
AFI (\$55,000 to \$64,999)	.206	.446	.214	1	.644	1.229	.513	2.943
AFI (\$65,000 to \$74,999)	.073	.447	.027	1	.870	1.076	.448	2.583
AFI (NOT sure but knows it is \$20,000 and Over)	.454	.507	.801	1	.371	1.575	.583	4.255
AFI (NOT sure but knows it is Under \$20,000)	.032	.636	.002	1	.960	1.032	.296	3.594

AFI (\$75,000 to \$99,999)	.378	.403	.882	1	.348	1.459	.663	3.212
AFI (\$100,000 and Over)	-.152	.373	.165	1	.684	.859	.413	1.786
Gender (Male is the Reference Category)	.129	.138	.869	1	.351	1.138	.867	1.492
Age in years at screening	-.007	.005	1.906	1	.167	.993	.983	1.003
Race (Mexican American Is the reference category)			104.456	5	.000			
Race (Other Hispanic)	-.292	.314	.869	1	.351	.746	.404	1.380
Race (Non-Hispanic White)	-.696	.258	7.281	1	.007	.498	.301	.827
Race (Non-Hispanic Black)	.166	.268	.382	1	.536	1.180	.698	1.995
Race (Non-Hispanic Asian)	-2.467	.331	55.676	1	.000	.085	.044	.162
Race (Other Race - Including Multi-Racial)	-1.072	.453	5.595	1	.018	.342	.141	.832
Education Level, EL (Less than 9th grade is the reference category)			9.333	4	.053			
EL (9-12th grade with no diploma)	.624	.293	4.523	1	.033	1.866	1.050	3.317
EL (High school graduate/GED or equivalent)	.393	.280	1.975	1	.160	1.481	.856	2.562
EL (Some college or AA degree)	.431	.277	2.412	1	.120	1.539	.893	2.650
EL (College graduate or above)	.043	.296	.021	1	.884	1.044	.584	1.866
Physical Activity Score	-.049	.016	9.302	1	.002	.952	.923	.983
Physical Activity Score by Total Phthalate Exposure (ng/mL)	.000	.000	1.227	1	.268	1.000	1.000	1.000
Constant	.918	.520	3.119	1	.077	2.503		

* Variable(s) entered on step 1: Total Phthalate Exposure (ng/mL), Marital status, Annual family income, Gender, Age in years at screening, Race, Education Level, Physical Activity Score, Physical Activity Score * Total Phthalate Exposure (ng/mL).

Cox & Snell R Square = 0.170; Nagelkerke R Square = 0.227.

The variables that are statistically significant ($p < 0.05$) in the model are shown in boldface.

Table 13

Logistic Regression for Total Phthalate Exposure excluding the effect of Physical

*Activity**

	B	S.E.	Wald	df	Sig.	Exp(B)	95% CI for EXP(B)	
							Lower	Upper
Total Phthalate Exposure (ng/mL)	.000	.000	.822	1	.365	1.000	1.000	1.000
Marital status (Married is the reference category)			15.257	5	.009			
Marital status (Widowed)	-.071	.275	.067	1	.796	.931	.543	1.596
Marital status (Divorced)	-.052	.254	.041	1	.839	.950	.577	1.562
Marital status (Separated)	-1.153	.404	8.150	1	.004	.316	.143	.697
Marital status (Never married)	-.577	.197	8.555	1	.003	.562	.382	.827
Marital status (Living with partner)	-.507	.272	3.463	1	.063	.602	.353	1.027
Annual family income, AFI (\$ 0 to \$ 4,999 is the reference category)			14.013	13	.373			
AFI (\$ 5,000 to \$ 9,999)	-.099	.404	.060	1	.807	.906	.410	2.001
AFI (\$10,000 to \$14,999)	.636	.388	2.694	1	.101	1.890	.884	4.041
AFI (\$15,000 to \$19,999)	.288	.395	.533	1	.465	1.334	.616	2.890
AFI (\$20,000 to \$24,999)	.623	.401	2.416	1	.120	1.865	.850	4.094
AFI (\$25,000 to \$34,999)	-.002	.358	.000	1	.996	.998	.494	2.015
AFI (\$35,000 to \$44,999)	.082	.376	.048	1	.827	1.086	.519	2.269
AFI (\$45,000 to \$54,999)	.243	.398	.372	1	.542	1.275	.584	2.782
AFI (\$55,000 to \$64,999)	.274	.442	.385	1	.535	1.315	.554	3.126
AFI (\$65,000 to \$74,999)	.131	.446	.087	1	.768	1.140	.476	2.732
AFI (NOT sure but knows it is \$20,000 and Over)	.532	.504	1.115	1	.291	1.703	.634	4.575
AFI (NOT sure but knows it is Under \$20,000)	.002	.634	.000	1	.997	1.002	.289	3.475
AFI (\$75,000 to \$99,999)	.455	.401	1.286	1	.257	1.576	.718	3.457
AFI (\$100,000 and Over)	-.106	.372	.080	1	.777	.900	.434	1.867

Gender (Male is the Reference Category)	.194	.136	2.026	1	.155	1.214	.930	1.585
Age in years at screening	-.004	.005	.590	1	.442	.996	.987	1.006
Race (Mexican American Is the reference category)			103.871	5	.000			
Race (Other Hispanic)	-.253	.311	.663	1	.415	.777	.422	1.427
Race (Non-Hispanic White)	-.702	.256	7.527	1	.006	.495	.300	.818
Race (Non-Hispanic Black)	.192	.266	.522	1	.470	1.212	.719	2.042
Race (Non-Hispanic Asian)	-2.409	.328	53.992	1	.000	.090	.047	.171
Race (Other Race - Including Multi-Racial)	-1.033	.451	5.243	1	.022	.356	.147	.862
Education Level, EL (Less than 9th grade is the reference category)			10.751	4	.030			
EL (9-12th grade with no diploma)	.635	.291	4.753	1	.029	1.887	1.066	3.341
EL (High school graduate/GED or equivalent)	.352	.277	1.613	1	.204	1.422	.826	2.448
EL (Some college or AA degree)	.390	.275	2.009	1	.156	1.477	.861	2.531
EL (College graduate or above)	-.030	.293	.010	1	.919	.971	.547	1.722
Constant	.432	.493	.766	1	.381	1.540		

* Variable(s) entered on step 1: Total Phthalate Exposure (ng/mL), Marital status, Annual

family income, Gender, Age in years at screening, Race, Education Level.

Cox & Snell R Square = 0.163; Nagelkerke R Square = 0.217.

The variables that are statistically significant ($p < 0.05$) in the model are shown in boldface.

Summary

This study investigated the relationship between exposure to phthalates and obesity, as well as whether or not the relationship between exposure to phthalates and obesity was influenced by other variables such as physical activity and socioeconomic status.

Three research questions were posed. The needed data for the study were obtained from the National Health and Examination Nutrition Survey, 2011-2012.

Binary logistic regression models were fitted to the null hypotheses and, in the process, the research questions posed were answered. The results show that I cannot reject the first null hypothesis and state that, based on the available information, on the average, exposure to Phthalate does not have a statistically significant effect on the odds of obesity (Table 9). In other words, the results of the fitted binary logistic regression model for the first research question showed that Total Phthalate Exposure is not statistically significant in the model ($p = 0.393$, Table 9).

On the other hand, based on the results for the second research question, I rejected the second null hypothesis and concluded that (although the interactions are weak) socio-demographic variables such as Race, Age, and Gender have a weak influence on the relationship between Total Phthalate Exposure and obesity, with only the influence from race been statistically significant ($p < 0.05$, Table 10).

However, the test of the third null hypothesis showed no statistical significance, such that the effect of the interaction between Physical Activity Score and Total Phthalate Exposure on obesity was not statistically significant ($p = 0.268$, Table 12) in the model. Based on the results for the third research question, I cannot reject the third null hypothesis and state that Physical Activity does not have influence on any possible relationship between Total Phthalate Exposure and obesity (Table 12 and 13) among adults 18 years and older in the US.

Chapter 5: Discussion, Conclusion, and Recommendations

The purpose of this study was to evaluate the relationship between exposure to phthalate chemicals and obesity in the United States. I examined whether or not there was an association between exposure to phthalates and obesity in the United States. I also investigated if SES (education level and income) and place of residence (zip code) influenced the relationship between exposure to phthalates and obesity. Further, I examined whether or not physical activity influenced the relationship between exposure to phthalates and obesity. Race/ethnicity, gender, and age were included as each research question was examined. Because the literature reviewed supported the contribution of these risk factors to obesity, this study built upon this paradigm while adjusting for potential confounders. Because about 30% of the population is obese (Flagel, Carroll, Ogden & Curtin, 2010) it would be pertinent to see if these statistics were reliable in the United States. I explored the single and collective impact of phthalates chemicals exposure, age, gender, race/ethnicity, physical activity, income, and education level on the prevalence of obesity. It is important to determine which of the obesity risk factors drive the increased incidence of obesity in the United States.

Inferential statistics were carried out using binary logistic regression modelling. I found that phthalate did not have a statistically significant effect on the odds of obesity and that (although the interactions are weak) sociodemographic variables such as race, age, and gender had a weak influence on the relationship between total phthalate exposure and obesity, with only the influence from race being statistically significant ($p <$

0.05). Furthermore, I found that the effect of the interaction between physical activity score and total phthalate exposure on obesity was not statistically significant ($p = 0.268$) among adults 18 years and older in the United States

Interpretation of Findings

For the last three decades, obesity rates in the United States and other Industrialized countries increased tremendously. (Hu, 2008). In developing countries, there is a shift from undernutrition to obesity, and obesity-related diseases like hypertension, type 2 diabetes and cardiovascular disease (Park, Juri, Seong, Kim, Cho, Baik, et al., 2012). To effectively, prevent obesity, the knowledge of the factors that impact obesity should be considered. Although dietary patterns and physical activity are known to cause obesity, endocrine disrupting chemicals like phthalates may impact obesity by altering hormonal balance or gene expression (Hatch et al., 2008). Many epidemiological studies conducted on the relationship between obesity and EDCs has limitations that necessitate further studies (Hu, 2008). Scholars have shown the link between exposures to phthalate chemicals and obesity (Plotnikoff et al., 2011). Obesity being one of the leading causes of long-term disability (Newbolt, 2010)), needs critical primary prevention in the United States.

Research Question 1

For the first research question, I found that total phthalate exposure was not statistically significant in the model ($p = 0.393$). On average, exposure to phthalates did

not have a statistically significant effect on the odds of obesity. Among the United States adults who are 18 years and above, exposure to phthalates alone by itself did not have any statistically significant influence on the odds of being obese. This result was in contrast to the findings in the literatures reviewed before this study (Tang-Peronard et al., 2011; Thayer et al., 2012). For instance, Tang-Peronard et al. (2011) showed that some obesogens like phthalates may play a role in the development of obesity. The findings of this study regarding the relationship between phthalate exposure and obesity was also in contrast with the results from the research conducted by Thayer et al. (2012) who concluded that 30% of obesity was associated with phthalates exposure.

Although studies reviewed before this research established that relationship exist between the exposure to phthalates and obesity (Fulcher & Gibb, 2014; Newbolt, 2010; Sargis et al., 2010). I found that among the United States adults who are 18 years and above, exposure to phthalates alone by itself did not have any statistically significant influence on the odds of being obese.

Research Question 2

I found that although the main effects of race and education level were statistically significant ($p < 0.05$) in the model, the main effect of gender ($p=0.450$) was not statistically significant in the model. The odds of being obese was influenced by an individual's race to a statistically significant level. Compared to the Mexican Americans, Non-Hispanic Whites and the Non-Hispanic, Asians had lower odds of being obese with an *OR* of 0.504 (95% *CI* of *OR* = (0.261, 0.975) and 0.076 (95% *CI* of *OR* = (0.033,

0.173). Compared to the Mexican Americans, Non-Hispanic Blacks had a higher odd of been obese ($OR=1.705$, 95% CI of $OR = (0.873, 3.327)$). Although I found that gender did not have any statistically significant effect on the odds of been obese, Ogden, Carrill, Curtin, McDowell, Tabak & Flegal (2006) had a different result. Ogden et al. observed that the prevalence of obesity has been increasing among women. In addition, Zhang et al. (2014) observed age-specific and sex-specific concentration-effect relationship between phthalate exposure and fat distribution in Chinese adolescents.

Furthermore, in the assessments of the effects of education level on the odds of obesity, I found that compared to the people with less than a ninth grade educational attainment, the people with 9th -12th grade educational attainment had higher odds of been obese ($OR=1.967$, 95% CI of $OR = (1.095, 3.533)$). On the other hand, the odds of obesity in the other educational attainment levels did not differ to a statistically significant extent from the odds of being obese for the people with less than a ninth grade educational attainment. Educational attainment may have little influence on an individual's odds of obesity. This does not match the findings of the previous studies on the association between educational attainment and the risk or odds of obesity (Hajian-Tilaki, & Haidari, (2009), showed that educational completion was inversely associated with general obesity in both sexes.

The interaction effect of race with total phthalate exposure was the only statistically significant variable ($p < 0.05$) in the model. Further, the removal of race, age, and gender from the second model made the effect of phthalate exposure on the odds of

obesity to approach statistical significance ($p = 0.077$). The statistical significance ($p < 0.05$) of the interaction effect of race with total phthalate exposure on obesity and the change in the statistical significance ($p = 0.077$) of the effect of phthalate exposure upon the removal of the sociodemographic variables from the model helped in answering the second research question. Based on the results, I rejected the null hypothesis and concluded that there was a weak interaction between sociodemographic variables such as race, age, and gender on the relationship between total phthalate exposure and obesity, with only the influence from race being statistically significant ($p < 0.05$). Overall, this was in line with the findings of previous studies. Dong et al. (2017) concluded that although phthalate exposure may be associated with obesity, associations and the potential gender and age differences were not consistent. The findings of the current study also match the results obtained by Trasande et al. (2013), who identified a race/ethnicity relationship between phthalates and obesity.

Research Question 3

For the third research question, the results show that physical activity has no statistically significant influence on the relationship between exposure to phthalates and obesity among adults 18 years and older in the U.S. The results of the fitted binary logistic regression models showed that while the main effects of Physical Activity Score was statistically significant ($p < 0.05$) in the models (Tables 17 and 18), the effect of the interaction between Physical Activity Score and Total Phthalate Exposure on obesity was not statistically significant ($p = 0.268$) in the model. These suggest that exposure to

phthalates does not mediate or moderate the effects of physical activity on an individual's odds of obesity and that the potential effects of the two factors (namely exposure to phthalates and physical activity) on obesity are independent. Nevertheless, Public health guidelines advocate increased physical activity as a key factor in the prevention and treatment of obesity despite little empirical evidence to support its effectiveness (Hajian-Tilaki & Haidari (2010). Although secular trends in activity levels are not consistent with trends in obesity prevalence nor do data from observational or clinical trials support a direct relationship between activity and excess weight gain, physical activity is crucial to maintenance of good health because, physical activity helps to improve overall health and fitness (Hajian-Tilaki & Heidari, 2010). The socio-ecological model suggests that in order to prevent a health problem like obesity, it is essential to act across several levels of the model at the same time. This will help to maintain preventive efforts over time than any single intervention (Morley & Pratte, 2013; Dahlberg & Krug, 2015).

Limitations of the Study

The limitations of this study involved the use of secondary data which was originally collected for another study (NHANES, 2011-2012). The rigidity of the process with questions fixed to maintain validity and reliability also limit this study (NHANES, 2011-2012). Another limitation is that this study exclusively focused on the U.S. noninstitutionalized population which is the population of interest (NHANES, 2011-2012). While this does not seem to be a limitation in itself, conclusions from this study

can only be generalized to the U.S. noninstitutionalized population. Also, the questionnaires may have been less objective as a result of several factors, such as recall bias, misunderstanding of questions, or socially desirable responses (NHANES, 2011-2012). In risk factors cross sectional survey, respondents' self-reported responses could include a tendency of giving socially acceptable answers rather than facts on objectionable behaviors (Salazar & DiClemate, 2006). Selective revealing and or suppression of information relevant to the study by the respondent or the researcher is also a cause of concern (Salazar & DiClemate, 2006). Researcher associated outcome biases, can introduce reporting bias (Salazar & DiClemate, 2006).

Recommendations

Based on the results of this study, several recommendations have been made for further study. A wider geographical scope is needed because this study was limited to American adults in the United States. The study should be extended to young children from different geographical regions. This was a small study population consisting of 118 participants. The results may not be a complete reflection of the general population. Additional comparative tests should be done on all the variables using a larger sample size. A study that uses a purposeful, instead of a convenience sample could yield better results. A qualitative research study will be of great importance for a more robust exploration of the relationship between phthalates exposure and obesity.

Implications

This study could contribute to the body of knowledge by influencing targeted educational interventions related to obesity and its risk factors. Also, it may direct practice, influence policies, and reduce health cost by reduced obesity incidence. This is an important study as it will address and illuminate an understudied area in primary prevention of obesity in the United states. Studies show that Americans have negative outcomes from obesity (Go, et al. 2013). Furthermore, the results of this study will guide obesity prevention by providing tailored or targeted education towards obesity.

Identification of the most important risk factors that drive the increased obesity occurrence will aid in primary prevention thus reducing the incidence of obesity in the United States. Primary obesity prevention is essential in the United States because of the numerous negative outcomes (Plotnikoff, Lightfoot, Spinola, Predy, & Barrett, 2010). It is hoped that the findings from this study will lead to greater awareness of obesity risk factors in the communities, and possibly lead to policy changes (Plotnikoff et al., 2010).

The study findings highlighted the importance of obesity education to patients by healthcare providers. This is especially important because, most obesity cases are only detected when patients visit the clinics for follow up with their primary care providers. Every clinic should have a lifestyle center where patient's questions can be addressed professionally. Obese patients should be referred to the Nutritional Counselling Units for advice and follow up. Planned community activities in community centers with different weekly topics on addressing some health issues like obesity would be beneficial. Faith-

based activities involving the religious groups in the communities are to be considered. All these are geared towards saturating the communities with educational materials. Obesity prevention can only be achieved when people understand the obesity risk factors.

The results of the current study support positive social change aimed at broadening the community educational programs on obesity. Understanding behaviors that positively influence health through the reduction of obesity risk factors is important. Also, the information from this research study will help in the establishment of effective programs that will reduce the prevalence of overweight and obesity. The identification of the impacts of phthalates exposure on obesity will help in primary prevention and reduction of the incidence of obesity in America. Primary obesity prevention is vital because weight loss is not easy to achieve once one gets obese. The findings will lead to greater awareness and implementation of obesity risk factor preventive measures in the communities, healthcare system, and policy change. According to Bienkowski (2012), there is poor implementation of obesity prevention strategies despite evidence of effectiveness of primary prevention and risk factor management.

Conclusion

Obesity is the leading cause of debilitation among Americans (Aungst, 2011). Obesity leads to increased risk for heart disease, cardiovascular events, hospitalizations and death (Aungst, 2011). Heaviness affects all racial and cultural groups in the world (Dahlberg, & Krug, 2015). The treatment of obesity affects the financial status of

America and other countries (CDC, 2016). Public health interventions to reduce obesity in the United States are both a moral and financial necessity. The reduction of unnecessary hospitalization and emergency room costs will result in redirecting those resources to other areas that can improve people's quality of life and contribute to greater economic stability. The Healthy People 2020 initiative targeted medical conditions like obesity and the social determinants of health (Aungst, 2011). To achieve optimal health outcomes, obesity and the related diseases must be considered and addressed (Aungst, 2011).

The results of this study showed that some variables like age and education were associated with the development of obesity. This information is useful in addressing the concerns about the exacerbation of obesity. Effective obesity education of patients by healthcare practitioners in primary care remains the key to reduced incidence of obesity.

References

Adams, K. F., Schatzkin, A., Harris, T. B., Kipnis, V., Mouw, T., Ballard-Barbash, R., ... & Leitzmann, M. F. (2006). Overweight, obesity, and mortality in a large prospective cohort of persons 50 to 71 years old. *New England Journal of Medicine*, 355(8), 763-778.

DOI: 10.1056/NEJMoa055643

Andreyeva, T., Kelly, I. R., & Harris, J. L. (2011). Exposure to food advertising on television: Associations with fast food and soft drink consumption and obesity.

Economics & Human Biology, 9(3), 221-233

<https://doi.org/10.1016/j.ehb.2011.02.004>

Aungst, R. B. (2011). Healthy people 2020. *Perspectives on Audiology*, 7(1), 29-33.,

doi:10.1044/poa7.1.29

Baillie-Hamilton, P. F. (2002). Chemical toxins: A hypothesis to explain the global

obesity epidemic. *The Journal of Alternative & Complementary Medicine*, 8(2),

185-192, <https://doi.org/10.1089/107555302317371479>

Barrett, J. R. (2015). Urinary biomarkers as exposure surrogates: Controlling for possible

bias. *Environmental Health Perspectives*, 123(4), A97,

<https://doi.org/10.3945/ajcn.2010.28701B>

Behl, M., Rao, D., Aagaard, K., Davidson, T. L., Levin, E. D., Slotkin, T. A. Thayer, K.

A. (2015). Evaluation of the association between maternal smoking, childhood

obesity, and metabolic disorders: A national toxicology program workshop

review. *Environmental Health Perspectives*, 121(2), 170. doi:10.1289/ehp.120540

Bengough, G., Castrignano, A., Pages, L., & Noordwijk, M. (2009). Sampling strategies,

scaling, and statistics. *Springer*, 56(6), 147-173. Retrieved from,

<https://doi.org/10.1146/annurev-publhealth-032315-021841>

Bertucci, M., Miller, A., Jaggi, S., & Wilding, S. (2010). Cutting the fat on healthcare:

An investigation of preventive healthcare and the fight on obesity. *Undergraduate*

Research Journal for the Human Sciences, 9(1). Retrieved from,

<http://www.kon.org/urc/v9/bertucci.html>

- Bienkowski (2012). *Chemical BPA Linked to Obesity in Children*. Environmental Health News. Retrieved from, www.sciencetificamerican.com
- Biro, F. M., & Wien, M. (2010). Childhood obesity and adult morbidities-. *The American journal of clinical nutrition*, 91(5), 1499S-1505S, <https://doi.org/10.3945/ajcn.2010.28701B>
- Boodman, S. G. (2011). Many Americans have poor health literacy. *The Washington Post*, 28. Retrieved from www.washingtonpost.com/Health
- Bray, G. A. (1985). Obesity: definition, diagnosis and disadvantages. *The Medical Journal of Australia*, 142(7 Suppl), S2. Retrieved from, <https://www.ncbi.nlm.nih.gov/pubmed/3884986>
- Bradley, R. H., & Corwyn, R. F. (2002). Socioeconomic status and child development. *Annual review of psychology*, 53(1), 371-399. Retrieved from, <https://www.annualreviews.org/doi/abs/10.1146/annurev.psych.53.100901.135233>
- Bronfenbrenner, U., & Condry Jr, J. C. (1970). *Two worlds of childhood: US and USSR*.
- Brown, R. J., De Banate, M. A., & Rother, K. I. (2010). Artificial sweeteners: a systematic review of metabolic effects in youth. *International Journal of Pediatric Obesity*, 5(4), 305-312, <https://www.tandfonline.com/doi/abs/10.3109/17477160903497027>
- Bleich, S. N., Pickett-Blakely, O., & Cooper, L. A. (2011). Physician practice patterns of obesity diagnosis and weight-related counseling. *Patient education and counseling*, 82(1), 123-129, 3 <https://doi.org/10.1016/j.pec.2010.02.018>

- Burris, S., Ashe, M., Levin, D., Penn, M., & Larkin, M. (2016). A transdisciplinary approach to public health law: The emerging practice of legal epidemiology. *Annual Review of Public Health, 37*, 135-148. Retrieved from, <https://doi.org/10.1146/annurev-publhealth-032315-021841>
- . Campbell, D. T., & Stanley, J. C. (1963). Experimental and quasi-experimental designs for research. *Handbook of research on teaching*, 171-246. Retrieved from, https://wagner.nyu.edu/files/doctoral/Campbell_and_Stanley_Chapter_5.pdf
- Casas, L., Fernandez, M. F., Llop, S., Guxens, M., Ballester, F., Olea, N., & Vryheid, M. (2011). Urinary concentrations of phthalates and phenols in a population of Spanish pregnant women. *Environment International, 37*(5), 858-866. Retrieved from, <https://doi.org/10.1016/j.envint.2011.02.012>
- Carter, R. C. (2002). The impact of public schools on obesity. *Journal of the American Medical Association, 288*(17), 2180. doi:10.1001/jama.288.17.2180
- Carson, V., Spence, J. C., Cutumisu, N., & Cargill, L. (2010). Association between neighborhood socioeconomic status and screen time among pre-school children: A cross-sectional study. *BMC Public Health, 10*(1), 367. doi:10.1186/14712458-10-367
- Cassel, K. D. (2010). Using the social-ecological model as a research and intervention framework to understand and mitigate obesogenic factors in Samoan populations. *Ethnicity & Health, 15*(4), 397-416. doi:10.1080/13557858.2010.481330
- Centers for Disease Control and Prevention. (2016). Childhood obesity facts. 2015. Available at www.cdc.gov/healthyschools/obesity/facts

- Chamorro-García, R., & Blumberg, B. (2014). Transgenerational effects of obesogens and the obesity epidemic. *Current Opinion in Pharmacology*, *19*, 153-158, <https://doi.org/10.1016/j.coph.2014.10.010>
- Chamorro-García, R., Kirchner, S., Li, X., Janesick, A., Casey, S. C., Chow, C., & Blumberg, B. (2012). Bisphenol A diglycidyl ether induces adipogenic differentiation of multipotent stromal stem cells through a peroxisome proliferator-activated receptor gamma-independent mechanism. *Environmental health perspectives*, *120*(7), 984, doi: [10.1289/ehp.1205063](https://doi.org/10.1289/ehp.1205063)
- Cha, E., Kim, K. H., Lerner, H. M., Dawkins, C. R., Bello, M. K., Umpierrez, G., & Dunbar, S. B. (2014). Health Literacy, Self-efficacy, Food Label Use, and Diet in Young Adults. *American Journal of Health Behavior*, *38*(3), 331–339. doi.org/10.5993/AJHB.38.3.2
- Chiellini, F., Ferri, M., Morelli, A., Dipaola, L., & Latini, G. (2013). Alternatives to phthalate plasticized poly (vinyl chloride) in medical devices applications. *Progress in Polymer Science*, *38*(7), 1067-1088. <https://doi.org/10.1016/j.progpolymsci.2013.03.001>
- Creswell, J. (2009). *Research design: Qualitative, quantitative, and mixed methods approach* (3rd ed.). Thousand Oaks, CA: Sage Publications. Retrieved from <https://books.google.com/books?hl=en&lr=&id=KGNADwAAQBAJ&oi>
- Dahlberg L. L., Krug, E. G. (2015). Obesity a global public health problem. World Report on Obesity and Health. Geneva, Switzerland: World Health Organization; 2002:1–56, <https://doi.org/10.1377/hlthaff.28.5.w822>.

- Dong, R., Zhou, T., Chen, J. et al. Arch Environ Contam Toxicol (2017) 73: 431.
- Dunton, G. F., Kaplan, J., Wolch, J., Jerrett, M., & Reynolds, K. D. (2010). Physical environmental correlates of obesity: A systematic review. *Obesity Reviews*, 10(4), 393-402. doi:10.1111/j.1467-789x.2009.00572.x.
- Facts, C. O. (2013). Centers for Disease Control and Prevention website. Available at <http://www.cdc.gov/healthyschools/obesity/facts>
- Faul, F., Erdfelder, E., Lang, A. G., & Buchner, A. (2007). G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, 39 (2), 171-191. Retrieved from, <https://link.springer.com/article/10.3758/BF03193146>
- Fayter, D., McDaid, C., & Eastwood, A. (2007). A systematic review highlights threats to validity in studies of barriers to cancer trial participation. *Journal of clinical epidemiology*, 60(10), 990-e1. Retrieved from, <https://doi.org/10.1016/j.jclinepi.2006.12.013>
- Field, A. (2009). *Discovering statistics using SPSS*. Sage publications. Retrieved from, <https://books.google.com/books?hl=en&lr=&id=4mEOw7xa3z8C&oi=fnd&pg>
- Finkelstein, E. A., Trogon, J. G., Cohen, J. W., & Dietz, W. (2009). Annual medical spending attributable to obesity: payer-and service-specific estimates. *Health affairs*, 28(5), w822-w831, Retrieved from, <https://doi.org/10.1377/hlthaff.28.5.w822>

- Flegal, K. M., Carroll, M. D., Ogden, C. L., & Curtin, L. R. (2010). Prevalence and trends in obesity among US citizens, 1999-2008. *Jama*, *303*(3), 235-241, doi:10.1001/jama.2009.2014
- Frankfort-Nachmias, C., & Nachmias, D. (2008). Sampling and sample designs. *Research methods in the Social Sciences (7th ed., pp. 161-185)*. New York, NY: Worth Publishers.
- Frerichs, L. (2016). Food literacy, Education and Communication. *Journal of the Academy of Nutrition and Dietetics*, .01.014 doi:10.1016/j.j.
- Fulcher, K., & Gibb, H. (2014). Setting the research agenda on the health effects of chemicals. *International journal of environmental research and public health*, *11*(1), 1049-1057, doi:[10.3390/ijerph110101049](https://doi.org/10.3390/ijerph110101049)
- Gittner L.S., Ludington-Hoe S.M., Haller, H.S. (2013). Utilizing infant growth to predict obesity status. *J. Pediatric. Child. Health*.49 (12), 564–574. doi: 10.1111/jpc.12283.
- Hao, C., Cheng, X., Xia, H., & Ma, X. (2012). The endocrine disruptor mono-(2-ethylhexyl) phthalate promotes adipocyte differentiation and induces obesity in mice. *Bioscience reports*, *32*(6), 619-629. Retrieved from, <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3558718/>
- Han, E., & Powell, L. M. (2010). Effect of food prices on the prevalence of obesity among young adults. *Public health*, *125*(3), 129-135, <https://doi.org/10.1016/j.puhe.2010.11.014>

- . Hatch, E. E., Nelson, J. W., Qureshi, M. M., Weinberg, J., Moore, L. L., Singer, M., & Webster, T. F. (2013). Association of urinary phthalate metabolite concentrations with body mass index and waist circumference: a cross-sectional study of NHANES data, 1999–2002. *Environmental Health*, 7(1), 27, <https://doi.org/10.1186/1476-069X-7-27>
- Hajian-Tilaki, K. O., & Heidari, B. (2009). Association of educational level with risk of obesity and abdominal obesity in Iranian adults. *Journal of Public Health*, 32(2), 202-209, doi.org/10.1093/pubmed/fdp083
- Hatch, E. E., Nelson, J. W., Qureshi, M. M., Weinberg, J., Moore, L. L., Singer M, et al. (2008). Association of urinary phthalate metabolite concentrations with body mass index and waist circumference: a cross-sectional study of NHANES data, 1999-2002. *Environ Health*. 7:27, <https://doi.org/10.1186/1476-069X-7-27>
- Heindel, J. J., Newbold, R., & Schug, T. T., (2015). Endocrine disruptors and obesity. *Nature Reviews Endocrinology*, 11(11), 653-661. Retrieved from, <https://www.nature.com/articles/nrendo.2015.163>
- Henderson, V. C., Kimmelman, J., Fergusson, D., Grimshaw, J. M., & Hackam, D. G. (2013). Threats to validity in the design and conduct of preclinical efficacy studies: a systematic review of guidelines for in vivo animal experiments. *PLoS Med*, 10(7), e1001489, doi.org/10.1371/journal.pmed.1001489

- Herrera, B. M., Keildson, S., & Lindgren, C. M. (2011). Genetics and epigenetics of obesity. *Maturitas*, 69(1), 41-49, <https://doi.org/10.1016/j.maturitas.2011.02.018>
- Holtcamp, W. (2012). Obesogens: An Environmental Link to Obesity. *Environ Health Perspect Environmental Health Perspectives*, 120(2), 62-68.
doi:10.1289/ehp.120-a62
- Hu, F. (2008). Measurements of Adiposity and Body Composition: Hu F, ed. Obesity Epidemiology. New York City: *Oxford University Press*, (3), 53–83. Retrieved from, <https://books.google.com/books?hl=en&lr=&id=uwgSDAAAQBAJ&oi>
- Ihantola, E. M., & Kihn, L. A. (2011). Threats to validity and reliability in mixed methods accounting research. *Qualitative Research in Accounting & Management*, 8(1), 39-58. Retrieved from, <https://www.emeraldinsight.com/doi/abs/10.1108/11766091111124694>
- Janesick, A. S., & Blumberg, B. (2016). Obesogens: an emerging threat to public health. *American journal of obstetrics and gynecology*, 214(5), 559-565. Retrieved from, <https://doi.org/10.1016/j.ajog.2016.01.182>
- Janesick, A., & Blumberg, B. (2011). Endocrine disrupting chemicals and the developmental programming of adiposeness and obesity. *Birth Defects Research Part C: Embryo Today: Reviews*, 93(1), 34-55 Retrieved from, <https://doi.org/10.1002/bdrc.20197>

- Jennings, A., Welch, A., Jones, A. P., Harrison, F., Bentham, G., Van Sluijs, E. M., & Cassidy, A. (2011). Local food outlets, weight status, and dietary intake. *American journal of preventive medicine*, 40(4), 405-410, doi.org/10.1016/j.amepre.2010.12.014
- Juonala, M., Magnussen, C. G., Berenson, G. S., Venn, A., Burns, T. L., Sabin, M. A., & Sun, C. (2011). Adult adiposity, and cardiovascular risk factors. *New England Journal of Medicine*, 365(20), 1876-1885. DOI: 10.1056/NEJMoa1010112
- Karnik, S., & Kanekar, A. (2015). Obesity: A Global Public Health Crisis. *School Nutrition and Activity Impacts on Well-Being*, 3-15. doi: 10.1201/b18227-3
- Kelishadi, R., Poursafa, P., & Jamshidi, F. (2013). Role of Environmental Chemicals in Obesity: A Systematic Review on the Current Evidence. *Journal of Environmental and Public Health*, 22 (34), 2011-2023, doi.org/10.1155/2013/896789
- Kim, S. H., and Park, M. J. (2014). Phthalate exposure and obesity. *Annals of Pediatric Endocrinology and Metabolism*, 4 (19), 69 - 75. Retrieved from, <https://www.thetimes.co.uk/article/obesity-is-fastest-growing-cause-of-death-around-the-world-bzk8vj5j5>
- Kim, K. E., Kim, S. H., Park, S., Khang, Y. H., Park, M. J. (2014). Changes in prevalence of obesity and underweight among Koreans: 1998-2008. *Korean J Obes.* (21), 228–235.

- Kuczmarski, M. F., Adams, EL., Cotugna, N., Pohlig, RT., Beydoun, MA., Zonderman, AB, et al. (2016) Health Literacy and Education Predict Nutrient Quality of Diet of Socioeconomically Diverse, Urban Adults. *J Epid Prev Med* 2(1): 115, doi: [10.19104/jepm.2016.115](https://doi.org/10.19104/jepm.2016.115)
- Kwack, L. (2010). Comparison of the short term toxicity of phthalate dieter and monoester in Sprague dawley male rats. *Toxicology Research* (10) 10.5487, doi: [10.5487/TR.2010.26.1.075](https://doi.org/10.5487/TR.2010.26.1.075)
- La Merrill, M. & Birnbaum, LS. (2011). Obesity and environmental chemicals. *Mt Sinai J Med*, 78 (1), 22-44, doi.org/10.1002/msj.20229
- Lam, L. T., & Yang, L. (2014). Is low health literacy associated with overweight and obesity in adolescents: an epidemiology study in a 12–16 years old population, Nanning, China, 2012. *Archives of Public Health*, 72(1), 11, doi.org/10.1186/2049-3258-72-11
- Lee, J. (2010). Odds ratio or relative risk for cross-sectional data? *International Journal of Epidemiology*, 23(1), 201-203. Retrieved from, http://samba.fsv.cuni.cz/~soukup/ADVANCED_STATISTICS/lecture2/texts/oddratio.pdf
- Lee, Y. S. (1994). Consequences of obesity. *Ann Acad Med Singapore*, 38(1), 75-77.
- Levin (2006). Study design III: Cross-sectional studies. *Evidence-based dentistry*, 7(1), 24. doi: 10.1038/sj.ebd.6400375

- Lind, P. M., Roos, V., Ronn, M., Johansson, L., Ahlstrom, H., Kullberg, J. et al., (2012). Serum concentrations of phthalate metabolites are related to abdominal fat distribution two years later in elderly women. *Environ Health*. 11(9), 21 -28 , doi.org/10.1186/1476-069X-11-2
- Lioy, P. J., Gennings, C., Hauser, R., Koch, H. M., & Kortenkamp, A. (2014). Changing trends in phthalate exposures. *Environmental health perspectives*, 122(10), A264. doi:10.1289/ehp.1408629
- Luke, A., & Cooper, S. (2013). Physical activity does not influence obesity risk: time to clarify the public health message. *International Journal of Epidemiology*, Volume 42, Issue 6, 1 December 2013, Pages 1831–1836, doi.org/10.1093/ije/dyt159
- Marijke de Cock, de Boer, M. R., Lamoree, M., Legler, J., & van de Bor, M. (2014). First year growth in relation to prenatal exposure to endocrine disruptors—a Dutch prospective cohort study. *International journal of environmental research and public health*, 11(7), 7001-7021, doi.org/10.1093/ije/dyt159
- Masters, R. K., Reither, E. N., Powers, D. A., Yang, Y. C., Burger, A. E., & Link, B. G. (2013). The impact of obesity on US mortality levels: the importance of age and cohort factors in population estimates. *American journal of public health*, 103(10), 1895-1901, [https://doi.org/10.1016/S0021-9150\(99\)00055-6](https://doi.org/10.1016/S0021-9150(99)00055-6)
- Mayo Clinic (2010). Obesity. Retrieved from <http://www.mayoclinic.com/health/-obesity/DS00698>

- MedCalc (2016). Logistic regression. Retrieved from,
<https://www.medcalc.org/manual/logistic-regression.php>
- Merrill, M. L., & Birnbaum, L. S. (2011). Obesity and Environmental Chemicals. *Mount Sinai Journal of Medicine: A Journal of Translational and Personalized Medicine*
Mt Sinai J Med, 78(1), 22-48. doi:10.1002/msj.20229
- Mitchell, J. A., and Byun, W. (2014). Sedentary behavior and health outcomes in adolescents. *American Journal of Lifestyle Medicine*, 8(3), 173-199,
doi.org/10.1177/1559827613498700
- Moisse, K. (2012). Plastic Chemical BPA Linked to Obesity. Retrieved from,
<http://abcnews.go.com/Health/Wellness/plastic-chemical-bpa-linked-childhood-obesity/story?id=17253096>
- Mond, J., Van den Berg, P., Boutelle, K., Hannan, P., & Neumark-Sztainer, D. (2011). Obesity, body dissatisfaction, and emotional well-being in late adolescence: findings from the project EAT study. *Journal of Adolescent Health*, 48(4), 373-378, doi.org/10.1016/j.jadohealth.2010.07.022
- Montoye, A. H., Pfeiffer, K. A., Alaimo, K., Betz, H. H., Paek, H. J., Carlson, J. J., & Eisenmann, J. C. (2013). Junk food consumption and screen time: association with adiposity. *American journal of health behavior*, 37(3), 395-403,
doi.org/10.5993/AJHB.37.3.12

- Morley, C. P., & Pratte, M. A. (2013). State-Level Tobacco Control and Adult Smoking Rate in the United States. *Journal of Public Health Management and Practice*, 19(6). doi:10.1097/phh.0b013e31828000de
- Nederkorn, C., Havermans, R. C., Giesen, J. C., & Jansen, A. (2011). High tax on high energy dense foods and its effects on the purchase of calories in a supermarket. An experiment. *Appetite*, 56(3), 760-765 doi.org/10.1016/j.appet.2011.03.002
- NCHS (National Center for Health Statistics) 2006a. Current NHANES Web Tutorial. Retrieved from, <http://www.cdc.gov/nchs/tutorials/currentnhanes/index.htm>
- NCHS (National Center for Health Statistics) 2006b. National Health and Nutrition Examination Survey. Retrieved from, <http://www.cdc.gov/nchs/nhanes.htm> [accessed 1 November 2018].
- Newbold, R. (2010). Impact of Environmental Endocrine-disrupting Chemicals on the Development of Obesity. *Hormones*, 9 (3), 206-217. Retrieved from, <http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.609.694&rep=rep1&type=pdf>
- Nyberg, K. A., Ramirez, A. G., & Gallion, K. (2011). *Addressing nutrition, overweight and obesity among Latino youth*. Robert Wood Johnson Foundation. Retrieved from, <https://www.rwjf.org/.../addressing-nutrition--overweight-and-obesity-among-latino-y>

- Obesity Statistics (2014). National Institute of Diabetes and Digestive and Kidney Diseases. Retrieved from <http://www.niddk.nih.gov/health-information/health-statistics/Pages/overweight-obesity-statistics.aspx>
- Obesity: Implications for Prevention and Treatment. American Diabetes Association Diabetes Care, 31(11), 2211-2221. doi: 10.2337/dc08-9024. *American Medical Association*, 288 (17), 2180. doi: 10.1001/jama.288.17.2180.
- Ogden, C. L., Flegal, K. M., Carroll, M. D., & Johnson, C. L. (2006). Trends in overweight among U.S. people, 1999-2000. *Jama*, 288(14), 1728-1732.
- and Theory (2013). Retrieved from, www.study.com
- Park, Juri, Seong H. Kim, Goo-Yeong Cho, Inkyung Baik, Nan H. Kim, Hong E. Lim, Eung J. Kim et al. "Obesity phenotype and cardiovascular changes." *Journal of hypertension* 29, no. 9 (2011): 1765-1772, doi: 10.1097/HJH.0b013e32834a50f3
- Plotnikoff, R. C., Lightfoot, P., Spinola, C., Predy, G., & Barrett, L. (2011). A Framework for Addressing the Global Obesity Epidemic Locally: Retrieved from, <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2483540/>
- Robinson, S. M., & Godfrey, K. M. (2008). Feeding practices in pregnancy and infancy: relationship with the development of overweight and obesity in childhood. *International Journal of Obesity*, 32(S6), S4, doi:10.1038/ijo.2008.201
- Russell-Mayhew, S., McVey, G., Bardick, A., & Ireland, A. (2012). Mental health, wellness, and childhood overweight/obesity. *Journal of obesity*, 2012, doi:10.1155/2012/281801
- . Salazar, L. F., Crosby, R. A., & DiClemente, R. J. (2006). Qualitative research strategies and methods for health promotion. *Research methods in health promotion*, 150-198.

Retrieved from,

<http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.609.694&rep=rep1&type=pdf>

- Saelens, B. E., Sallis, J. F., Frank, L. D., Couch, S. C., Zhou, C., Colburn, T., & Glanz, K. (2012). Obesogenic neighborhood environments, child and parent obesity: the Neighborhood Impact on Kids study. *American journal of preventive medicine*, 42(5), e57-e64, doi.org/10.1016/j.amepre.2012.02.008
- Sargis, R. M., Johnson, D. N., Choudhury, R. A., & Brady, M. J. (2010). Environmental endocrine disruptors promote adipogenesis in the 3t3 \square 11 cell line through glucocorticoid receptor activation. *Obesity*, 18(7), 1283-1288. Retrieved from, <https://doi.org/10.1038/oby.2009.419>
- Seidell, J. C., & Halberstadt, J. (2015). The global burden of obesity and the challenges of prevention. *Annals of Nutrition and Metabolism*, 66(Suppl. 2), 7-12, <https://doi.org/10.1159/000375143>
- Selassie, M., & Sinha, A. C. (2011). The epidemiology and aetiology of obesity: a global challenge. *Best Practice & Research Clinical Anaesthesiology*, 25(1), 1-9. doi.org/10.1016/j.bpa.2011.01.002
- Schmidt, C. O., & Kohlmann, T. (2009). When to use the odds ratio or the relative risk? *International journal of public health*, 53(3), 165-167. Retrieved from, <https://link.springer.com/article/10.1007%2Fs00038-008-7068-3>
- Schug, T. T., Janesick, A., Blumberg, B., & Heindel, J. J. (2011). Endocrine disrupting chemicals and disease susceptibility. *The Journal of steroid biochemistry and molecular biology*, 127(3), 204-215, doi.org/10.1016/j.jsbmb.2011.08.007

- Sharma, M. (2011). Dietary education in school-based obesity prevention programs. *Advances in Nutrition: An International Review Journal*, 2(2), 207S-216S. Endocrine-disrupting chemicals and obesity development in humans: A review. *Obesity reviews*, 12(8), 622-636.
- Smerieri, A., Testa, C., Lazzeroni, P., Nuti, F., Grossi, S., Montanini, L. et al., (2015). Di-(2-ethylhexyl) phthalate metabolites in urine show are-related changes and associations with adiposity and parameters of insulin sensitivity. *Pub Med*, 10 (2): e0117831. doi: 10.1371/journal.pone.0117831.
- Singh, S., & Li, S. S. (2011). Phthalates: toxicogenomics and inferred human diseases. *Genomics*, 97(3), 148-157, doi.org/10.1016/j.ygeno.2010.11.008
- Song, Y., Hauser, R., Hu, F. B., Franke, A. A., Liu, S., & Sun, Q. (2014). Urinary concentrations of bisphenol A and phthalate metabolites and weight change: a prospective investigation in US women. *International journal of obesity*, 38(12), 1532. Retrieved from, <https://www.nature.com/articles/ijo201463>
- Su, J. G. (2015). An online tool for obesity intervention and public health. *BMC public health*, 16(1), 131, doi.org/10.1186/s12889-016-2797-3
- Tang- Péronard, J. L., Andersen, H. R., Jensen, T. K., & Heitmann, B. L. (2011). Endocrine- disrupting chemicals and obesity development in humans: a review. *Obesity Reviews*, 12(8), 622-636., doi: 10.1111/j.1467-789X.2011.00871
- Tirosh, A., Shai, I., Afek, A., Dubnov-Raz, G., Ayalon, N., Gordon, B., ... & Rudich, A. (2011). Adolescent BMI trajectory and risk of diabetes versus coronary

disease. *New England Journal of Medicine*, 364(14), 1315-1325. DOI:

10.1056/NEJMoa1006992

Thayer, K. A., Heindel, J. J., Bucher, J. R., & Gallo, M. A. (2012). Role of environmental chemicals in diabetes and obesity: A National Toxicology Program workshop review. *Environmental Health Perspective*, 120(6), 779-789,

doi: [10.1289/ehp.1104597](https://doi.org/10.1289/ehp.1104597)

Trasande, L., Zoeller, R. T., Hass, U., Kortenkamp, A., Grandjean, P., Myers, J. P., & Skakkebaek, N. E. (2015). Estimating burden and disease costs of exposure to endocrine-disrupting chemicals in the European Union. *The Journal of Clinical Endocrinology & Metabolism*, 100(4), 1245-

1255 <https://doi.org/10.1210/jc.2014-4324>

Trasande, L., Attina, T. M., Sathyanarayana, S., Spanier, A. J., & Blustein, J. (2013). Race/ethnicity-specific associations of urinary phthalates with childhood body mass in a nationally representative sample. *Environmental health*

perspectives, 121(4), 501, doi: [10.1289/ehp.1205526](https://doi.org/10.1289/ehp.1205526)

Vander Wal, J. S., & Mitchell, E. R. (2011). Psychological complications of pediatric obesity. *Pediatric Clinics*, 58(6), 1393-1401. ,

<https://doi.org/10.1016/j.pcl.2011.09.008>

Vogt, W. P. (Ed.). (2007). *SAGE quantitative research methods*. Sage. Retrieved from <http://www.sagepub.com/reference/quantitative-research-methods>

Wang, H., Zhou, Y., Tang, C., He, Y., Wu, J., Chen, Y., & Jiang, Q. (2013). Urinary phthalate metabolites are associated with body mass index and waist circumference in Chinese school children. *PloS ONE*, 8(2), e56800. doi: 10.1371/journal.pone.0056800

World Health Organization. (2014). Health literacy: the solid facts. 2013. URL: http://www.euro.who.int/__data/assets/pdf_file/0008/190655/e96854.pdf [accessed 2014-10-27] [WebCite Cache ID 6Tdo2QPb8]

World Health Organization (WHO). (2017). Health literacy. The solid facts. *Health*. Retrieved from, <http://www.euro.who.int/en/health-topics/environment-health/health/urban-health/publications/2013/health-literacy-the-solid-facts>

Zhang Y, Meng X, Chen L, Li D, Zhao L, Zhao Y, & Shi, H. et al. (2014) Age and Sex-Specific Relationships between Phthalate Exposures and Obesity in Chinese Children at Puberty. *PLoS ONE* 9(8): e104852. Retrieved from, <https://doi.org/10.1371/journal.pone.0104852>

Appendix A: Title of Appendix

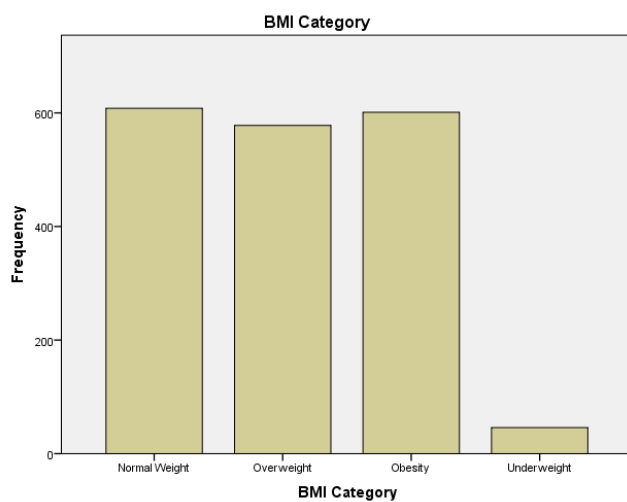


Figure A1. Frequency distribution of BMI Category



Figure A2. Frequency distribution of marital status

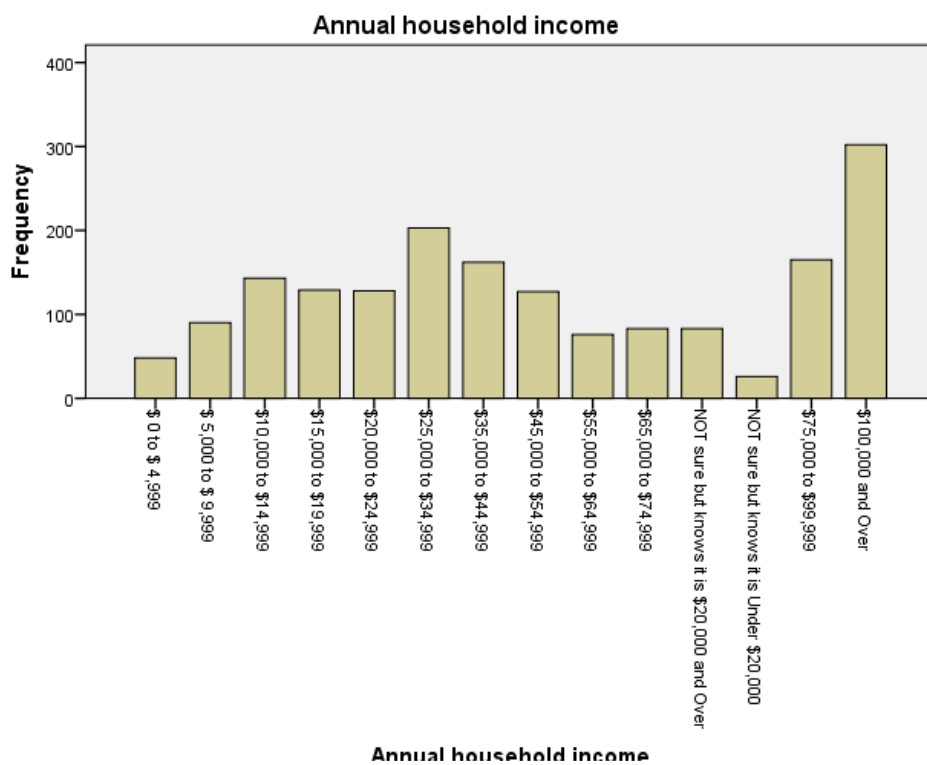


Figure A3. Frequency distribution of annual household income

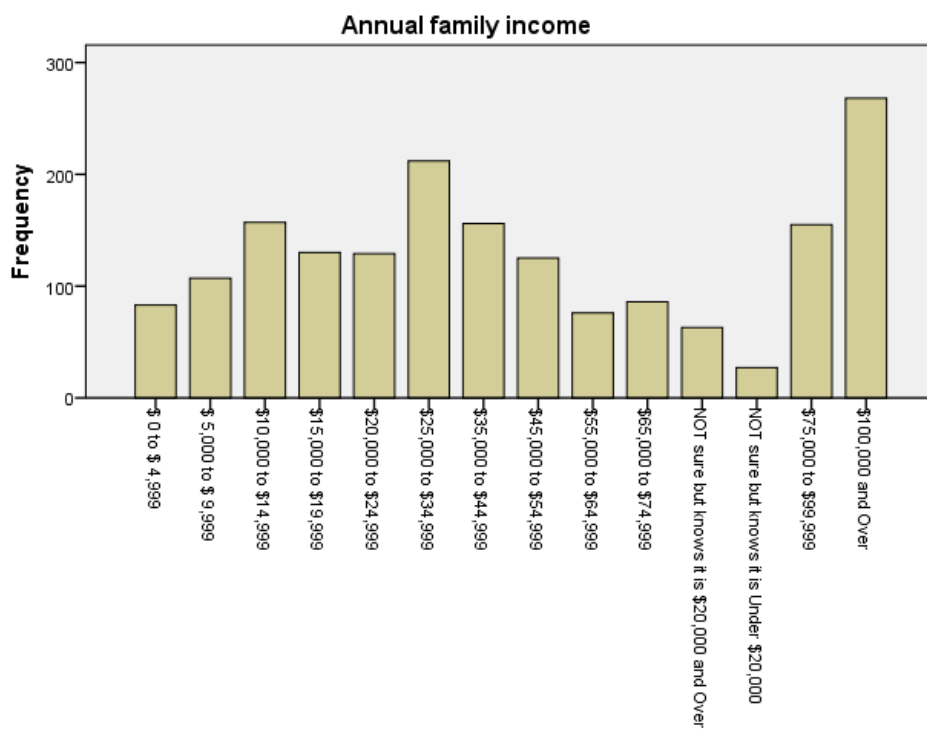


Figure A4. Frequency distribution of annual family income

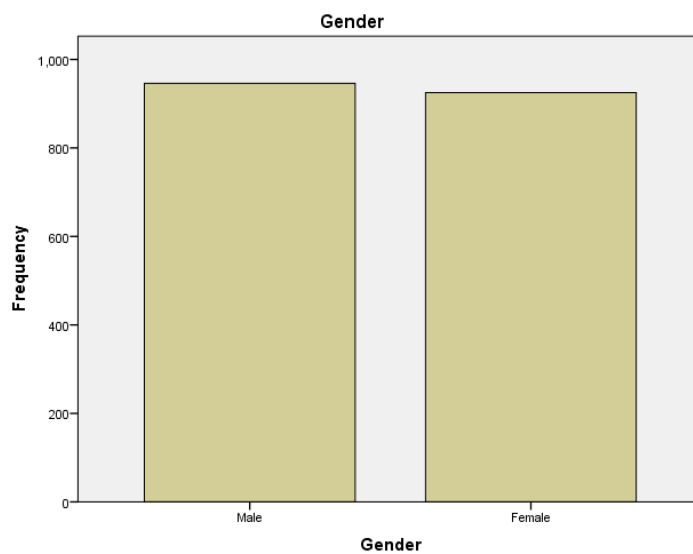


Figure A5. Frequency distribution of gender

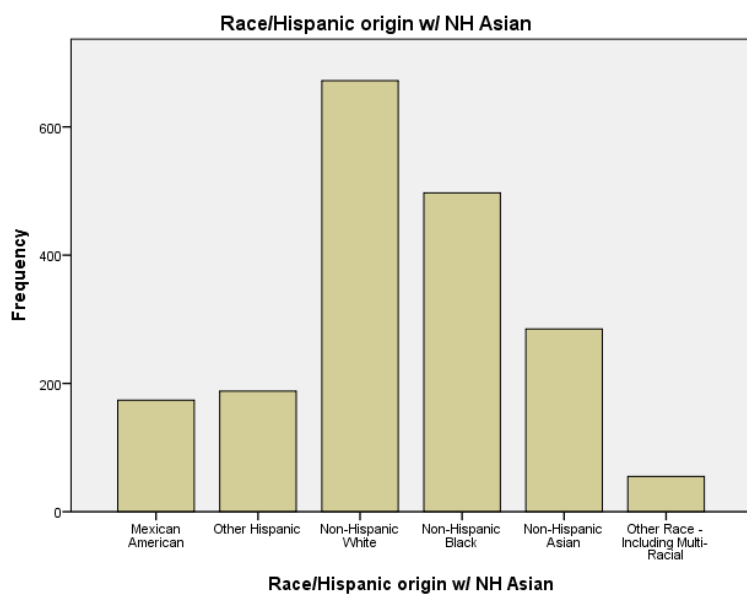


Figure A6. Frequency distribution of race

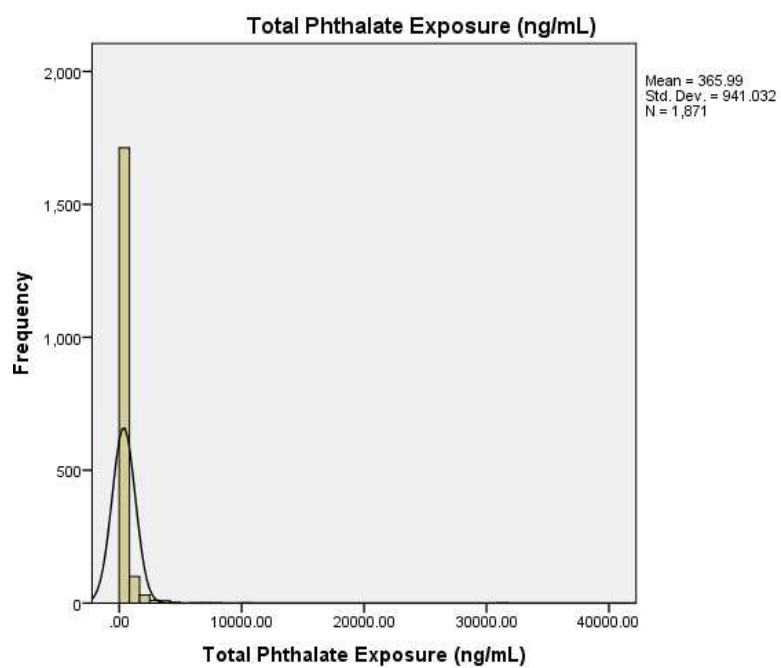


Figure A7. Distribution of total phthalate exposure (ng/mL)

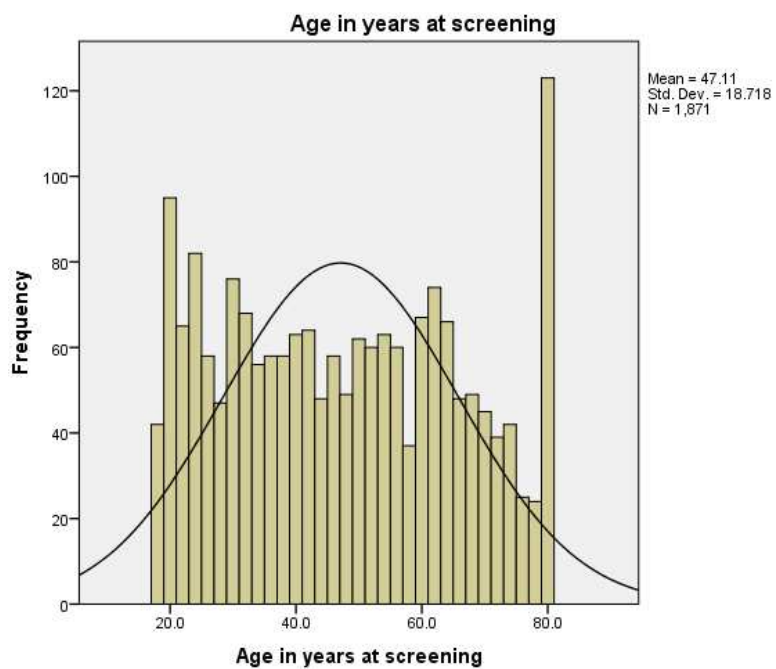


Figure A8. Distribution of age in years at screening

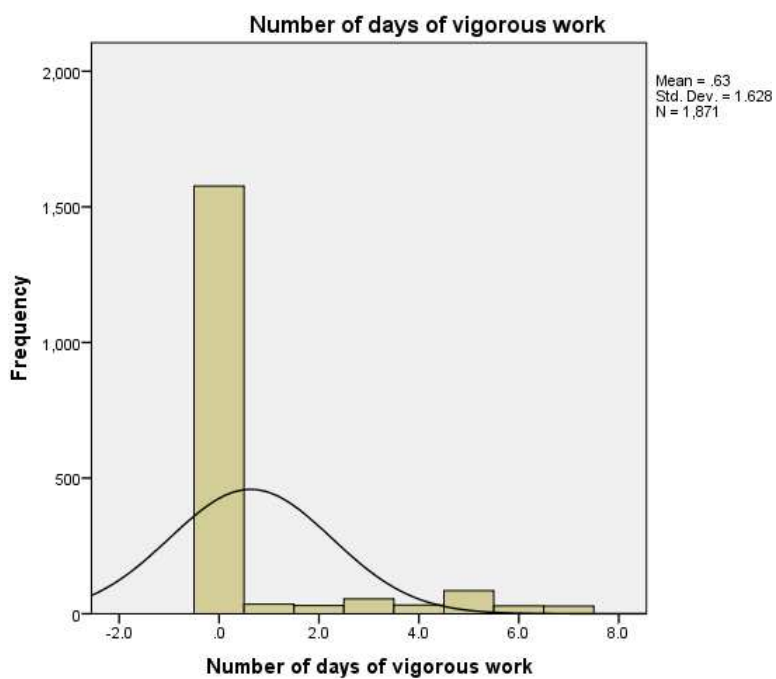


Figure A9. Distribution of the number of days of vigorous work

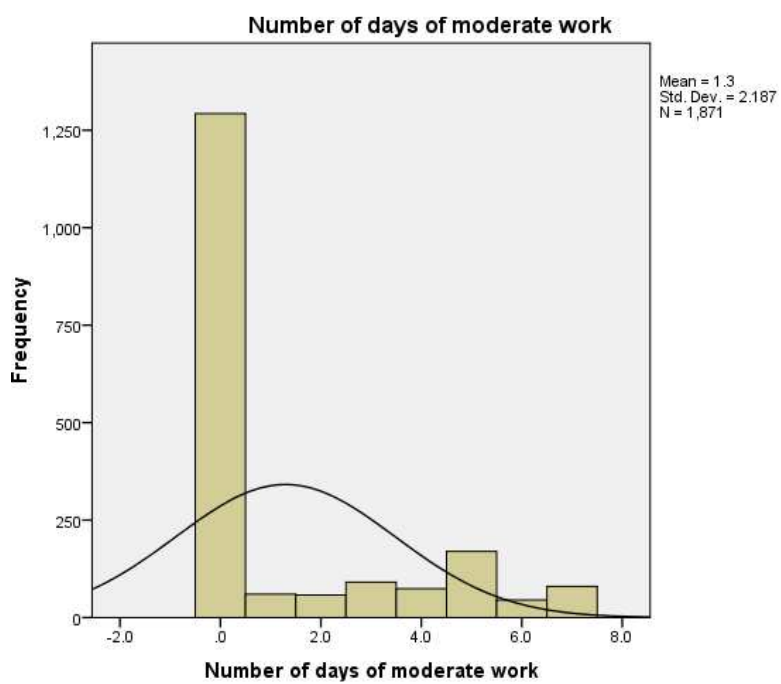


Figure A10. Distribution of the number of days of moderate work

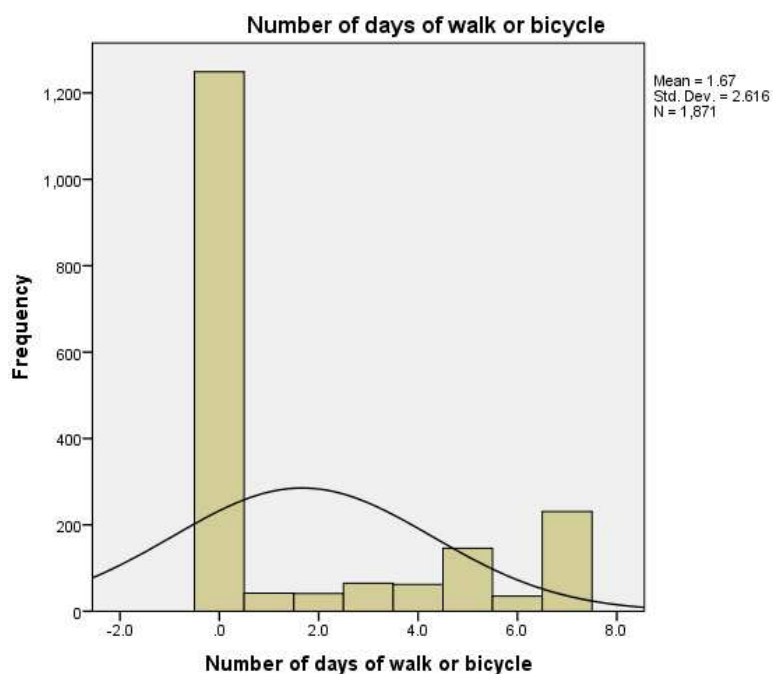


Figure A11. Distribution of the number of days of walk or bicycle

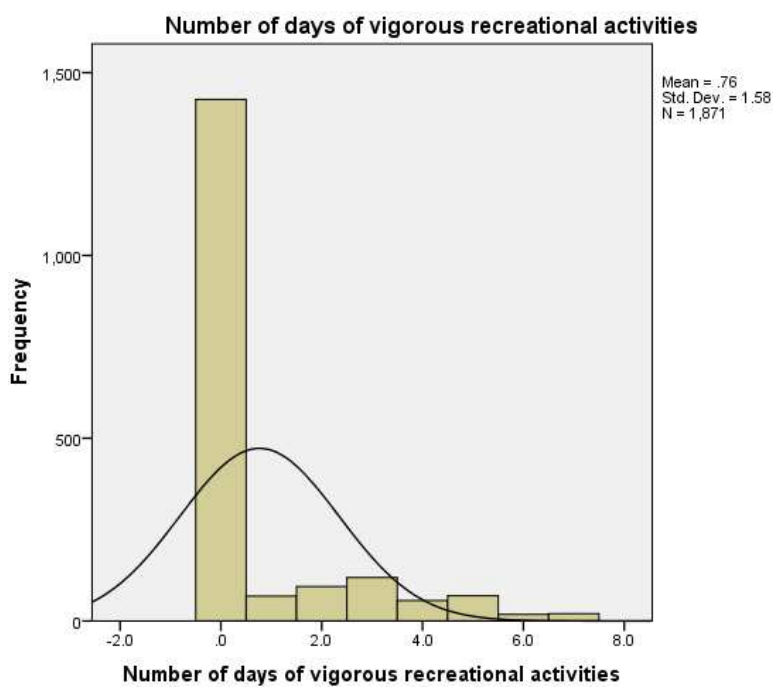


Figure A12. Distribution of the number of days of vigorous recreational activities

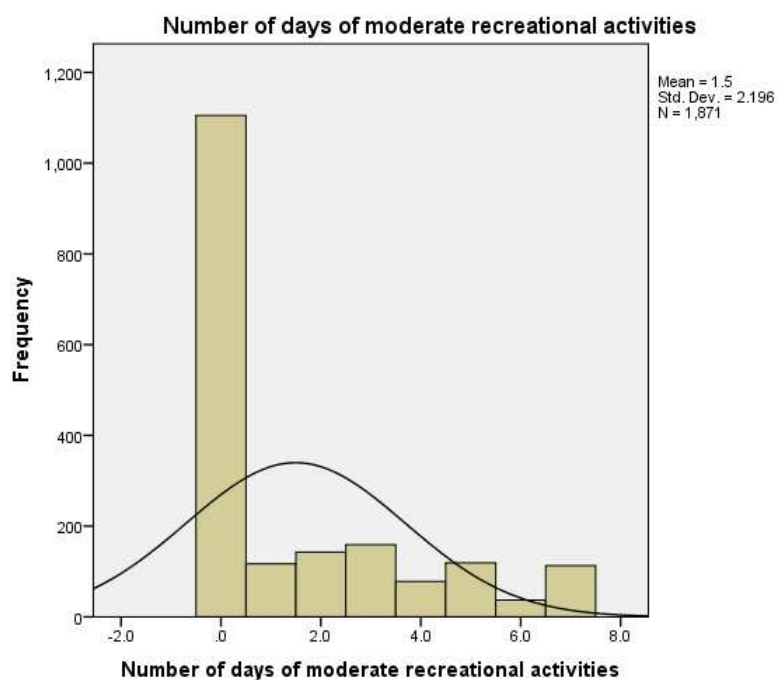


Figure A13. Distribution of the number of days of moderate recreational activities

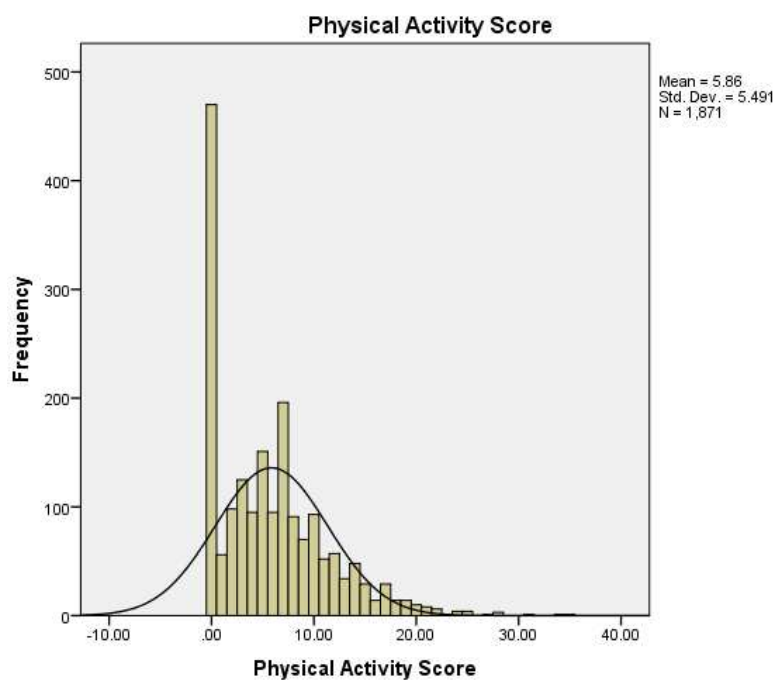


Figure A14. Distribution of the physical activity score