The Influence of Race/Ethnicity and Alcohol Use on High Blood Pressure and Diabetes

# A Dissertation presented for partial fulfillment of requirements for the Doctor of Philosophy Degree Public Health

Kent State University

VANESSA J. MARSHALL

December 2014

ProQuest Number: 10306818

All rights reserved

INFORMATION TO ALL USERS The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if material had to be removed, a note will indicate the deletion.



ProQuest 10306818

Published by ProQuest LLC (2017). Copyright of the Dissertation is held by the Author.

All rights reserved. This work is protected against unauthorized copying under Title 17, United States Code Microform Edition © ProQuest LLC.

> ProQuest LLC. 789 East Eisenhower Parkway P.O. Box 1346 Ann Arbor, MI 48106 – 1346

Copyright © 2014 by Vanessa J. Marshall ALL RIGHTS RESERVED

#### ABSTRACT

Alcohol use disorders persist as a public health challenge. Few studies focus on the role that alcohol use consumption contributes to the development of medical and behavioral outcomes that may influence susceptibility to chronic diseases among minority populations, in particularly high blood pressure and diabetes. Due to the limited evidence regarding racial/ethnic differences in risk and protective factors as it relates to alcohol use disorders across the life span and chronic diseases, little is known to inform the development of effective prevention strategies. The purpose of this study was to investigate the influence of race/ethnicity and alcohol use on high blood pressure and diabetes. A secondary data analysis from the 2008-2012 US National Survey on Drug Use and Health (NSDUH) was conducted. Weighted multivariable logistic regression models tested the moderating effects of race/ethnicity on the relationship between alcohol use in the self-reported outcomes of high blood pressure and diabetes diagnoses, adjusting for known risk and protective factors. Participants' alcohol use was categorized as nondrinkers, regular drinkers, and alcohol use disorder. After adjusting for risk and protective factors, the relationship between alcohol use and high blood pressure showed some differential protective effects for Non-Hispanic White and Non-Hispanic African American categories. For the outcome diabetes, adjusted models displayed protective effects of alcohol use that varied substantially across race/ethnicity categories. These findings support a significant interaction between race/ethnicity and alcohol use on high blood pressure and diabetes diagnoses. This study highlights the social ecological approach for understanding these complex determinants of alcohol use and cardiovascular outcomes across racial/ethnic populations. Study findings emphasize the importance of training public health professionals in prevention science on racial/ethnic variations of risk and protective factors and the association between alcohol use and chronic diseases. This study offers implications for public health professionals to provide preventive screening information to patients consuming alcohol and routinely assess psychosocial factors for determining and promoting healthy lifestyle behavior change.

Keywords: alcohol use, race/ethnicity, high blood pressure, diabetes, health disparities

ii

Dissertation written by

Vanessa J. Marshall

B.A., Psychology, Kent State University, 1995 M.A., Sociology, Cleveland State University, 2002 Ph.D., Kent State University, 2014

# Approved by

\_\_Jeffrey S. Hallam\_\_\_\_\_, Chair, Doctoral Dissertation Committee Jeffrey S. Hallam, Ph.D., CHES

\_\_Scott Grey\_\_\_\_\_, Member, Doctoral Dissertation Committee Scott Grey, Ph.D.

\_\_Margaret Stephens\_\_\_\_\_, Member, Doctoral Dissertation Committee Margaret Stephens, Ph.D.

\_\_Angela Neal-Barnett\_\_\_\_\_, Member, Doctoral Dissertation Committee Angela Neal-Barnett, Ph.D.

Accepted by

\_Jeffrey Hallam\_\_\_\_\_, Department Chair Jeffrey Hallam, Ph.D., CHES

\_Sonia Alemagno\_\_\_\_\_\_, Dean, College of Public Health\* Sonia Alemagno, Ph.D.

# **DEFINITION OF TERMS**

Alcohol Use Disorders: medical conditions that doctors can diagnose when a patient's alcohol consumption causes harm or stress and classified as either alcohol abuse or alcohol dependence (National Institute of Alcohol Abuse and Alcoholism [NIAAA], 2011)

Alcohol dependence: (also known as alcoholism) a chronic disease that includes symptoms such as craving, loss of control, withdrawal or tolerance (NIAAA, 2011)

Alcohol abuse: a pattern of drinking that results in harm to one's health and lifestyle such as relationships, school, and work (NIAAA, 2011)

Alcohol Related Health Disparities: the usage of alcohol interferes with daily lifestyle activities and causes problems such as health and behavioral (NIAAA, 2011)

Binge Drinking: a form of alcohol abuse (consumes more than 4-5 drinks in one setting) (NIAAA, 2011)

Cardiovascular Disease: includes heart and blood vessel disease and consists of high blood pressure, heart disease, atherosclerosis, and/or diabetes (Centers for Disease Control and Prevention [CDC], 2012)

Mental Disorders: behavioral pattern that causes distress or disability and generally defined by a combination of how a person feels or acts. Some commonly recognized categories are anxiety and depression (CDC, 2012)

Mental Health: state of well-being when an individual can cope with the stressors of life. http://www.cdc.gov/mentalhealth/basics.htm

Psychosocial Factors: combination of medical, behavioral and environmental factors (Everson-Rose & Lewis 2005); factors that affect a person psychologically or socially <u>http://psychologydictionary.org/psychosocial-factors/</u>

Race/Ethnicity: Alcohol research consistently uses multiple terms for race and ethnic populations. For instance, Blacks also may be referenced as African Americans or African descent, Whites as Caucasians or European descent, and Hispanics as Latinos or Latin American descent. For purposes of this study, African Americans/Blacks are defined as persons of African descent residing in the US; Caucasians/Whites are defined as persons of European descent residing in the US; and Hispanics/Latino are defined as persons of Latin American descent residing in the US (CDC, 2012; NIAAA, 2011;Substance Abuse and Mental Health Services Administration [SAMHSA], 2011)

Social Support: the perception that one is cared for by people and part of a supportive social network. It can be measured as the perception that one has assistance available such as family, friends, coworkers, organizations (Gottlieb & Bergen 2010); two demographic indicators often used as proxy measures of support including marital status and whether patients were living alone will be used (Frasure-Smith et al.,2000)

# LIST OF ABBREVIATIONS

Abbreviation	Description
CDC	Centers for Disease Control and Prevention
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition
NIAAA	National Institute on Alcohol Abuse and Alcoholism
NHLB	National Heart, Lung, and Blood Institute
NIDA	National Institute on Drug Abuse
NIH	National Institutes of Health
SAMHSA	Substance Abuse and Mental Health Services Administration
WHO	World Health Organization

# **ACKNOWLEDGEMENTS**

Understand to achieve anything requires faith, belief, vision, patience, hard work, determination, and dedication. I can do all things through Christ who strengthens me ~Philippians 4:13. In everything by prayer and supplication, with thanksgiving, let your requests be made known to God; and the peace of God, which surpasses all understanding, will guard your hearts and minds through Christ Jesus ~Philippians 4:6-7. The Lord is my light and my salvation ~ Psalm 27

I would like to thank the many people who have helped me to successfully complete this milestone of my life. I would like to extend a most sincere and grateful thanks to my parents, for their love, support, words of encouragement and wisdom. Thanks to my grandparents, aunts, uncles, brother, cousins, nephew, godchildren and friends for their support.

I would like to thank my committee members (Dr. Jeffrey Hallam, Dr. Scott Grey, Dr. Margaret Stephens, Dr. Angela Neal-Barnett) for their guidance, support and feedback throughout this endeavor. I would also like to thank my colleagues at Kent State University, Howard University, Case Western Reserve University, Cleveland State University, University of Connecticut, National Institutes of Health and Centers for Disease Control and Prevention.

TABLE OF	CONTENTS
----------	----------

Description	Page
Abstract	ii
List of Abbreviations	iii
Definitions of Terms	iv
Acknowledgements	V
List of Table	ix
Chapter I INTRODUCTION	10
Chronic Diseases	10
Link between Alcohol Use and Cardiovascular Related Chronic Diseases	11
Problem Statement	
Purpose	
Research Questions	
Specific Aim	
Hypotheses	16
Public Health Significance	16
Innovation	17
Chapter II REVIEW OF THE LITERATURE	19
Chronic Diseases and Health Disparities	
Alcohol Use: A Public Health Problem	21
Alcohol Related Health Disparities	
Race/Ethnic Disparities of Alcohol Use	24
Chronic Diseases Relation to Alcohol Use	

Cardiovascular Risk Factors	27
Link between Cardiovascular Disease and Alcohol Use	27
High Blood Pressure in High Risk Populations	29
Diabetes in High Risk Populations	31
Biopsychosocial Interactions of Alcohol Use on High Blood Pressure and Diabetes	32
Link between High Blood Pressure and Alcohol Use	
Link between Diabetes and Alcohol Use	
Stress, Chronic Diseases and Alcohol Use	
Racism and Discrimination as Risk Factors for Chronic Diseases	
Social Support, Chronic Diseases and Alcohol Use	
Social Ecological Framework and Alcohol Use	40
Conclusions	45
Chapter III METHODOLOGY	48
Data Source	49
Participants	49
Procedure	50
Measures	51
Demographic Section	52
Study Variables	52
Data Analysis Plan	55
Chapter IV RESULTS	59
Demographic Analyses	60
Unadjusted Interaction between Race/Ethnicity and Alcohol Use on High Blood Pressu	re61

Unadjusted Interaction between Race/Ethnicity and Alcohol Use on Diabetes	
Adjusted Full Model for High Blood Pressure	64
Adjusted Reduced Model for High Blood Pressure	64
Adjusted Full Model for Diabetes	69
Trend Analysis Effect for High Blood Pressure and Diabetes	71
Chapter V DISCUSSION	72
Objective of Study	72
Summary of Findings	78
Unadjusted Interaction Models on High Blood Pressure and Diabetes	79
Adjusted Models for High Blood Pressure and Diabetes	79
Socio-ecological Theoretical Approach for Prevention	80
Limitations	83
Implications of for Public Health Care Professionals	85
Future Direction	87
Conclusions	87
REFERENCES	89
APPENDIX A: SAS Data Code	119

# LIST OF TABLES

Table 1. Demographic Variables	68
Table 2. Alcohol Categories and Outcome Variables	69
Table 3. Race/Ethnicity and Outcome Variables	69
Table 4. High Blood Pressure Logistic Regression Models	70
Table 5. Diabetes Logistic Regression Models	71

#### **CHAPTER I**

#### **INTRODUCTION**

#### **Chronic Diseases**

Chronic diseases including cardiovascular disease, cancer, lung disease, diabetes mellitus and hypertension account for most of the mortality in the world (Yach, Hawkes, Gould, & Hofman, 2004; World Health Organization [WHO], 2012). In the United States, approximately 117 million adults have one or more chronic diseases (Ward, Schiller & Goodman, 2014). Moreover, these chronic diseases share essential modifiable behavioral risk factors such as alcohol use, tobacco use, sedentary lifestyle, and poor diet. Chronic diseases are referred to as non-communicable diseases and degenerative diseases with a prolonged temporal course (Centers for Disease Control and Prevention [CDC], 2012; Remington, Brownson & Wegner, 2010).

Literature suggest there is an increased of substance abuse (i.e. alcohol, illegal drugs and/or prescribed medications) in people diagnosed with chronic diseases (Dickey, Normand, Weiss, Drake, & Azeni, 2002). For example, the use of alcohol may assist a person by a means of self-medication of coping with stress, depression or anxiety from chronic diseases. Traditional risk factors (i.e. lack of physical activity, obesity, diet) do not completely account for the total risk of chronic diseases. It is essential to identify potentially modifiable environmental and behavioral risk factors for chronic diseases. All too often, patients and providers overlook risk factors such as alcohol use, level of social support, and mental health well-being when associated with chronic diseases.

To better understand the context of health disparities, it is important to understand and recognize different populations. It is not clear what accounts for the health disparities in chronic diseases. One explanation may be the reported risk factors and disease in populations with disparities in healthcare utilization and socioeconomic status (Kuller, 2004). The variations in epidemiological disease patterns across racial/ethnic populations are due both to genetic, environmental, and lifestyle behaviors (Cooper, Kaufman, & Ward, 2003). Some of the greatest health disparities in public health are found in the area of chronic diseases such as high blood pressure and diabetes. For instance, Black men had significantly higher rates of hospitalizations for high blood pressure than did White men (CDC, 2012). According to the Office of Minority Health [OMH] (2013), African Americans were 2.2 times as likely as non-Hispanic Whites to die from diabetes. There remains an increased susceptibility to chronic diseases that results from risk factors and psychosocial factors including social and cultural changes, and stressful life events. Unfortunately, there are 68 million people diagnosis with high blood pressure, 79 million with pre-diabetes, and 25.8 million with diabetes in the US (CDC, 2012). Diabetes and high blood pressure have an increased risk for a number of serious complications including renal disease, heart disease, stroke and congestive heart failure (Klatsky 2007; Wilson, 1998). For instance, compared to Non-Hispanic Whites, Non-Hispanic African American adults are 1.7 times and Hispanics are 2 times to have diabetes (Healthy People 2020).

# Link between Alcohol Use and Cardiovascular- Related Chronic Diseases

Chronic diseases such as hypertension and diabetes are the result of multiple risk factors including alcohol use. Researchers suggests cardiovascular disease and diabetes display a J or U shaped relationship implying characteristics of both beneficial and detrimental consequences of alcohol use (Goldberg & Soleas, 2001; Howard, Arnsten, & Gourevitch, 2004; Rehm, et al.,

2010; Roerecke & Rehm, 2012). It is well-known that the consumption of large quantities of alcohol is hazardous to the health and may induce cardiomyopathy and arrhythmias (CDC,2012). However consuming alcohol, even low amounts, increases the risk of developing chronic health problems. There are several interpretational problems with the data from ecological, case control, and prospective cohort studies about the evidence for a preventive effect of alcohol consumption for chronic diseases specifically cardiovascular disease (Foppa, Fuchs, & Duncan, 2001; Svardsudd, 1998). For instance, data from prospective cohort studies suggest a beneficial effect from all types of alcohol (Klatsky and Armstrong 1993; Stampfer, Colditz, Willett, Speizer & Hennekens, 1988) whereas, data from case control studies suggest no beneficial effect at all (Kaufman, Rosenberg, Helmrick, & Shapir, 1985; Rosenberg et al., 1981). There are many possible mechanisms for the preventive effect of alcohol including effects of the coagulation system (Hendriks, Veenstra, Velthuis-te Wierike, Schaafasma & Kluft, 1994; Ridker, Vaughan, Stampfer, Glynn & Hennekens, 1994), improved insulin sensitivity (Kiechl et al., 1996), and antioxidative effects (Fuchs & Chambless, 2007).

There is a multifaceted relationship between alcohol use and metabolic syndrome (risk of type 2 diabetes, hypertension and obesity; Alkerwi et al., 2009). Two studies suggest moderate alcohol use is associated with lowered risk of myocardial infarction (Fuchs et al., 1995; Garg, Wagener, & Madans, 1993). Furthermore, two meta-analyses suggest there is an inverse association with improved outcomes related to reduced cardiovascular health outcomes and light to moderate alcohol use (Costanzo, Di Castelnuovo, Donati, Iacoviello, & de Gaetano, 2010; Ronksley, Brien, Turner, Mukamal, & Ghali, 2011). However, there are inconsistencies regarding the extent of the benefits of low to moderate alcohol use in relation to cardiovascular disease (Fillmore, Stockwell, Chikritzhs, Bostrom, & Kerr, 2007; Harris et al., 2007). Evidence suggests a number of risk factors for cardiovascular disease include alcohol consumption,

obesity, diabetes mellitus, hypertension, high cholesterol level, diet, physical activity, cigarette smoking, age, gender and family history (Mendis, Puska, & Norrving, 2011). Researchers suggest alcohol consumption is a modifiable risk factor for cardiovascular disease, such as hypertension (Carter-Edwards, Godette, White, & Tyson, 2009; Niskanen, et al., 2004; Whelton, et al., 2002). The role of alcohol consumption and diabetes is unclear. Research shows both negative (Ajani, Hennekens, Spelsberg, & Manson, 2000) and positive (Holbrook, Barrett-Connor, Wingard, 1990; Kao, Puddey, Boland, Watson, & Brancati, 2001; Saremi, Hanson, Tulloch-Reid, Williams, & Knowler, 2004) association between alcohol consumption and diabetes.

However, it might be that the amount and pattern of alcohol consumed differs between people in different levels of socioeconomic status and psychosocial factors such as social support and stress may influence alcohol use. Investigations into psychosocial factors including the influence of environmental conditions, social support, stress, and cardiovascular diseases are significantly associated with increased risk of cardiovascular morbidity and mortality (Everson-Rose & Lewis, 2005). Multiple social and psychological influences affect individuals' health. There remains limited empirical research that focuses on the interrelationships among psychosocial factors and other risk factors and how they jointly affect chronic diseases. The association between alcohol consumption, confounding variables that may vary by gender, age and race/ethnicity, and the incidence of some chronic diseases is unknown. This work has been limited largely to European descent samples; it is unclear whether associations between alcohol use, psychosocial factors and chronic disease outcomes specifically high blood pressure and diabetes vary by race/ethnicity. Some of the most important challenges in this area include addressing health disparities and identifying the impact of psychosocial and risk factors of chronic diseases in minority populations. This study allows an exploratory approach of risk

factors with alcohol outcomes between race/ethnicity and chronic diseases, specifically high blood pressure and diabetes. The conceptual approach is informed by the health and social impacts of alcohol outcomes and related health disparities. This study presents a conceptual framework for the mechanisms linking the influences of race/ethnicity and alcohol use to persons diagnosed with a chronic disease, specifically high blood pressure and diabetes. Findings from the review of literature characterizing the association between alcohol use and biopsychosocial and socioecological factors in persons with chronic diseases posited to influence the relation of moderators for health behavior change. The conceptual approach recognizes that alcohol relatedhealth disparities do exist but what types of moderators and mediators are influential remains limited. Some protective factors such as social support, resiliency, marital status, mental health well-being, religiosity, and drinking norms may mitigate the impact of alcohol use.

Epidemiological evidence shows that African Americans tend to consume lower amounts of alcohol relative to other populations; however, African Americans experience greater alcohol related disorders relative to other populations (Barr, Farrell, Barnes, & Welte, 1993; Godette et al., 2009; Herd 1991; National Institute of Alcohol Abuse and Alcoholism [NIAAA], 2001; Scott et al., 2008). Few studies focus on the role that alcohol use contributes to the development of medical and behavioral outcomes that may influence susceptibility to chronic diseases among minority populations. For instance, research suggest among the 37.5 million persons with substance use disorders or serious psychological distress in the US, 6.0 million had hypertension, 5.3 million had asthma, 2.0 million had diabetes, and 1.4 million had heart disease (Hans, Gfroerer, Batts, & Colliver, 2011). There remains a lack of research to better understand the impact of alcohol on chronic disease and the myriad factors that may interact with alcohol to modify chronic disease risk among racial/ethnic populations.

#### **Problem Statement**

Alcohol use disorders persist to be a public health challenge, specifically across age, race, ethnicity, and sex. There remains a significant gap in the literature that limits the understanding of how African Americans suffer disproportionately from alcohol related problems relative to their low prevalence of alcohol consumption and associated social determinants including socioeconomic and psychosocial factors. Moreover, there are key determinants that may serve as protective factors from alcohol use consumption and chronic diseases among African Americans. Due to the limited evidence regarding race/ethnicity differences in risk and protective factors as it relates to alcohol use disorders across the life span, little is known to inform the development of effective prevention strategies. Few studies have probed the psychosocial factors (i.e. combination of medical, behavioral and/or environmental factors) that likely to underlie these disparities. This study seeks to answer the following research questions:

- How does race/ethnicity and the level of alcohol use effect high blood pressure and diabetes?
- 2. Are there racial/ethnic variations that may interact with alcohol use to modify the risk of high blood pressure and diabetes?
- 3. Are racial/ethnic differences in the relationship between alcohol use, high blood pressure and diabetes specifically related to age, sex, income, education, social support or mental health well-being?

The answers to these questions will help to guide future public health intervention and clinical research to address the burden of risk factors associated with high blood pressure and diabetes among race/ethnic populations. The purpose of this study to investigate the influence of race/ethnicity and alcohol use on high blood pressure and diabetes. The specific aim is to investigate how alcohol use affects high blood pressure and diabetes differences across

race/ethnic variations including African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites. This study includes four null hypotheses:

H0<sub>1</sub>: There are no differences in the relationship between alcohol use and high blood
pressure among African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites.
H0<sub>2</sub>: There are no differences in the relationship between alcohol use and diabetes among
African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites.

H0<sub>3</sub>: There are no differences in the relationship between alcohol use and high blood pressure among African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites when controlling for income, education, sex, age, mental health well-being and social support. H0<sub>4</sub>: There are no differences in the relationship between alcohol use and diabetes among African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites when controlling for income, education, sex, age, mental health well-being and social support.

# **Public Health Significance**

The influence of race/ethnicity and alcohol use on high blood pressure and diabetes is not well understood. In the United States, minority populations such as African Americans and Hispanics experience the health impact of alcohol use disorders disproportionately when compared to the Caucasian population (Cateano, 1997; Chartier & Caetano 2010; Dawson 1998; Mulia, Ye, Zemore, & Greenfield, 2008; Mulia, Ye, Greenfield, & Zemore, 2009). There is limited evidence for how alcohol consumption differentially affects race/ethnic differences of chronic diseases and the need to identify various mechanisms to eliminate alcohol related health disparities. These disparities may be explained by socioeconomic status, geographic location, drinking behaviors, and variations in alcohol metabolizing genes (Chartier, Hesselbrock &

Hesselbrock, 2013; Chartier & Caetano 2011; Kalu et al., 2012; Marshall et al., 2013; Marshall et al., 2014; Scott & Taylor, 2008).

Researchers suggest there may be other factors that may explain alcohol related health disparities besides racial/ethnic differences in alcohol consumption (Chartier, Vaeth & Caetano 2014a; Chartier & Caetano 2010; Herd, 1994; Jones-Webb et al., 1997; Mulia et al., 2009). Furthermore, alcohol related health disparities may be explained by psychosocial factors. Understanding risk and protective factors across race/ethnicity populations could lead to the development of effective alcohol prevention interventions. Accordingly, this research has significant implications for health care professionals to promote a better culturally competent understanding of alcohol use and psychosocial factors impact on chronic diseases which may help with healthy behavior change. This study will advance the public health field in social determinants of alcohol use and chronic diseases specifically high blood pressure and diabetes among race/ethnicity.

# Innovation

This project is innovative in several ways. First, the project seeks to address the underinvestigated area of alcohol use and chronic diseases specifically high blood pressure and diabetes among racial and ethnic populations. Substantive differences exist in alcohol-related consequences across persons of African descent. According to the 2010 US Census, there are 38.9 million (12.3%), African American/Blacks non-Hispanic in the US (Humes, Jones, & Ramirez, 2011) Moreover, African Americans are disproportionately experiencing health disparities of chronic disease with high incidence and prevalence rates (CDC,2012). The growth of chronic diseases remains an epidemic public health problem worldwide. While a number of studies have examined alcohol use and cardiovascular disease, few have expanded their scope of

inquiry to examine the level of alcohol use effect between high blood pressure and diabetes across race/ethnicity controlling for various confounding factors. Second, the project will explore this complex interaction of alcohol use as a risk factor with chronic diseases such as high blood pressure and diabetes and psychosocial factors to make the study of this important area significant for health disparities research. Third, the project will examine the current perspectives of the influence of race/ethnicity and alcohol use in high blood pressure and diabetes with a goal to shape future interventions. Furthermore, this project will provide the foundation for future health and behavioral prevention of chronic diseases and alcohol use in public health. Due to the limited evidence regarding race/ethnicity differences in risk and protective factors as it relates to alcohol use disorders across the life span, little is known to inform the development of effective prevention strategies. The proposed study will help with public health interventions to prevent alcohol related health disparities by addressing these risk factors across race/ethnic variations and future development of policy, merging public health, behavioral and medical systems, and implementation of brief intervention addressing alcohol use and chronic diseases for health care professionals. This research serves as an important step in the direction to eliminate alcohol related health disparities.

#### **CHAPTER II**

#### **REVIEW OF LITERATURE**

The purpose of this chapter is to review and assess the existing literature on health disparities of alcohol use on high blood pressure and diabetes. The scope of this review includes the link between alcohol use and cardiovascular outcomes including cardiovascular disease, high blood pressure, diabetes and psychosocial factors (i.e. social support, mental health well-being). The purpose of this study is to investigate the influence of race/ethnicity and alcohol use on high blood pressure and diabetes. The literature review will focus on the main specific aim of how alcohol use affects high blood pressure and diabetes differences across African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites populations. Secondly, the review will assess the role of social-contextual factors and other variables related to alcohol use outcome and health disparities. Thirdly, the review will evaluate the biopsychosocial interactions of alcohol use on high blood pressure and diabetes. Lastly, the review will summarize the social ecological theoretical framework which may contribute to the improvement of current strategies and evaluations to alcohol use related health disparities and chronic diseases specifically high blood pressure and diabetes. Overall, a foundation of knowledge will be provided to identify the gap in literature regarding disproportionate rates of morbidity and mortality in response to stress related health conditions such as diabetes, cardiovascular disease, high blood pressure and alcohol related health disparities. Findings should inform intervention development for prevention on the individual and community levels to eliminate health disparities in the impact of chronic diseases and alcohol use.

#### **Chronic Diseases and Health Disparities**

According to the Centers for Disease Control and Prevention (CDC, 2012), chronic diseases such as diabetes, obesity, hypertension, substance abuse, heart disease, cancer and depression are now responsible for over 75% of health care costs in the US. Nearly one in every two adults is affected by at least one chronic disease (CDC, 2012). Furthermore, minority race/ethnic populations in the US bear an even higher burden and prevalence of morbidity and mortality for chronic diseases. For instance, African American adults are twice as likely than White adults to have been diagnosed with diabetes by a physician (OMH, 2013). In addition, African Americans are 1.4 times more likely to have high blood pressure compared to Whites (OMH, 2013). Moreover, the root causes especially affecting urban communities and racial/ethnic populations are the higher rates of chronic diseases (Frist, 2005). Providing prevention and intervention services to reduce the prevalence and incidence of chronic diseases can impact the United States' health care costs and budget tremendously (DeVoe, Tillotsom, Lesko, Wallace, & Angier, 2011). The CDC (2012) suggests health disparities reflect gaps in the quality of health and health care that mirror differences in socioeconomic status, racial and ethnic background, and education level. These disparities may stem from many factors, including accessibility of health care, increased risk of disease from occupational exposure, and increased risk of disease from underlying genetic, ethnic, or familial factors (CDC,2012).

People will experience significantly diverse levels of health. Historically, health disparities have been documented since the early 20<sup>th</sup> century in the United States (LaViest, 2005; Williams & Sternthal, 2010). Most health disparities includes shorter life expectancy resulting from higher rates of cancer, cardiovascular disease, diabetes, infant mortality, sexually transmitted diseases, HIV/AIDS, immunizations, or mental health problems (CDC, 2012). Some

determinant factors that may contribute to health disparities include environmental conditions, access and quality of care, health behaviors, racial/ethnic discrimination, mental health wellbeing, stress, geographic location, economic, gender, education, age, and income.

Health disparities are a social injustice and economic issue that impact all race/ethnic populations. The economic impact of health disparities can affect the individual and the community including lost income and labor productivity and lack of access and lower quality care (LaViest, 2005). Healthy People 2020 acknowledges that there are social determinants of health to address for elimination of health disparities. Social determinants of health are considered the social and economic conditions under which people may live that determines their health status (HealthyPeople, 2020). Social determinants of health can influence an individual as well as communities.

In order to address health problems and eliminate health disparities effectively, health care professionals must examine socio-cultural factors and understand beliefs about health within a context of populations. Planning and implementing interventions that are applicable and acceptable within the cultural framework of populations is very essential. There remain health disparities in general however; there are gaps in the literature to address alcohol related health disparities.

#### **Alcohol Use: A Public Health Problem**

Alcohol use disorders remain a significant risk factor for the global burden of disease (Rehm et al., 2003; World Health Organization [WHO], 2006). The relationship between alcohol use disorders and health outcomes is multidimensional and complex. Alcohol consumption is identified as the fifth most important preventable risk factor for global health (WHO, 2009). According to the National Prevention Strategy, preventing excessive alcohol use is a major priority to reduce the burden of leading causes of morbidity and mortality in the United States. About three in ten US adults drink alcohol at levels that elevate their risk for physical, mental health, and social problems (National Institute on Alcohol Abuse and Alcoholism [NIAAA], 2011). According to the CDC (2011), excessive alcohol use is the third preventable cause of death in the US. Furthermore, excessive alcohol use costs the US about \$185 billion each year for health care and criminal justice expenses (CDC, 2011). For instance, 40-60 % of trauma center patients are under the influence of illicit drugs or alcohol (Substance Abuse and Mental Health Services Administration [SAMSHA], 2009). Ultimately, alcohol use can predispose a population to a myriad of detrimental health and psychosocial outcomes such as cardiovascular disease, stress and depression.

# Alcohol related health disparities

The contribution of alcohol use disorders to health disparities is a growing concern for public health. The National Institutes of Health [NIH], 2012 defines health disparities as differences in the incidence, prevalence, mortality, and burden of diseases and other adverse health conditions that exist among specific population groups in the United States. Multiple factors such as poverty, inadequate access to health care and educational inequalities are influenced by health disparities (Alegria, et al., 2002; Diez Roux, 2012; Wells, Klap, Koike, & Sherbourne, 2001).

According to American Psychiatric Association (1994), alcohol use disorders including alcohol abuse and alcohol dependence are defined as maladaptive patterns of alcohol consumption manifested by symptoms leading to clinically significant impairment or distress. Alcohol abuse is a pattern of drinking that results in harm to one's health and lifestyle such as relationships, school, and work. Moreover, alcohol abuse can lead to alcohol dependence in which an individual continues to consume alcohol despite significant harm. Additionally, alcohol dependence is the presence of physiological symptoms such as withdrawal, cravings and tolerance. Furthermore, alcohol use disorders are diagnosed medical conditions, specifically when a patient's alcohol consumption pattern causes harm or distress (NIAAA, 2011).

During 2013, substantive changes have occurred for substance-related and addictive disorders (American Psychiatric Association [APA], 2013). For instance, according to Diagnostic and Statistical Manual of Mental Disorders (DSM-V), substance use disorder combines the DSM-IV categories of substance abuse and substance dependence into a single disorder measured on a spectrum from mild to severe (APA, 2013). Furthermore, each specific substance is addressed as a separate use disorder (e.g. alcohol use disorder). Previously a diagnosis of alcohol abuse required only one symptom however, now a mild alcohol use disorder in DSM-IV requires two to three symptoms (APA, 2013). For the purpose of this study, the data were collected during the years of 2008 to 2012 and the data analysis will refer to the DSM-IV definitions.

Alcohol use disorders are associated with a wide range of psychosocial and health consequences such as traffic injuries, liver damage, cancers, and depression (Chartier & Caetano, 2011; NIAAA, 2011). The scope of these consequences is attributable to differences in the patterns of alcohol consumption, social determinant factors, and genetic vulnerability. Alcohol use disorders can complicate the assessment and management of many medical and psychiatric conditions (NIAAA, 2011).

Alcohol use remains a major public health problem and risk factor associated with development of diseases. Many risk factors are produced by behaviors that may lead to chronic diseases. Furthermore, part of today's high medical costs, morbidity and mortality rates can be attributed to behavioral risk factors that influence chronic diseases. Because many of these

behavioral risk factors are modifiable and preventable, future research is needed to assess this burden to implement a healthy behavior for the social environment.

#### **Race/Ethnic Disparities of Alcohol Use**

Epidemiologic information about alcohol use disorders is essential to provide evidence for etiological research and prevention. The prevalence of alcohol use and consequences vary between populations. For example, Blacks and Hispanics have higher rates of complete abstinence from alcohol than Caucasians (NIAAA, 2011). Furthermore, Blacks and Hispanics show fewer alcohol use disorders than Caucasians, even though there are high rates of alcohol related health disparities among minorities (NIAAA, 2011). Moreover, there remains a gap in the literature to address the higher rates of alcohol related health disparities between populations that exhibit fewer alcohol use disorders. Contributing factors that may account for the epidemiologic variations are cultural such as perceptions, attitudes, and norms. In the US, minority differences in alcohol use disorders including onset, persistence, and recurrence vary across gender and age (Grant et al., 2012). Alcohol consumption varies by ethnic populations during adolescence and young adulthood (Cartier & Caetano 2011). Furthermore, research suggests there are strong effects of ethnicity on the development of alcohol consumption behaviors from ages 18 to 37 (Muthen & Muthen, 2000). Research studies confirm when compared to a Caucasian sample, alcohol consumption is lower among Blacks and Asians, similar among Hispanics, and higher among Native Americans (Gilman et al., 2008; Grant 1997). Compared to Caucasians, Blacks and Hispanics are found to be significantly more likely to have persistent DSM-IV alcohol dependence diagnosis (Dawson, 2005; Grant, 1997).

There are many social and cultural factors such as beliefs, norms, attitudes and acculturation that effect alcohol use disorders and related health disparities. Literature confirms

there are numerous factors such as individual, cultural, historical, and environmental characteristics that shape alcohol consumption patterns among minorities (Caetano, Clark, & Tam 1998; Chartier & Catetano 2010; Mulia et al., 2008; Romley et al., 2007). Furthermore, environmental factors such as neighborhood levels contribute to alcohol related health disparities among ethnic populations (Freisthler et al., 2003; LaViest, 2011). Research literature reveals there are biological factors that also effect alcohol use disorders and related health disparities. Alcohol metabolizing genes plays a major role with the effect on alcohol consumption behaviors (Enoch 2013; Zakhari 2006). For instance, ADH 1B1\*3 allele exhibits many characteristics including protection against development of alcoholism and birth defects for African Americans (Arfsten et al., 2004; Edenberg et al., 2006; Ehler et al., 2003; Ehlers et al., 2001; Marshall et al., 2014; McCarver et al., 1997; Scott and Taylor 2007). Overall there are many cultural, environmental, and biological factors that influence race/ethnic alcohol related disparities. However, there is a gap of literature that examines psychosocial factors and alcohol use related to health disparities and chronic diseases. With the widespread use of alcohol consumption and chronic diseases, there remains a need of understanding the social ecological framework including micro and macro conditions in this disease burden of health disparities.

#### **Chronic Diseases Relation to Alcohol Use**

Chronic diseases include various diseases and conditions related to alcohol use (Shield, Parry, & Rehm, 2014). For instance, liver diseases have the most evident association with alcohol consumption. However, the association is less clear for other chronic diseases such as high blood pressure, diabetes, HIV/AIDS, cancer, and stroke across race/ethnicity, age and sex. Some challenges faced with understanding the association of alcohol consumption with chronic diseases may include confounding variables (Breslow et al., 2006; Naimi et al., 2005; Smothers

and Bertolucci, 2001), methodological conventions (Fillmore et al., 2006), and the measurement of alcohol (Mukamal et al., 2003; Rehm et al., 2006). Studies of chronic diseases and alcohol use that measures the drinking patterns (ex. quantity, frequency) and average volume are still needed specifically across race/ethnicity, age, and sex.

Alcohol consumption has adverse and chronic health consequences including a wide range of injuries both intentional and unintentional. In contrast, there is substantial evidence of beneficial effects of moderate alcohol use such as for cardiovascular disease (Corrao et al., 2000; Mukamal and Rimm 2001). Moreover, even though beneficial effects of moderate alcohol use have been suggested for ischemic stroke, diabetes, high blood pressure evidence still remains uncertain (Ashley et al., 2000). There remains a gap in the literature addressing whether alcohol uses at a specific level confer any protective effects for chronic diseases such as diabetes, stroke, or high blood pressure and are there any differences across race/ethnicity, sex and age. Still fewer studies investigated psychosocial and physiological measures to represent mediating and moderating factors for cardiovascular risk outcomes and alcohol use.

Despite the US Preventive Services Task Force (2013) recommendation to provide screening and/or counseling for alcohol, tobacco, and illicit drug use, many healthcare professionals face challenges providing early intervention, motivating change of behavior, facilitating access to treatment when appropriate and improving quality of care with at risk users. Overall training in substance use related diagnosis, treatment, and prevention with chronic disease management has been inadequate for physicians (Fiellin et al., 2002). To address the demand of prevention and intervention in various medical and behavioral settings, healthcare professionals must explore the social determinants of health specifically the social and environmental factors that place populations at significant disadvantage with regard to health and disease. For example, solutions may include focusing on education, prevention and awareness

early during childhood, using family health history and genograms, improving health literacy, environmental enhancements, community collaboration in research, and acknowledging historical legacy of discrimination on health.

#### **Cardiovascular Risk Factors**

There are major cardiovascular risk factors including hypertension, cigarette smoking, obesity, physical inactivity, dyslipidemia, diabetes mellitus, stroke, end stage renal disease, chronic kidney disease, metabolic syndrome, age, discrimination and family history. Appropriate lifestyle modifications are strongly recommended for patients. For example, Joint National Committee (JNC) on Prevention, Detection, Evaluation, and Treatment of high blood pressure (2003), seventh report recommends specific public health interventions such as the Dietary Approaches to Stop Hypertension (DASH) which includes decreasing calories, weight reduction, saturated fat, and salt intake, increasing physical activity, moderate alcohol intake of no more than two drinks per day. However, smoking and alcohol cessation should be recommended to improve cardiovascular health. The combination of diabetes and high blood pressure significantly increases the risk of cardiovascular complications and end stage renal disease.

# Link of Alcohol Use and Cardiovascular Diseases

Racial/ethnic disparities in cardiovascular risk outcomes are some of the most widely studied and consequential foundations of health disparities in the US. For example, compared with Whites, African-Americans and Hispanics have higher rates of high blood pressure and diabetes and disproportionately higher rates of complications (Douglas 2005; Lackland and Keil 1996).

There still remain many unknown factors that generate and drive these disparities after adjusting for a wide range of socioeconomic, behavioral and health risk factors.

An inverse association between alcohol consumption and risk of coronary artery disease is well documented (Klatsky 1994; Marmot 1991; Moore 1986; O'Keefe et al., 2007; Rimm et al. 1991). In addition, alcohol consumption has been associated with the increase in high density lipoprotein cholesterol (Rimm et al., 1999). Research suggests the risk of coronary heart disease decreases in a downward linear direction with alcohol consumption up to three drinks a day (Fuchs et al., 1995; Rimm et al., 1991). However, alcohol itself does not necessarily mean there is a cause of lower risk for an association between moderate alcohol use and coronary heart disease. There may be other risk factors that could account for some of the association between alcohol and lower risk of coronary heart disease such as diet, exercise, stress, social support, and smoking. Literature suggests diet influences mortality of coronary heart disease (Huijbregts et al., 1977). Alcohol use varies with dietary habits. Dietary intake has been considered in some studies of alcohol consumption and coronary disease (Colditz et al., 1991; Jones et al. 1982; Thomason et al. 1988). For example, after adjusting for dietary risk factors there was an inverse association found between alcohol and coronary heart disease in the Nurses' Health Study (Stampfer et al., 1988). Moreover, Kleiner and colleagues (2004) found a significant inverse relationship between alcohol consumption and body mass index. However, Grucza and colleagues (2010) found a link between familial alcoholism risks and obesity. As for physical activity, the risk for coronary heart disease is proportionately lower at higher exercise levels (Berlin and Colditz, 1990).

The relationship between alcohol use and cardiovascular risk factors such as high blood pressure, diabetes and heart disease may be modulated by various factors including environmental, psychosocial and genetic factors. There is some evidence that race/ethnic groups

differ in the protective effect of alcohol consumption associated with a reduced risk for cardiovascular diseases (Freiberg et al., 2009; Fuchs et al., 2001, 2004; Kerr et al., 2011; Mukamal et al., 2010; Pletcher et al., 2005; Sempos et al., 2003; Steffens et al., 2006). However, there remains limited data to explain whether the relationship of alcohol use and cardiovascular risk factors differs among subgroups including race/ethnicity and sex (Rimm et al. 1991; Stampfer et al., 1988; Taylor et al., 2009) and adjustment for confounding variables such as stress and social support. In addition, an important limitation of some research studies is that few included race/ethnic groups or failed to emphasize possible differences of racial/ethnicity. Despite the health disparities for the development of diabetes and high blood pressure, little evidence is available to delineate the relationship of alcohol use to these specific chronic diseases across race/ethnic populations, lifespan and sex.

# **High Blood Pressure in High Risk Populations**

According to the CDC (2013), blood pressure is the measurement force against the wall of arteries as the heart pumps blood through the body. High blood pressure is also known as hypertension and can be categorized into two stages, 1) when systolic blood pressure is 140-159 mmHg or diastolic blood pressure is 90-99; 2) when systolic blood pressure is 160 mmHg or higher or diastolic blood pressure is 100 mmHg or higher (CDC, 2013). Furthermore, a doctor will diagnose a persons who has a blood pressure of 140/90 mmHg or higher over time. However, if a person is diagnosed with another chronic disease such as diabetes or chronic kidney disease, then a blood pressure of 130/80 mmHG or higher is considered as high blood pressure (National Heart, Lung, and Blood Institute [NHLB], 2014). Additionally high blood pressure can be identified either as primary or secondary. When high blood pressure has no known cause then it is identified as primary, essential or idiopathic hypertension (National Heart,

Lung, and Blood Institute [NHLB], 2014). In contrast, secondary hypertension is identified when another condition causes high blood pressure (National Heart, Lung, and Blood Institute [NHLB], 2014). High blood pressure usually has no signs or symptoms and also known as the silent deadly killer (NHLB, 2014). Some people only learn that they have high blood pressure after experiencing problems such as stroke, kidney disease, and cardiovascular disease. There are numerous complications of high blood pressure including enlarged heart, narrowing of blood vessels, and loss of vision (NHLB, 2014). The presence or absence of coexisting comorbidities plays a major role with elevation of the level of systolic and/or diastolic blood pressure.

The prevalence of high blood pressure varies with race/ethnicity, sex and age. For instance adults diagnosed with high blood pressure are 23% between the ages of 20 and 74 years, 75% women and 64% men aged 75 and older, and African Americans have the highest prevalence of high blood pressure in the United States at all ages and in both sexes (NHLB, 2014). Furthermore, high blood pressure tends to develop at an earlier age and to be more severe in African Americans compared to other race/ethnic populations. Literature suggests African-Americans exhibit a greater increase in target organ damage compared to other racial/ethnic populations (NHLB, 2014). High blood pressure remains a greater challenge for the African American community than for the rest of the US nation. There are many factors responsible for the disparities in the incidence, prevalence, and detection of high blood pressure. The awareness of risk factors can be useful in the design and development of intervention and prevention programs, and policies targeted within race/ethnic populations. An understanding of the unique sociocultural aspects of race/ethnic populations will improve cardiovascular outcomes and maximize effective prevention.

#### **Diabetes in High Risk Populations**

Diabetes is a growing public health epidemic worldwide. Diabetes is a chronic disease in which the body doesn't make enough insulin or use insulin properly then blood glucose levels are above normal (CDC, 2013). Diabetes is considered a disorder of metabolism in which the way the body uses digested food for energy. Diabetes is categorized into two main types including type 1 also known as juvenile diabetes (when the body doesn't make enough insulin) and type 2 also known as adult-onset diabetes (the body doesn't use insulin properly) (CDC, 2013). Similar to high blood pressure, diabetes also is known as a silent deadly killer (NHLB, 2014). Some symptoms of diabetes include increased thirst, increased hunger, increased urination, unexplained weight loss, fatigue, blurred vision or sores that do not heal (CDC, 2013). There are numerous complications of diabetes including damaged nerves and blood vessels, heart disease, stroke, kidney disease, amputations, and oral diseases. Diabetes is diagnosed by a doctor using blood tests including an A1C ( hemoglobin A1c), fasting plasma glucose (FPG) test , or oral glucose tolerance test (OGTT) (CDC, 2013).

According to CDC (2011), there are about 20.9 million (9.3% Blacks, 9.2% Hispanics, 6.5% Asians, 5.9% Whites) of people diagnosed with diabetes. For Blacks and Hispanics, diabetes remains a public health crisis in prevalence and incidence of cases. Furthermore, diabetes continues to be a physical, social, psychological, and economic burden. Psychosocial assessments can assist in identifying potential risks and concerns for diabetes. Research suggests examining variables such as cultural background and social support network (ex. family, friends, peers) heavily influences health behaviors and diabetes management among African Americans (Leeman, 2008; Jones et al., 2008).

#### **Biopsychosocial Interactions of High Blood Pressure and Diabetes**

Chronic diseases constitute a major health problem worldwide. Health is considered a state of physical, mental and social well-being. This perspective involves the interplay of biological, psychological and social interactions of a person's life which is also known as the biopsychosocial model (Engle, 1980; Schwartz 1982). As for the role of biological factors, this includes the genetic and inherited characteristics. Next, psychological factors include the behavior and mental processes involving motivation, emotion and cognition. Lastly, social factors include the social environment in which people interact and form relationships such as community, culture and society. The challenge is to understand how health is affected by the interplay of these biopsychosocial interactions and to recognize that behavior is complex.

There is a required genetic predisposition and an environmental influence for chronic diseases such as high blood pressure and diabetes. While biological and genetic differences may influence the distribution of high blood pressure and diabetes within a population, socioeconomic and psychosocial factors may have a greater influence resulting in a disproportionate burden of diseases. There is still no identified genetic basis for the excess prevalence of high blood pressure and diabetes in the African-American community. However, there are several risk factors (i.e. lifestyle, environmental) for high blood pressure and diabetes that have been identified with important differences among racial/ethnic populations. Moreover, there remains a gap of unknown risk factors to explore. This implies that it is not genetics and biological influences alone but there may be a gene-environment interaction that is responsible for the higher incidence and prevalence of high blood pressure and diabetes among African American communities. The explanation for racial/ethnic differences in high blood pressure and diabetes probably involves a complex interaction between environmental factors such as diet, stress, discrimination, socioeconomic status or residential segregation. For example, LaViest

and colleagues (2011) found that racial differences in social environments explain a meaningful portion of variance in disparities and when social factors are equalized the racial disparities are minimized. Having high blood pressure and diabetes may lead to an increase susceptibility to mental health problems such as depression, anxiety and stress. Individuals diagnosed with chronic diseases such as diabetes or high blood pressure are more likely to come from families with a history of that particular chronic disease. Supportive aspects of family may play a role in the prevention and management of chronic disease by increasing risk factor awareness. There is a gap in the literature examining the degree of admixture incorporating socioeconomic status, social support, stress, mental health well-being and coping mechanisms related to chronic diseases and alcohol use among racial/ethnic population. Multiple risk factors are undoubtedly involved in the causation of chronic diseases such as high blood pressure and diabetes in populations. However, the independent contributions of some of these risk factors in explaining racial/ethnic differences require clarification and further exploration in order for effective and evidence based prevention programs to be developed and implemented in communities as well address to eliminate health disparities.

#### Link between High Blood Pressure and Alcohol Use

High blood pressure accounts for the greatest portion of disparities in years of lost life (Wong et al., 2002). The mechanisms responsible for the development of chronic diseases, specifically high blood pressure and diabetes, and association of alcohol use consumption and racial/ethnic variations have yet to be fully explained. Many research studies show a positive relation between alcohol consumption and blood pressure (Criqui et al. 1981; Dyer et al. 1977; Elliott et al., 1987; Klatsky et al., 1986; Klatsky 1985; Klatsky et al. 1977; Maheswaran et al., 1991; Potter and Beevers 1984; Saunders et al 1981; Wannamethee et al. 1991). For instance, one study
demonstrated that alcohol has an association with blood pressure independent of body mass index, smoking, sodium and potassium (Marmot et al., 1994). Some prospective studies have shown that the risk of developing high blood pressure increases with increasing alcohol use (Fuchs et al., 2001; Nakanishi et al., 2001). Heavy alcohol use has been clearly associated with increased blood pressure; however the effects of light to moderate alcohol use on blood pressure are still inconsistent (Klatsky et al., 1986; Wakabayshi 2008). Moreover, little is known about the diagnosis of high blood pressure and the association of alcohol use across race/ethnicity, age, and sex (Beulens et al., 2007; Beilin and Puddey 2006). The study of the effects of alcohol use on various levels of blood pressure may therefore help elucidate mechanisms for effective interventions.

#### Link between Diabetes and Alcohol Use

Diabetes is a chronic disease with long term consequences that may include cardiovascular symptoms, amputations, loss of vision and kidney failure (ADA,2008). There are several factors which increase the risk of diabetes including lack of physical activity, obesity, smoking, alcohol use, and family history. With the use of dietary pattern measurements, a protective effect of alcohol on diabetes was found (Imamura et al., 2009). There remains an unclear specific dose-response relationship with alcohol consumption and diabetes. For instance, some literature has suggested a U-shaped relationship (e.g. protective effect) of moderate alcohol consumption and diabetes (Baliunas et al., 2009; Carlsson et al., 2005; Conigrave et al., 2003; Howard et al., 2004; Klatsky 2007; Koppes, et al., 2005; Zilkens, et al., 2003;) which is analogous to previously studies examining cardiovascular diseases (Di Castenuovo et al., 2002; Maclure et al., 1993). Kerr and Ye (2010) found regular lower quantity of alcohol use may be protective against adult onset of diabetes and no protection for hypertension and heart problems. As for increases of alcohol use over a four year time period, Joosten and colleagues (2011) found an association

with lower risk of diabetes among initially rare and light male drinkers. Prospective research studies such as the Atherosclerosis Risk in Communities (ARIC) Study has examine the clinical and subclinical atherosclerotic diseases in four US community based cohort of middle aged 45-64 women and men (Fuchs et al.,2004). Biological, environmental and psychosocial mechanisms may explain the apparently effects of alcohol use on diabetes and high blood pressure. However, confounding factors may be also partly responsible for the effects too. Studies on the interaction between confounding factors leading to diabetes and high blood pressure are necessary. Exploring the relationship between alcohol drinking patterns and the associated risk of chronic diseases across race/ethnicity, age and sex remains a needed area of investigation.

#### Stress, Chronic Diseases and Alcohol Use

The focus on stress in relation to health and mental well-being appreciates the fact that many people remain prosocial in the face of stressing conditions that place various demands on the human body systems which predispose them to chronic diseases. Researchers suggest African Americans experience disproportionate rates of morbidity and mortality in response to stress related health conditions such as diabetes, cardiovascular disease, high blood pressure when compared to Whites (Greenwood et al., 1996; Smedley et al., 2002; Sternthal et al., 2011). Growing evidence links stress to cardiovascular risk outcomes, including high blood pressure, diabetes and heart disease (Dimsdale 2008). Research suggests that increased cardiovascular and blood pressure between race/ethnic groups (Livingston and Marshall, 1990). Researchers suggest that chronic stress is an important determinant of racial/ethnic disparities in health (Sternthal et al., 2011). For instance, Hicken and colleagues (2014), investigated the association between anticipatory stress and hypertension prevalence in Black, Hispanic, and White Adults. This study found that Blacks reported higher levels of stress of racism-related vigilance than both

Whites and Hispanics is associated with hypertension for Blacks. In addition, this study found that stress of racism-related vigilance was positively associated with hypertension prevalence for Blacks but not Whites. Most studies reporting efforts of chronic stressors on cardiovascular disease focus on topics such as job stress, discrimination, or relationships. For instance, the INTERHEART study investigated the relation of chronic stressors to incidence of myocardial infarction while adjusting for gender, age, smoking, and geographic region (Yusuf et al., 2004). This study found participants who reported stress had a greater than 2.1 times of risk for developing myocardial infarction.

Stress is a psychosocial and modifiable risk factor that influences health. Stressors cause adverse effects on the human body. Moreover, exposures to stress are considered very toxic resulting in various responses (i.e. behavioral, physiological) that influence disease. For instance, stressful events influence disease risk by causing negative affective states such as anxiety and depression (McEwen 1998). Furthermore, behavioral changes occurring as coping responses to stressors such alcohol use also influence disease risk. Literature supports a link between psychological stress and disease (ex. depression, cardiovascular disease, HIV/AIDS, cancer) (Cohen et al., 2007). The development of interventions that can reduce stress will modify disease risk. Social ecological including individual and community level approaches have been shown to influence self-efficacy, well-being, resilience to minimize stress and unhealthy behaviors (ex. excessive alcohol use) that increase risk for chronic disease (Woods-Giscombe and Black, 2010). Other theories that explain stress related disparities include the allostatic load (McEwen, 2012). The concept of allostatic load refers to the accumulation of physiological disturbances as a result of repeated stressors in daily life (Geronimus et al., 2006; McEwen 1998; Stewart 2006). Exposure to prolonged psychological risk factors such as stress, depression or anxiety may alter an individual's physiological responses causing morbidity and mortality effects of chronic

diseases. The frequency of stressors increases the adverse health outcomes of chronic diseases such as high blood pressure and diabetes. Duru and colleagues (2012) examined whether allostatic load is associated with Black-White mortality disparities independent of sociobehavioral risk factors. Results suggest Blacks had higher allostatic load scores than Whites and the allostatic load burden partially explained higher mortality among Blacks independent of socioeconomic and health behaviors. The increase in blood pressure in response to stress may contribute to increase cardiovascular risk. Some common causes of stress are life experiences, social status, income, discrimination, relationship breakdown, care giving, ageing, and occupational. There is a large variation between individuals in the reaction to stress.

### **Racism and Discrimination as Risk Factors for Chronic Diseases**

Interpersonal, institutional and structural racial bias may play a role in the central cause of racial and health disparities in the United States. The persistence of racial bias is evident in the health care system of the United States. For instance, Smedley and colleagues (2002) completed the Institute of Medicine Study, Unequal Treatment: Confronting Racial and Ethnic Disparities in Healthcare. Results from this study found that some health care providers were influenced by a patient's race which further created a barrier to access to health care and poorer health outcomes. Access to health care still remains unequal and as a result racial and health disparities persist. For example, the National Healthcare Disparities Report (2007) found that racial disparities in access to health care and health status continued to exhibit gaps between African Americans and Caucasians. In the response to empirical research studies and reports, several initiatives have been issued to address racial and health disparities (i.e. Healthy People and the National Prevention Strategy). Various reasons have been offered to explain the continuation of racial and health disparities including socioeconomic status and health insurance access. Even

when research studies have controlled for confounding factors, racial and health disparities still persist. There remains a gap in the literature to other plausible answers for the continuation of theses disparities that exist. This current study begins to explore and mainly focus on psychosocial factors such as social support and mental health well-being (ex. stress, anxiety, depression) and the influence of race/ethnicity and alcohol use on high blood pressure and diabetes.

Race/ethnicity is viewed as a socio-cultural factor that may denote differential exposure to stressors. Research suggests that racial discrimination is an important risk factor undermining the health of Blacks (Krieger 2012; Lukachko et al., 2014; Williams &Mohammed 2009). Racism may appear in various forms including interpersonal to institutional interactions. Racial discrimination predicts alcohol use, smoking and illicit drug use (Pascoe & Rickman, 2009). Literature suggests African Americans are hyper-responsive to stress (Taherzadeh et al., 2010; Thomas et al., 2006). Stress is significantly associated with blood pressure in the African American population (Brydon et al., 2010; Deter et al., 2006; Spruill 2010). Psychosocial factors such as stress, anxiety or depression are significantly associated with blood pressure in the African American population. The influence of race/ethnicity and alcohol use on high blood pressure and diabetes has rarely focused on issues related to health disparities and psychosocial factors. This study will begin to explore this gap in the literature.

#### Social Support, Chronic Diseases and Alcohol Use

By investigating how psychosocial factors such as a stress moderator for persons diagnosed with chronic diseases may be social support can serve as a buffering function for risky behaviors such as alcohol use is important for public health prevention. Research suggests various forms of social support (e.g. family, friend, support groups, romantic partner) buffer the effect of alcohol use (Jarnecke and South, 2014). Social relationships influence alcohol use. Some studies suggest adolescent alcohol use is associated with poor family social support (Mason and Windle, 2001; Miles et al., 2005) where as positive family social support predicts lower levels of problem behaviors (i.e. substance use) in adulthood (Bailey et al., 2011). Social support is measured by a respondent's self-reported description of the perception that one is cared for by people such as family, friends, coworkers, self-help groups, organizations, churches, and clinics. Literature suggests there are two demographic indicators often used as proxy measures of support including marital status and whether patients were living alone (Frasure-Smith et al., 2000). There are some studies that investigate marital relationship as a primary form of social support for families and the relationship to hypertension (Baker et al., 1999; Tobe et al., 2005). However, just examining marital status may have limiting generalizability for some populations. Therefore, examining whether an adult family member living in the household may address the non-marital form of social support that may not be present however equivalent as a source of strength. Moreover, several studies reveal that coping mechanisms such as religiosity/spirituality, parental influences and perceived social support from family and friends reduce risk factors such as alcohol and illicit drug use, cigarette smoking and risky sexual behavior (Marshal & Chassin 2000; Poulson et al., 1998; Turner-Musa & Lipscomb 2007). For instance, literature show that support received from family and friends increases well-being and life satisfaction (Stice et al., 2004). Some research demonstrates that low social support is associated to hypertension (Dressler & Bindon 2000; Uchino, Cacioppo & Kiecolt-Glaser 1996). Knowing who provides social support to an individual is essential for prevention. Literature findings suggest that cardiovascular health benefits are associated with marriage and cohabiting adults (Baker et al., 1999; Gallo et al., 2003; Gump et al., 2001). Positive and well-adjusted marriages with frequent spousal interactions may play a major role with contributing to health and mental benefits of social

support. Additionally, the role of social support in coping with emotional problems is important. The family has a significant role of influence on the health and mental well-being of its individual members (Brittain, Taylor & Wu 2010; Lelinneth, Barnes, De La Cruz, Williams & Rogers 2006). Understanding the role social support such as family influences as a health promoting unit is important for prevention. Therefore, research and interventions that utilize the importance of social support may target healthy behavior change for persons diagnosed with chronic diseases and consuming alcohol. Although social support is intended to be positive and helpful for individuals, it can sometimes result in negative experiences. The potential for negative health and mental effects from social relationships may increase the risk of chronic diseases. Social relationships have the potential for both health promoting and damaging effects.

## **Social Ecological Framework and Alcohol Use**

Epidemiological research studies have documented variation in patterns of alcohol consumption and differential consequences of alcohol use across race/ethnic populations (Chartier and Caetano, 2010; Mulia et al., 2009). For instance, Blacks and Hispanics are disproportionately disadvantaged by medical and psychosocial problems from alcohol consumption (Chartier, Vaeth, & Caetano 2014a). However, compared to other race/ethnic populations, Whites have a greater risk of alcohol use disorders (Chartier, Vaeth, & Caetano 2014a). Alcohol use is associated with a broad range of adverse medical and psychosocial consequences, both acute (e.g., motor vehicle accidents, violence) and chronic (e.g., liver cirrhosis, heart cardiomyopathy, high blood pressure, diabetes, stroke, cardiovascular disease) (Chartier, Vaeth, & Caetano 2014a; Chartier et al., 2014b). The main scope of these problems is attributable to multilevel factors that may affect contextual differences in individual (e.g. socioeconomic status, age), social (e.g. culture, beliefs, norms, social support, stressors,

policies), biological (e.g., genetic variations, metabolism), physical (e.g. built environment, alcohol outlets) (Chartier et al., 2014b). The concept of a multilevel perspective seems to be better ecological approach for understanding this phenomenon. For instance, Chartier and colleagues (2014b), reviewed and provided a multilevel perspective on factors associated with drinking and alcohol-related problems in racial ethnic/groups. Results included many factors such as biological pathways to alcohol problems, gene x stress interactions, neighborhood disadvantage, stress, and access to alcohol, and alcohol drinking cultures and contexts (Chartier et al., 2014b). These differences are important as they may provide information about cultural, social, or biological differences associated with risk and resiliency. Furthermore, Warnecke and colleagues (2008) emphasized the creation of new paradigms to explore the determinants of health disparities using a multilevel approach of combing population, social and behavioral, clinical and biological theory and methods to address the burden of morbidity and mortality of diseases.

The epidemiological evidence of alcohol-related health disparities remains to be complex based on disproportionate rates of alcohol related medical and psychosocial problems leading to alcohol use disorders. For instance, studies suggest there is a lower prevalence of alcohol use disorders among African Americans and Hispanics (Chartier and Caetano, 2010; Grant et al., 2012) however; there continues to be race/ethnic disparities in morbidity and mortality associated with alcohol use for these populations. There is a need to continue focus on areas of emphasis in research including incidence, prevalence and patterns of alcohol consumptions across and within race/ethnicity.

The health disparities observed for persons with chronic diseases such as diabetes and high blood pressure at different socio-ecological levels may be explained by differences individual level determinants including social support, stress, or mental health well-being. It is

not clear whether the influences of race/ethnicity on alcohol use outcomes observed in the general populations are the same for persons with chronic diseases. To understand why psychosocial factors such as a lack of social support, stress or negative perceived mental health well-being may lead to poor health outcomes of chronic diseases, researcher must identify the pathway through which socio-cultural factors influences race/ethnicity of alcohol use. The pathway which race/ethnicity moderates alcohol use on high blood pressure and diabetes may be direct. For example, social cultural factors of specific race/ethnicity can consume alcohol as coping mechanism, celebrational or health benefit which may help the person experience improved psychological and medical well-being and fewer negative emotions (i.e. stress, anxiety, depression) and more positive emotions (i.e. self-efficacy, optimism). Social support may affect the physical symptoms to adhere to medication regime and healthier lifestyles such as increase physical activity and diet to manage the chronic disease. However, race/ethnicity can moderate effects of alcohol use on high blood pressure and diabetes through intermediate outcomes such as sociodemographic factors (i.e. age, sex, income, education) access to care, social support, and stress that could then affect health or that could contribute to better health outcomes. For example, social support could lead to greater understanding of healthy lifestyle to manage chronic disease and this in turn may facilitate and improve patient mental health wellbeing. By considering both the socio-cultural and individual contexts of persons diagnosed with chronic diseases, the potential for achieving health behavior change and prevention may be successful (Green, Richard & Potvin 1996). Acknowledging the diversity of population health requires identifying social determinants of health such as socioeconomic status (i.e. income, education), social support networks, access to health care, environment and genetic factors to address healthy behavior change. Furthermore, incorporating theoretical frameworks in health

promotion and behavior change will help address the health disparities at the micro and macro levels.

Some of the dominant theoretical models used in public health and health promotion are based on social psychology by emphasizing the role of social and cultural influences in health behavior by adopting an ecological perspective. According to the Social Ecology Framework, behavior is affected by multiple levels of influences including intrapersonal, interpersonal, organizational and community factors (Glanz et. al 2008). Furthermore, social ecology framework involves understanding health problems within the broader context of community than just limiting to individual determinants for intervention and prevention. Bronfenbrenner (1979) was a prominent researcher who utilized the ecological systems theory and considered the individual, community, organization and culture to be nested factors. This model divides factors into four levels including macro, micro, meso and exo, which describes influences such as community and individual to development that occurs in different systems (Glanz et. al 2008). There are many effects from various relationships between and among different levels. Personal beliefs and attitudes are generally most proximal influences on personal behaviors.

Many researchers interested in alcohol use research have as a goal understanding the determinants. Frequently more than one theory is needed to effectively address the issue of health behavior, promotion, and prevention for alcohol use disorders. Theories have been proposed at a variety of levels including individual, interpersonal, group, community, and organizational levels. Furthermore, theories may overlap and should be integrated within the planning stages of the program. Theories represent an attempt to organize empirical observations into a logical order that may have causal influences on alcohol use behavior (Rivers, 1994). Social determinants of health (e.g. role of policy making, social factors, health services, individual behaviors, and biology and genetics) for alcohol use disorders should be

integrated with the planning, promotion, intervention, prevention, and evaluation of programs to maintain a successful healthy behavior change and sustainability. For this study, the social determinants of health represent the following: at the individual level factors such as mental health well-being (ex. stress, depression, anxiety), interpersonal level factors such as social support (ex. marital status, family, friends, people living in the household over 18 years old), organizational level factors such as income and community level factors such as environment. As the recognition of the magnitude of alcohol use disorders continue to increase, theories provide supportive evidence for change to this public health concern. Understanding the mechanisms for early onset of alcohol use such as the risk and protective factors are critical in designing effective prevention programs.

When using theories to explain a phenomenon, constructs are central to theories and understanding any type of behavior. For instance, the social learning theory organizes constructs such as self-efficacy and outcome expectancies as a focus for intervention. Self-efficacy may be associated with behavior change such as alcohol abuse and/or dependence. Glanz and colleagues (2008) have argued that the health professional's ability to apply theories and models of health behavior is one of the most critical skills needed for successful health behavior change and prevention.

During the past decade, there have been increased efforts to develop intervention and prevention programs for substance use disorders that are based on empirical research and theory. Most theories of substance misuse attribute variability in rates of alcohol and/or drugs to situational variables such as cues and emotional circumstances (Bandura, 1977) or individual variables that influence reinforcing and modeling such as outcome expectancies (Leonard and Blane, 1999). A number of theory-based processes appear to protect individuals from initiating

substance use and progressing toward abuse and/or dependence. These processes involve various constructs such as modeling, bonding, and self-efficacy.

Expectations of the effect of alcohol may contribute to the development of alcohol use disorders. Alcohol related expectancies include both positive effects and/or negative effects. Research findings suggest that positive expectancies are related to drinking problems and behaviors (Brown et al., 1987; Hesselbrock et al., 1987). Some studies suggest that there are cognitive factors that play a role in drinking decisions (Goldman et al., 1991; Greenfield, 2009). The value of theory to the understanding of alcohol use disorders is vital for developing effective intervention and prevention programs.

## Conclusions

Research has shown differential effects of alcohol use across race/ethnic populations (Chartier and Cateano 2010; Chartier, Vaeth, & Caetano 2014a). For example, national surveys show race/ethnic variations across epidemiological alcohol frequency and patterns differences associated with greater risk for adverse effects. However, few research investigations have explored the potential variability across race/ethnic populations and the relationship of alcohol use and chronic diseases such as cancers, diabetes, and hypertension. There is limited evidence on how alcohol consumption differentially affects produces race/ethnic variations with persons diagnosed with chronic diseases. Some of the major challenges for previous alcohol research include an important limitation of not including an adequate sample size of race/ethnic groups or failing to emphasize possible differences within the data analyses. Literature has well documented that African Americans consume lower rates of alcohol but experience more alcohol related problems compared to Whites ( Dauber et al., 2009; Horton 2007; Skidmore et al., 2012; Wu et al., 2011). There are multiple factors associated with racial/ethnic disparities in alcohol

outcomes including biological pathways (e.g. genetic variants; alcohol metabolism), social context (e.g. culture, alcohol laws/policies, exposure to stressors), individual (e.g. socioeconomic status), and physical context (i.e. alcohol outlets) (Cartier et al., 2014b). There remain gaps in the literature to explain racial/ethnic variations in alcohol use and cardiovascular risk outcomes. To elucidate alcohol related health disparities, research must focus on the determinants of disparate health and social outcomes across populations. The current study expands the literature on alcohol use and chronic diseases, specifically high blood pressure and diabetes by exploring racial/ethnic variations; the specific aim is to investigate how alcohol use affects high blood pressure and diabetes differences across race/ethnicity. Understanding these complex determinants of alcohol outcomes and chronic diseases for racial/ethnic populations will inform the development of culturally sensitive targeted interventions and improve infrastructure and capacity building in communities to reduce alcohol related health disparities.

The fundamental purpose of public health care is to improve the health of populations. The health of populations is influenced by various factors such as lifestyle, genetics, and environment. Preventative services that foster the implementation of healthy lifestyles demonstrate the greatest potential for the health of populations. In conclusion, researchers should continue to address the challenges to contextual research with the use of theoretical framework to identify specific attributes of constructs public health problems such as alcohol use disorders. The generality of family, peer, and neighborhood social contexts are significantly implicated factors that influences alcohol use disorders. By incorporating theories and constructs into the conceptualization of alcohol consumption patterns and related health disparities, there is the potential for a better understanding and developments of interventions that targets the underlying causes and provide effective behavioral change. National alcohol datasets continues

to demonstrate a richness, representativeness, and large sample size that enable researchers to address alcohol use disorders in various populations such as understudied minorities.

#### **CHAPTER III**

#### METHODOLOGY

The purpose of this chapter is to outline and describe the research strategy for this project. An in-depth explanation will be provided for the selected data source, participants, data collection, measures, study variables and statistical methods. To accomplish the aim of this study, a secondary data analysis using the National Survey of Drug Use and Health ([NSDUH], years 2008- 2012) was conducted to investigate the influence of race/ethnicity and alcohol use on high blood pressure and diabetes. The data set was selected using the Alcohol Epidemiologic Data Directory, June 2012 version that contains a current listing of surveys and other relevant data suitable for epidemiologic research on alcohol (NIAAA, 2012). This directory is created and updated by the Alcohol Epidemiologic Data System (AEDS) that is operated by CSR, under contract to the National Institute on Alcohol Abuse and Alcoholism (NIAAA, 2012). The criteria and process used to select this data set was initially reviewing proposed aims, hypotheses and constructs then assessing data availability including number of completed responses for completed interviews specifically alcohol use (ex. prevalence, incidence, patterns and consequences of regular use, abuse and dependence) and oversampling of African American/Black and Hispanic/Latino populations, and annual data collection. These criteria were selected because they will allow adequate sample size for race/ethnic population and alcohol use to analyze the proposed specific aim and hypotheses.

**Specific Aim:** The specific aim of this study is to investigate how alcohol use affects high blood pressure and diabetes differences across race/ethnic variations, including African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites.

## Hypotheses:

- H0<sub>1</sub>: There are no differences in the relationship between alcohol use and high blood pressure among African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites.
- H0<sub>2</sub>: There are no differences in the relationship between alcohol use and diabetes among African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites.
- H0<sub>3</sub>: There are no differences in the relationship between alcohol use and high blood pressure among African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites when controlling for income, education, sex, age, mental health well-being and social support.
- H0<sub>4</sub>: There are no differences in the relationship between alcohol use and diabetes among African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites when controlling for income, education, sex, age, mental health well-being and social support.

**Research Design:** The research design for this study is a cross-sectional study of a large, nationally representative sample using the NSDUH that combines data from the 2008- 2012 samples.

**Participants:** The NSDUH is designed to provide national and state-level estimates of the use of tobacco products, alcohol, illicit drugs (including non-medical use of prescription drugs) and mental health in individuals aged 12 years and older residing in the United States and the District of Columbia (SAMSHA, 2012). For this study, the analyses are delimited to individuals aged 18 to 50 years and older who fall into the three most frequently reported race/ethnicity categories of

Non-Hispanic Caucasian/White, Non-Hispanic African American/Black and Hispanic/Latino. **Data Source:** The NSDUH is a self-report survey conducted annually in the United States. Prior to the year 2002, the survey was previously titled National Household Surveys on Drug Abuse. There are important methodological differences in the NSDUH studies conducted after 2002 that that result in prevalence estimates that cannot be compared. Some of the methodological differences includes: sample weighting procedures that utilize population data from the 2000 census, new imputation methods for missing variables and the use of incentive payments that has increased the service response rate. The current NSDUH uses a four stage, multivear sampling design. In the first stage, states are geographically partitioned into equal size regions according to population. In the second stage, a sample of census tracts was taken within state sampling regions that had been stratified by socio-economic status and race. These census tracts were then used to form geographic clusters of adjacent census tracts referred to as segments. Segments were then randomized to two-year periods such that half the segments would be resampled within the next two year period. The third stage of sample selection determined the number of dwelling units (any freestanding home, condominium or townhome, apartment building, college dormitory, homeless shelter, or billet on a military base) within each segment to be sampled by the most prevalent age group. Finally, in the fourth stage an interviewer visited each dwelling unit and selected between zero and two persons to complete the survey (Morton et. al., 2013).

**Funding:** The NSDUH is funded by the United States DHHS, SAMSHA, and the Center for Behavioral Health Statistics and Quality (283-2004-00022) (SAMSHA, 2012).

**Data Collection:** Data were collected and prepared for the NSDUH by Research Triangle Institute, Research Triangle Park (RTI), North Carolina. Each respondent received an incentive payment of \$30.00 for completing the interview. There were three modes of computer assisted interviews (CAI), featuring interactive and bilingual data collection: an audio computer-assisted self-interview (ACASI), a computer-assisted personal interview (CAPI) and a computer-assisted self-interview (CASI), implemented to maximize the validity of responses to sensitive questions. Consistency checks, standardization of missing values, and creation of an online analysis version with question checking for out of range codes were completed. To protect the confidentiality of respondents, RTI employed a patented statistical disclosure limitation method called MASSC that combines amalgamation, probabilistic subsampling and substitution, and optimal sample weight calibration to introduce uncertainty into any attempt to identify any individual and their confidential responses (Singh, Yu and Wilson, 2004). The data for years 2008-2012 were downloaded from the SAMHDA (website www.icpsr.umich.edu/icpsrweb/SAMHDA/) and extracted from the interviews to conduct secondary data analysis. No special authorization was required to access the free public data. A second data set was created to merge the 5 years of data and using only proposed variables for data analysis as well as the sample weights and sample design variables needed to correctly estimate variances and standard errors.

**Institutional Review Board:** The Kent State University Institutional Review Board approved this study protocol (IRB Protocol #13-555)

**Measures:** The NSDUH primarily measures the prevalence and correlates of drug use in the United States. Information is provided on the use of alcohol, tobacco and illicit drugs of people aged 12 years and older in US households. Survey questions include substance abuse treatment history, perceived need for treatment, Diagnostic and Statistical Manual (DSM) of Mental Disorders defined substance abuse and mental health related disorders, demographic information, health care access and coverage, problems resulting from the use of drugs, illegal activities,

social environment, social support, attitudes and beliefs (SAMSHA, 2012). See below for specific items:

#### **Study Variables**

The study variables used in this study includes demographic characteristics, chronic disease, mental health well-being, social support and alcohol use. This section provides a description of how the variables are measured and operationally defined.

*Demographic Characteristics:* Five variables were used to characterize the sample and classified into the following categories: (1) race/ethnicity (Caucasian/White, African American/Black, Hispanic/Latino); (2) sex (male, female); (3) income (less than \$20,000, \$20,000-\$49,999, \$50,000-\$74,999, \$75,000-more); (4) education (less than high school, high school graduate, some college, college graduate); and (5) age (18-25 years old; 26-34 years old; 35-49 years old; 50 or older).

*Chronic Diseases:* High blood pressure is defined as the measurement of force against the wall of arteries as the heart pumps blood through the body (CDC, 2012). High blood pressure can be categorized into two stages, 1) when systolic blood pressure is 140-159 mmHg or diastolic blood pressure is 90-99; 2) when systolic blood pressure is 160 mmHg or higher or diastolic blood pressure is 100 mmHg or higher (CDC, 2012). Diabetes is defined as when the body cannot make enough insulin or use insulin properly then blood glucose levels are above normal (CDC, 2012). The survey question provides the participant a list of health conditions asked the participant to "Please read the list, and type in the numbers of any of these conditions that a doctor or other medical professional has ever told you that you had." On the list, the two questions for the outcomes of interest are "Ever had diabetes" and "Ever had high blood pressure." The responses are limited to yes or no. Therefore, the outcome variables high blood pressure and diabetes are binary variables.

*Mental Health Well-Being:* Mental health well-being is defined as the state of well-being when an individual can cope with the stressors of life (CDC, 2012). The mental health well-being variable assesses if the respondent felt depressed (ex. state of feeling sad), anxious (ex. a state of fear or nervousness characterized by uneasiness) or emotionally stressed (ex. a state of mental tension and worry caused by problems in your life, work, etc. and something that causes strong feelings of worry or anxiety) during a specific time period. The survey question asks the participant, "Now think about the past 12 months. Was there a month in the past 12 months when you felt more depressed, anxious, or emotionally stressed than you felt during the past 30 days?" The response is limited to yes or no.

*Social Support:* Social support is measured by a respondent's self-reported description of the perception that one is cared for by people such as family, friends, coworkers, self-help groups, organizations, churches, and clinics. Published research suggests there are two demographic indicators often used as proxy measures of support including marital status and whether patients were living alone will be used (Frasure-Smith et al., 2000). Two questions will be used to measure social support. The first asks about marital status and the responses are limited to married, widowed (spouse deceased), divorced or separated, and never married. The second survey question asks if the participant has family members that reside in the household aged 18 or older. The responses are limited to yes or no. The two variables will be used as proxy measures for the social support variable and considered as two independent variables for the data analyses.

*Alcohol Use:* Alcohol use is defined as consuming at least one drink of any type of alcoholic beverage, and it excludes people who only take a sip or two from a drink. Alcohol use is measured by the respondent's self- reported consumption of a can or bottle of beer; a wine cooler or a glass of wine, champagne, or sherry; a shot of liquor; or a mixed drink with liquor in it.

Alcohol use is grouped into three categories non-drinkers, regular drinkers, and alcohol use disorders (alcohol abuse and dependence combined). Below are the operational definitions of the categories of alcohol use.

1) No alcohol use for non-drinkers was defined as by the respondent's self- report of no consumption of alcohol.

2) Alcohol use for regular drinkers was defined as no more than 4 drinks on any single day and no more than 14 drinks per week for men; and no more than 3 drinks on any single day and no more than 7 drinks per week for women. The question assessing alcohol use for regular drinkers was, "On the days that you drank during the past 30 days, how many drinks did you usually have?"

3) Alcohol use disorder was determined given that respondents met criteria for both alcohol abuse and alcohol dependence according to DSM-IV criteria. Questions assessing alcohol abuse criteria in the NSDUH included: (1) "Did drinking alcohol cause you to have serious problems like this [neglecting children, missing work or school, doing a poor job at work or school; losing a job or dropping out of school] at home, work, or school?"; (2) "Did you regularly drink alcohol and then do something where being drunk might have put you in physical danger?"; (3) "Did drinking alcohol cause you to do things that repeatedly got you in trouble with the law?"; (4) "Did you have any problems with family or friends that were probably caused by your drinking?" AND "Did you continue to drink alcohol even though you thought it caused problems with family or friends?" All abuse criteria questions were asked with regard to the past 12-month period. Questions assessing dependence criteria for alcohol included: (1) "Was there a month or more when you spent a lot of your time getting or drinking alcohol?" OR "Was there a month or more when you spent a lot of time getting over the effects of the alcohol you drank?"; (2) "Were you able to keep to the limits you set, or did you often drink more than you intended to?"; (3)

"Did you need to drink more alcohol than you used to in order to get the effect you wanted?" OR "Did you notice that drinking the same amount of alcohol/had less effect than it used to?"; (4) "Were you able to cut down or stop drinking alcohol?"; (5) "Did you continue to drink alcohol even though you thought it was causing you to have problems with your emotions, nerves, or mental health? "OR "Did you continue to drink alcohol even though you thought it was causing you to have physical problems?"; (6) "Did drinking alcohol cause you to give up or spend less time doing these types [working, going to school, taking care of children, etc.] of important activities?" An additional withdrawal criteria question was included to assess alcohol dependence: "Did you have 2 or more of these symptoms [sweating or feeling that your heart was beating fast, having your hands tremble, having trouble sleeping, vomiting or feeling nauseous, seeing, hearing, or feeling things that weren't really there, feeling like you couldn't sit still, feeling anxious, having seizures or fits] the same time that lasted for longer than a day after you cut back or stopped drinking alcohol?" All dependence criteria questions were asked regarding the past 12-month period. NSDUH classified individuals responding "yes" to at least one out of four abuse criteria or "yes" to at least three out of the seven dependence criteria as having a past year alcohol according to Alcohol Use Disorders Identification Test (AUDIT) (Saunders et al., 1993; Bohn et al., 1995).

## **Data Analyses:**

To test the hypothesis of the study, a number of statistical procedures were conducted using SAS 9.3 (Cary, NC). A key consideration in the analysis of the data was the complex sampling procedures used to collect the NSDUH data. To appropriately weigh respondents to reflect the population of the United States and to correctly calculate p-values and standard errors, SAS survey analysis procedures SURVEYFREQ and SURVEYLOGISTIC were employed in

conjunction with sampling weights (divided by the number of years, five), stratum and replicate variables provided in the NSDUH to account for the multistage survey design (SAS Institute, 2011). In addition, this study utilizes a subpopulation of the United States who are identified as falling into one of three race/ethnicity categories (Caucasian/White, African-American/Black and Hispanic/Latino) and aged 18 or greater, as noted earlier in the participants section. To correctly calculate p-values and standard errors, a "domain analysis," in which the subpopulation is analyzed within the sample population, was conducted using the TABLES and DOMAIN statements in SURVEYFREQ and SURVEYLOGISTIC procedures.

The data analysis was completed in four steps. The first step consisted of data screening in which cross tabulations of the raw unweighted data was conducted in order to assure that for each combination of independent variable and dependent variable, there are at least five subjects per cell, which would permit more advanced statistical analysis. In the second step, weighted frequencies for the independent variables was calculated by the three categories of race/ethnicity in order to understand the distribution of the other independent variables across the three groups in the United States. The third step of the data analysis tested the first and second hypothesis that the relationship between alcohol use and the outcome variables of high blood pressure and diabetes does not change across racial/ethnic groups. It was accomplished by creating two weighted logistic regression models in which the outcome variables were regressed on the independent variables of the categories of alcohol use (non-drinkers, regular drinkers and alcohol use disorders), race/ethnicity (Caucasian/White, African-American/Black and Hispanic/Latino) and an interaction between alcohol use and race/ethnicity as stated in the following formula:

logit (outcome) =  $\beta_1$  alcohol use +  $\beta_2$  race/ethnicity +  $\beta_3$  alcohol use\*race/ethnicity The hypothesis was tested by determining if the interaction coefficient  $\beta_3$ , was statistically significant at a criteria of p < 0.05. If the null hypothesis was rejected, then comparisons of the

odds of the outcome across the three alcohol use categories within each race/ethnicity was conducted by calculating odds ratios with 95% confidence intervals. To control for the inflation of type I error that comes from multiple comparisons, p-values and 95% confidence intervals were adjusted using a simulation method that estimates the true p-value from a simulated multivariate t distribution (Edwards and Barry, 1987).

The fourth step in the data analysis was conducted to test the second and third hypothesis that the relationship between alcohol use and the outcome variables of high blood pressure and diabetes does not change across racial/ethnic groups when controlling for potential confounding variables of income, education, sex, age, mental health well-being and social support. This was accomplished with two weighted multivariable logistic regression models in which the outcome variables were regressed on the independent variables of alcohol use, race/ethnicity, an interaction between alcohol use and race/ethnicity, and the potential confounding variables listed above as stated in the following formula:

logit (outcome) =  $\beta_1$  alcohol use +  $\beta_2$  race/ethnicity +  $\beta_3$  alcohol use\*race/ethnicity +  $\beta_4$ sex +  $\beta_5$  age +  $\beta_6$  income +  $\beta_7$  education +  $\beta_8$  mental health well-being +  $\beta_9$  marital status +  $\beta_{10}$  family >18 in household

As in the third step, the two hypotheses were tested by determining if the interaction coefficient  $\beta_3$  was statistically significant, and if the null hypothesis is rejected, odds ratios with 95% confidence intervals comparing the odds of the outcome across the three alcohol use categories within each race/ethnicity was conducted. Odds ratios, 95% confidence intervals and p-values for covariates are also calculated and reported. To create more parsimonious models, covariates which had p-values greater than 0.157 were eliminated from the models to create a "reduced" model.

In addition to these four analysis steps, a fifth step was conducted to determine if time in which the survey data have been collected has an impact on the interaction term that tests the hypothesis of differing relationships of alcohol use on the outcomes across race/ethnicity. This step, multivariable logistic regression models regressing the independent variables, and covariates was created, but with an additional categorical variable of the year (2008, 2009, 2010, 2011, in 2012) in which the survey was conducted. These models also included an interaction term with alcohol use, race/ethnicity and year. If this three-level interaction was statistically significant, then the relationship of alcohol use on the outcomes across race/ethnicity would also change over time.

#### **CHAPTER IV**

#### RESULTS

The purpose of this chapter is to present the completed data analysis results for this study. This chapter will initially review the demographic characteristics of the population derived from the weighted results. The data were weighted to account for the NSDUH (2008-2012) survey's complex sample design developed to measure drug use in the civilian, non-institutionalized population of the United States and the District of Columbia. These weighting procedures improve the external validity of population estimates. Secondly, to test the first and second hypotheses, the interaction between the categories of race/ethnicity and alcohol use on each of the outcome variables of high blood pressure and diabetes was conducted using a Wald Chi-Square test. The Wald Chi-Square test is a Type III test that determines if any of the nine groups formed by the interaction between the categories of race/ethnicity and alcohol use is statistically different from any other category. If the Wald test is statistically significant, multiple comparisons of the odds ratios (with associated 95% Confidence Intervals) comparing alcohol groups within race/ethnicity categories is presented. Thirdly, to test the third and fourth hypotheses, multivariable logistic regression analysis with full and reduced models was conducted using the following covariates (age, sex, education, income, mental well-being, social support). Lastly, a post-hoc data analysis was conducted to rule out the presence of a change in the interaction between race/ethnicity and alcohol use over time.

## Demographics by race/ethnicity

The demographic characteristics of the sample include the unweighted and weighted distributions are listed in Table 1. Of the total 5 years combined sample for NSDUH 2008-2012 there were unweighted (N=172,863) adults used for this study. The unweighted frequency of participants for each year included 2008 N= 34,153; 2009 N= 34,260; 2010 N= 35,358; 2011 N= 35,206; 2012 N= 33,886. In terms of the weighted prevalence across the race/ethnicity categories utilized in the study, the Non-Hispanic White category represented 72.7% of the population, and Non-Hispanic Black/African-American and Hispanic categories represented 12.3% and 15.0% of the population, respectively. The estimated frequency of the distribution of age groups differed across the race/ethnicity categories, with the largest age group in the Non-Hispanic White category being aged 50 or older years old at 47.4% and Non-Hispanic Black/African-American category at 35.8%. However the Hispanic category largest age group was aged 35-49 years old at 31.6%. The Non-Hispanic White category also self-reported a higher percentage of being depressed, anxious, or emotionally stressed during the past 30 days (32.1%) compared to the Non-Hispanic Black/African-American (29.5%) and Hispanic (27.4%) categories. As for marital status, 57.6% of Non-Hispanic White category self-reported being married compared to the Non-Hispanic Black/African-American category at 33.5% and Hispanics category at 49.6%. However, a larger percentage of the Hispanic category (85.0%) self-reported family members in the household over 18 years or older, compared to the Non-Hispanic White category (79.3%) and Non-Hispanic Black/African-American category (73.3%). The distribution of alcohol use among race/ethnic categories showed that the majority of population consumes alcohol. The prevalence of non-drinkers self-reported among Non-Hispanic White category was 26.0%, Non-Hispanic Black/African-American category at 38.2%, and Hispanic category at 37.7%. The prevalence of the alcohol drinkers was larger among Non-Hispanic White category at 66.5% compared to NonHispanic Black/African-American category at 55.2% and Hispanic category at 54.3%. For the alcohol use disorders groups, the Hispanic category (7.8%) self-reported similar to Non-Hispanic White category (7.4%) compared to Non-Hispanic Black/African-American category (6.5%). The self-reported diagnosis of chronic disease for the US population during the years of 2008-2012 was 8.31% for diabetes and 24.3% for high blood pressure for this study. Across the alcohol categories, the non-drinkers (30.18%), drinkers (22.63%), and AUD (16.74%), participants self-reported a diagnosis of high blood pressure (see Table 2). However, across the alcohol categories, the non-drinkers (14.34%), drinkers (6.58%), and AUD (3.37%) participants self-reported a diagnosis of diabetes (see Table 2). Across the race/ethnicity categories, the Non-Hispanic Black/African-American category self-reported the highest percentage of high blood pressure (29.5%) and diabetes (11.5%) diagnoses (see Table 3).

#### Unadjusted interaction between race/ethnicity and alcohol use on high blood pressure

For testing the first hypotheses, the Wald test showed a significant interaction between race/ethnicity and alcohol use (Wald Chi-Square = 11.75; DF=4; p-value 0.0194) for the high blood pressure outcome. The unadjusted multiple comparisons of odds ratios across alcohol and race/ethnicity interaction categories on the outcome of high blood pressure are shown in the first column of Table 4. Specifically, within each race/ethnicity category, the odds ratios and 95% confidence intervals comparing the odds of reporting high blood pressure across alcohol groups are reported. Examining within Non-Hispanic White category across the alcohol groups, the drinkers compared to non-drinkers had a reduced odds for high blood pressure (OR= 0.619; 95% CI= 0.585-0.656). There was also a reduced odds for alcohol use disorders compared to drinkers (OR= 0.708; 95% CI= 0.629-0.798), and; a greater reduction in odds for alcohol use disorders compared to non-drinkers (OR= 0.439; 95% CI= 0.390-0.494) for high blood pressure within the

Non-Hispanic White category. Within the Hispanic category, drinkers compared to non-drinkers had a reduced odds for high blood pressure (OR=0.800; 95% CI 0.681-0.941) that was significant. There was a reduced odds for high blood pressure for within the Hispanic category across alcohol groups of alcohol use disorders compared to drinkers (OR=0.667; 95% CI= 0.510-0.873) and alcohol use disorders compared to non-drinkers (OR= 0.534; 95% CI= 0.383-0.745). Within the Non-Hispanic Black/African American category, the results showed a reduced odds of high blood pressure across all alcohol groups, including the drinkers compared to non-drinkers (OR= 0.681; 95% CI= 0.587-0.777), alcohol use disorders compared to drinkers (OR= 0.453; 95% CI= 0.514-0.863), and alcohol use disorders compared to non-drinkers (OR= 0.453; 95% CI= 0.348-0.590). The model fit statistics were (DF=4; -2Log L = 229915387; AIC = 229915405).

## Unadjusted interaction between race/ethnicity and alcohol use on diabetes

For testing the second hypotheses, the Wald test of effects showed a significant interaction between race/ethnicity and alcohol use (Wald Chi-Square= 12.25; DF=4; p value = .0156) on the outcome of self-reported diabetes. The unadjusted multiple comparisons of odds ratios across alcohol and race/ethnicity interaction categories on the outcome of diabetes are shown in Table 5 in the first column. Investigating within the Non-Hispanic White category across the alcohol groups, there was a reduced odds for the drinkers compared to non-drinkers (OR= 0.416; 95% CI= 0.377-0.458) and alcohol use disorders compared to drinkers (OR= 0.454; 95% CI= 0.342-0.602) for diabetes. In contrast, there a greater reduction in odds for alcohol use disorders compared to non-drinkers (OR=0.189; 95% CI= 0.145-0.245) for diabetes within the Non-Hispanic White category. Within the Hispanic category across the alcohol groups, there was reduced odds for the drinkers compared to non-drinkers (OR= 0.325-0.513) and alcohol use disorders compared to drinkers (OR=0.479; 95% CI= 0.266-0.863). However, there was a reduction in odds for alcohol use disorders compared to non-drinkers (OR= 0.196; 95% CI=0.113-0.340) within the Hispanic category. The Non-Hispanic White and Hispanic categories showed a similar pattern with results for comparing the odds of reporting diabetes across alcohol categories. Within the Non-Hispanic Black/African American category, drinkers compared to non-drinkers (OR= 0.495; 95% CI=0.409-0.600) and alcohol use disorders compared to non-drinkers (OR= 0.354; 95% CI= 0.214-0.586) had a larger reduction in the odds for diabetes than for alcohol use disorders compared to drinkers (OR= 0.715; 95% CI=0.429-1.193). In general, diabetes had an odds ratio closer to one with alcohol use disorders compared to drinkers in the Non-Hispanic Black/African American category. The model fit statistics showed (DF=4; -2Log L = 1020274228; AIC = 10274246).

Overall, the findings for the effect of alcohol use differs across the categories of race/ethnicity with the outcome variables. Compared to non-drinkers, regular drinkers had almost half the odds of high blood pressure and diabetes, whereas alcohol use disorders' participants had smaller odds of self-reported diabetes. Among non-drinkers, the Non-Hispanic Black/African-American category had higher odds of high blood pressure and diabetes than Non-Hispanic White and Hispanic categories, and Non-Hispanic White category had higher odds of high blood pressure than the Hispanic category. For regular drinkers, the Non-Hispanic Black/African-American category had higher odds of high blood pressure and diabetes than Non-Hispanic White and Hispanic categories, and the Non-Hispanic White category also had higher odds of high blood pressure than the Hispanic category. In alcohol use disorders' participants, the Non-Hispanic Blacks/African-American and Non-Hispanic White categories had triple the odds of high blood pressure than the Hispanic category. Non-Hispanic White categories had triple the

higher odds of diabetes than the Hispanic category. These findings are indicative of the statistically significant interaction between race/ethnicity and alcohol categories on high blood pressure and diabetes diagnoses.

#### Adjusted Full Model for High Blood Pressure

The full multivariable logistic regression model containing all covariates on the outcome high blood pressure, presented in the second column of table 4, is statistically significant (Wald chi-square = 16661.52; DF=23; p-value < .0001). For testing the third hypotheses, the Type III analysis of effects showed a significant interaction between race/ethnicity and alcohol use (Wald Chi-Square = 22.23; DF=4; p-value= .0002). Only one covariate, family in the household >18 years old, was not statistically significant (Wald Chi-Square= 2.47; DF=1; p-value = 0.1156). All other variables demonstrated very low p-values (p-value <.0001) so a reduced model was created as discussed below.

## Adjusted Reduced Model for High Blood Pressure

After removing the non-significant variable, the reduced adjusted multivariable logistic regression model containing for the outcome high blood pressure reduced model is statistically significant (Wald chi-square test statistics = 16431.90; DF=22; p-value <.0001) (see the third column of Table 4). For testing the third hypotheses using the high blood pressure reduced model, the Type III analysis of effects showed a significant interaction between race/ethnicity and alcohol use (Wald Chi-Square = 22.06; DF=4; p-value < .0002). Covariates showing statistically significant effects on self-reported high blood pressure include: income (Wald Chi-Square = 16.85; DF=3; p-value = 0.0008), sex (Wald Chi-Square = 44.19; DF=1; p-value = <.0001), age (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 3; p-value= <.0001), education (Wald Chi-Square = 6957.26; DF= 6957.26

Square = 99.82; DF= 3; p-value = <.0001); marital status (Wald Chi-Square = 68.09; DF=3; p-value = <.0001); mental health well-being (Wald Chi-Square= 24.17; DF=1; p-value = <.0001) on high blood pressure. The model fit statistics showed (DF=22; -2Log L = 202255176; AIC = 202255222).

Investigating within the Non-Hispanic White category across the alcohol groups, the significant alcohol groups included drinkers compared to non-drinkers (OR= 0.861; 95% CI= 0.807-0.918) and alcohol use disorders compared to non-drinkers (OR= 1.186; 95% CI= 1.030-1.364) for high blood pressure. In contrast, the alcohol use disorders compared to non-drinkers (OR=1.020; 95% CI= 0.887-1.174) for high blood pressure within the Non-Hispanic White category was non-significant when controlling for covariates. Within the Hispanic category for the adjusted reduced model on high blood pressure, all alcohol group comparisons were nonsignificant when controlling for covariates, with 95% confidence intervals that include 1: the drinkers compared to non-drinkers (OR= 1.116; 95% CI= 0.931-1.337), alcohol use disorders compared to drinkers (OR=0.990; 95% CI= 0.713-1.374) and alcohol use disorders compared to non-drinkers (OR= 1.105; 95% CI=0.754-1.618) within the Hispanic category for high blood pressure. Within the Non-Hispanic Black/African American category, drinkers compared to non-drinkers (OR= 0.972; 95% CI=0.859-1.099) and alcohol use disorders compared to drinkers (OR= 0.784; 95% CI= 0.605-1.016) had a reduced odds for high blood pressure that was nonsignificant. However; within the Non-Hispanic Black/African American category, the alcohol use disorders compared to non drinkers (OR= 0.762; 95% CI=0.582-0.998) was significant. The model fit statistics showed (DF=22; -2Log L = 2002255176; AIC = 202255222).

#### Adjusted Full Model for Diabetes

The adjusted full multivariable logistic regression model containing for the outcome diabetes model (shown in the second column of Table 5) is statistically significant (Wald chi-square = 9995.13; DF=23; p-value <.0001). For testing the fourth hypotheses, the Type III analysis of effects showed a non-significant interaction between race/ethnicity and alcohol use (Wald Chi-Square = 6.05; DF=4; p value= .1951). However the following covariates were statistically significant: income (Wald Chi-Square = 79.59; DF=3; p-value = <.0001), sex (Wald Chi-Square = 53.56; DF=1; p-value = <.0001) , age (Wald Chi-Square = 1965.67; DF= 3; p-value= <.0001), education (Wald Chi-Square = 11.72; DF= 3; p-value= .0084); marital status (Wald Chi-Square = 21.55; DF=3; p-value= <.0001); mental health well-being (Wald Chi-Square = 11.73; DF=1; pvalue= 0.0006), family in the household >18 years old (Wald Chi-Square = 35.19; DF= 1; pvalue= <.0001), race (Wald Chi-Square = 74.60; DF= 2; p-value <.0001), and alcohol categories (Wald Chi-Square = 120.31; DF = 2; p-value= <.0001) on diabetes.

Overall, these results were very different from unadjusted models of the first two hypotheses for both outcome variables across the race/ethnicity categories. First, the addition of covariates substantially reduces the effects of alcohol use on both outcome variables, which can be seen in Tables 4 and 5 as the odds ratios across the race/ethnicity categories are much closer to 1 in the adjusted models compared to the unadjusted models. This was particularly evident for the outcome self-reported high blood pressure. Among Non-Hispanic Whites, higher levels of alcohol use always show a protective effect in the unadjusted model, but in the adjusted model the protective effect remains only for drinkers compared to non-drinkers. Among Hispanics, statistically significant protective effects for the AUD group compared to the drinkers and nondrinkers seen in the unadjusted model disappear in the adjusted model. And among Non-

Hispanic Blacks, the only protective effect that remains is when the AUD group is compared to the nondrinker group. For the outcome self-reported diabetes, protective effects of higher levels of alcohol use remain in effect in the adjusted models, but all the odds ratios move closer to 1, indicating a much smaller effect size.

## Trend Analysis Effect for high blood pressure and diabetes

A post-hoc data analysis was conducted to rule out the presence of a change in the interaction between race/ethnicity and alcohol use over time. Two extended models for each of the dependent variables were completed to examine a trend analysis across the 2008-2012 years of data. Data showed non- significant interaction between race/ethnicity categories, alcohol groups and years on diabetes (p-value =0.0495) and high blood pressure (p-value =0.9437). This date indicated that there is not sufficient evidence to conclude that there is a change in the interaction between race/ethnicity and alcohol use over time for the diabetes outcome and high blood pressure.

	Non-Hispanic Whites		Non-Hispanic Black/ African-Ameri	icans	Hispanics	
Variables	Unweighted n	Weighted	Unweighted n	Weighted n (%)	Unweighted n	Weighted n (%)
Alcohol Use	8	8	8	8 ()	8	8 ()
Non-Drinkers	25687	39955209 (25.95%)	7830	9909750 (38.15%)	9987	11955466 (37.74%)
Drinkers	80352	102376994 (66.51%)	13489	14318954 (55.13%)	16069	17192358 (54.27%)
Alcohol Use Disorder	14153	11582133 (7.52%)	2064	1743672 (6.71%)	3232	2526470 (7.97%)
Age				~ /		· · · · ·
18-25	55863	19771627 (12.84%)	12543	4662532 (17.95%)	16369	6340712 (20.01%)
26-34	16592	21174852 (13.75%)	3381	4544712 (17.49%)	5100	7140906 (22.54%)
35-49	25549	39881420 (25.91%)	4533	7441860 (28.65%)	5416	10013502 (31.61%)
50 or older	22188	73086437 (47.48%)	2926	9323272 (35.89%)	2403	8179174 (25.82%)
Gender						
Male	56767	74346225 (48.30%)	10111	11596487 (44.64%)	13630	15991850 (50.48%)
Female	63425	79568111 (51.69%)	13272	14375889 (55.35%)	15658	15682444 (49.51%)
Income						
Less than \$20,000	25666	21950164 (14.26%)	9112	8199948 (31.57%)	8763	8192541 (25.86%)
\$20,000-\$49,000	38670	47273236 (30.71%)	8527	9446862 (36.37%)	12815	13419091 (42.36%)
\$50,000-\$74,999	21143	28670625 (18.62%)	2753	3746270 (14.42%)	3734	4288294(13.53%)
\$75,000- or more	34713	56020310 (36.39%)	2991	4579296 (17.63%)	3976	5774368 (18.23%)
Education						
Less than high school	14712	15994429 (10.39%)	4381	4709923 (18.13%)	9180	10965096 (34.61%)
High school graduate	38429	47649774 (30.95%)	8850	9073990 (34.93%)	9968	9274083 (29.27%)
Some college	36374	40308219 (26.18%)	7107	7513947 (28.93%)	7270	7066669 (22.31%)
College graduate	30677	49961914 (32.46%)	3045	4674516 (17.99%)	2870	4368446 (13.79%)
Marital Status						
Married	47479	88734170 (57.65%)	4485	8706226 (33.52%)	9769	15713386 (49.60%)
Widowed	3379	10314703 (6.70%)	552	1505262 (5.79%)	399	1118296 (3.53%)
Divorced/Separated	11448	20814453 (13.52%)	2375	4658541 (17.93%)	2379	4252774 (13.42%)
Never married	57886	34051009 (22.12%)	15971	11102348 (42.74%)	16741	10589838 (33.43%)
Family in Household 18 yrs. or o	lder					
No	27981	31968337 (20.77%)	6241	6838200 (26.32%)	4596	4670098 (14.74%)
Yes	92211	121945999 (79.22%)	17142	19134176 (73.67%)	24692	27004195 (85.25)
Mental Health Well-Being						
No	76971	104420488 (67.84%)	15638	18293765 (70.43%)	20109	22972732 (72.52%)
Yes	43221	49493848 (32.15%)	7745	7678612 (29.56%)	9179	8701562 (27.47%)

# **Table 1: Demographic Variables**

## Table 2. Alcohol Categories and Outcome Variables

		Drinker		Alcohol Use Disorder	
No	Yes	No	Yes	No	Yes
36,004	7,500	96,160	13,750	17,656	1,793
43157624 (69.81%)	18662801 (30.18%)	103588011 (77.36%)	30300296 (22.63%)	13197211 (83.25%)	2655063 (16.74%)
39,990	3,514	105,805	4,105	19,089	360
52951187 (85.65%)	8869238 (14.34%)	125076227 (93.41%)	8812080 (6.58%)	15316601 (96.62%)	535673 (3.37%)
	No 36,004 43157624 (69.81%) 39,990 52951187 (85.65%)	Non-Drinker   No Yes   36,004 7,500   43157624 (69.81%) 18662801 (30.18%)   39,990 3,514   52951187 (85.65%) 8869238 (14.34%)	Non-Drinker No   No Yes No   36,004 7,500 96,160   43157624 (69.81%) 18662801 (30.18%) 103588011 (77.36%)   39,990 3,514 105,805   52951187 (85,65%) 8869238 (14.34%) 125076227 (93.41%)	Non-Drinker Drinker   No Yes No Yes   36,004 7,500 96,160 13,750   43157624 (69.81%) 18662801 (30.18%) 103588011 (77.36%) 30300296 (22.63%)   39,990 3,514 105,805 4,105   52951187 (85,65%) 8869238 (14.34%) 125076227 (93.41%) 8812080 (6.58%)	Non-Drinker Drinker   No Yes No Yes No   36,004 7,500 96,160 13,750 17,656   43157624 (69.81%) 18662801 (30.18%) 103588011 (77.36%) 30300296 (22.63%) 13197211 (83.25%)   39,990 3,514 105,805 4,105 19,089   52951187 (85.65%) 8869238 (14.34%) 125076227 (93.41%) 8812080 (6.58%) 15316601 (96.62%)

## Table 3. Race/Ethnicity and Outcome Variables

	Non-Hispanic Whites		Bla	Non-Hispanic ck/African-America	ns	Hispanics
Outcome	No	Yes	No	Yes	No	Yes
High Blood Pressure						
Unweighted n	103,150	17,042	19,493	3,890	27,177	2,111
Weighted n (%)	114199693 (74.19%)	39714642 (25.80%)	18236126 (70.21%)	7736250 (29.78%)	27507026 (86.84%)	4167268 (13.15%)
Diabetes						
Unweighted n	114,867	5,325	22,033	1,350	27,984	1,304
Weighted n (%)	141494554 (91.93%)	12419781 (8.06%)	22931092 (88.29%)	3041284 (11.70%)	29323725 (91.22%)	2755925 (8.70%)
# Table 4. High Blood Pressure Logistic Regression Models

	<b>Unadjusted Interaction Effect</b>	Adjusted Full Model	Adjusted Reduced Model
Variable	OR (95% CI)	OR (95% CI)	OR (95% CI)
Non-Hispanic Whites			
Drinkers compared to Non-Drinkers	0.619 (0.585-0.656)*	0.862 (0.808-0.919)*	0.861 (0.807-0.918)*
AUD compared to Drinkers	0.708 (0.629-0.798)*	1.186 (1.030-1.366)*	1.186 (1.030-1.364)*
AUD compared to Non-Drinkers	0.439 (0.390-0.494)*	1.022 (0.888-1.176)	1.020 (0.887-1.174)
Hispanics			
Drinkers compared to Non-Drinkers	0.8 (0.681-0.941)*	1.117 (0.933-1.337)	1.116 (0.931-1.337)
AUD compared to Drinkers	0.667 (0.510-0.873)*	0.992 (0.716-1.374)	0.990 (0.713-1.374)
AUD compared to Non-Drinkers	0.534 (0.383-0.745)*	1.107 (0.758-1.618)	1.105 (0.754-1.618)
Non-Hispanic Black/African-Americans			
Drinkers compared to Non-Drinkers	0.681 (0.597-0.777)*	0.974 (0.860-1.102)	0.972 (0.859-1.099)
AUD compared to Drinkers	0.666 (0.514-0.863)*	0.782 (0.604-1.012)	0.784 (0.605-1.016)
AUD compared to Non-Drinkers	0.453 (0.348-0.590)*	0.761 (0.581-0.997)*	0.762 (0.582-0.998)*
Age		<b>D</b> (	<b>D</b> (
18-25		Ref	Ref
26-34		2.199 (1.981-2.441)	2.181 (1.966-2.419)
35-49		5.088 (4.714-5.492)*	5.036 (4.661-5.440)*
50 or older		15.583 (14.362-16.909)*	15.390 (14.209-16.671)*
Gender		Def	Daf
Male Formala		Kel 0.820 (0.702 0.880)*	
Income		0.839 (0.793-0.889)*	0.848 (0.808-0.890)*
Less than \$20,000		1.023 (0.957-1.093)	1.011 (0.947-1.080)
\$20,000-\$49,000		Ref	Ref
\$50,000-\$74,999		0.948 (0.882-1.020)	0.952 (0.886-1.023)
\$75,000- or more		0.854 (0.790-0.924)*	0.860 (0.796-0.929)*
Education			
Less than high school		Ref	Ref
High school graduate		1.166 (1.079-1.260)*	1.161 (1.075-1.254)*
Some college		1.147 (1.055-1.247)*	1.140 (1.049-1.238)*
College graduate		0.912 (0.824-1.010)	0.903 (0.818-0.997)*
Marital Status			
Married		1.062 (0.984-1.146)	1.091 (1.018-1.169)*
Widowed		1.482 (1.331-1.651)*	1.474 (1.323-1.643)*
Divorced/Separated		Ref	Ref
Never married		0.913 (0.846-0.984)*	0.916 (0.849-0.987)*
Family in Household 18 yrs. or older			
No		Ref	
Yes		1.061 (0.986-1.143)	removed
Mental Health Well-Being		D.C.	D.C
No		Ref	Ret
Yes		1.142 (1.083-1.203)*	1.141 (1.082-1.202)*
FIT STATISTICS	4	22	22
DF	4	23	22
-2 Log L	229915387	202243972	202255176
AIC	229915405	202243924	202255222

Note: Ref= Reference Group; OR= Odds Ratios; CI=Confidence Intervals; DF= Degrees of Freedom; AIC= Akaike Information Criterion; -2Log L= Negative Two Log Likelihood AUD= Alcohol Use Disorder; \* The OR is significant at the .05 level

# Table 5. Diabetes Logistic Regression Models

Variable         OR (95% CL)         OR (95% CL)           Non-Hispanic Whites         0.416 (0.377-0.458)*         0.597 (0.539-0.662)*           AUD compared to Non-Drinkers         0.454 (0.342-0.602)*         0.687 (0.512-0.921)*           AUD compared to Non-Drinkers         0.189 (0.145-0.245)*         0.410 (0.310-0.544)*           Hispanics         Drinkers compared to Non-Drinkers         0.408 (0.325-0.513)*         0.586 (0.458-0.749)*           AUD compared to Non-Drinkers         0.479 (0.266-0.863)*         0.665 (0.338-1.309)         AUD compared to Non-Drinkers           AUD compared to Non-Drinkers         0.495 (0.409-0.600)*         0.718 (0.589-0.876)*         AUD compared to Non-Drinkers           AUD compared to Non-Drinkers         0.495 (0.409-0.600)*         0.718 (0.589-0.876)*         AUD compared to Non-Drinkers         0.495 (0.409-0.600)*         0.718 (0.589-0.876)*           AUD compared to Non-Drinkers         0.495 (0.409-0.600)*         0.718 (0.589-0.876)*         AUD compared to Non-Drinkers         0.495 (0.409-0.600)*         0.718 (0.589-0.876)*           AUD compared to Non-Drinkers         0.495 (0.409-0.600)*         0.718 (0.589-0.876)*         AUD compared to Non-Drinkers         0.354 (0.214-0.586)*         0.566 (0.354-0.903)*           Age         I8-25         Ref         Iso 255 (4.571-6.040)*         Iso 255 (4.571-6.040)*         Iso 255 (4.571-6.040)* <th></th> <th><b>Unadjusted Interaction Effect</b></th> <th>Adjusted Full Model</th>		<b>Unadjusted Interaction Effect</b>	Adjusted Full Model
Non-Hispanic Whites         0.416 (0.377-0.458)*         0.597 (0.539-0.662)*           Drinkers compared to Non-Drinkers         0.454 (0.342-0.602)*         0.687 (0.512-0.921)*           AUD compared to Non-Drinkers         0.189 (0.145-0.245)*         0.410 (0.310-0.544)*           Hispanics              Drinkers compared to Non-Drinkers         0.408 (0.325-0.513)*         0.586 (0.458-0.749)*           AUD compared to Drinkers         0.470 (0.266-0.863)*         0.586 (0.338-1.309)           AUD compared to Non-Drinkers         0.495 (0.409-0.600)*         0.718 (0.589-0.876)*           AUD compared to Non-Drinkers         0.495 (0.409-0.600)*         0.586 (0.354-0.903)*           Age          2.143 (1.827-2.514)         5.255 (4.571-6.040)*           Age          2.143 (1.827-2.514)         5.255 (4.571-6.040)*           Gender         Ref         1.5275 (1.3.212-17.660)*         Ref	Variable	OR (95% CL)	OR (95% CL)
Drikers compared to Non-Drinkers         0.416 (0.377-0.458)*         0.557 (0.530-0.62)*           AUD compared to Drinkers         0.454 (0.342-0.602)*         0.687 (0.512-0.921)*           AUD compared to Non-Drinkers         0.498 (0.325-0.513)*         0.586 (0.458-0.749)*           Hispanics         0.498 (0.325-0.513)*         0.586 (0.458-0.749)*           AUD compared to Non-Drinkers         0.498 (0.325-0.513)*         0.586 (0.458-0.749)*           AUD compared to Non-Drinkers         0.498 (0.130-0.340)*         0.390 (0.208-0.732)*           Non-Hispanic Black/African-Americans         0.196 (0.113-0.340)*         0.390 (0.208-0.732)*           Non-Hispanic Black/African-Americans         0.495 (0.429-1.193)         0.787 (0.481-1.289)           AUD compared to Non-Drinkers         0.354 (0.214-0.586)*         0.566 (0.354-0.03)*           Age         26-34         2.143 (1.827-2.514)           35-49         5.255 (4.571-6.040)*         5275 (13.212-17.660)*           Gender         Ref         866 (0.761-0.854)*           Male         Ref         5275 (13.212-17.660)*           S50,000-S74,090         Ref         530,000-S74,099           S75,000- or more         0.520 (0.583-0.729)*         0.511 (0.828-1.059)           S75,000- or more         0.520 (0.583-0.729)*         0.520 (0.583-0.729)*	Non-Hispanic Whites		
AUD compared to Drinkers     0.454 (0.342-0.602)*     0.687 (0.512-0.921)*       AUD compared to Non-Drinkers     0.189 (0.145-0.245)*     0.410 (0.310-0.544)*       Hispanics     0.408 (0.325-0.513)*     0.586 (0.458-0.749)*       AUD compared to Drinkers     0.479 (0.266-0.863)*     0.665 (0.338-1.309)       AUD compared to Non-Drinkers     0.495 (0.409-0.600)*     0.718 (0.589-0.876)*       AUD compared to Non-Drinkers     0.495 (0.409-0.600)*     0.778 (0.481-1.289)       AUD compared to Non-Drinkers     0.354 (0.214-0.586)*     0.566 (0.354-0.903)*       Age     Ref     1.412 (0.287-0.514)     1.525 (4.571-0.604)*       Joard     S.255 (4.571-0.604)*     5.255 (4.571-0.604)*       S0 or older     Ref     Ref       Female     0.806 (0.761-0.854)*     1.604)*       Income     Income     Income       Less than \$20,000     Ref     Ref       S50,000-S74,999     0.931 (0.850-1.019)     937 (0.850-1.019)       S75,000- or more     0.911 (0.825-1.006)     Son older       Less than high school     Ref     Net	Drinkers compared to Non-Drinkers	0.416 (0.377-0.458)*	0.597 (0.539-0.662)*
AUD compared to Non-Drinkers       0.189 (0.145-0.245)*       0.410 (0.310-0.544)*         Hispanics       Drinkers compared to Non-Drinkers       0.408 (0.325-0.513)*       0.586 (0.458-0.749)*         AUD compared to Drinkers       0.479 (0.266-0.863)*       0.665 (0.338-1.309)         AUD compared to Non-Drinkers       0.196 (0.113-0.340)*       0.390 (0.208-0.732)*         Non-Hispanic Black/African-Americans       0.495 (0.409-0.600)*       0.718 (0.589-0.876)*         Drinkers compared to Non-Drinkers       0.715 (0.429-1.193)       0.787 (0.481-1.289)         AUD compared to Non-Drinkers       0.354 (0.214-0.586)*       0.566 (0.354-0.903)*         Age       Ref       1.43 (1.827-2.514)         52-54       525 (4.571-6.040)*       5255 (1.571-6.040)*         50 or older       Ref       826         Male       Ref       826         Kernale       0.806 (0.761-0.854)*       1.600*         S0.000-574.999       0.331 (0.850-1.019)       5275 (13.212-17.660)*         S75,000- or more       0.652 (0.583-0.729)*       826         Less than S20,000       Ref       1.014 (1.028-1.265)*         S30.000-S74.999       0.331 (0.850-1.019)       575,000- or more       0.652 (0.583-0.729)*         Education       Ref       0.940 (0.826-1.057)       0.940 (	AUD compared to Drinkers	0.454 (0.342-0.602)*	0.687 (0.512-0.921)*
Hispanics       U         Drinkers compared to Don-Drinkers       0.408 (0.325-0.513)*       0.586 (0.458-0.749)*         AUD compared to Drinkers       0.479 (0.266-0.863)*       0.665 (0.338-1.309)         AUD compared to Non-Drinkers       0.190 (0.113-0.340)*       0.390 (0.208-0.732)*         Non-Hispanic Black/African-Americans       0.495 (0.409-0.600)*       0.718 (0.589-0.876)*         Drinkers compared to Non-Drinkers       0.715 (0.429-1.193)       0.787 (0.481-1.289)         AUD compared to Non-Drinkers       0.354 (0.214-0.586)*       0.566 (0.354-0903)*         AUD compared to Non-Drinkers       0.354 (0.214-0.586)*       0.566 (0.354-0903)*         Age       Ref       1.43 (1.827-2.514)       5.255 (4.571-6.040)*         50 or older       5.275 (13.212-17.660)*       5.275 (13.212-17.660)*         Gender       Ref       Ref       8         Kenale       Non-Drinkers       0.391 (0.850-1.019)       5.275 (13.212-17.660)*         S20,000-54.9900       Ref       5.275 (13.212-17.660)*       Ref         S20,000-54.9900       Ref       5.200 (0.581-0.57)       0.391 (0.850-1.019)         \$75,000- or more       0.520 (0.583-0.729)*       0.511 (0.850-1.019)       0.552 (0.583-0.729)*         Educatiot       No       Ref       0.970 (0.862-0.914)*	AUD compared to Non-Drinkers	0.189 (0.145-0.245)*	0.410 (0.310-0.544)*
Drikers compared to Non-Drinkers         0.408 (0.325-0.513)*         0.586 (0.488-0.749)*           AUD compared to Non-Drinkers         0.479 (0.266-0.863)*         0.665 (0.338-1.309)           AUD compared to Non-Drinkers         0.196 (0.113-0.340)*         0.390 (0.208-0.732)*           Non-Hispanic Black/African-Americans         0.475 (0.409-0.600)*         0.718 (0.589-0.876)*           Drinkers compared to Non-Drinkers         0.495 (0.409-0.600)*         0.718 (0.589-0.876)*           AUD compared to Drinkers         0.495 (0.409-0.600)*         0.718 (0.589-0.876)*           AUD compared to Drinkers         0.495 (0.409-0.600)*         0.718 (0.589-0.876)*           AUD compared to Drinkers         0.495 (0.409-0.600)*         0.718 (0.481-1.289)           AUD compared to Drinkers         0.354 (0.214-0.586)*         0.576 (0.481-1.289)           AUD compared to Drinkers         0.354 (0.214-0.586)*         0.525 (4.571-6.040)*           Age         Ref         2.143 (1.827-2.514)         5.525 (1.3212-17.660)*           Gender         No         Ref         1.5275 (13.212-17.660)*           Male         Ref         0.930 (0.671-0.854)*         1.014 (1.028-1.265)*           Status         0.930 (0.800 - 0.701         Ref         0.931 (0.850-1.019)           \$75,000- or more         0.931 (0.851-1.05)         0.931 (0	Hispanics		
AUD compared to Drinkers       0.479 (0.266-0.863)*       0.665 (0.338-1.309)         AUD compared to Non-Drinkers       0.196 (0.113-0.340)*       0.390 (0.208-0.732)*         Non-Hispanic Black/African-Americans       0.495 (0.409-0.600)*       0.718 (0.589-0.876)*         Drinkers compared to Non-Drinkers       0.495 (0.409-0.600)*       0.718 (0.589-0.876)*         AUD compared to Non-Drinkers       0.495 (0.409-0.600)*       0.718 (0.589-0.876)*         AUD compared to Non-Drinkers       0.495 (0.409-0.600)*       0.787 (0.481-1.289)         AUD compared to Non-Drinkers       0.354 (0.214-0.586)*       0.566 (0.354-0.903)*         Age       Ref       1.425       1.43 (1.827-2.514)         35-49       5.255 (1.3.212-17.660)*       5257 (13.212-17.660)*         Gender       Ref       86       1.5275 (13.212-17.660)*         Gender       Ref       86       1.5275 (13.212-17.660)*         S0 or older       1.41(1.028-1.265)*       82.000       82.000-874.99       9.931 (0.850-1.019)       9.755.000-0r more       0.652 (0.583-0.729)*         Education       Ref       1.41(1.028-1.265)*       82.000       82.000       82.000       9.940 (0.836-1.057)       0.652 (0.583-0.729)*         Education       Less than high school       Ref       1.324 (1.201-1.55)*       1.324 (1.201-1.	Drinkers compared to Non-Drinkers	0.408 (0.325-0.513)*	0.586 (0.458-0.749)*
AUD compared to Non-Drinkers       0.196 (0.113-0.340)*       0.390 (0.208-0.732)*         Non-Hispanic Black/African-Americans       0.495 (0.409-0.600)*       0.718 (0.589-0.876)*         Drinkers compared to Non-Drinkers       0.495 (0.409-0.600)*       0.718 (0.589-0.876)*         AUD compared to Non-Drinkers       0.354 (0.214-0.586)*       0.718 (0.827-2.514)         Age       Ref       2.143 (1.827-2.514)         5.255 (d.571-6.040)*       5.255 (1.5212-17.660)*       Gender         Gender       Ref       0.806 (0.761-0.854)*         Income       Ref       8.86       8.86         Kes than \$20,000       Ref       8.80.01       Ref         §20,000-6374,999       0.931 (0.850-1019)       0.552 (0.583-0.729)*       Education         Less than high school       Ref       0.911 (0.825-1.006)       Some college       0.940 (0.836-1.057)         College graduate       0.911 (0.825-1.006)       Some college       0.940 (0.836-1.057)       College graduate       0.941 (0.825-1.06)         No	AUD compared to Drinkers	0.479 (0.266-0.863)*	0.665 (0.338-1.309)
Non-Hispanic Black/African-Americans       0.495 (0.409-0.600)*       0.718 (0.589-0.876)*         AUD compared to Non-Drinkers       0.715 (0.429-1.193)       0.787 (0.481-1.289)         AUD compared to Non-Drinkers       0.354 (0.214-0.586)*       0.566 (0.354-0.903)*         Age       Ref       2.143 (1.827-2.514)       5.255 (4.571-6.040)*         35-49       2.525 (4.571-6.040)*       5.255 (4.571-6.040)*         50 or older       15.275 (13.212-17.660)*         Gender       Ref       Ref         Male       Ref       8.869         Eess than \$20,000       Ref       0.806 (0.761-0.854)*         S20,000-\$49,000       Ref       9.31 (0.850-1.019)         \$75,000- or more       0.931 (0.850-1.019)       \$75,000-0.719,99         \$75,000- or more       0.931 (0.850-1.019)       \$75,000-0.939,90         \$75,000- or more       0.931 (0.852-1.006)       \$60         S0 or older       0.931 (0.850-1.019)       \$75,000-0.939,90         \$75,000- or more       0.940 (0.036-1.057)       \$61         Less than high school       Ref       \$120 (0.83-1.057)         \$75,000- or more       0.941 (0.825-1.006)       \$50         Some college       0.940 (0.036-1.057)       \$120 (0.862-0.914)*         Marited <t< td=""><td>AUD compared to Non-Drinkers</td><td>0.196 (0.113-0.340)*</td><td>0.390 (0.208-0.732)*</td></t<>	AUD compared to Non-Drinkers	0.196 (0.113-0.340)*	0.390 (0.208-0.732)*
Drikers compared to Non-Drinkers         0.495 (0.409-0.600)*         0.718 (0.589-0.876)*           AUD compared to Non-Drinkers         0.715 (0.429-1.193)         0.787 (0.481-1.289)           AUD         0.566 (0.534-0.903)*         0.566 (0.534-0.903)*           Age         Ref         2.143 (1.827-2.514)           35-49         5.255 (4.571-6.040)*         5.255 (4.571-6.040)*           50 or older         15.275 (13.212-17.660)*         600           Gender         Ref         806 (0.761-0.854)*           Income         Ref         806 (0.761-0.854)*           S20,000-S74,990         0.931 (0.850-1.019)         575,600-0.019)           \$75,000-or more         0.931 (0.852-1.005)         600           Education         Ref         806 (0.761-0.857)*           Golder         0.931 (0.850-1.019)         575,000-0 more         0.931 (0.850-1.019)           \$75,000-or more         0.931 (0.850-1.019)         575,000-0 more         0.931 (0.852-1.006)           Gender         0.931 (0.852-1.006)         0.900 (0.832-0.071)         0.900 (0.832-0.071)           Goldege graduate         0.901 (0.832-1.006)         0.900 (0.832-0.071)         0.901 (0.832-1.006)           Marited         1.012 (0.891-1.148)         1.324 (1.120-1.565)*         1.324 (1.120-1.565)*	Non-Hispanic Black/African-Americans		
AUD compared to Drinkers       0.715 (0.429-1.193)       0.787 (0.481-1.289)         AUD compared to Non-Drinkers       0.354 (0.214-0.586)*       0.566 (0.354-0.903)*         Age       Ref         26-34       2.143 (1.827-2.514)         35-49       5.255 (4.571-6.60)*         50 or older       15.275 (13.212-17.660)*         Gender       Ref         Male       Ref         Female       0.806 (0.761-0.854)*         Income       1.141(1.028-1.265)*         \$20,000-\$49,000       Ref         \$20,000-\$49,000       Ref         \$20,000-\$49,000       Ref         \$50,000-\$74,999       0.931 (0.850-1.019)         \$75,000- or more       0.652 (0.583-0.729)*         Education       Ref         Less than high school       Ref         High school graduate       0.940 (0.836-1.057)         College graduate       0.940 (0.836-1.057)         Married       1.012 (0.891-1.148)         Married       1.012 (0.891-1.148)	Drinkers compared to Non-Drinkers	0.495 (0.409-0.600)*	0.718 (0.589-0.876)*
AUD compared to Non-Drinkers     0.354 (0.214-0.586)*     0.566 (0.354-0.903)*       Age     Ref       18-25     Ref       26-34     2.143 (1.827-2.514)       35-49     5.255 (4.571-6.040)*       50 or older     5.255 (4.571-6.040)*       6ender     Ref       Male     Ref       Female     0.806 (0.761-0.854)*       Income     1.141(1.028-1.265)*       \$20,000-\$49,000     Ref       \$50,000-\$74,999     0.931 (0.850-1.019)       \$75,000- or more     0.931 (0.850-1.019)       \$75,000- or more     0.931 (0.825-1.006)       Some college     0.940 (0.836-1.057)       College graduate     0.940 (0.836-1.057)       College graduate     0.940 (0.836-1.057)       College graduate     0.940 (0.836-1.057)       Married     1.012 (0.891-1.148)       Midowed     1.324 (1.120-1.565)*       Married     1.012 (0.891-1.148)       Midowed IS yrs. or older     Ref       No     Ref       Yes     1.349 (1.249-1.556)*       Married     1.012 (0.691-1.168)       Married     1.034 (1.249-1.556)*       Married     1.394 (1.249-1.556)*       No     Ref       Yes     1.162 (1.066-1.266)*       Yes     1.162 (1.066-1.266)* </td <td>AUD compared to Drinkers</td> <td>0.715 (0.429-1.193)</td> <td>0.787 (0.481-1.289)</td>	AUD compared to Drinkers	0.715 (0.429-1.193)	0.787 (0.481-1.289)
Age         Ref           18-25         2.143 (1.827-2.514)           35-49         5.255 (4.571-6.040)*           50 or older         15.275 (13.212-17.660)*           Gender         Ref           Male         Ref           Female         0.806 (0.761-0.854)*           Income         Ref           S50,000-574,990         Ref           \$20,000-549,000         Ref           \$20,000-574,999         0.931 (0.850-1.019)           \$75,000- or more         0.931 (0.850-1.019)           \$75,000- or more         0.931 (0.850-1.019)           \$75,000- or more         0.931 (0.850-1.019)           \$70 college graduate         0.911 (0.825-1.006)           Some college         0.940 (0.836-1.057)           College graduate         0.911 (0.825-1.006)           Some college         0.940 (0.836-1.057)           College graduate         0.911 (0.825-1.006)           Married         1.012 (0.891-1.148)           Widowed         1.324 (1.120-1.565)*           Divorced/Separated         8ef           No         Ref           Yes         0.907 (0.892-0.914)*           Harried         1.92 (0.826.0108)*           No         Ref     <	AUD compared to Non-Drinkers	0.354 (0.214-0.586)*	0.566 (0.354-0.903)*
I8-25         Ref           26-34         2.143 (1.827-2.514)           35-49         2.254 (5.71-6.040)*           50 or older         15.275 (13.212-17.660)*           Gender         Ref           Female         0.806 (0.761-0.854)*           Income         Ref           22,000-549,000         Ref           \$20,000-549,000         Ref           \$75,000- or more         0.931 (0.850-1.019)           \$75,000- or more         0.652 (0.583-0.729)*           Education         Ref           High school graduate         0.931 (0.825-1.019)           Some college graduate         0.940 (0.836-1.057)           College graduate         0.940 (0.836-1.057)           Married         1.012 (0.891-1.148)           Widowed         1.324 (1.120-1.56)*           No         Ref           Yes         1.99 (1.249-1.556)*           Mertial Health Well-B	Age		
26-34       2.143 (1.827-2.514)         35-49       5.255 (4.571-6.040)*         5.255 (4.571-6.040)*       5.255 (1.5212-17.660)*         Gender       Ref         Male       Ref         Female       0.806 (0.761-0.854)*         Income       1.141(1.028-1.265)*         \$20,000-\$49,000       Ref         \$50,000-\$74,999       0.931 (0.850-1.019)         \$75,000 or more       0.931 (0.825-1.006)         \$75,000 or more       0.931 (0.825-1.006)         \$75,000 or more       0.931 (0.825-1.006)         \$76,000 or more       0.931 (0.825-1.006)	18-25		Ref
35-49       5.255 (4.571-6.040)*         50 or older       15.275 (13.212-17.660)*         Gender       Ref         Male       Ref         Female       0.806 (0.761-0.854)*         Income       1.141(1.028-1.265)*         S20,000-S74,999       0.931 (0.850-1.019)         \$75,000- or more       0.652 (0.583-0.729)*         Education       Ref         Less than ligh school       Ref         High school graduate       0.911 (0.825-1.006)         Some college       0.940 (0.836-1.057)         College graduate       0.940 (0.836-1.057)         College graduate       0.940 (0.836-1.057)         Koldowed       1.324 (1.120-1.565)*         Married       1.012 (0.891-1.148)         Widowed       1.324 (1.120-1.565)*         Divorced/Separated       Ref         No       Ref         No       Ref         Yes       1.394 (1.249-1.556)*         Mental Health Well-Being       1.162 (1.066-1.266)*         Fit Statistics       1.162 (1.066-1.266)*         Fit Statistics       1.20274228       109653289         AIC       120274246       109653337	26-34		2.143 (1.827-2.514)
50 or older       15.275 (13.212-17.660)*         Gender       Ref         Male       Ref         Female       0.806 (0.761-0.854)*         Income       1.141(1.028-1.265)*         \$20,000-\$49,000       Ref         \$20,000-\$49,000       Ref         \$50,000-\$74,999       0.931 (0.850-1.019)         \$75,000 or more       0.652 (0.583-0.729)*         Education       Ref         Less than high school       Ref         High school graduate       0.911 (0.825-1.006)         Some college       0.940 (0.836-1.057)         College graduate       0.940 (0.836-1.057)         College graduate       0.940 (0.836-1.057)         Married       1.012 (0.891-1.148)         Married       1.012 (0.891-1.148)         Widowed       1.324 (1.120-1.565)*         Divorced/Separated       Ref         No       Ref         Yes       0.907 (0.809-1.018)         Family in Household       18 yrs. or older         No       Ref         Yes       1.162 (1.066-1.266)*         Fit Statistics       Inference         DF       4       23         -2 Log L       120274228       109653337 <td>35-49</td> <td></td> <td>5.255 (4.571-6.040)*</td>	35-49		5.255 (4.571-6.040)*
Gender         Ref           Female         0.806 (0.761-0.854)*           Income         1.141 (1.028-1.265)*           \$20,000-\$49,000         Ref           \$20,000-\$54,990         0.931 (0.850-1.019)           \$50,000 or more         0.931 (0.850-1.019)           \$75,000 or more         0.911 (0.825-1.006)           Some college         0.911 (0.825-1.006)           Some college         0.911 (0.825-1.006)           Some college         0.940 (0.836-1.057)           College graduate         0.911 (0.825-1.006)           Some college         0.940 (0.836-1.057)           College graduate         0.910 (0.820-9.014)*           Mariced         1.012 (0.891-1.148)           Mariced         1.012 (0.891-1.148)           Widowed         1.324 (1.120-1.565)*           Divorced/Separated         Ref           No         Ref           Yes         1.394 (1.249-1.556)*           Mental H=alth Well-Being         Ref           Yes         1.162 (1.066-1.266)*           Fit Statist=         Intereteeeeeeeee	50 or older		15.275 (13.212-17.660)*
Male         Ref           Female         0.806 (0.761-0.854)*           Income         1.141(1.028-1.265)*           \$20,000-\$379,000         Ref           \$20,000-\$74,999         0.931 (0.850-1.019)           \$75,000- or more         0.652 (0.583-0.729)*           Education         Ref           Less than high school         Ref           High school graduate         0.911 (0.825-1.006)           Some college         0.940 (0.836-1.057)           College graduate         0.940 (0.836-1.057)           College graduate         0.970 (0.682-0.914)*           Martied         1.012 (0.891-1.148)           Widowed         1.324 (1.120-1.565)*           Divorced/Separated         Ref           No         Ref           Yes         1.394 (0.249-1.556)*           Hental Health Well-Being         Ref           Yes         1.394 (1.249-1.556)*           Fit Statistics         Ref           Jose         Ref           Yes         1.394 (1.249-1.556)*           Mental Health Well-Being         Ref           Yes         1.394 (0.21066-1.266)*           Fit Statistics         IDF         4           DF         4         <	Gender		
Female       0.000 (.0.101-0.0534)*         Income       1.141(1.028-1.265)*         \$20,000-\$49,000       Ref         \$50,000-\$74,999       0.931 (0.850-1.019)         \$75,000- or more       0.652 (0.583-0.729)*         Education       Ref         Itigh school graduate       0.911 (0.825-1.006)         Some college       0.910 (0.835-1.006)         College graduate       0.910 (0.825-1.006)         Some college       0.940 (0.836-1.057)         College graduate       0.9940 (0.836-1.057)         College graduate       0.790 (0.682-0.914)*         Martial Status       1.012 (0.891-1.148)         Widowed       1.324 (1.120-1.565)*         Divorced/Separated       Ref         Nowed       1.324 (1.120-1.565)*         Family in Household 18 yrs. or older       Ref         No       Ref         Yes       1.394 (1.249-1.556)*         Mental Health Well-Being       Ref         Yes       Ref         Yes       1.304 (1.260)*         Fit Statistics       IDF         JDF       4       23         -2 Log L       120274228       109653289         AIC       120274264       109653337 </td <td>Male</td> <td></td> <td>Ref</td>	Male		Ref
Income       1.141(1.028-1.265)*         \$20,000-\$349,000       Ref         \$50,000-\$74,999       0.931 (0.850-1.019)         \$75,000- or more       0.652 (0.583-0.729)*         Education       Ref         High school graduate       0.911 (0.825-1.006)         Some college       0.940 (0.836-1.057)         College graduate       0.940 (0.836-1.057)         College graduate       0.990 (0.682-0.914)*         Married       1.012 (0.891-1.148)         Married       1.012 (0.891-1.148)         Married       1.012 (0.891-1.148)         Midowed       1.324 (1.120-1.565)*         Divorced/Separated       Ref         Never married       0.907 (0.809-1.018)         Family in Household 18 yrs. or older       Ref         No       Ref         Yes       1.012 (1.066-1.266)*         Fit Statistics       I1.62 (1.066-1.266)*         Fit Statistics       I1.62 (1.066-1.266)*         DF       4       23         -2 Log L       120274228       109653289         AIC       120274246       109653337	Female		0.806 (0.761-0.854)*
Less than is 20,000       Ref         \$20,000-\$74,999       0,931 (0.850-1.019)         \$75,000-or more       0.652 (0.583-0.729)*         Education       Ref         Less than high school       Ref         High school graduate       0.911 (0.825-1.006)         Some college       0.940 (0.836-1.057)         College graduate       0.940 (0.836-1.057)         Married       1.012 (0.891-1.148)         Married       1.324 (1.120-1.565)*         Micowed       1.324 (1.120-1.565)*         Divorced/Separated       Ref         No       Ref         Yes       1.394 (1.249-1.556)*         Mental Health Well-Being       Ref         Yes       1.162 (1.066-1.266)*         Fit Statistics       120274228         DF       4       23         -2 Log L       120274246       109653337	Loss then \$20,000		1 1/1/1 028 1 265)*
S20,000-S74,999         0.931 (0.850-1.019)           \$50,000-\$74,999         0.931 (0.850-1.019)           \$50,000-\$74,999         0.652 (0.583-0.729)*           Education         Ref           Less than high school         Ref           High school graduate         0.911 (0.825-1.006)           Some college         0.940 (0.836-1.057)           College graduate         0.940 (0.836-1.057)           College graduate         0.790 (0.682-0.914)*           Married         1.012 (0.891-1.148)           Widowed         1.324 (1.120-1.565)*           Divorced/Separated         Ref           Never married         0.907 (0.809-1.018)           Family in Household 18 yrs. or older         Ref           Yes         1.394 (1.249-1.556)*           Mental Health Well-Being         Ref           No         Ref           Yes         1.162 (1.066-1.266)*           Fit Statistics         I           DF         4         23           -2 Log L         120274228         109653289           AIC         120274246         109653337			$P_{\rm of}$
\$350,000-374,979       0.531 (0.530-1.019)         \$75,000- or more       0.652 (0.583-0.729)*         Education       Ref         Less than high school       Ref         High school graduate       0.911 (0.825-1.006)         Some college       0.940 (0.836-1.057)         College graduate       0.907 (0.682-0.914)*         Married       1.012 (0.891-1.148)         Married       1.012 (0.891-1.148)         Widowed       1.324 (1.120-1.565)*         Divorced/Separated       Ref         Never married       0.907 (0.809-1.018)         Family in Household       18 yrs. or older         No       Ref         Yes       1.394 (1.249-1.556)*         Mental Health Well-Being       1.394 (1.249-1.556)*         Yes       1.162 (1.066-1.266)*         Fit Statistics       Image: Statistics         DF       4       23         -2 Log L       120274228       109653327	\$20,000-\$47,000 \$50,000 \$74,000		0.021 (0.850, 1.010)
Education       Ref         Less than high school       Ref         High school graduate       0.911 (0.825-1.006)         Some college       0.940 (0.836-1.057)         College graduate       0.790 (0.682-0.914)*         Martial Status       1.012 (0.891-1.148)         Married       1.012 (0.891-1.148)         Widowed       1.324 (1.120-1.565)*         Divorced/Separated       Ref         Never married       0.907 (0.809-1.018)         Family in Household       18 yrs. or older         No       Ref         Yes       1.394 (1.249-1.556)*         Mental Health Well-Being       Ref         Yes       1.162 (1.066-1.266)*         Fit Statistics       U         DF       4       23         -2 Log L       120274228       109653289         AIC       120274246       109653337	\$30,000-\$77,777		0.931(0.830-1.019) 0.652 (0.583-0.729)*
Less than high school         Ref           High school graduate         0.911 (0.825-1.006)           Some college         0.940 (0.836-1.057)           College graduate         0.790 (0.682-0.914)*           Married         1.012 (0.891-1.148)           Married         1.012 (0.891-1.148)           Widowed         1.324 (1.120-1.565)*           Divorced/Separated         Ref           Never married         0.907 (0.809-1.018)           Family in Household 18 yrs. or older         Ref           No         Ref           Yes         1.394 (1.249-1.556)*           Mental Health Well-Being         Ref           Yes         1.162 (1.066-1.266)*           Fit Statistics         U           DF         4           Q3         23           -2 Log L         120274228           AIC         120274246	Education		0.052 (0.565-0.725)
High school graduate       0.911 (0.825-1.006)         Some college       0.940 (0.836-1.057)         College graduate       0.790 (0.682-0.914)*         Married       1.012 (0.891-1.148)         Widowed       1.324 (1.120-1.565)*         Divorced/Separated       Ref         Never married       0.907 (0.809-1.018)         Family in Household       18 yrs. or older         No       Ref         Yes       1.394 (1.249-1.556)*         Mental Health Well-Being       Ref         Yes       1.162 (1.066-1.266)*         Fit Statistics       4       23         JPF       4       23         -2 Log L       120274228       109653289         AIC       120274246       109653337	Less than high school		Ref
Some college       0.940 (0.836-1.057)         College graduate       0.790 (0.682-0.914)*         Marital Status       1.012 (0.891-1.148)         Married       1.012 (0.891-1.148)         Widowed       1.324 (1.120-1.565)*         Divorced/Separated       Ref         Never married       0.907 (0.809-1.018)         Family in Household       18 yrs. or older         No       Ref         Yes       1.394 (1.249-1.556)*         Mental Health Well-Being       Ref         Yes       1.162 (1.066-1.266)*         Fit Statistics       1.162 (1.066-1.266)*         Fit Statistics       23         AIC       120274228       109653289	High school graduate		0.911 (0.825-1.006)
College graduate       0.790 (0.682-0.914)*         Marital Status       .012 (0.891-1.148)         Married       1.012 (0.891-1.148)         Widowed       1.324 (1.120-1.565)*         Divorced/Separated       Ref         Never married       0.907 (0.809-1.018)         Family in Household       18 yrs. or older         No       Ref         Yes       1.394 (1.249-1.556)*         Mental Health Well-Being	Some college		0.940 (0.836-1.057)
Marital Status       1.012 (0.891-1.148)         Married       1.012 (0.891-1.148)         Widowed       1.324 (1.120-1.565)*         Divorced/Separated       Ref         Never married       0.907 (0.809-1.018)         Family in Household       18 yrs. or older         No       Ref         Yes       1.394 (1.249-1.556)*         Mental Health Well-Being       1.162 (1.066-1.266)*         Fit Statistics       1.162 (1.066-1.266)*         Fit Statistics       4       23         OF       4       23         -2 Log L       120274228       109653289         AIC       120274246       109653337	College graduate		0 790 (0 682-0 914)*
Married       1.012 (0.891-1.148)         Widowed       1.324 (1.120-1.565)*         Divorced/Separated       Ref         Never married       0.907 (0.809-1.018)         Family in Household       18 yrs. or older         No       Ref         Yes       1.394 (1.249-1.556)*         Mental Health Well-Being       Ref         Yes       1.394 (1.249-1.556)*         Fit Statistics       1.162 (1.066-1.266)*         Fit Statistics       23	Marital Status		0.770 (0.002 0.711)
Widowed       1.324 (1.120-1.565)*         Divorced/Separated       Ref         Never married       0.907 (0.809-1.018)         Family in Household       18 yrs. or older         No       Ref         Yes       1.394 (1.249-1.556)*         Mental Health Well-Being       1.394 (1.249-1.556)*         Yes       1.394 (1.249-1.556)*         Mental Health Well-Being       Ref         Yes       1.162 (1.066-1.266)*         Fit Statistics       1.162 (1.066-1.266)*         Fit Statistics       23         AC       120274228       109653289         AIC       120274246       109653337	Married		1.012 (0.891-1.148)
Divorced/Separated         Ref           Never married         0.907 (0.809-1.018)           Family in Household         18 yrs. or older           No         Ref           Yes         1.394 (1.249-1.556)*           Mental Health Well-Being         Ref           Yes         1.162 (1.066-1.266)*           Fit Statistics         1           DF         4         23           -2 Log L         120274228         109653289           AIC         120274246         109653337	Widowed		1.324 (1.120-1.565)*
Never married         0.907 (0.809-1.018)           Family in Household 18 yrs. or older         Ref           No         Ref           Yes         1.394 (1.249-1.556)*           Mental Health Well-Being         Ref           Yes         1.162 (1.066-1.266)*           Fit Statistics         1.162 (1.066-1.266)*           Fit Statistics         1.120274228           AIC         120274246         109653337	Divorced/Separated		Ref
Family in Household 18 yrs. or older         No       Ref         Yes       1.394 (1.249-1.556)*         Mental Health Well-Being       Ref         No       Ref         Yes       1.162 (1.066-1.266)*         Fit Statistics       23         DF       4       23         -2 Log L       120274228       109653289         AIC       120274246       109653337	Never married		0.907 (0.809-1.018)
No       Ref         Yes       1.394 (1.249-1.556)*         Mental Health Well-Being       Ref         No       Ref         Yes       1.162 (1.066-1.266)*         Fit Statistics       23         DF       4       23         -2 Log L       120274228       109653289         AIC       120274246       109653337	Family in Household 18 yrs. or older		
Yes       1.394 (1.249-1.556)*         Mental Health Well-Being       Ref         No       Ref         Yes       1.162 (1.066-1.266)*         Fit Statistics       23         DF       4       23         -2 Log L       120274228       109653289         AIC       120274246       109653337	No		Ref
Mental Health Well-Being         Ref           No         Ref           Yes         1.162 (1.066-1.266)*           Fit Statistics         23           DF         4         23           -2 Log L         120274228         109653289           AIC         120274246         109653337	Yes		1.394 (1.249-1.556)*
No         Ref           Yes         1.162 (1.066-1.266)*           Fit Statistics         23           DF         4         23           -2 Log L         120274228         109653289           AIC         120274246         109653337	Mental Health Well-Being		
Yes         1.162 (1.066-1.266)*           Fit Statistics         23           DF         4         23           -2 Log L         120274228         109653289           AIC         120274246         109653337	No		Ref
Fit Statistics         4         23           DF         4         23           -2 Log L         120274228         109653289           AIC         120274246         109653337	Yes		1.162 (1.066-1.266)*
DF423-2 Log L120274228109653289AIC120274246109653337	Fit Statistics		
-2 Log L120274228109653289AIC120274246109653337	DF	4	23
AIC 120274246 109653337	-2 Log L	120274228	109653289
	AIC	120274246	109653337

Note: Re⊨ Reference Group; OR= Odds Ratios; CI=Confidence Intervals; DF= Degrees of Freedom; AIC= Akaike Information Criterion; -2Log L= Negative Two Log Likelihood AUD= Alcohol Use Disorder; \* The OR is significant at the .05 level

# CHAPTER V DISCUSSION

The purpose of this chapter is to discuss the results regarding the influence of race/ethnicity and alcohol use on high blood pressure and diabetes. This section provides an interpretation of findings and how the results relate to the existing literature on health disparities of alcohol use on high blood pressure and diabetes. A contribution to answering the research questions is addressed and will help guide future public health intervention and clinical research to address the risk factors associated with high blood pressure and diabetes among race/ethnic populations. Future public health implications are provided to inform intervention development for prevention on the individual and community levels to eliminate health disparities in the impact of high blood pressure, diabetes, and alcohol use.

The objective of this study was to investigate the relationship of race/ethnicity and alcohol use on high blood pressure and diabetes. Within this representative sample of US adults, the data show a significant interaction in the role of race/ethnicity differences of alcohol consumption with persons diagnosed with high blood pressure or diabetes. This study rejected the following three null hypotheses: H0<sub>1</sub>: There are no differences in the relationship between alcohol use and high blood pressure among African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites; H0<sub>2</sub>: There are no differences in the relationship between alcohol use and diabetes among African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites; H0<sub>3</sub>: There are no differences in the relationship between alcohol use and high blood pressure among African Americans/Whites; H0<sub>3</sub>: There are no differences in the relationship between alcohol use and high blood pressure among African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites; H0<sub>3</sub>: There are no differences in the relationship between alcohol use and high blood pressure among African Americans/Blacks, Hispanics/Latinos and Caucasians/Whites when controlling for income, education, sex, age, mental health well-being and social support. However, the fourth hypothesis, which specified that there are no differences in the relationship between alcohol use and diabetes among across race/ethnicity categories after controlling for covariates, failed to be rejected.

A substantial body of literature has confirmed findings about the J-shaped relation where low to moderate drinkers are at reduced risk (protective effect) compared to non-drinkers, while alcohol use abuse and/dependence are at an increased risk in cardiovascular disease. However, this confirmation has been mainly found in European descent populations. This study begins to address our understanding of race/ethnicity alcohol–related health disparities. Findings suggest there are racial/ethnic differences between alcohol use and chronic diseases, specifically high blood pressure and diabetes. Previous literature shows inconsistencies of a protective effective among African-American/ Blacks between alcohol use and cardiovascular disease. For example, researchers did not find evidence of a protective effect among Blacks (Sempos et al.,

2003;Mukamal et al., 2010); however, other researchers found a protective effect among Whites (Rehm et al., 1995) and cardiovascular disease. This study is unique because it addresses two of the most common chronic diseases (high blood pressure and diabetes) diagnosed within the minority populations. The selected factors that were controlled were mental health well-being, social support, and demographic characteristics. Differential effects of beverage type, tobacco smoking, use of illegal drugs, and diet were not controlled for in this study because these data were not available given the data set used. Additionally, the differentiated effect of lifetime abstainers and former drinkers within the non-drinker category was not assessed.

A major finding of this study revealed that alcohol use as a risk factor was different across race/ethnicity. Moreover, the prevalence of these risk factors was unique to each group. Results showed significant demographic characteristics between the race/ethnicity categories. For instance, the group showing the highest percentage of drinkers was Non-Hispanic White participants and they also reported experiencing stress, depression or anxiety. In contrast, Non-Hispanic African-American/Black participants reported the highest group of non-drinkers and lowest percentage of experiencing stress, depression or anxiety. Being diagnosed with high blood pressure and diabetes is overall more frequent among minority populations (CDC, 2011). Selfreported diagnoses of high blood pressure and diabetes were observed to be highest among the African-Americans population for this study. These findings are consistent with other literature (OMH, 2009).

The role of socioeconomic and psychosocial factors on high blood pressure and diabetes is noteworthy. Factors such as socioeconomic status (e.g. education and income) could be affecting the race/ethnic differences, alcohol use, and chronic diseases examined in this study. For instance, a person's socioeconomic status may be predictor of chronic diseases (Braveman,

Cubbin, Egerter et al., 2010). The present study showed major disparities with income and education among racial/ethnic categories. For example, Non-Hispanic White group reported the highest levels of education and income. The Hispanic group reported the lowest level of education. These findings are comparable with other published results suggesting social equalities still remain as an epidemic in the US (Healthy People, 2020; CDC, 2012).

# Unadjusted interaction between race/ethnicity and alcohol use on high blood pressure and diabetes

Alcohol continues to be the most widely used substance of abuse in the US (CDC, 2011). Alcohol use has complex physiological, behavioral and social interrelationships. Moreover, the consumption of alcohol may be beneficial or harmful in its influence on physical and mental health outcomes. There are many factors that make people more sensitive to the effects of alcohol. Results showed that the categories of race/ethnicity and alcohol use categories did differ with the outcome variables demonstrating statistically significant interaction between race/ethnicity and alcohol groups on high blood pressure and diabetes diagnoses. The present study findings support how alcohol use is a major risk factor for chronic diseases such as high blood pressure and diabetes (Rubin, 1989). For investigating the unadjusted interaction of differences between race/ethnicity and alcohol use on high blood pressure, the Non-Hispanic White participants showed a J-shaped curve (protective effect) for alcohol use on high blood pressure; however, the Hispanic and Non-Hispanic Black/African-American categories did not. These findings are consistent with other studies that found a positive relationship between alcohol consumption and blood pressure (Criqui et al. 1981; Dyer et al. 1977; Elliott et al., 1987; Klatsky et al., 1986; Klatsky 1985; Klatsky et al. 1977; Maheswaran et al., 1991; Potter and

Beevers 1984; Saunders et al 1981; Wannamethee et al. 1991) and coronary heart disease and Jshaped for alcohol use (Goldberg & Soleas 2001; Howard, Arnsten, & Gourevitch, 2004; Rehm et al., 2010; Roerecke & Rehm 2012) among the Non-Hispanic White population. In contrast, the unadjusted interaction between race/ethnicity and alcohol use on diabetes, the Non-Hispanic White and Hispanic participants showed a similar pattern when comparing the odds of reporting diabetes across alcohol categories. Diabetes had an odds ratio closer to one with alcohol use disorders compared to drinkers in the Non-Hispanic Black/African American category. Potential explanations for these findings may be explained by the biopsychosocial interactions including that biological, psychological and social factors all play a significant role in the context of chronic disease. For instance, the resilience to minimize stressors such as discrimination and racism are important for the moderating effects of race/ethnicity and alcohol use on chronic diseases.

# Adjusted models for high blood pressure and diabetes

This study demonstrated that there are racial/ethnic variations that interact with alcohol use on persons diagnosed with high blood pressure and diabetes. The addition of covariates substantially reduces the effects of alcohol use on both diabetes and high blood pressure. However, in the adjusted full model for high blood pressure the variable "family members in the household over 18 years old" was not significant. This exploratory study results show a statistically significant finding that race/ethnicity moderates alcohol use on high blood pressure while controlling for covariates. Within the Non-Hispanic African American/Black category, the odds of AUD reporting a diagnosis of high blood pressure is lower compared to non-drinkers when controlling for covariates. Within the Hispanic category, there were no differences across the alcohol drinking groups reporting diagnosis of high blood pressure when controlling for covariates. Within the Non-Hispanic White category, data showed the odds of reporting diagnosis of high blood pressure reduced when compared with drinkers and non-drinkers alcohol groups. Furthermore the AUD compared to drinkers showed a harmful effect within the Non-Hispanic White category. These results for the Non-Hispanic White category demonstrated the Jshaped phenomenon and supported literature regarding cardiovascular disease (Goldberg & Soleas 2001; Howard, Arnsten, & Gourevitch, 2004; Rehm et al., 1995 Rehm et al., 2010; Roerecke & Rehm 2012). Results demonstrated that there are differences across race/ethnicity and alcohol use on high blood pressure when controlling for age, education, income, sex, marital status proxy measure of social support, and mental health well-being. As the proportion of race/ethnic populations in the US increases, it is important to understand socio-cultural factors that exist within race/ethnicity and influential variables such as demographic characteristics, social support and mental health well-being for chronic disease prevention intervention programs. There are several explanations to support the data from this study. Various explanations can support data from this study. First of all, the risk factor of alcohol use is thought to be associated with psychosocial factors such as stress and lack of social support. Furthermore the risk factor of alcohol use can be influenced at any ecological level (ex. individual, family, community). Secondly, the protective factor of a positive mental health well-being and increase level of social support are thought to facilitate positive cardiovascular outcomes by operating as a buffering effect between individuals and the consumption of alcohol. Consuming alcohol may increase stress, anxiety, depression and lack of social support which exacerbates cardiovascular outcomes such as high blood pressure and diabetes. The protective influences of mental health well being and increase level of social support may outweigh the negative impact of exposure to

alcohol use across race/ethnic groups. This study illustrated that the risk factor of alcohol use seems to become a cardioprotective factor when used at low to moderate use for some race/ethnic groups when controlling for education, sex, income, marital status, and mental health well-being when diagnosed with high blood pressure. The development of sustainable preventive interventions that have the potential to impact social-cultural factors across race/ethnicity is essential. Incorporating perspective of various disciplines including public health should be utilized.

This study raises several key questions for future research on alcohol-related health disparities and chronic diseases specifically high blood pressure and diabetes. Further investigation is needed to examine within each of the race/ethnicity subgroups. This study explores the role of social environment including social support and mental health well being as protective factors. Investigating these protective factors as key determinants for alcohol use and chronic disease across race/ethnicity is essential in developing primary prevention interventions. The racial/ethnic differences of alcohol use remain an important factor to understanding the health disparities of alcohol use. Preventive interventions should focus on enhancing protective factors (mental health well-being and social support) as a mechanism both to reduce alcohol related health disparities to prevent high blood pressure and diabetes. Given the significant interaction of racial/ethnic differences of alcohol use across high blood pressure and diabetes, it remains unclear to what extent a common set of individual level risk and protective factors that may evolve conceptually to predict alcohol use among adults of different social-cultural origin. Only a modest proportion of variance in alcohol use was explained by the models. Furthermore, the variables assessed for this study mainly focused on individual level psychosocial factors. Other multilevel factors such as discrimination, neighborhood factors may play an important role

to the alcohol related health disparities. Another factor that may help to understand the protective effect among Blacks and Whites with high blood pressure diagnoses. Within an ecological framework, there is a need to move beyond the individual level factors and consider family factors (ex. lack of social support) that may be increasing the vulnerability for alcohol use. Moreover, how these various factors interact with socio-cultural factors. Results from this study suggest alcohol as a risk factor may manifest differently due to cultural norms and values. There is a need to address the interaction between other risk factors, socio-cultural context and alcohol use. Social and contextual factors may provide the understanding of the mechanisms associated with this study's observed differences of the Non-Hispanic White and Non-Hispanic African/American/Black groups diagnosed with high blood pressure. The social ecological model provides multilevel constructs that are helpful in determining a variety of risk factors that may account of alcohol related health disparities. The investigation of social determinants and their relationship to cardiovascular outcomes remains important. The present study found significant differences in the influence of race/ethnicity and alcohol use on high blood pressure and diabetes. However when controlling for demographic variables, social support, and mental health well-being, these variables were only significant for high blood pressure and not diabetes. Integrating of mental health promotion and chronic disease prevention should address the needs of these populations. Treating of mental health (stress, anxiety, depression), alcohol use, and chronic diseases independent of one another will not address the alcohol related health disparities.

The pathogenesis of diabetes and high blood pressure is multi-factorial and complex, influenced by genetic, environmental and psychosocial factors. Several non-modifiable risk factors were examined including sex and age for this study. However, there are also several

modifiable risk factors that have an influence on these chronic diseases such as alcohol use, mental health well-being, and social support. Despite the non-modifiable and modifiable risk factors examined in this study, there remain other factors to consider. Even when all the selected risk factors are considered together they cannot explain all the moderating factors of race/ethnicity differences in alcohol use on diabetic and hypertensive patients. It is likely that genetic and environmental factors also play an important role. Diabetes and high blood pressure remain progressive diseases especially within ethnic minority populations. Moreover, there is a large proportion of patients that are undiagnosed however exhibits a chronic disease (CDC, 2011).

#### Socio-ecological Theoretical Approach for Prevention

There remain important factors to consider for prevention on the individual and community levels to eliminate health disparities related to chronic diseases and alcohol use. The alcohol use and high blood pressure associations are different across race/ethnicity groups. Psychosocial factors that disproportionately impact ethnic populations have been proposed as a mechanism that increases their vulnerability to chronic diseases. More importantly, the impact of mental health well-being on high blood pressure is found to be significantly stronger among the Non-Hispanic African-American/Black category than Non-Hispanic White and Hispanic categories in this study. These results point to the important role that social support (marital status and family in household over 18 years old) may play as buffers to the negative impacts associated with alcohol use and high blood pressure. Mental health well-being including stress, anxiety and/or depression provides an important conceptual and physiological link between individual level characteristics of physical health outcomes such as high blood pressure and diabetes. This study

addresses a key shortcoming in the public health and health disparities literature. The findings from this study link the psychosocial context to health outcomes which may be important in providing information that will better help guide intervention, prevention and policies specifically aimed to address alcohol related health disparities. Furthermore, the results presented are essential for health care professionals to better ensure culturally, efficacious and effective interventions to improve health of diverse populations. Alcohol related harm is determined by the amount and pattern of drinking. There remains a large health and social burden caused by the harmful use of alcohol (WHO, 2014); however, researchers and policymakers must continue to examine the alcohol effects related to health and social burden among populations who consume low to moderate use of alcohol. It is essential that a distinction between use of alcohol levels should be made. The models direct path of race/ethnicity influences on alcohol use on high blood pressure and diabetes suggests that social support and mental health well-being are not the only two mechanisms underlying the health outcomes. Examination of coping responses should be used as a further inquiry. Overall, the results add to the address the gap in the literature and the inconsistent findings.

It is well established that excessive alcohol consumption is associated with chronic diseases such as hypertension, diabetes, and liver disease. However, whether or not low to moderate levels of alcohol use are associated with chronic diseases specifically high blood pressure and diabetes remains controversial. Some studies show that a single moderate dose of alcohol can lower the blood pressure in hypertensive patients that are habitual drinkers (Kawano et al., 1992). For the past 30 years there has been many scientific articles linking a favorable effect of drinking alcohol at a low and moderate level. However, there are inconsistent results as to the influential factors of alcohol use and chronic diseases. For example, Semba and colleagues

(2014) show that resveratrol (a polyphenol found in red wine, grapes, chocolate) does not have a substantial influence on inflammation, cardiovascular disease, cancer or longevity.

This study is not suggesting a recommendation to start consuming alcohol as a method of cardiovascular prevention. Furthermore, the possibility of residual confounding cannot be ruled out. For example, some residual factors to consider are racism/discrimination, coping strategies, smoking tobacco, dietary, physical activity, medications for treatment of disease, family history and obesity. However, it is unlikely that residual confounding could fully explain the influencing factors. There still remain many unknown factors that generate and drive these disparities after adjusting for the selected covariates.

By identifying the relative contribution of these risk factors to alcohol related health disparities, this study has helped to inform what is needed in interventions seeking to address chronic diseases. Furthermore, it allows health care professionals to build upon existing protective factors for racial/ethnic populations. This study investigated whether psychosocial moderating factors help explain the influence of race/ethnicity of alcohol use on high blood pressure and diabetes. Critical gaps in knowledge have been addressed with investigating the influence of race/ethnicity and alcohol use on high blood pressure and diabetes.

Thus in spite of numerous studies, the role of mental health factors in association of high blood pressure and diabetes remains unclear. Statistical adjustment of these covariates that are correlated both with race/ethnicity, alcohol use and high blood pressure diminishes racial differences except family members in the household over 18 years old. It is possible that participants in the present sample who report marital status and having family members over 18 years old are more likely to assume caregiving responsibilities for their family members which may become detrimental to their own diagnosis of a chronic disease. Nevertheless as for

prevention, such intervention might include reduction of alcohol use and increase social support and mental health well-being. Furthermore, decreasing stressors that may be culturally driven such as discrimination and acculturation should be used for prevention strategies. Overall, the influence of race/ethnicity on alcohol outcomes plays an important role in elimination alcohol related health disparities. Prior evidence indicates inconsistent results between alcohol use on high blood pressure and diabetes when race/ethnicity is examined. The models for this study support that race/ethnicity differences moderates alcohol classification for both high blood pressure and diabetes.

## Limitations

This study's findings must be interpreted with caution due to certain limitations. First, the research design for this study was cross-sectional. A casual inference cannot be concluded because it is unknown what came first between the diagnoses of high blood pressure or alcohol use. The concept of confounding can also be addressed as an inferential problem in construct validity with using proxy measures. The association of alcohol use and high blood pressure and its magnitude was different when adjusting for the covariates. Recall bias and self-reporting of data should also be considered. Most notably, one cannot exclude the possibility that other existing risk factors may also moderate and mediate the outcome variables. Nevertheless, it is yet unknown which psychological, physiologic, and/or environmental factors may influence this study did not assess important variables including obesity and body mass index. These variables were not provided in the data set. Social support is derived increasingly from many ways such as family, friends, or community. This study only measured social support from a family

perspective. The supportive behavior on the part of spouse and other family members in the household may be helpful in preventing alcohol use and dealing with mental health issues. This is an impressive difference and deserves attention from the public health field. Again, the social support variable emerges as especially important for chronic diseases. Adequate social support interactions among people are essential and may protect against progression of chronic diseases such as high blood pressure and alcoholism. Although one must be cautious about interpreting the effect of stress, depression and anxiety. Alcohol use is related to depression. This may exhibit escapist drinking in which one with high levels of stress and low social support use alcohol as a coping mechanism. This measure had moderating effects on some variables but not with regard to other variables. What remains is to consider possible mechanisms for the other risk and protective factors. Given that factors such as depression, anxiety, stress have been linked to poorer health outcomes, understanding the relationship between these psychosocial factors among race/ethnicity and chronic disease outcomes is important for interventions.

Despite the limitations, this study demonstrated many strengths including using nationally representative data. The importance of eliminating health disparities in cardiovascular risk outcomes such as high blood pressure and diabetes is equally vital through primary and secondary prevention with addressing risk factors such as alcohol use. This study highlighted a need to better understand race/ethnicity differences in mechanisms and epidemiology linking psychosocial risk factors and alcohol use to high blood pressure and diabetes.

#### **Implications for Public Health Care Professionals**

This study has several implications for public health professionals providing preventive screening information to patients who have been diagnosed with a chronic disease and who have

or have not been screened for alcohol use. The results from this study indicate that there are significant relationships between high blood pressure and diabetes across race/ethnicity. Certain socio-cultural factors do influence the alcohol use. Routinely assessing psychosocial factors such as family support and mental health well-being with alcohol use outcomes among chronic diseases is important for prevention. Assessing these variables as a part of patient-provider communication or brief survey should include questions about social ecological factors including behavioral and environmental factors for determining healthy lifestyle behaviors.

A combined effort of health care professionals, stakeholders and the community residents produces a synergistic effect to deal with the challenge of alcohol use disorders within the communities. Literature suggests an estimated 89% of 17.9 million Americans with an alcohol use disorder do not perceive a need for treatment and therefore do not seek care (Clark et al., 2008). Effective interventions typically involve behavioral counseling technique such as motivational interviewing and use of other resources to assist patients in undertaking advised behavior change. One main approach included with brief intervention is motivational interviewing which encourages individuals to change their behavior using empathy rather than confrontation (Rollnick et al., 2008). Alcohol screening and brief intervention offers an evidence base and cost effective approach (Bradley et al, 1993, Fleming et al., 2002).

It is vital to understand alcohol use disorders across the life span, various race/ethnic populations because of the health disparities impact and begin to address and alleviate the consequences of this problem on the individual and community level. Knowledge must be obtained and applied in developing culturally competent and relevant prevention strategies to maintain a healthy behavior change of any substance use. The priority setting for brief interventions of alcohol use disorders includes the process of addressing the demand for

prevention and intervention in primary healthcare setting. Future research is needed to help guide prevention, intervention, and treatment programs at state, federal, and local levels. Agencies such as the CDC and SAMSHA should continue to collaborate with the Task Force on Community Preventive Services to assess public health interventions and policies designed to prevent excessive alcohol use and related harms.

Lastly, prevention programs such as the screening brief intervention and referral to treatment (SBIRT) training demonstrates a standardized method of addressing alcohol use disorders for adult patients in health care venues (Marshall et al., 2012; SAMSHA, 2012). Most health care professionals confront the consequences of substance abuse among patient care but often lack the expertise to provide behavior change (Geller, 1989). Overall training in substance use related diagnosis, treatment, and prevention with chronic disease management has been inadequate for physicians (Fiellien et al., 2002). To address the demand for prevention and intervention in healthcare setting, health care professionals must have the education and training to effectively identify substance use problems, provide behavioral counseling, and coordinate treatment. Accessibility is the key to shaping health behavior. Changes in health behavior are more likely to occur when changes in attitudes, beliefs, perception, and knowledge occur. Overall, theories and models are comprehensive guides for effective health behavior, promotion, and prevention and should be used for planning, implementation, and evaluation of programs (Glanz, 2008). In addition, collaboration of multi-discipline professionals is required to lower the excessive alcohol use costs the US spends each year for alcohol related health, mental and social care. Increase funding for brief intervention and prevention programs is needed in order to decrease the social determinant factors of the harmful effects of alcohol dependency.

## **Future Direction**

Health disparities must be eliminated through comprehensive, coordinated and sustained public health efforts directed at the public health core, assessment, and policy levels in order to promote positive health behavior and prevent disease. The prevention of health disparities requires intervention strategies that address the allocation of public health resources, environment and access and quality of care.

In conclusion, the US nation has taken enormous steps to eliminate health disparities (Healthy People, 2020). The Affordable Care Act addresses various goals such as transforming health care to allow access for all race/ethnicity populations, strengthen the infrastructure of health care professionals, advance health status with increasing community programs and applicable policy decisions, advance scientific knowledge and innovation, increase efficiency and accountability with programs (Healthy People, 2020). Health disparities can have a wide range of adverse impacts on the course of medical and mental illness, social, and environment. Consistent with other findings reported in the literature, health disparities requires culturally appropriate public health initiatives, community support, and equitable access to quality health care. Programs must target population specific differences in risk factors for determinants of health and develop prevention and intervention programs with a specific population focus. Policy is needed to develop strategies for sustainability to include evaluation, guality monitoring of interventions, collaborations and partnerships with various organizations, data collection and program management for eliminating health disparities (Healthy People, 2020). Additionally, diversifying the workforce with improving cultural competence is needed. Social determinants of health including the role of policy making, social factors, health services, individual behaviors, and biology and genetics should be integrated with the planning, promotion, intervention,

prevention, and evaluation of programs to maintain a successful healthy behavior change and sustainability.

#### REFERENCES

- Ashley, M.J., Rehm, J., Bondy, S., Single, E., & Rankin, J. (2000). Beyond ischemic heart disease: Are There Other Benefits From Drinking Alcohol? *Contemporary Drug Problems*, 27 (1), 735-777.
- Babor, T.F., McRee, B.G., Kassebaum, P.A., Grimaldi, P.L., Ahmed, K., Bray, J. (2007).
  Screening, Brief Intervention, and Referral to Treatment (SBIRT): Toward a Public
  Health Approach to Management of Substance Abuse. *Substance Abuse*, 28 (3), 7-30.
- Baker, B., Helmers, K., O'Kelly, B., Sakinofsky, I., Abelsohn, A., Tobe, S. (1999). Marital cohesion and ambulatory blood pressure in early hypertension. American Journal of Hypertension 12 (2), 227-230.
- Bailey, J.A., Hill, K. G., Meacham, M.C., Young, S.E., & Hawkins, J. (2011).Strategies for characterizing complex phenotypes and environments: general and specific family environmental predictors of young adults tobacco dependence, alcohol use disorder, and co-occurring problems. *Drug and Alcohol Dependence*, 118 (1), 444-451.
- Baliunas, D.O., Taylor, B.J., Irving, H., Roerecke, M., Patra, J., Mohapatra, S., & Rehm, J. (2009). Alcohol as a risk factor for type 2 diabetes: a systematic review and meta-analysis. *Diabetes Care* 32 (11), 2123-2132.

Bandura, A. (1977). Social learning theory. Englewood Cliffs, NJ: Prentice-Hall.

- Barr, K.E., Farrell, M.P., Barnes, G.M., & Welte, J.W. (1993). Race, class and gender differences in substance abuse: evidence of middle class/underclass polarization among black males. *Social Problems* 40 (2), 316-327.
- Berlin, J.A. & Colditz, G.A. (1990). A meta-analysis of physical activity in the prevention of coronary heart disease. *American Journal of Epidemiology* 132 (4), 612-628.

- Beilin, L.J., & Puddey, I.B. (2006). Alcohol and hypertension: an update. *Hypertension* 47 (1), 1035-1038.
- Beulens, J.W., Rimm, E.B., Axherio, A., Spiegelman, D, Hendriks, H.F., & Mukamal,K.J.(2007). Alcohol consumption and risk for coronary heart disease among men withhypertension. *Annual Internal Medicine* 146 (1), 100-109.
- Bohn, M.J., Babor, T.F., Kranzler, H.R. (1995). The alcohol use disorders identification test
- (AUDIT): validation of a screening instrument for use in medical settings. *Journal Studies of Alcohol* 56 (4), 423-32.
- Bradley K.A., Donovan D.M., Larson E.B. (1993). How much is too much? Advising patients about safe levels of alcohol consumption. *Archives of Internal Medicine* 153 (24), 2734-2740.
- Braveman, P.A., Cubbin, C., Egerter, S., Williams, D.R., & Pamuk, E. (2010). Socioeconomic
   Disparities in Health in the United States: What the Patterns Tell Us. *American Journal* of Public Health 100 (S1), S186-S196.
- Breslow, R.A., Guenther, P.M., & Smothers, B.A. (2006). Alcohol drinking patterns and diet quality: the 1999-2000 National Health and Nutrition Examination Survey. *American Journal of Epidemiology*, 163 (4), 359-366.
- Brittain K., Taylor J.Y., Wu, C. Y., (2010). Family adaptability among urban African American women with hypertension. *Journal for Nurse Practitioners*. 6 (10), 786-793.
- Bronfenbrenner, U. (1979). *The ecology of human development*. Cambridge, MA: Harvard University Press.
- Brown, S.A., Christiansen, B.A., & Goldman, M.S., (1987). The Alcohol Expectancy Questionnaire: An instrument for the assessment of adolescent and adult alcohol expectancies. *Journal of Studies on Alcohol and Drugs*, 48 (5), 483-491.

- Brydon, L., Strike, P.C., Bhattacharyya, M.R., Whitehead, D.L., McEwan, J., Zachary, I., & Steptoe, A. (2010). Hositility and physiological responses to labatory stress in acute coronary syndrome patients. *Journal of Psychosomatic Research*, 68 (2), 109-116.
- Caetano, R. (2003). Alcohol-Related Health Disparities and Treatment-Related Epidemiological Findings Among Whites, Blacks, and Hispanics in the United States. *Alcoholism Clinical Experimental Research*, 27 (8), 1337-1339.
- Caetano, R. (1997) Prevalence, Incidence and Stability of Drinking Problems among Whites,
  Blacks and Hispanics: 1984-1992. *Journal of Studies on Alcohol and Drugs*, 58 (6), 565-572.
- Caetano, R., Clark, C., & Tam, T. (1998). Alcohol consumption among racial/ethnic minorities: theory and research. *Alcohol Health Research World*, 22(4),233-241.
- Caetano, R., & Clark, C.L. (1999). Trends in situational norms and attitudes toward drinking among whites, blacks, and hispancis: 1984-1995. *Drug and Alcohol Dependence*, 54(1), 45-56.
- Carlsson, S., Hammar, N., & Grill, V. (2005). Alcohol consumption and type 2 diabetes:
  meta-analysis of epidemiological studies indicates a U-shaped relationship. *Diabetologia* 38 (6),1051-1054.
- Carter-Edwards, L., Godette, D.C., White, S.S., & Tyson, W. (2009). A conceptual Framework for studying alcohol intake and blood pressure on historically black college and university campuses. *Journal of Drug Education*, 39 (2),149-165.
- Chartier, K.G., Vaeith, P.A.C., Caetano, R. (2014a). Focus on: ethnicity and the social and health harms from drinking. *Alcohol Research*, 35(2):229-237.

- Chartier, K.G., Scott, D.M., Wall, T.L., Covault, J., Karriker-Jaffe, K.J., Mills, B.A., Luczak,
  S.E., Caetano, R., & Arroyo, J.A. (2014b). Framing Ethnic Variations in Alcohol
  Outcomes From Biological Pathways to Neighborhood Context. *Alcoholism Clinical Experimental Research*, 30 (1), 1-8.
- Chartier, K.G., Hesslebrock, M.N., Hesselbrock, V.M. (2013). Ethnicity and gender comparisons of health consequences in adults with alcohol dependence. *Substance Use Misuse*, 48 (3), 200-210.
- Chartier, K.G, & Caetano, R. (2011). Trends in alcohol services utilization from 1991-1992 to
   2001-2002: ethnic group differences in the U.S. population. *Alcoholism Clinical Experimental Research*, 35(8),1485-1497.
- Chartier, K., & Caetano, R. (2010). Ethnicity and health disparities in alcohol research. *Alcohol Research & Health*, 33(1-2), 152-160.

Centers of Disease of Control and Prevention (CDC) (2011)

Retrieved from

http://www.cdc.gov/chronicdisease/resources/publications/aag/alcohol.htm

- Centers for Disease Control and Prevention (CDC) (2010). A Closer Look at African American Men and High Blood Pressure Control: A Review of Psychosocial Factors and Systems-Level Interventions. Atlanta: U.S. Department of Health and Human Services.
- Centers for Disease Control and Prevention (CDC). (2012). Chronic disease prevention and health promotion. Atlanta: National Center for Chronic Disease Prevention and Health Promotion. Retrieved from <u>http://www.cdc.gov/chronicdisease/overiveiw/index.him</u>.

Centers of Disease of Control and Prevention (CDC) (2013)

Retrieved from http://www.cdc.gov/chronicdisease

- Cohen, S., Janicki-Deverts, D., & Miller, G.E. (2007). Psychological Stress and Disease. Journal of American Medical Association, 298(14),1685–1687.
- Colditz, G.A., Giovannucci, E., Rimm, E.B., Stampfer, M.J., Rosner, B., Speizer, F.E.,Gordis, E., & Willett, W.C. (1991). Alcohol intake in relation to diet and obesity amongwomen and men. *American Journal of Clinical Nutrition*, 54(1), 49-55.
- Conigrave, K.M., & Rimm, E.B. (2003). Alcohol for the prevention of type 2 diabetes mellitus? *Treatments in Endocrinology*, 2 (3), 145-152.
- Cooper, R.S., Kaufman, J.S., Ward, R. (2003). Race and Genomics. *New England Journal of Medicine*, 348(12), 1166-1170.
- Costanzo, S., Di Castelnuovo, A., Donati, M.B., Iacoviello, L., & de Gaetano, G. (2010). Alcohol consumption and mortality in patients with cardiovascular disease: a metaanalysis. *Journal of the American College of Cardiology*, 55(13), 1339-1347.
- Corrao, G., Rubbiati, L., Bagnardi, V., Zambon, A., & Poikolainen, K. (2000). Alcohol and coronary heart disease: a meta-analysis. *Addiction*, 95(10), 1505-1523.
- Criqui, M.H., Wallace, R.B., Mishkel, M., Barrett-Conno, r E., & Heiss, G. (1981). Alcohol consumption and blood pressure the lipid research clinics prevalence study. *Hypertension*, 3 (1), 557-565.
- Dauber, S., Hogue, A., Paulson, J.F., & Leiferman, J.A. (2009). Typologies of alcohol use in White and African American adolescent girls. *Substance Use & Misuse*, 44(8), 1121-1141.
- Dawson, D.A., Grant, B.F., Stinson, F.S., Chou, P.S., Huant, B., Ruan, W.J.(2005). Recovery from DSM-IV alcohol dependence: United States, 2001-2002. *Addiction* 100 (1), 281-292.

- Dawson, D.A. (1998). Beyond black, white and hispanic: race, ethnic origin and drinking patterns in the United States. *Journal of Substance Abuse*, 10 (4), 321-339.
- Deter, H.C., Micus, C., Wagner, M., Sharma, A.M., & Buchholz, K. (2006). Salt sensitivity, anxiety, and irritability predict blood pressure increase over five years in healthy males. *Clinical and Experimental Hypertension*, 28(1), 17-27.
- DeVoe, J.E., Tillotsom, C.J., Lesko, S.E., Wallace, L.S., & Angier, H. (2011). The case for synergy between a usual source of care and health insurance coverage. *Journal of General Internal Medicine*, 26(99), 1059-66.
- Di Castelnuovo, A., Rotondo, S., Iacoviello, I., Donati, M.B., & De Gaetano, G. (2002). Meta-analysis of wine and beer consumption in relation to vascular risk. *Circulation* 105 (1), 2836-2844.
- Dickey, B., Normand, S. T., Weiss, R.D., Drake, R.E., Azeni, H (2002). Medical morbidity, mental illness, and substance use disorders. *Psychiatric Services*, (53),7, 861-867.
- Diez Roux, A.V. (2012). Conceptual approaches to the study of health disparities. *Annual Review of Public Health*, 33(1),41-58.
- Dismsdale, J.E. (2008). Psychological stress and cardiovascular disease. *Journal of the American College of Cardiology*, 51(13), 1237-1246.
- Douglas, J.G. (2005). Clinical guidelines for the treatment of hypertension in African American. *American Journal of Cardiovascular Drugs*, 5(1), 1-6.
- Duru, O.K., Harawa, N.T., Kermah, D., & Norris, K.C. (2012). Allostatic load burden and racial disparities in mortality. *Journal of National Medical Association*, 104 (1-2), 89-95.
- Dressler, W, Bindon, J. (2000). The health consequences of cultural consonance: cultural

dimensions of lifestyle, social support, and arterial blood pressure in an African American community. *American Anthropologist*, 102(2),244-260.

- Dyer, A.R., Stamler, J., Paul, O., Berkson, D.M., Lepper, H., McKean, R.B., Shekelle, R.B., Lindberg, H.A., & Garside, D. (1977). Alcohol consumption, cardiovascular risk factors and mortality in two Chicago epidemiologic studies. *Circulation*, 56 (1),1067-1074.
- Edenberg, H.J., Xuei, X., Chen, H.J., Tiam, H., Wetherill, L.F., Dick, D.M., Almasy, L., Bierut, L., Bucholz, K.K., Goate, A., Hesslebrock, V., Kuperman, S., Nurnberger, J.,
- Porjesz, B., Rice, J., Schuckit, M., Tischfield, J., Begleiter, H., & Foroud, T. (2006).
  Association of alcohol dehydrogenase genes with alcohol dependence: a comprehensive analysis. *Human Molecular Genetics*, 15(1),1539-1549.
- Edwards, D. and Berry, J. J. (1987). The Efficiency of Simulation-Based Multiple Comparisons, *Biometrics*, 43 (1),913–928.
- Ehlers, C.L., Gilder, D.A., &Harris, L., Carr, L. (2001). Association of the ADH2\*3 allele with a negative family history of alcoholism in African American young adults. *Alcoholism Clinical Experimental Research*, 25 (12), 1773-1777.
- Ehlers, C.L., Carr, L., Betancourt, M., & Montane-Jaime, K. (2003). Association of the ADH2\*3 allele with greater alcohol expectancies in African American young adults. *Journal of Studies on Alcohol and Drugs*, 64(2),176-181.
- Elliott, P., Fehily, A.M., Sweetnam, P., & Yarnell, J.W.G. (1987). Diet, alcohol, body mass, and social factors in relation to blood pressure: the caerphilly heart study. *Journal of Epidemiology Community Health*, 41 (1), 37-43.
- Engel, G.L (1980). The clinical application of the biopsychosocial model. *American Journal of Psychiatry*, 137 (1), 535-544.

- Enock, M.A (2013). Genetic influences on the development of alcoholism. *Current psychiatry reports*, 15 (11), 412-427.
- Everson-Rose, S.A., & Lewis, T.T. (2005). Psychosocial factors and cardiovascular diseases. *Annual Review Public Health*, 26(1), 469-500.
- Fiellin, D., Butler, R., D'Onofrio, G., Brown, R., & O'Connor, P. (2002). The physician's role in caring for patients with substance use disorders; Implications for medical education and training. *Substance Abuse*, 23(1), 207-212.
- Fields, L.E., Burt, V.L., Cutler, J.A., Hughes, J., Roccella, E.J., Sorlie, P. (2004). The burdern of adult hypertension in the United States 1999 to 2000: a rising tide. Hypertension 44:398-404.
- Fillmore, K.M., Stockwell, T., Chikritzhs, T., Bostrom, A., & Kerr, W. (2007). Moderate alcohol use and reduced mortality risk: systematic error in prospective studies and new hypotheses. *Annals of Epidemiology*, 17(5), 16-23.
- Fillmore, K.M., Kerr, W.C., Stockwell, T.R., Chikritzhs, T., Bostrom, A. (2006). Moderate alcohol use and reduced mortality risk: systematic error in prospective studies. *Addiction Research & Theory* 14 (2), 101-132.
- Fleming M.F., Mundt M.P., French M.T., Manwell L.B., Stauffacher E.A., Barry K.L. (2002).
   Brief physician advice for problem drinkers: long term efficacy and benefit-cost analysis.
   *Alcoholism, Clinical and Experimental Research* 26, (1), 36-43.
- Foopa, M., Fuchs, F.D., Duncan, B.B. (2001). Alcohol and atherosclerosis. *Arq Bras Cardiology*, 76 (1), 171-176.

- Frasure-Smith, N., Lesperance, F., Gravel, G., Masson, A., Juneau, M., Talajic, M.,
  & Bourassa, M.G. (2000). Social support, depression, and mortality during the first year after myocardial infarction. *Circulation*, 101 (1), 1919-1924.
- Freiberg, M.S., Chang, Y.F., Kraemer, K.L., Robinson, J.G., Adams-Campbell, L.L., & Kuller, L.L. (2009). Alcohol consumption, hypertension, and total mortality among women. *American Journal of Hypertension*, 22(11), 1212-1218.
- Freisthler, B., Gruenewald, P.J., Treno, A.J., &Lee, J. (2003). Evaluating alcohol access and the alcohol environment in neighborhood areas. *Alcoholism Clinical Experimental Research*, 27(3), 477–484.
- Frist, WH (2005). Overcoming disparities in US healthcare. Health Affairs, 24(2), 445-451.
- Fuchs, F. D., &Chambles, L.E. (2007). Is the cardioprotective effect of alcohol real? *Alcohol*, 47(1), 399-402.
- Fuchs, F.D., Chambless, L.E., Folsom, A. R., Eigenbrodt, M.L., Duncan, B.B., Gilbert, A.,
  & Szklo, M. (2004). Association between alcoholic beverage consumption and incidence of coronary heart disease in whites and blacks: the Atherosclerosis Risk in Communities Study. *American Journal of Epidemiology*, 160(5), 466-474.
- Fuchs, F.D., Chambless, L.E., Whelton, P.K., Nieto, F.J., & Heiss, G. (2001). Alcohol consumption and the incidence of hypertension: the Atherosclerosis Risk in Communities Study. *Hypertension*, 37 (1), 1242-1250.
- Fuchs, C.S., Stampfer, M.J., Colditz, G.A., Giovannucci, E.L., Manson, J.E., Kawachi, I.,
  Hunter, D.J., Hankinson, S.E., Hennekens, C.H., Rosner, B, Speizer, F. E, & Willett,
  W.C. (1995). Alcohol consumption and mortality among women. *New England Journal* of Medicine, 332(1), 1245-1250.

- Gallo, L.C., Troxel, W.M., Matthews, K.A., Kuller, L.H. (2003). Marital status and quality in middle-aged women: associations with levels and trajectories of cardiovascular risk factors. *Health Psychology*, 22 (5), 453-463.
- Garg, R., Wagener, D.K., & Madans, J.H. (1993). Alcohol Consumption and Risk of Ischemic Heart Disease in Women. *Archives of Internal Medicine*, 153(10), 1211-1216.
- Geller,G., Levine, D.M., Mammon, J.A., Moore, R.D., Bone, L.R., Stokes, E.J. (1989).
  Knowledge, attitudes and reported practices of medical students and house staff regarding the diagnosis and treatment of alcoholism. *Journal of the American Medical Association*, 261 (21), 3115-3120.
- Geronimus, A.T., Hicken, M., Keene, D., & Bound, J. (2006). "Weathering" and age patterns of allostatic load scores among Blacks and Whites in the United State. *American Journal of Public Health*, 96(5), 826-837.
- Glanz, K., Rimer, B., & Viswanath, K. (2008). Health Behavior and Health Education:Theory, Research, and Practice Fourth Edition. San Francisco, CA: Jossey-Bass.
- Gilman, S.E., Breslau, J., Conron, K.J., Koenen, K.C., Subramanian, S.V., & Zaslavsky,
  A.M. (2008). Education and race-ethnicity differences in the lifetime risk of alcohol
  Dependence. *Journal of Epidemiology Community Health*, 62 (3), 224-230.
- Godette, D.C., Edwards, E., Ford, C.L., Strunin, L., Heeren, T., Kwachi, I (2009). Social status, gender and alcohol-related problems: the black young adult experience. *Ethnicity & Health*, 14 (5) 479-496.
- Goldberg, D.M., & Soleas, G.J. (2001). Beverage alcohol consumption as negative risk factor coronary heart disease: biochemical mechanisms. Alcohol in Health and Disease by

Agarwal DP, Seitz HK. Marcel Dekker, Inc. New York.

- Goldman, M.S., Brown, S. A., Christiansen, B.A., & Smith, G.T. (1991) Alcoholism and memory; broadening the scope of alcohol-expectancy research. *Psychological Bulletin*, 110 (1), 137-146.
- Gottlieb, B.H., & Bergen, A.E. (2010). Social support concepts and measures. *Journal of Psychosomatic Research*, 69(1), 511-520.
- Grant, J.D., Verges, A., Jackston, K.M., Trull, T.J., Sher, K.J., & Bucholz, K.K. (2012). Age and ethnic differences in the onset, persistence and recurrence of alcohol use disorder. *Addiction*, 107(4),756-765.
- Grant, B.F. (1997). Prevalence and correlates of alcohol use and DSM-IV alcohol
  dependence in the United States: results of the National Longitudinal Alcohol
  Epidemiologic Survey. *Journal of Studies on Alcohol and Drugs*, 58(5),464-473.
- Green, L.W., Richard, L., Potvin, L. (1996). Ecological foundations of health promotion. *American Journal of Health Promotion*. 10 (1), 270-281.
- Greenfield T.K., Harford T.C., &Tam T.W. (2009) Modeling cognitive influences on drinking and alcohol problems. *Journal of Studies on Alcohol and Drugs*, 70 (1), 78-86.
- Greenwood, D.C., Muir, K.R., Parkham, C.J., & Madeley, R.J. (1996). Coronary heart disease: a review of the role of psychosocial stress and social support. *Journal of Public Health*, 18(2), 221-231.
- Grucza, R.A., Krueger, R.F., Racette, S.B., Norberg, K.E., Hipp, P.R., & Bierut, L.J. (2010). The emerging link between alcoholism risk and obesity in the United States. *Archives* of General Psychiatry, 67(12), 1301-1308.

- Gump, B.B., Polk, D.E., Kamarck, T.W., Shiffman, S.M. (2001). Partner interactions are associated with reduced blood pressure in the natural environment: ambulatory monitoring evidence from a healthy, multiethnic adult sample. Psychosomatic medicine, 63 (3), 423-433.
- Hans, B., Gfroerer, J., Batts, K.R., & Colliver, J. (2011). Co-occurrence of selected chronic physical conditions and alcohol, drug, or mental health problems and health care utilization among persons aged 18 to 64 in the United States. *CBHSQ Data Review*, (1), 1-13.

HealthyPeople 2020 Retrieved from http://www.healthypeople.gov/2020/default.aspx

- Hendriks, H.F., Veenstra, J., Velthuis-te Wierik, E.J., Schaafasma, G., &Kluft, C. (1994).
  Effect of moderate dose of alcohol with evening meal on fibrinolytic factos. *British Medical Journal*, 308(1), 1003-1006.
- Herd, D. (1991). Drinking patterns in black population, In: Clark, EB., Hildton M.E (EDS.),Alcohol in America: Drinking Practices and Problems, New York State University Press,New York.
- Herd, D. (1994). Predicting drinking problems among black and white men: results from a national survey. *Journal of Studies on Alcohol and Drugs*, 55(1),61-71.
- Hesselbrock, V.M., O'Brien, J., Weinstein, M.A., & Carter-Menendez, N.C. (1987). Reasons for drinking and alcohol use in young adults at high risk and at low risk for alcoholism. *British Journal of Addiction*, 82(12),1335-1339.
- Hicken, M.T., Lee, H., Morenoff, J., House, J.S., & Williams, D.R. (2014). Racial/ethnic disparities in hypertension prevalence: reconsidering the role of chronic stress. *American Journal of Public Health*, 104(1), 117-123.

- Holbrook, T.L., Barrett-Connor, E., Wingard, D.L. (1990). A prospective population-based study of alcohol use and non-insulin-dependent diabetes mellitus. *American Journal of Epidemiology*, 132(5), 902-909.
- Horton, E.G. (2007) Racial differences in the effects of age of onset on alcohol consumption and development of alcohol-related problems among makes from mid-adolescence to young adulthood. *Journal of Ethnic Substance Abuse*, 6(1), 1-13.
- Howard, A.A., Arnsten, J.H., & Gourevitch, M.N. (2004). Effect of alcohol consumption on diabetes mellitus: a systematic review. *Annual Internal Medicine*, 140(1), 211-219.
- Huijbregts, P., Feskens, E., Rasanen, L., Fidanza, F., Nissinen, A., Menotti, A., & Kromhout,
  D. (1997). Dietary pattern and 20 year mortality in elderly men in Finland, Italy, and the
  Netherlands: longitudinal cohort study. *British Medical Journal*, 315(7099), 13-17.
- Humes, K.R., Jones, N.A., & Ramirez, R.R. (2011). Overview of race and Hispanic origin:
  2010 (Census Brief C2010BR-02). Washington, DC: US Department of Commerce,
  Economics and Statistics Administration, US Census Bureau.
- Imamura, F., Lichtenstein, A.H., Dallal, G.E., Meigs, J.B., & Jacques, P.F. (2009).Confounding by dietary patterns of the inverse association between alcohol consumption and type diabetes risk. *American Journal of Epidemiology*, 170 (1), 37-45.
- Jarnecke, A.M., South, S.C. (2014). Genetic and environmental influences on alcohol use problems: moderation by romantic partner support but not family or friend support. *Alcohol clinical and experimental research*, 38 (2), 367-375.
- Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) (2003). *Hypertension*,42:1206. Retrieved from <u>http://www.nhlbi.nih.gov/guidelines/hypertension/</u>

- Jones, R.A., Utz, S.W., Williams, I.C, Hinton, I., Alexander, G., Moore, C., Blankenship, J., Steeves, R., & Oliver, N. (2008). Family interactions among African Americans with Type 2 diabetes. *The Diabetes Education*, 34(2), 318-26.
- Jones, B.R., Barrett-Connor, E., Criqui, M.H., & Holdbrook, M.J.(1982). A community study of calorie and nutrient intake in drinkers and nondrinkers of alcohol. *American Journal of Clinical Nutrition*, 35 (1), 135-139.
- Jones-Webb, R., Hsiao, C.Y., Hannan, P., Caetano, R. (1997). Predictors of increases in alcoholrelated problems among black and white adults: results from the 1984 and 1992 National Alcohol Surveys. *American Journal of Drug Alcohol Abuse*, 23 (2), 281-299.
- Kalu, N., Ramchandan, V.A., Marshall, V., Scott, D., Ferguson, C., Cain, G., & Taylor, R.
   (2012). Heritability of level of response and association with recent drinking history in nonalcohol-dependent drinkers *Alcohol Clinical Experimental Research*, 36(6),1034-41.
- Kao, W.H., Puddey, I.B., Boland, L.L., Watson, R.L., & Brancati, F.L. (2001). Alcohol consumption and the risk of type 2 diabetes mellitus: Atherosclerosis risk in communities study. *American Journal of Epidemiology*, 154(8),748-757.
- Kaufman, D.W., Rosenberg, L., Helmrich, S.P, & Shapir, S. (1985). Alcoholic beverages and myocardial infarction in young men. *American Journal of Epidemiology*, 121(1), 585-554.
- Kerr, W.C., &Ye, Y. (2010) Relationship of life-course drinking patterns to diabetes, heart problems and hypertension among those 40 and older in the 2005 US National Alcohol Survey. *Journal of Studies on Alcohol and Drugs*, 71 (1), 515-525.
- Kerr, W.C., Greenfield, T.K., Bond, J., Yu, Y., & Rehm, J. (2011). Racial and EthnicDifferences in All-Cause Mortality Risk According to Alcohol Consumption Patterns in

the National Alcohol Surveys. American Journal of Epidemiology, 174(7),769-778.

- Kiechl, S., Willeit, J., Poewe, W., Egger, G., Oberhollenzer, F., Muggeo, M., & Bonora, E. (1996). Insulin sensitiviety and regular alcohol consumption: large, prospective, crosssectional population study. *British Medical* Journal, 313(1), 1040-1044.
- Klatsky, A.L.(2007). Alcohol, cardiovascular diseases and diabetes mellitus. *Pharmacological* Research, 55 (3), 237-247.
- Klatsky, A.L. (1994). Epidemiology of coronary heart disease influence of alcohol. *Alcohol Clinical Experiment Research*, 18(1),88-96.
- Klatsky, A.L. Friedman, G.D., Abraham, B.S., & Gerard, M.J. (1977). Alcohol consumption and blood pressure: Kaiser Permanente multiphasic health examination data. *New England Journal of Medicine*, 296 (1), 1194-2000.
- Klatsky, A.L. (1985). Blood pressure and alcohol consumption In: Bulpitt CJ, ed. Handbook of hypertension, Vol 6. Epidemiology of hypertension, Amsterdam: Elsevier Science 159- 174.
- Klatsky, A.L., Friedman, G.D., & Armstrong, M.A.(1986). The relationships between alcoholic beverage use and other traits to blood pressure: a new Kaiser Permanente Study. *Circulation*, 73(1), 628-636.
- Kliener, K.D., Gold, M.S., Fost-Pineda, K., Lenzbrunsman, B., Perri., M.G., & Jacobs, W.S. (2004). Body mass index and alcohol use. *Journal of Addictive Diseases, 23(3)*, 101-118.
- Koppes, L.L., Dekker, J., Hendriks, H.F., Bouter, L.M., & Heine, R.J. (2005) Moderate alcohol consumption lowers the risk of type 2 diabetes: a meta-analysis of prospective observational studies. *Diabetes*, 28(3),719-725.

- Krieger, N. (2012). Methods for the scientific study of discrimination and health: an ecosocial approach. *American Journal of Public Health*, 102(5), 936-944.
- Kuller, L.H. (2004). Ethnic differences in atheroschlerosis, cardiovascular disease and lipid metabolism.
- Lackland, D.T., & Keil, J.E. (1996). Epidemiology of hypertension in Africans Americans. *Semi Nephrology*, 16 (1), 63-70.
- LaVeist, T., Pollack, K., Thorpe Jr, R., Fesahazion, R., & Gaskin, D. (2011). Place, not race: disparities dissipate in southwest Baltimore when blacks and whites live under similar conditions. *Health Affairs*, 30(10), 1880-1887.
- LaViest, T. (2005). *Minority Populations and Health: An Introduction to Health Disparities in the United States*. San Francisco, CA Jossey-Bass.
- Leeman, J., Skelley, A.H., Burns, D., Carlon, J., & Soward, A. (2008). Tailoring a diabetes intervention for use with older, rural African American women. *The Diabetes Education*, 34(2): 310-317.
- Leonard K.E. and Blane, H.T. (1999). *Psychological theories of drinking and alcoholism*. New York, NY: Guilford Press.
- Lelinneth, N., Barnes, M., De La Cruz, N.G., Williams, P.N., Rogers, J., (2006). Public health perspectives on the family: an ecological approach to promoting health in the family and community. *Family and Community Health*, 29(1), 28-42.
- Livingston, I.L.,& Marshal, R. (1990). Cardiac reactivity and elevated blood pressure levels among young African Americans: The importance of stress. *The Urban League Review* 13, (1), 77-91.

- Lukachko, A., Hatzenbuehler, ML., & Keyes, KM (2014). Structural racism and myocardial infarction in the United States. *Social Science Medicine*, 103 (1), 42-50.
- Maclure, M. (1993). Demonstration of deductive meta-analysis: ethanol intake and risk of myocardial infarction. *Epidemiology Review*, 15 (1), 328-351.
- Maheswaran, R., Gill, J.S., Davies, P., & Beevers, D.G. (1991). High blood pressure due to alcohol. A rapidly reversible effect. *Hypertension*, 17 (1),787-792.
- Marmot, M.G., Elliott, P., Shipley, M.J., Dyer, A.R., Ueshima, H., Beevers, D.G., Stamler, R., Kesteloot, H., Rose, G., & Stamler, J. (1994). Alcohol and blood pressure: the INTERSALT Study. *British Medical Journal*, 308 (6939), 1263-1267.
- Marmot, M., & Brunner, E. (1991). Alcohol and cardiovascular disease: the status of the Ushaped curve. *British Medical Journal*, 303 (6802), 565-568.
- Marshal, M.P., Chassin L. (2000). Peer influence on adolescent alcohol use: the moderating role of parental support and discipline. *Applied Developmental Science*, 4 (2), 80-88.
- Marshall, V.J., Kalu, N., Kwagyan, J., Scott, D.M., Cain, G.E., Hill, K., Hesselbrock, V.,
  Ferguson, C., & Taylor, R.E. (2013). Alcohol dependence and health care utilization in
  African Americans. *Journal of National Medical Association*, 105 (1), 42-49.
- Marshall, V.J., McLaurin-Jones, T.L., Kalu, N., Kwagyan J., Scott, D.M., Cain, G., Greene,
  W., Adenug, a B., & Taylor, R.E. (2012). Screening, Brief Intervention, and Referral to
  Treatment: Public Health Training for Primary Care. *American Journal Public Health*, 102 (8), e30-36.
- Marshall, V.J., Ramchandan, R.A., Kalu, N., Kwaygan, J., Scott, D.M., Ferguson, C.L., Taylor, R.E. (2014). Evaluation of the influence of ADH polymorphisms on alcohol elimination rates Measured Using the Alcohol Clamp. *Alcohol Clinical Experiment*
Research, 38(1), 51-59.

- Mason, W.A., & Windle, M. (2001). Family, religious, school and peer influences on Adolescent alcohol use: a longitundinal study. *Journal of Studies on Alcohol and Drugs*, 62(1):44-53.
- McCarver, D.G., Thomasson, H.R., Martier, S.S., Sokol, R.J, Li, T (1997). Alcohol dehydrogenase 2\*3 allele protects against alcohol-related birth defects among African Americans. Journal of Pharmacology and Experimental Therapeutics, 283 (3), 1095-1101.
- McEwen, B.S (2012). Brain on stress: how the social environment gets under the skin. *Proceedings of National Academy of Sciences of the United States of American*, 109 (Suppl 2), 17180-17185.
- McEwen, B.S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine*, 338(3), 171-179.
- Mendis, S., Puska, P., & Norrving, B. (2011). Global Atlas on cardiovascular disease prevention and control. World Health Organization in collaboration with the World Heart Federation and the World Stroke Organization: France.
- Miles, D. R., Silberg, J. L., Pickens, R.W, Eaves, L.J. (2005). Familial influences on alcohol use in adolescent female twins: testing for genetic and environmental interactions. *Journal* on Studies of Alcohol and Drugs, 66(1), 445-451.
- Morton KB, Martin PC, Shook-Sa, BE, Chromy JR, Hirsch EL (2013). 2012 National Survey on Drug Abuse and Health Sample Design Report. RTI International, North Carolina downloaded from: <u>http://www.samhsa.gov/data/2k12/NSDUH2012MRB/Index.aspx</u>

- Mulia, N., Ye, Y., Greenfield, T.K., & Zemore, S.E. (2009). Disparities in alcohol-related problems among white, black and Hispanic Americans. *Alcohol Clinical Experiment Research*, 33(4), 654-662.
- Mulia, N., Yu. Y., Zemore, S.E., & Greenfield, T.K. (2008). Social Disadvantage, Stress, and
   Alcohol Use Among Black, Hispanic, and White Americans: Findings from 2005 U.S.
   National Alcohol Survery. *Journal on Studies of Alcohol and Drugs*, 69(6),824-833.
- Mukamal, K.J, Chen, C. M., Rao, S.R., & Breslow, RA (2010). Alcohol consumption and cardiovascular mortality among US adults, 1987-2002. *Journal of American College of Cardiology*, 55(13), 1328-1335.
- Mukamal, K.J., & Rimm, E.B.(2001) Alcohol's effects on the risk for coronary heart disease. *Alcohol Research Health*, 25(4), 255-261.
- Mukamal, K.J., Conigrave, K.M., Mittleman, M.A., Camargo, C.A., Stampfer, M.J., &
  Willett, W.C. (2003). Roles of drinking pattern and type of alcohol consumed in
  coronary heart disease in men. *New England Journal of Medicine*, 348(1), 109–118.
- Muthen, B.O., & Muthen, L.K. (2000). The development of heavy drinking and alcoholrelated problems from ages 18 to 37 in a US national sample. *Journal of Studies on Alcohol and Drugs*, 61(2), 290- 300.
- Naimi, T.S., Brown, D. W., Brewer, R.D., Giles, W.H., Mensah, G., Serdula, M.K., Mokdad, A.H., Hungerford, D.W., Lando, J, Naimi, S., & Stroup, D.F. (2005). Cardiovascular risk factors and confounders among nondrinking and moderate-drinking US adults. *American Journal Preventive Medicine*, 28(4), 369-373.

Nakanishi, N., Yoshida, H., Nakarmura, K., Suzuki, K., & Tatara, K. (2000). Alcohol consumption and risk for hypertension in middle-aged Japanese men. *Journal of Hypertension*, 19(5), 851-855.

National Institutes of Health (NIH) (2012) Retrieved from http://www.nih.gov/

National Healthcare Disparities Report (2007) Agency for Healthcare Research and Quality Retrieved from <u>www.ahrq.gov</u> <u>http://archive.ahrq.gov/qual/qrdr07.htm#nhdr</u>

National Institute on Alcohol Abuse and Alcoholism (NIAAA) (2011)

Retrieved from <u>http://www.niaaa.nih.gov/</u>

- National Institute of Alcohol Abuse and Alcoholism (NIAAA) (2001). Forecast for the *future: Strategic plan to address health disparities*. Rockville: National Institute of Health.
- National Heart, Lung, and Blood Institute (NHLB) (2014)

Retrieved from http://www.nhlbi.nih.gov/health/health- topics/topics/hbp/names.html

National Prevention Strategy (2012) Retrieved from

http://www.surgeongeneral.gov/initiatives/prevention/strategy/index.html

Niskanen, L., Laaksonen, D.E., Nyyssonen, K., Punnonen, K., Valkonen, V.P., Fuentes, R., Tuomainen, T.P., Salonen, R., & Salonen, J.T. (2004). Inflammation, abdominal obesity, and smoking as predictors of hypertension. *Hypertension*, 44(4),859–865.

Office of Minority Health (2013) Retrieved from http://www.minorityhealth.hhs.gov/

- O'Keefe, J.H., Bybee, K.A., & Lavie, C.J. (2007). Alcohol and Cardiovascular Health. Journal of American College of Cardiology, 50(11), 1009-1014.
- Pascoe, E.A., Richman, L.S. (2009). Racial discrimination and health: a meta-analytic review. *Psychological Bulletin*, 135, (1), 531-554.

- Pletcher, M.J. Varsoy, P., Kiefe, C.L., Lewis, C.E., Sidney, S., & Hulley, S.B. (2005).
  Alcohol consumption, binge drinking, and early coronary calcification: finding from the coronary artery risk development in young adults (CARDIA) study. *American Journal of Epidemiology*, 161(5), 423-433.
- Potter, J.F., & Beevers, D.G. (1984) Pressor effect of alcohol in hypertension. *Lancet*, 2 (1): 119-122.
- Poulson, R.L., Eppler, M.A., Satterwhite, T.N., Wuensch, K.L., Bass, L.A.(1998). Alcohol consumption, strength of religious beliefs, and risky sexual behavior in college students. *Journal of American College Health*, 46 (5), 227-232.

PsychologyDictionaryOrg Retrieved from http://psychologydictionary.org/psychosocial-factors/

- Rehm, J., Baliunas, D., Borges, G.L., Graham, K., Irving, H., Kehoe, T., Parry, C.D.,
  Patra, J., Popova, S., Poznyak, V., Roerecke, M., Room, R., Samokhvalov, A.V., &
  Taylor, B. (2010). The relation between different dimensions of alcohol consumption
  and burden of disease: an overview. *Addiction* 105(5):817-843.
- Rehm, J., Taylor, B., & Room, R. (2006). Global burden of disease from alcohol, illicit drugs and tobacco. *Drug Alcohol Review*, 25(6),503-513.
- Rehm, J., Room, R., Monterio, M., Gmel, G., Graham, K., Rehn, N., Sempos, C.T., & Jernigan, D., (2003). Alcohol as a risk factor for global burden of disease. *Europe Addiction Research*, 9(4),157-64.
- Remington PL, Brownson RC, Wegner MV (2010). *Chronic Disease Epidemiology and Control*. 3<sup>rd</sup> Edition.American Public Health Association, Washington DC.
- Ridker, P. M., Vaughan, D. E., Stampfer, M. J., Glynn, R.J., & Hennekens, C. H. (1994) Association of moderate alcohol consumption and plasma concentration of endogenous

tissue-type plasminogen activator. *Journal of American Medical Association*, 272(1),929-933.

- Rimm, E.B., Willams, P., Fosher, K., Criqui, M., & Stampfer, M.J. (1999). Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors. *British Medical Journal*, 319(1), 1523-1528.
- Rimm, E.B., Giovannucci, E.L., Willett, W.C., Colditz, G.A., Acherio, A., Rosner, B., & Stampfer, M.J. (1991). Prospective study of alcohol consumption and risk of coronary disease in men. *Lancet* 388 (1): 464-468.
- Rivers, P.C. (1994) *Alcohol and Human Behavior Theory, Research and Practice*. New Jersey: Prentice Hall.
- Roerecke, M., & Rehm, J. (2012). Alcohol intake revisited: risks and benefits. *Current Atherosclerosis Reports*, 14(6),556-562.
- Rollnick S., Miller, W.R, Butler, C.C. (2008). Motivational Interviewing in Health Care. New York: Guildford Press.
- Romley, J.A., Cohen, D., Ringel, J., & Sturm, R. (2007). Alcohol and environmental justice: the density of liquor stores and bars in urban neighborhoods in the United States. *Journal of Studies on Alcohol and Drugs*, 68(1),48–55.
- Ronksley, P.E., Brien, S.E., Turner, B.J., Mukamal, K.J., & Ghali, W.A. (2011). Association of alcohol consumption with selected cardiovascular disease outcomes: a systematic review and meta-analysis. *British Medical Journal*, 3429(1), 1-13.
- Rubin , E. (1989). How alcohol damages the body. Alcohol Health Res World, 13:322-333.
- Saremi, A., Hanson, R.L., Tulloch-Reid, M., Williams, D.E., & Knowler, W.C. (2004). Alcohol consumption predicts hypertension but not diabetes. *Journal of Studies on*

Alcohol and Drugs, 65(2),184-190.

SAS 9.3 [Computer program]. Cary, NC; SAS Institute, Inc.; 2012.

- SAS Institute, Inc. (2011) SAS/STAT 9.3 User's Guide: Survey Data Analysis (Book Excerpt). SAS Publishing Cary, NC.
- Saunders, J.B., Aasland, O.G., Babor, T.F., de la Fuente, J.R., Grant, M. (1993). Development of the alcohol use disorders identification test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption-II. *Addiction*, 88 (6), 791-804.
- Saunders, J.B., Beevers, D.G., & Paton, A. (1981). Alcohol-induced hypertension. *Lancet*, 2(1), 653-656.
- Schmidt, L.A., Yu, Y., Greenfield, T.K., & Bond, J. (2007) Ethnic disparities in clinical severity and services for alcohol problems: results from the national alcohol survey. *Alcohol Clinical Experiment Research*, 31(1),48-56.
- Scott, D.M. & Taylor, R.E. (2007). Health related effects of genetic variations of alcoholmetabolizing enzymes in African-Americans. *Alcohol Researc Health*, 1(1),18-21.
- Scott, D.M., Williams, C.D., Cain, G.E., Kwagyan, J., Kalu, N., Ehlers, C.L., Hesselbrock, V., & Taylor, R.E. (2008). Clinical course of alcohol dependence in African Americans.
   *Journal of Addictive Disease*, 27(4),43-50.
- Schwartz G.E. (1982). Testing the biopsychosocal model: the ultimate challenge facing behavioral medicine? *Journal of Consulting and Clinical Psychology*, 50, 1040-1053.
- Semba R.D., Ferrucci L., Bartali, B., Uripi-Sarda, M., Zamora-Ros, R., Sun, K., Cherubini A., Bandinelli, S., Andres-Lacueva C. (2014). Resveratrol Levels and All-Cause Mortality in Older Community- Dwelling Adults. Journal American Medical Association 1:1-8

- Sempos, C.T., Rehm, J., Wu, T., Crespo, C.J., & Trevisan, M. (2003). Average volume of alcohol consumption and all-cause mortality in african Americans: the NHEFS cohort. *Alcohol Clinical Experiment Research*, 27(1),88-92.
- Shield, K.D, Parry, C., & Rehm, J (2014). Chronic diseases and conditions related to alcohol use. *Alcohol Research*, 35 (2): 155-171.
- Singh A, Yu F, Wilson DH. (2004). Measures of information loss and disclosure risk under MASSC treatment of micro-data for statistical disclosure limitation. *Proceedings of the* 2004 Joint Statistical Meetings, American Statistical Association, Section on Survey Research Methods, Toronto, Canada (pp. 4374-4381). Alexandria, VA: American Statistical Association.
- Skidmore, J.R., Murphy, J.G., Martens, M., & Dennhardt, A,A, (2012). Alcohol–related Consequences in African American and European American college students. *Journal of Ethnic Substance Abuse*, 11(1), 174:191.
- Smedely, B.D., Stith, A.Y., & Nelson A.R., eds. (2002). Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care. Washington, DC: National Academy Press.
- Smothers, B., & Bertolucci, D. (2001). Alcohol consumption and health-promotion behavior in a US household sample: leisure-time physical activity. *Journal of Studies on Alcohol and Drugs*, 62(4), 467-476.
- Spruill, T.M. (2010). Chronic psychosocial stress and hypertension. *Current Hypertension Reports*, 12(1), 10-16.
- Stampfer, M.J., Colditz, G.A., Willett, W.C., Speizer, F.E., & Hennekens, C. H. (1988). A prospective study of moderate alcohol consumption and the risk of coronary disease and

stroke in women. New England Journal of Medicine, 319(1), 267-273.

- Steffens, A.A., Moreira, L.B., Fuch, S.C., Wiehe, M., Gus, M., Fuchs F. D. (2006). Incidence of hypertension by alcohol consumption: is it modified by race? *Journal of Hypertension*, 24(1), 1489-1462.
- Sternthal, M.J., Slopen, N., & Williams, D.R. (2011). Racial disparities in health: how much does stress really matter? *Du Bois Review*, 8(1), 95-111.
- Stewart, J.A. (2006). The detrimental effects of allostatsis: allostatic load as a measure of cumulative stress. *Journal of Physiology Anthropology*, 25(1), 133-145.
- Svardsudd, K.(1998). Moderate alcohol consumption and cardiovascular diseases: is there evidence for a preventive effect? *Alcohol Clinical Experiment Research*, 22(7),307-314S.
- Substance Abuse and Mental Health Services Administration (SAMSHA). (2012) Retrieved from <u>http://www.samhsa.gov/</u>
- Substance Abuse and Mental Health Data Archive (SAMHDA) Retrieved from www.icpsr.umich.edu/icpsrweb/SAMHDA/
- Substance Abuse and Mental Health Services Administration (SAMSHA). (2009) Results from the 2008 National Survey on Drug Use and Health: National Findings. NSDUH Series H-36, HHS Publication No. SMA 09-4434. Rockville, MD: SAMSHA
- Taherzadeh, Z., Brewster, L.M., van Montfrans, G.A., & VanBavel, E. (2010). Function and structure of resistance vessels in black and white people. Journal of Clinical *Hypertension*, 12(6), 431-438.
- Taylor, B., Irving, H.M., Baliunas, D., Roerecke, M., Patra, J., Mohapara, S., & Rehm, J.(2009). *Addiction*, 104(1), 1981-1990.

- Thomas, K.S., Nelesen, R.A., Malcaren, V.L., Ziegler, M.G., & Dimsdale, J.E. (2006). Ethnicity, perceived discrimination, and vascular reactivity to phenylephrine. *Psychosomatic Medicine*, 68(5), 692-697.
- Thomason, M., Fulton, M., & Elton, R.A. (1988). Alcohol consumption and nutrient intake in middle-aged Scottish men. *American Journal of Clinical Nutrition*, 47(1), 139-145.
- Tobe, S. W., Kiss, A., Szalai, J.P., Perkins, N., Tsigoulis, M., Baker, B. (2005). Impact of job and marital strain on ambulatory blood pressure results from the double exposure. American Journal of Hypertension 18(8), 1046-1051.
- Turner-Musa, J., Lipscomb,L. (2007). Spirtuality and social support on health behaviors of african american undergraduates. *American Journal of Health Behavior*, 31 (5): 495-501.
- Uchino, B., Cacioppo, J., Kiecolt-Glaser, J. (1996). The relationship between social support and physiological processes: areive with emphasis on underlying mechanisms and implications for health. *Psychologic Bulletin*, 119, (3),488-531.
- U.S. Preventive Services Task Force. Recommendations (2013), Retrieved from http://www.uspreventiveservicestaskforce.org/recommendations.htm
- U.S. Department of Health & Human Services, Agency for Healthcare Research & Quality, National Healthcare Disparities Report 2007 1-2 (2008). Available at http://archive.ahrq.gov/qual/nhdr07.pdf.
- Wakabayashi, I. (2008). Influence of gender on the association of alcohol drinking with blood pressure. *American Journal of Hypertension*, 21(12):1310-1317.
- Wannamethee, G., & Shaper, A.G. (1991). Alcohol intake and variations in blood pressure by day of examination. *Journal of Human Hypertension*, 5 (2), 59-67.

- Ward BW, Schiller JS, Goodman RA. Multiple chronic conditions among US adults: a 2012 update. *Preventing Chronic Diseases*, 2014, 11,(1), 1-5.
- Warnecke, R.B., Oh, A., Breen, N., Gehlert, S., Paskett, E., Tucker, K.L., Lurie, N.,
  Rebbeck, T.,Goodwin, J., Flack, J., Srinivasan, S., Kerner, J., Heurtin-Roberts, S.,
  Abeles, R., Tyson, F.L., Patmios, G., & Hiatt, R.A. (2008). Approaching health
  disparities from a population perspective: the National Institutes of Health Centers
  for Population Health and Health Disparities. *American Journal Public Health*,
  98 (9),1608-1615.
- Wells, K., Klap, R., Koike, A., & Sherbourne, C. (2001). Ethnic disparities in unmet need for alcoholism, drug abuse, and mental health care. *American Journal of Psychiatry*, 158(12), 2037-2032.
- Whelton, P.K., He, J., Appel, L.J., Culter, J.A., Havas, S., Kotchen, T.A., Roccella, E.J.,
  Stout, R., Vallbona, C., Winston, M.C., Karimbakas, J., & National High Blood
  Pressure Education Program Coordinating Committee (2002). Primary prevention
  of hypertension:clinical and public health advisory from The National High Blood
  Pressure Education Program. *Journal of American Medical Association*, 288(15),
  1882-1888.
- Williams, D. R, & Sternthal, M (2010). Understanding racial-ethnic disparities in health: sociological contributions. *Journal of Health and Social Behavior*, 51(S), s15-s27.
- Williams, D. R, & Mohammed, S.A. (2009). Discrimination and racial disparities in health: evidence and needed research. *Journal of Behavioral Medicine*, 32(1), 20-47.
- Wilson, P.W.(1998). Diabetes mellitus and coronary heart disease. *American Journal of Kidney Dis*ease, 32(5 Supp 3), S89-S100.

- Woods-Giscombe, C.L., & Black, A.R. (2010) Mind-body interventions to reduce risk for health disparities related to stress and strength among African American women: The potential of mindfulness-based stress reduction, loving-kindness, and the NTU therapeutic framework. *Complementary Health Practice Review*, 15 (1), 115-131.
- Wong, M.D., Shapiro, M.F., Boscardin, W.J., & Ettner, S.L (2002). Contribution of major diseases to disparities in mortality. *New England Journal of Medicine* 347 (20),1585-1592.
- World Health Organization (2014) Global Status Report on Alcohol and Health 2014. Retrieved from <a href="http://www.who.org">www.who.org</a>
- World Health Organization (2012). Assessing national capacity for the prevention and control of NCDs: report of the 2010 global survey Retrieved from <a href="http://www.who.int/chp/en/">http://www.who.int/chp/en/</a>
- Wu, L.T., Woody ,G.E., Yang, C., Pan, J.J., & Blazer, D.G. (2011) Racial/ethnic variations in substance related disorders among adolescents in the United States. *Archives General Psychiatry*, 68(1), 1176-1185.
- Xin, X.,He, J., Frontini., M.G., Ogden, L.G., Motsamai, O.I., Whelton, P.K. (2001). Effects of alcohol reduction on blood pressure: a meta-analysis of randomized controlled trials. Hypertension 38:1112-1117.
- Yach, D., Hawkes C., Gould, C., & Hofman K. (2004). The global burden of chronic diseases Journal of American Medical Association, 291(21), 2616-2622.
- Yusuf, S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, McQueen M, Budaj A, Pais P, Varigos J, Lisheng L. & Interheart Study Investigators (2004). Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the Interheart study): case-control study. *Lancet*, 364(9438), 937-952.

- Zakhari, S. (2006). Overview: How is alcohol metabolized by the body? *Alcohol Research* & *Health*, 29(4),245-254.
- Zilkens, R.R.,& Puddey, I.B.(2003). Alcohol and cardiovascular disease: more than one paradox to consider. Alcohol and type 2 diabetes: another paradox? *Journal of Cardiovascular Risk*, 10 (1), 25-30.

## **APPENDIX A**

## SAS CODE

Program: combined analysis2 Created By: Vanessa Marshall and Scott Grey Infiles: NSDUH08\26701 NSDUH09\29621 NSDUH10\32722, NSDUH11\34481, NSDUH12\34933. Outfiles: alc.comb Usage: SAS 9.3. Code uses the DOMAIN statement and NOMCAR option to create a subset of subjects that match study inclusion criteria and preserve the correct sample weighting and variance calculations. Results from zero domain ONLY should be considered. Purpose: Conduct weighted analysis of five years of NSDUH data. /\*\* \*\*/ Import Data **PROC CIMPORT** INFILE="C:\Users\Public\Documents\Scott Grey Work Documents\Grey Work Files \Alc data\NSDUH08\26701-0001-Data.stc" LIB=WORK; RUN: **PROC CIMPORT** INFILE="C:\Users\Public\Documents\Scott Grey Work Documents\Grey Work Files \Alc data\NSDUH09\29621-0001-Data.stc" LIB=WORK; RUN; **PROC CIMPORT** INFILE="C:\Users\Public\Documents\Scott Grey Work Documents\Grey Work Files \Alc data\NSDUH10\32722-0001-Data.stc" LIB=WORK; RUN: PROC CIMPORT INFILE="C:\Users\Public\Documents\Scott Grey Work Documents\Grey Work Files \Alc data\NSDUH11\34481-0001-Data.stc" LIB=WORK; RUN; **PROC CIMPORT** INFILE="C:\Users\Public\Documents\Scott Grey Work Documents\Grey Work Files \Alc data\NSDUH12\34933-0001-Data.stc" LIB=WORK; RUN; \*\*/ /\*\* Create Combined Dataset /\*\*\*\* Keep variables used for analyses or weighting \*\*\*\*/ DATA WORK.data08 (KEEP = income irsex newrace2 irmarit cataq3 edfam18 educcat2 dstworst alcrec abusealc depndalc hbplif diablif analwt c vestr verep year); SET work.Da26701p1;

```
year = 2008; /* Create year var */
RUN;
DATA WORK.data09
  (KEEP = income irsex newrace2 irmarit catag3 edfam18 educcat2
   dstworst alcrec abusealc depndalc hbplif diablif analwt c vestr
verep year);
 SET work.Da29621p1;
 year = 2009; /* Create year var */
RUN;
DATA WORK.data10
  (KEEP = income irsex newrace2 irmarit catag3 edfam18 educcat2
   dstworst alcrec abusealc depndalc hbplif diablif analwt c vestr
verep year);
 SET work.Da32722p1;
 year = 2010; /* Create year var */
RUN;
DATA WORK.data11
  (KEEP = income irsex newrace2 irmarit catag3 edfam18 educcat2
   dstworst alcrec abusealc depndalc hbplif diablif analwt c vestr
verep year);
 SET work.Da34481p1;
 year = 2011; /* Create year var */
RUN;
DATA WORK.data12
  (KEEP = income irsex newrace2 irmarit cataq3 edfam18 educcat2
   dstworst alcrec abusealc depndalc hbplif diablif analwt c vestr
verep year);
 SET work.Da34933p1;
 year = 2012; /* Create year var */
RUN;
/*** Merge to combine data set in permanent directory Alc data ***/
libname alc "C:\Users\Public\Documents\Scott Grey Work Documents\
Grey Work Files\Alc data\";
DATA alc.comb;
 SET WORK.data08 WORK.data09 WORK.data10 WORK.data11 WORK.data12;
RUN;
/** Create variables needed for analysis **/
/* Create categories for alcohol use */
DATA alc.comb; SET alc.comb;
```

```
120
```

```
alc cat = 0;
 IF (alcrec < 3) THEN alc cat = 1;
 IF (abusealc EQ 1) OR (depndalc EQ 1) THEN alc cat = 2;
/* Create new variable for high blood pressure to exclude unknowns */
hbplif2 = .;
 IF (hbplif = 1) THEN hbplif2 = 1;
 IF (hbplif = 0) THEN hbplif2 = 0;
/* Create new variable for diabetes to exclude unknowns */
diablif2 = .:
 IF (diablif = 1) THEN diablif2 = 1;
 IF (diablif = 0) THEN diablif2 = 0;
/* Create new variable for age to exclude 12-17 year olds */
catag3age= .;
 IF (catag3 = 2) THEN catag3age = 1;
 IF (catag3 = 3) THEN catag3age = 2;
 IF (catag3 = 4) THEN catag3age = 3;
 IF (catag3 = 5) THEN catag3age = 4;
/* Create new variables for DSTWORST and EDFAM18 */
dstworst2 = .;
  IF (dstworst = 1) THEN dstworst2 = 1;
  IF (dstworst = 2) THEN dstworst2 = 0;
edfam18 2 = .;
  IF (edfam18 = 0) THEN edfam18 2 = 1;
  IF (edfam18 = 1) THEN edfam18 2 = 0;
/* Create new variable for NEWRACE2 to exclude other racial groups */
race = \cdot;
 IF (newrace2 = 1) THEN race = 0;
 IF (newrace2 = 2) THEN race = 1;
 IF (newrace2 = 7) THEN race = 2;
RUN;
/**** Create new person weighing variable to correct for using 5 yrs
data ****/
DATA alc.comb; SET alc.comb;
 newwt c = analwt c/5;
RUN;
/**** Create domain variable to subset data for correct weighing ****/
DATA alc.comb; SET alc.comb;
domain = 0;
  IF (hbplif2 = .) OR (diablif2 = .) OR (catag3age = .)
 OR (dstworst2 = .) OR (edfam18 2 = .) OR (race = .)
 THEN domain = 1;
RUN;
```

```
/* Look at missing data and created variables by domain variable */
DATA alc.comb; SET alc.comb;
 MISS N = CMISS(OF alc cat -- race);
RUN;
PROC SORT DATA = alc.comb; BY domain; RUN;
PROC FREQ DATA = alc.comb;
     TABLES MISS N alc cat irsex race income irsex catag3age irmarit
educcat2
          edfam18 2 dstworst2 year;
     BY domain;
RUN;
/* Re-format categorical variables */
proc format;
value alcoholcat 0= "no use"
               1 = "use"
                   2 = "abuse or dep.";
value race
            2="Hispanic"
             1="Black"
                0="White";
             1="< $20"
value money
             2="$20-$40"
                3="$41-$75"
                4="$75-more";
             1="18-25"
value age
             2="26-34"
             3="35-49"
             4="50";
value relationship
                   1="married"
                   2="widowed"
                   3="divorced/sep"
                   4="never mar.";
run;
**/
/**
         Weighted Demmographics
PROC SORT DATA = alc.comb; BY vestr verep; RUN;
ODS PDF FILE= 'C:\Users\sgrey\Documents\Work
Docs\KSU\Alc data\weighted5.pdf';
```

```
TITLE "Weighted Demmographics";
PROC SURVEYFREQ DATA = alc.comb;
  FORMAT alc cat alcoholcat.; FORMAT race race.; FORMAT income
money.;
  FORMAT cataq3age age.; FORMAT irmarit relationship.;
  TABLES domain* (hbplif2 diablif2 alc cat race income irsex catag3age
                 irmarit educcat2 edfam18 2 dstworst2 year)
                 / NOSPARSE NOCELLPERCENT NOSTD ROW;
                      /* Look only at 0 results! */
  STRATA vestr;
  CLUSTER verep;
  WEIGHT newwt c;
RUN;
TITLE "Weighted Demmographics by Alcat and Race";
PROC SURVEYFREQ DATA = alc.comb;
  FORMAT alc cat alcoholcat.; FORMAT race race.; FORMAT income
money.;
  FORMAT catag3age age.; FORMAT irmarit relationship.;
  TABLES domain*alc cat* (hbplif2 diablif2)
         domain*race*(hbplif2 diablif2)
         domain*race*(alc cat income irsex catag3age irmarit educcat2
                     edfam18 2 dstworst2 year)
         / NOSPARSE NOCELLPERCENT NOSTD ROW;
            /* Look only at 0 results! */
  STRATA vestr;
  CLUSTER verep;
  WEIGHT newwt c;
RUN:
/**
             Weighted Analysis
                                       **/
/********* Hi BP Outcome ***********/
/** Main effects and interaction only **/
TITLE "Hi BP Logistic Regression with Main Effects and Interaction";
PROC SURVEYLOGISTIC DATA=alc.comb NOMCAR;
  FORMAT alc cat alcoholcat.; FORMAT race race.;
  CLASS alc cat race / DESC PARAM=GLM;
  MODEL hbplif2(event='1') = alc cat|race/ EXPB;
  LSMEANS alc cat*race / ILINK CL ADJUST=SIMULATE;
  SLICE alc cat*race / SLICEBY=race ODDSRATIO CL ADJUST=SIMULATE;
  SLICE alc cat*race / SLICEBY=alc cat ODDSRATIO CL ADJUST=SIMULATE;
  DOMAIN domain; /* Look only at 0 domain results! */
  STRATUM vestr;
  CLUSTER verep;
  WEIGHT newwt c;
```

RUN;

```
/** Main effects, interaction and covars, FULL MODEL **/
TITLE "Hi BP Logistic Regression with Main Effects, Interaction
       and Covars, FULL MODEL";
PROC SURVEYLOGISTIC DATA=alc.comb NOMCAR;
   FORMAT alc cat alcoholcat.; FORMAT race race.; FORMAT income
money.;
   FORMAT catag3age age.; FORMAT irmarit relationship.;
   CLASS alc cat race income irsex catag3age irmarit educcat2
edfam18 2
         dstworst2
         / DESC PARAM=GLM;
  MODEL hbplif2(event='1') = alc cat|race income irsex catag3age
irmarit
         educcat2 edfam18 2 dstworst2/ EXPB;
   LSMEANS alc cat*race / ILINK CL ADJUST=SIMULATE;
   SLICE alc cat*race / SLICEBY=race ODDSRATIO CL ADJUST=SIMULATE;
   SLICE alc cat*race / SLICEBY=alc_cat ODDSRATIO CL ADJUST=SIMULATE;
   DOMAIN domain; /* Look only at 0 domain results! */
   STRATUM vestr;
  CLUSTER verep;
  WEIGHT newwt c;
RUN;
TITLE "Hi BP Logistic Regression with Main Effects, Interaction and
Covars,
       EXTENDED MODEL";
PROC SURVEYLOGISTIC DATA=alc.comb NOMCAR;
   FORMAT alc cat alcoholcat.; FORMAT race race.; FORMAT income
money.;
   FORMAT catag3age age.; FORMAT irmarit relationship.;
   CLASS alc cat race income irsex catag3age irmarit educcat2
edfam18 2 dstworst2
         year / DESC PARAM=GLM;
  MODEL hbplif2(event='1') = alc cat|race|year income irsex cataq3age
irmarit
         educcat2 edfam18 2 dstworst2/ EXPB;
   DOMAIN domain; /* Look only at 0 domain results! */
   STRATUM vestr;
  CLUSTER verep;
  WEIGHT newwt c;
RUN;
/** Main effects, interaction and covars, REDUCED MODEL **/
TITLE "Hi BP Logistic Regression with Main Effects, Interaction and
Covars,
       REDUCED MODEL";
```

```
PROC SURVEYLOGISTIC DATA=alc.comb NOMCAR;
```

```
FORMAT alc cat alcoholcat.; FORMAT race race.; FORMAT income
money.;
   FORMAT catag3age age.; FORMAT irmarit relationship.;
   CLASS alc cat race income irsex catag3age irmarit educcat2
dstworst2
         / DESC PARAM=GLM;
  MODEL hbplif2(event='1') = race|alc cat income irsex catag3age
irmarit
        educcat2 dstworst2/ EXPB;
  LSMEANS race*alc cat / ILINK CL ADJUST=SIMULATE;
   SLICE race*alc cat / SLICEBY=race ODDSRATIO CL ADJUST=SIMULATE;
   SLICE race*alc_cat / SLICEBY=alc cat ODDSRATIO CL ADJUST=SIMULATE;
  DOMAIN domain; /* Look only at 0 domain results! */
  STRATUM vestr;
  CLUSTER verep;
  WEIGHT newwt c;
RUN;
/********* Diabetes Outcome **********/
/** Main effects and interaction only **/
TITLE "Diabetes Logistic Regression with Main Effects and
Interaction";
PROC SURVEYLOGISTIC DATA=alc.comb NOMCAR;
   FORMAT alc cat alcoholcat.; FORMAT race race.;
   CLASS alc cat race / DESC PARAM=GLM;
  MODEL diablif2(event='1') = alc_cat|race/ EXPB;
  LSMEANS alc cat*race / ILINK CL ADJUST=SIMULATE;
   SLICE alc cat*race / SLICEBY=race ODDSRATIO CL ADJUST=SIMULATE;
   SLICE alc cat*race / SLICEBY=alc_cat ODDSRATIO CL ADJUST=SIMULATE;
  DOMAIN domain; /* Look only at 0 domain results! */
   STRATUM vestr;
  CLUSTER verep;
  WEIGHT newwt c;
RUN;
/** Main effects, interaction and covars, FULL MODEL **/
TITLE "Diabetes Logistic Regression with Main Effects, Interaction and
Covars,
       FULL MODEL";
PROC SURVEYLOGISTIC DATA=alc.comb NOMCAR;
   FORMAT alc cat alcoholcat.; FORMAT race race.; FORMAT income
money.;
   FORMAT catag3age age.; FORMAT irmarit relationship.;
   CLASS alc cat race income irsex catag3age irmarit educcat2
edfam18 2 dstworst2
        / DESC PARAM=GLM;
   MODEL diablif2(event='1') = alc cat|race income irsex catag3age
irmarit
         educcat2 edfam18 2 dstworst2/ EXPB;
```

```
LSMEANS alc cat*race / ILINK CL ADJUST=SIMULATE;
   SLICE alc cat*race / SLICEBY=race ODDSRATIO CL ADJUST=SIMULATE;
   SLICE alc cat*race / SLICEBY=alc cat ODDSRATIO CL ADJUST=SIMULATE;
DOMAIN domain; /* Look only at 0 domain results! */
   STRATUM vestr;
   CLUSTER verep;
   WEIGHT newwt c;
RUN;
TITLE "Diabetes Logistic Regression with Main Effects, Interaction and
Covars,
      EXTENDED MODEL";
PROC SURVEYLOGISTIC DATA=alc.comb NOMCAR;
   FORMAT alc cat alcoholcat.; FORMAT race race.; FORMAT income
money.;
   FORMAT catag3age age.; FORMAT irmarit relationship.;
   CLASS alc cat race income irsex catag3age irmarit educcat2
edfam18 2 dstworst2
        year / DESC PARAM=GLM;
   MODEL diablif2(event='1') = alc_cat|race|year income irsex
catag3age irmarit
         educcat2 edfam18 2 dstworst2/ EXPB;
   DOMAIN domain; /* Look only at 0 domain results! */
   STRATUM vestr;
  CLUSTER verep;
   WEIGHT newwt c;
RUN;
ODS PDF CLOSE;
```