2016

Effect of Genetic Background Combined with Excessive Media Screen Time on Markers of Cardiovascular Risk in United States Youth Aged Newborn to 20 Years

Maria Moroni
Walden University

Walden University

College of Health Sciences

This is to certify that the doctoral dissertation by

Maria Moroni

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

Review Committee
Dr. Richard Jimenez, Committee Chairperson, Public Health Faculty
Dr. Ronald Hudak, Committee Member, Public Health Faculty
Dr. Ernest Ekong, University Reviewer, Public Health Faculty

Chief Academic Officer
Eric Riedel, Ph.D.

Walden University
2016

Abstract

Effect of Genetic Background Combined with Excessive Media Screen Time on Markers of Cardiovascular Risk in United States Youth Aged Newborn to 20 Years

by

Maria Moroni

MA, University of Pisa, Italy, 1997

BA, University of Pisa, Italy, 1994

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Epidemiology

Walden University

August 2016

Abstract

Time with media screens (television, computers, videogames, cell phones, and tablets) is the primary activity of youth, second only to sleeping, and represents a major risk factor for cardiovascular diseases (CVD). Additionally, the populations with highest rates of screen time are also those most at risk of CVD from genetic predisposition (i.e., Blacks, Hispanics). The purpose of this descriptive, correlational study, based on cross-sectional analysis of archived data from the 2009 – 2010 NHANES for United States youth, newborn to 20 years old, was to determine whether the combination of media screen time with genetic background is a better predictor of CVD than either factor alone. The theoretical framework was the social ecological theory of disease distribution. The relationship between media screen time, genetic background, and CVD risk factor was determined using binary logistic regression. Results of this study indicated that the relationship between ethnicity, gender, and type/duration of exposure to media screen is important to predict the CVD risk factors C-reactive protein (CRP), triglycerides, and diastolic blood pressure. Interventions that limit exposure total screen time will reduce the risk of increased blood pressure among all races. However, culturally relevant intervention should be designed specifically for non-Hispanic Blacks, other Hispanics, and other race. These ethnicities have the highest propensity to increase in blood pressure, CRP, and triglycerides and also spend the largest amount of time in front of the media screen. Results from this study may help to promote policies and initiatives to limit screen time that are culturally relevant and more focused.

Effect of Genetic Background Combined with Excessive Media Screen Time on Markers of Cardiovascular Risk in Youth Aged Newborn to 20 Years

by

Maria Moroni

MA, University of Pisa, Italy, 1997

BA, University of Pisa, Italy, 1994

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Epidemiology

Walden University

August 2016

Acknowledgments

I wish to extend special thanks to the committee members, Dr Jimenez and Dr Hudak, for helping me complete this work and providing guidance throughout the entire process.

Table of Contents

List of Tables

List of Figures

Chapter 1: Introduction to the Study

An increase in media technology use has paralleled the increase in the obesity rate and sedentary life in the last 30 years, bringing with it a variety of physical, psychological, and health issues (Rosen et al., 2014). Media entertainment has become the primary activity of U.S. youth when not asleep, with major negative consequences on physical and mental health (Parkes, Sweeting, Wight, & Henderson, 2013). Age and socioeconomic status (SES) are among the highest risk factors for engaging in activities on the media screen (Carson, Rosu, & Janssen, 2014). Moreover, families of lower SES status and ethnic minorities are already at a higher risk of health problems, including obesity and cardiovascular diseases (CVD), and lower quality of care (Rideout, Foehr, & Roberts, 2010). Teenagers, who have access to all varieties of technology and are the primary targets of media tech companies, are the age group most at risk (Rideout, Foehr, & Roberts, 2010).

Increased risk of obesity, CVD, and dyslipidemia are only some of the health outcomes for youth, 2 to 18 years old, exposed to an excessive amount of media screen time (Rosen et al., 2014). Excessive screen time is conducive to lower social skills, lower academic achievement, mental illnesses, and violent behavior (Rideout, 2011). Poorer behavioral conduct, such as aggression, anxiety, depression, social isolation, and ADHD is observed even if programs are designed specifically for youth and aggravated in the case of programs with violent or sexually explicit content (Parkes et al., 2013).

The American Pediatric Association recommended that involvement with media entertainment (consisting of videogames, computers, television, music, and other audio) be limited to two 2 hours per day for school age youth and avoided for youth less than

under two 2 years of age (as cited in Vanderloo, 2014). Instead, on average, youth in the United States spend up to 7 hours per day in front of a media screen (Rideout et al., 2010). Multitasking (i.e., searching the Internet or texting while watching television or videos) increases the active screen time to over 10 hours (Rideout et al., 2010). According to Vanderloo (2014) and Grøntved et al. (2014), screen time behavior learned during childhood and early adolescence increases the risk for obesity and CVD in adults.

The implications for positive social change of this study, in which I aimed to understand whether excessive media screen time can enhance the genetic predisposition to risk factors, include the development of ethnic and culturally relevant policies to reduce the risk of CVD. In this chapter, I provide a brief summary of the literature related to the effect of genetic background combined with excessive media screen exposure on markers of CVD risk in youth aged newborn to 20 years. I also introduce the problem, the purpose, and the research questions that I addressed in this study, within the context of the social ecological theory, and describe the study design, its assumptions, scope and limitations, and significance.

Background

The prevalence of obesity among youth has tripled in the past 10 years, reaching 20 in youth aged 6 to 19 years (Rosen et al., 2014). The epidemic of obesity and presence of CVD risk factors already in preschool age youth has been related to an increase in sedentary life at the expense of physical activity (Rideout et al., 2010). Electronic media entertainment is assumed to be largely responsible for this trend (Rideout et al., 2010), which represents a serious threat for psychological, behavioral, and physical health issues. Extensive exposure to media screen time leads to a sedentary life,

obesity, lack of sleep, low cardio-respiratory fitness, and altered lipid profiles, thus increasing the risk for CVD (Grøntved et al., 2014; Rideout et al., 2010; Rosen et al., 2014). Psychosocial and psychiatric problems such as depression, lower self-efficacy, and conduct disorders have been linked to increased Internet and television use, and videogame playing (Busch, Manders, & de Leeuw, 2013), possibly through direct modification of neurotransmitter release and brain structure and anatomy, in addition to questionable program content (Hong et al., 2013; Takeuchi et al., 2013). Screen time was found to predict aggression, social isolation among youth, shorter ability to focus, use of marijuana and alcohol, smoking, bullying, poor nutritional behaviors, lower physical activity, and skipping school (Busch et al., 2013; Parkes et al., 2013, Rosen et al., 2014).

The number of hours that youth spend on electronic media has surpassed any other activity in their lives (Rideout, 2011). The most common sedentary activity among preschool age youth is watching television or playing videogames for over 2 hours daily. This tendency occurs both in home-based and school-based childcare settings (Vanderloo, 2014). Type of childcare has a significant, direct association with the cumulative time spent with media screen, which ranges from 3.2 to 4.2 hours in childcare centers and Head Start programs to 5.5 hours in home-based care centers (Tandon, Zhou, Lozano, & Christakis, 2011). The extent of the problem is aggravated during the transition to teenager years, due to easier access to media technology (Rideout, 2011). Behavior learned during childhood will determine the type of lifestyle during adulthood and will carry along risk factors for medical conditions that will manifest later on in life (Vanderloo, 2014).

Type and extent of media use are gender and demographics dependent (Garcia-Continente, Pérez-Giménez, Espelt, & Nebot Adell, 2014; Herrick, Fakhouri, Carlson, & Fulton, 2014). After age, SES is the second strongest risk factor for spending time on media screen-associated activities (Carson et al., 2014). Excessive media screen time is prevalent among ethnic minorities and individuals with lower SES. Association between media use and ethnicity persisted after controlling for demographic factors (Rideout et al., 2010), with Hispanics and Blacks spending up to about 13 hours in front of the screen, and Whites about 8.5 hours (Rideout et al., 2010). In addition, according to Garcia-Continente et al. (2014), screen-related sedentary behavior was associated with living in a low SES neighborhood, eating unhealthy food, and not reading books frequently. The main mediators of media screen viewing were the presence of a television in the bedroom, lower access to outdoor activities, home environment, and parents' screen time habits (Appelhans et al., 2014; Tandon et al., 2011).

The association between screen time and lower SES spans across modifiable behavioral factors, such as having a television in the bedroom, having fewer opportunities for an active lifestyle, and parental lack of knowledge of the negative impact of electronics on youth's health (Martin, 2011; Stamatakis, Hamer, & Dunstan, 2011; Tandon et al., 2012). Parents often neglect to set rules and guidelines to limit exposure to media screen, either because they are not aware of the impact of electronic media on their youth's health or because parents themselves are excessively drawn to the media screen, and prefer a sedentary lifestyle to a more active and healthy conduct of living (Veldhuis, van Grieken, Renders, HiraSing, & Raat, 2014). Several studies assessing the effect of family rules, personal (demographics, parental cognition, parental behavior), and physical

environment factors (television, computer, and console in the bedroom) on television use and computer use among youth aged 5 to18 years found that personal and environmental factors together explained most of the association with screen time (Carson et al., 2014; Gingold, Simon, & Schoendorf, 2014). Parental cognition and screen time habits alone explained 38 of the association (Carson et al., 2014).

Increased risk for health issues depends on cultural and behavioral determinants as well as on genetic predisposition. The similarities in risk factors associated with both excessive media screen time and CVD suggest that media screen time may exacerbate the risk in populations with a genetic predisposition to CVD. However, it appears that no study has specifically addressed whether media screen time in association with genetic background (i.e., gender, ethnicity) increases the risk of CVD. Although genetic predisposition cannot be changed, interventions can be developed to educate and protect populations at higher risk.

In addition to modifiable behavioral factors, excessive screen time pushes the energy metabolism towards obesogenic trends and impacts major molecular pathways towards proinflammatory, proatherogenic outcomes, increasing the risk of CVD regardless of daily physical activity (Berentzen et al., 2014; Börnhorst et al., 2015; Martin, 2011; Stamatakis et al., 2011). Excessive media screen time and consequent sedentary behavior elevate blood pressure, cholesterol, triglycerides, high density lipoprotein (HDL), low density lipoprotein (LDL), fibrinogen, metabolic syndrome, C-reactive protein (CRP), and lower CVD fitness (Berentzen et al., 2014; Börnhorst et al., 2015; Martin, 2011; Stamatakis et al., 2011). Increased levels of body mass index (BMI), CRP, and HL cholesterol explained about 25 of the relationship between television

viewing and CVD risk (Stamatakis et al., 2011). The association between biomarkers of CVD and diabetes with television viewing differed from that with work-related sedentary activity (Pinto Pereira, Ki, & Power, 2012). Both direct (genetic) and indirect (behavioral) risks factors for CVD and media screen time were found increased among ethnic minorities and people with lower SES.

Interventions targeted to raise awareness of the problem of excessive screen time and to educate parents, caregivers, and youth about the physical, mental, and health risks that derive from the unhealthy use of media electronics are needed. Understanding the relationship between excessive screen time, the molecular mediators of vascular damage, and genetic predisposition to CVD is important to establish proper interventions for the prevention of CVD problems. In this study, I specifically tested the relationship between genetic background, exposure to media screen, and risk factors for CVD, which provided insights on the associations between risk factors and genetic background in youth exposed to excessive time of media screen viewing. Such findings may assist in the development of policies to reduce the risk of CVD by limiting exposure to media screen. In particular, interventions targeted to raise awareness of the problem and to educate parents, caregivers, and youth about the physical, mental, and health risk that derive from the unhealthy use of media electronics are needed.

Problem Statement

Genetic background and behavioral factors are relevant for the development of diseases (Corona et al., 2013). Risk factors of CVD include obesity, inflammation, high blood pressure, dyslipidemia, gender, age, and ethnicity. The same risk factors associated with CVD are also induced by excessive media screen time. Although a large body of

evidence supports the link between genetic determinants and excessive screen time, taken singularly, with CVD, the combined effect of genetics with exposure to media screen time has not been thoroughly investigated. Therefore, the problem being addressed is the relationships between genetic and behavioral risk factors in youth exposed to excessive screen time. Environmental and genetic determinants, as well as excessive exposure to media screen, increase the expression of risk factors of CVD diseases (i.e., inflammatory biomarkers, dyslipidemia, and blood pressure) (Rosen et al., 2014; Taylor, 2015). Media screen time may negatively affect CVD health through enhancement of inflammation, dyslipidemia, and hypertension (American Association of Pediatrics, 2012; Berentzen et al., 2014; Chinapaw, Proper, Brug, van Mechelen, & Singh, 2011).

The distribution of genetic determinants of CVD time is uneven across ethnicities (Morris & Keith, 2009). The tendency to devote a disproportionate length of time in front of a media screen and to have a television in the bedroom is highest among youth of low SES and minorities, populations already at higher risk for CVD (Kurian & Cardarelli, 2007; Rideout, 2011). Although previous researchers have analyzed in depth the relationship between risk of CVD and genetic background or CVD and excessive exposure to media screen time, the synergy between genetic back ground and excessive exposure to media screen to increase risk of CVD has not been thoroughly explored.

Purpose of the Study

Previous researchers have assessed either the genetic predisposition to CVD or the consequences of disproportionate exposure to media screen on biomarkers of CVD (Mathews et al., 2014; Pinto Pereira et al., 2012; Stamatakis et al., 2011; Väistö et al., 2014; World Health Federation, 2015). The purpose of this quantitative study was to

determine the effect of genetic background combined with excessive media screen time on markers of CVD risk in United States youth aged newborn to 20 years. Newborn is defined here as less than 1 year old. Therefore, the age range in the research questions is expressed as 0 to 20 years old. The independent variables are media screen time, gender, and race/ethnicity. The dependent variables are CRP, lipids, and blood pressure.

Research Questions and Hypotheses

Risk factors of CVD are CRP, lipids, and blood pressure. The research questions that I answered are as follows:

1. RQ1. For U.S. youth 0 to 20 years old, what is the relationship between media screen time, genetic background (gender, race/ethnicity), and CRP?

   Null hypothesis. There is no relationship between media screen time, genetic background (gender, race/ethnicity), and CRP in U.S. youth aged 0 to 20 years old.

   Alternative hypothesis. There is a relationship between media screen time, genetic background (gender, race/ethnicity), and CRP in U.S. youth aged 0 to 20 years old.

2. RQ2. For U.S. youth 0 to 20 years old, what is the relationship between media screen time, genetic background (gender, race/ethnicity), and lipids (HDL, LDL, and triglycerides)?

   Null hypothesis. There is no relationship between media screen time, genetic background (gender, race/ethnicity), and lipids in U.S. youth aged 0 to 20 years old.

Alternative hypothesis. There is a relationship between media screen time, genetic background (gender, race/ethnicity), and lipids, in U.S. youth aged 0 to 20 years old.

3. RQ3. For U.S. youth 0 to 20 years old, what is the relationship between media screen time, genetic background (gender, race/ethnicity), and blood pressure (diastolic and systolic)?

   Null hypothesis. There is no relationship between media screen time, genetic background (gender, race/ethnicity), and blood pressure in U.S. youth aged 0 to 20 years old.

   Alternative hypothesis. There is a relationship between media screen time, genetic background (gender, race/ethnicity), and blood pressure in U.S. youth aged 0 to 20 years old.

The variables were categorical or continuous.

Theoretical and/or Conceptual Framework for the Study

The determinants at the origin of specific phenomena must be understood for the development of proper evidence-based interventions. The social ecological theory proposes that personal relationships, the physical environment, settings, and policies determine an individual's behavior and health (Carson & Janssen, 2012). As the interaction between the environment and the individual may cause epigenetic changes that vary according to the genetic make-up of the individual, the effect of the environment (i.e., exposure to media screen) on youth of various genetic backgrounds deserves immediate attention.

Theoretical Foundation

The theoretical framework for this study is the social ecological theory of disease distribution. The theory was introduced in the late 70s by Bronfenbrenner, a developmental psychologist, who postulated that in order to understand human development, the entire ecological system surrounding the individual as well as relevant biological and genetic aspects involved in the development must be taken into consideration (Brofenbrenner, 1977). The social ecological theory thus accounts for the complex interaction between individuals, the social and physical environment, and biological processes (Krieger, 2011). The individual is placed in subsystems, each of which influences and is influenced by the individual, and that support and guide human development. The subsystems consist of the microsystem, the mesosystem, the exosystem, the macrosystem, and the chronosystem. These systems cover the interaction of the individual with the immediate environment (microsystem), the interplay among several microsystems (mesosystem), and spheres of influence not immediately in contact with the individual but indirectly relevant to the development (exosystem). The macrosystem and the chronosystem represent higher levels of influence; they consist of laws, moral and cultural values, economical and political events, and changes along a person's life. The individual's genetic composition and its physical expression mediate how people react to stressors and adversity (Van Cleve & Akçay, 2014).

A detailed explanation of the social ecological theory and its subsystems is presented in Chapter 2. The relevance of this theory to the study of the interaction between genetic background and excessive screen time consists in the recognition that both genetics and the environment play an important role in the development of CVD

complications. The interaction between the individual and the environment is bidirectional, meaning that as the surroundings impact the health status of people, depending on their genetic make-up, people should modify surroundings in order to achieve better health. The complex interaction of excessive screen time with health, resulting in the displacement of physical activity by a media-based sedentary life, in a metabolic switch to obesogenic pathways, and in alteration of brain structure and physiology, demands simultaneous interventions at all systems' levels impacting the life of the individual. This concept is supported by the fact that intervention for preventing obesity is now starting to involve state regulations for school-based initiatives promoting healthy nutrition and physical activity.

Conceptual Framework

Individual genetic predisposition and attitudes, demographics, sociocultural and environmental determinants, and parental beliefs influence the child's attitude towards media screen time and its impact on health and development. The behavioral component of media screen-triggered onset of CVD risk factors is tightly tied to ethnicity and SES, as indicated by the higher prevalence of excessive media screen time among ethnic minorities and individuals of lower SES. Research has shown that African-American, Latino, and youth from families with low SES status spend more time in front of the screen than White youth, and that low active play and extended television viewing are positively correlated with age, gender, race/ethnicity, and BMI (Anderson, Economos, & Must, 2008; Rideout, 2011). Therefore, an interaction between genetic background, social- and physical-environment, and health outcome must be postulated and further explored in the context of excessive exposure to media screen time.

Nature of the Study

To explore the relationship between genetic backgrounds, media screen time, and risk factors for CVD, I used a quantitative approach that used archived data from the National Health and Nutrition Examination Survey (NHANES, 2011a-d). The independent variables were media screen time, gender, and ethnicity. The dependent variables were CRP, lipids, and blood pressure.

The research design was a descriptive, correlational design based on cross-sectional analysis of data from the 2009 – 2010 NHANES. The choice of the dataset was dependent upon data availability. The study was limited to youth, aged 0 to 20 years, for whom demographic data (age, gender, ethnicity), laboratory data (lipids, CRP, blood pressure), and behavioral data (media screen time) were known.

Definitions

The independent variables were media screen time, gender, and ethnicity. The dependent variables were CRP protein, lipids, and blood pressure. Gender (boys, girls) and ethnicity (Mexican American, other Hispanic, non-Hispanic White, non-Hispanic Black, and other race) were operationalized by NHANES as nominal and categorical, respectively (NHANES, 2011a).

According to the NHANES website, the race/ethnicity reflected individuals' self-identification as Mexican American, other Hispanic, non-Hispanic white, non-Hispanic Black, and other non-Hispanic race including non-Hispanic multiracial (NHANES, 2011a).

Media screen time was also operationalized as categorical, according to NHANES (2011b), and consisted of the number of hours the individual watched TV or

videos in the past 30 days and the number of hours the individuals used computers in the past 30 days. In addition, I generated and operationalized four novel categorical variables: total media screen time (i.e., total number of hours individual watched TV or videos plus used computer), as well as screen time (TV or videos, computer, and total) according to APA recommendations (more or less than 2 hours) (Fakhouri et al., 2014). Dependent variables were risk factors of CVD, such as CRP protein, a biomarker for acute inflammation, lipids (HDL, LDL, and triglycerides), and blood pressure (systolic and diastolic; NHANES, 2011c). Both dependent and independent variables were defined on the NHANES webpage (CDC, 2014). Variables are described in more detail in Chapter 3.

Assumptions

The major assumption of the study is that exposure to excessive media screens is unhealthy for one's health. Although a very large amount of evidence points to the negative effect of media screen time on physical and mental health, as well as social and academic skills, the studies were only descriptive and did not prove the cause-effect relationship between the two variables. The assumption that media screen time may increase CVD risk factors must be made in order to assess the relationship between the combined effect of media screen time and genetic background with CVD.

Scope and Delimitations

The scope of the study was to begin to explore the association between genetic background (i.e., gender, ethnicity) and screen time with risk factors of CVD. Baseline profiles of lipids and inflammatory markers (CRP) are distributed differently across gender and ethnicities (Adult Treatment Panel III, 2002; Morris & Keith, 2009). The

same risk factors of CVD associated with genetic predisposition to CVD (dyslipidemia, hypertension, and CRP) are found elevated in individuals exposed to long hours of media screen time (Hardy, Denney-Wilson, Thrift, Okely, & Baur, 2010; Stamatakis et al., 2011). Exploring whether media screen time impacts the genetic predisposition to risk factors is critical to formulate targeted, relevant policies for the prevention of CVD.

The population used here was limited to youth 0 to 20 years old. Choice of age range was determined by the availability of data for the variables in the study. The external validity of the study is therefore limited to youth of this age range. Generalizability to the population at large (worldwide) cannot be extended from the results deriving from the NHANES dataset on this topic because of the multifaceted behavioral, nutritional, and cultural components that characterize the development of CVD risk factors and that are found largely different across nations and geographical locations. Finally, the number of risk factors of CVD as well as parameters for genetic background is limited, due to lack of data availability.

Limitations

The main limitations of the study consist of the type of design, self-reporting for some of the questions in the survey, answers provided by substitutes on behalf of study subjects age 16 and less, use of convenience sampling instead of random sampling, lack of control over potential confounders, and the use of a dataset that is over 5 years old. Potential confounders for CVD in youth, not included in this study, are family history, smoking, obesity, sedentary activity, and unhealthy eating (World Heart Federation, 2015b). Nevertheless, the current study, although not exhaustive, provides initial basic information over the relationship between genetic background, screen time, and risk

factors for CVD and has generated sufficient preliminary results to warrant further research.

Cross sectional studies, such as the one used here, do not allow for the identification of a cause-effect relationship. Convenience sampling in place of random sampling can pose severe limitations over the generalizability of findings, while a lack of control over confounders may lead to erroneous conclusions. Content and construct validities of the NHANES survey have been corroborated through multiple research efforts and by comparison to other national surveys. Limitations to the study are included as part of the conclusion to the findings of this study.

Significance

A clear understanding of the health effect of excessive media screen time is necessary to develop meaningful policies. Youth who spend long hours in front of the media screen are three to four times more likely to become obese with respect to those who meet the 2-hour daily limit and display higher levels of biomarkers for CVD (Martin, 2011).

The potential link between media screen time, ethnicity, and risk of CVD is concerning since the same ethnicities that are already at higher risk for CVD complications (i.e., Blacks, Hispanics) also spend more time in front of media screens and receive a lower level of medical care. The current study is in alignment with the concept that the association of TV viewing with markers of poor health status is independent of physical activity and supports the idea that attempts to reduce the burden of chronic diseases (i.e., obesity, diabetes) in high-risk populations must target recreational screen viewing behaviors using culturally relevant interventions.

Interventions must target not only youth, but parents, caregivers, home, and educational settings as well. Growing evidence that risk factors of CVD disease are detectable already during childhood (Faienza et al., 2013) supports the notion that preventive interventions to limit behavioral determinants of CVD should start at a very early age. Parental education over the harmful effect of excessive screen time on youth's health may solicit parental involvement in demanding regulations over accessibility to media screens, better quality of television and videogames program contents, elimination of screen-based educational material in preschool child care settings, and restriction of screen-based teaching within scholastic curricula for school and homework.

Summary

Screen time habits developed during childhood track into adulthood are tightly linked to poor physical and mental health as well as limited social and academic skills (Grøntved et al., 2014; Martin, 2011; Rosen et al., 2014). Media screen time is excessively used by ethnic minorities and people at lower SES, which categories are already at risk of lower health quality (Fakhouri, 2011). Choice of the type of media entertainment is affected by age, gender, and ethnicity (Rideout, 2011). Exposure to media screens starts already before the child reaches 2 years of age and occurs both in home settings as well as in childcare centers, predisposing youth at increased risk of poor health during their adulthood (Grøntved et al., 2014). Parental screen time habits and unawareness over the consequence of excessive screen time play a role in the amount of time that youth are exposed to media screens.

Excessive media screen time increases the levels of risk factors for CVD (dyslipidemia, blood pressure, inflammation). The same parameters are expressed at

different levels among ethnicities due to variations in genetic and epigenetic features across populations. Natural baseline levels are elevated among those ethnicities who also spend the longest hours in front of the screen. The concern is that excessive media screen time further increases the risk of CVD among people already at higher risk of CVD because of their genetic makeup. In this study, I explored if the combination of screen time with genetic background was a better predictor of CVD than either component alone. Results from this study may help to promote policies and initiatives to limit screen time that are culturally relevant and more focused.

In Chapter 2, I discuss in more detail the current literature that establishes the relevance of the problem and the theoretical foundation of the current study.

Chapter 2: Literature Review

Many youth spend close to 7 hours per day in front of media screen. The uncontrolled use of tablets and smart phones for recreational purposes, multitasking with smart phones while watching television, and working with computers on classwork and homework increases this estimate to over 10 hours per day (Rideout et al., 2010).

Excessive media screen time has serious consequences on the health of the child (Rosen et al., 2014). The mechanisms by which screen time impacts health are multiple and likely to act synergistically. Mechanisms include displacement of physical activity by sedentary life, increased consumption of high calorie and low nutrient junk food, alteration of metabolic pathways balance towards oxidative stress, and modification of nervous system impulses and brain architecture. This last effect triggers the release of neurotransmitters responsible for addictive behavior and shrinkage of the part of the brain regulating cognitive functions and centers of self-control (Dong, Yanbo, & Xiao, 2013; Hong et al., 2013; Takeuchi et al., 2013; Weng et al., 2013).

One of the health consequences of excessive media screen time is the increased risk of CVD (Rosen et al., 2014). The association between excessive media screen time and CVD is only partially mediated by the lack of physical activity (Wilson, McNeal, & Blackett, 2015). The displacement of an active life with a sedentary life can lead to an obesogenic caloric shift, hormone imbalance, hypertension, and dyslipidemia; however, the link between excessive media screen time and increased risk of CVD remains after adjusting for physical activity.

Besides behavioral determinants, CVD has a genetic component, which supports the evidence of a differential burden of the disease among ethnicities (CDC, 2015a). The

genetic component of CVD is well established, as illustrated by the higher chance of developing a CVD complication for individuals with family history of CVD. Recently, single nucleotide polymorphisms in the genetic sequence for susceptible genes for increased risk for CVD have been identified and found to be differently distributed across ethnic groups (Ozaki &Tanaka, 2015).

Many of the genetic risk factors for CVD, manifesting as a phenotypic response of the genome to the environment and measured at different levels among ethnic groups and minorities (i.e., abnormal lipid profile, markers of inflammation, hypertension) are also found to be associated with prolonged exposure to excessive media screen time (Hardy et al., 2010; Stamatakis et al., 2011). Similarly, many of the modifiable risk factors of CVD (i.e., cultural factors, social inequities, high prevalence of television in the youth's bedroom) are more prevalent among ethnic groups and minorities and are associated with high exposure to media screen time (Appelhans et al., 2015; Dennison, Erb, & Jenkins, 2002; Myers, Gibbons, Arnup, Volders, & Naughton, 2015; Ohira et al., 2012). It is therefore of the utmost importance to understand if exposure to excessive screen time increases the risk of CVD through biological mediators that are already increased in vulnerable populations due to genetic predisposition, life stressors, or environmental conditions. In this study, I investigated whether there is an association between screen time, genetic background (i.e., ethnicity, gender, and family history), lipid profile, and/or inflammatory markers, thereby increasing the risk of developing CVD.

In this chapter, I discuss how the social ecological theory can be used as a framework for this study and how behavioral determinants and biomarkers of CVD are

linked to excessive exposure to media screen time. Particularly, I discuss the epidemiology (person, time, and place) of excessive screen time, demographics and socioeconomic determinants, and adverse health outcomes. Among the adverse health outcomes, I then focus on CVD, risk factors (hypertension, lipids, and inflammatory biomarkers), genetic and social determinants, and review studies describing CVD biomarkers found increased in youth exposed to excessive screen time.

Literature Search Strategy

To conduct a literature review of influences of screen time on CVD risk, I searched six databases: PubMed, Medline, CINAHL, Dissertation Database, Cochrane Database of Systematic Reviews, and Academic Search Complete. Search terms were *screen time* OR *television* OR *cardiovascular disease* OR *social ecological theory*, alone or in combination with *epidemiology* OR *youth* OR *inflammation* OR *ethnicity* OR *genetics* OR *risk factors* OR *lipids* OR *health* OR *socioeconomic status*. Searches were limited to articles published in English; all available dates were searched for completeness, but I focused on the most recent sources (2010-2015) unless fundamental research work had been done prior to these dates. I selected for inclusion studies reporting on predictors or correlates of screen time and CVD risk among the population at large, or among specific ethnicities, as well intervention or review articles on CVD diseases and their risk factors.

Theoretical Foundation

The theoretical framework for this study is the social ecological theory of disease distribution. The social ecological theory takes into consideration the multifaceted interaction between individuals, the community, and economical and societal factors, and

focuses on social and biological processes across political and economical interests responsible for social inequities (Krieger, 2011). Individual lifestyle changing behaviors, which neglect the environmental and societal influence on health and illnesses, are not suitable for the disease prevention of health issues fueled by enormous economic interests and are deeply rooted at the community, institutional, and political level. Indeed, successful interventions, focusing on nutrition and physical activity that have occurred in schools, have adopted a social ecological approach. As people are a product of their environment, the effect of the environment (exposure to media screen) on youth of various genetic backgrounds deserves immediate attention.

Bronfenbrenner's (1977) social ecological paradigm was first introduced in the 1970s to include environmental systems (i.e., real-life settings and real-life implications) in research revolving around youth's development. In the later revision of his theory, psychologist Bronfenbrenner recognized the contribution of biological and genetic characteristics of an individual during development (Bronfenbrenner & Ceci, 1994). Throughout the development of the paradigm, formalized as theory in the 1980s, Bronfenbrenner refined the concept of the reciprocal interaction between an individual and the people, objects, and environment in the proximity. In the social ecological theory, it is proposed that the proximal environment changes through the lifespan of the individual and modulates the individual's behavior (Brofenbrenner, 1977).

According to Brofenbrenner (1977), the environment, or system, comprises of the microsystem, the mesosystem, the exosystem, the macrosystem, and the chronosystem. The microsystem is the immediate environment and includes immediate relationships and interactions (i.e., family, caregivers, school, daycare). The interaction of the child with

the environment is bidirectional and impacts the way the child will grow, mature, and respond to people and to events. A positive and nurturing relationship will provide a positive influence to the child's development and how the child will behave with her proximal environment. A child's personality and temperament are, however, predisposed by his or her biological and genetic makeup. The mesosystem represents the interplay between the various microsystems in the child's life. Harmony between microsystems leads to a harmonious development, while dissent leads to stress and developmental issues (Atkins, 2015). The exosystem includes levels with which the child may not interact often but still represent an area of influence on the child's growth, such as parents' workplaces, extended family, and community. The macrosystem is the largest and most distant set of people and values, which have the most influence on a child's life and are represented by government's rules, moral and cultural values, the economy, and conflicts. Finally, the chronosystem encompasses all stages in a child's life and is related to changes in the child's environment.

Health issues caused by excessive screen time have roots in social, genetic, and cultural elements. The social ecological theory embraces the complicated network of interactions between environment (i.e., physical, social, cultural), hierarchical society, (individuals, groups, communities), and psyche and physiology (brain maturation, endocrine system, and metabolic homeostasis; Atkins, Rusch, Metha & Lakind, 2015). Social ecology represents a holistic approach to the study of human nature in its natural environment and recognizes the importance of the individual's innate features in the relationship with the environment. Therefore, this approach is suitable to understand the relationship between excessive screen time, social levels, and genetic determinants.

The relationship between the exposure to adverse environments, low SES, and development of social and health problems is well documented (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2011; Hong, Kral, Espelage, & Allen-Meares, 2012; McCullough, Pedersen, Schroder, Tabak, & Carver, 2013; Ungar, Ghazinour, & Richter, 2013). Although the prevalence of health problems is larger in high-risk families, the environment in which a child lives is not sufficient to explain the variation in the response of the child to his or her environment. Additional concepts, such as the individual's genotype and phenotype, that is, the makeup of genes in the DNA and their physical expression, have been suggested as mediators of how people react to stressors and adversity, as well as their response to the availability of resources and positive support (Van Cleve & Akçay, 2014).

The social ecological theory assumes that environment, society, and human nature must be considered simultaneously when attempting to explain human behavior and health (Bronfenbrenner & Ceci, 1994). The theory has been applied primarily to initiatives dealing with youth nutrition and physical activities and provides a framework for preventive initiatives, which include youth as well as their proximal and distal environment and their genetic makeup (Golden & Earp, 2012). Indeed, according to Martinez-Vizcaino et al. (2015), successful planning for interventions to prevent obesity and promote physical fitness should include youth and the environment (i.e., the mesosystem and exosystem represented by parents, teachers, and the school's community) where the initiative was implemented to prevent and control for potential barriers to the promotion of physical activity. The model to evaluate the efficacy of intervention should differentiate between genders, due to the physiological diversity in

patterns of weight and height, triceps skinfold thickness, and body fat between boys and girls. The strategy for the analysis should take into account origin of the youth, SES, neighborhood, school, environment, and genetics (gender).

Besides social determinants, genetics play an important role in the development of CVD complications. Wells (2012) recognized significant differences in body composition (fat/lean ratio, fat distribution, lean mass composition and metabolism, and adipose tissue biology) among ethnicities and suggested a link between cardio-metabolic risk and body composition. According to Wells (2012), adiposity contributes to the individual's metabolic load, which, if it overwhelms the homeostatic capability of the system, can elevate cardio-metabolic risk. Within this context, ethnic differences represent the variability of the metabolic buffering system and might play in an important role in the variability of CVD risk. This observation finds support in a study by Yin, Moore, Johnson, Vernon, Grimstvedt, and Gutin (2012). Yin et al. applied the socio-ecological approach to simultaneously examine both micro- and macro-level factors of adiposity in youth. Fat content was measured using dual-energy x-ray absorptiometry, while psychosocial and demographic variables were collected using surveys. The association between body fat and CVD fitness was dependent on gender, athletic competence, and type of neighborhood.

The enormous influence of the environment on the individual's phenotype is confirmed by epigenetic changes during people's life, starting already before birth. For example, the development of a healthy individual is supported already during gestation in the first months of life by a supportive environment that can strengthen the developing individual and build the foundation for lifelong health (Branchi & Cirulli, 2014). In

contrast, severe stress can alter the brain's architecture and lead to increased susceptibility for psychopathology (Lo & Zhou, 2014). Just like severe stress impacts the brain architecture, screen time can modify the brain anatomy. Alarmingly, exposure to an excessive amount of screen time has been demonstrated to impact brain volume and development (Dong et al., 2013; Hong et al., 2013; Lin et al., 2012; Takeuchi et al., 2013; Weng et al., 2013; Yuan et al., 2013). Changes occur particularly in the brain's frontal lobe, the region of the brain that undergoes dramatic modifications during puberty and through early adulthood, and that is responsible for motor and cognitive function, problem solving, spontaneity, memory, language, initiation, judgment, impulse control, violent behavior, and social and sexual behavior. Television viewing and videogame playing affect youth's verbal abilities and other physical, cognitive, and emotional development (Hummer, Kronenberger, Wang, Anderson, & Mathews, 2014; Tekeuchi et al., 2013). In youth with screen addiction, internet addiction was found to be responsible for brain atrophy, reduced cortical thickness, and dopamine release (Dong et al., 2013; Hong et al., 2013; Lin et al., 2012; Weng et al., 2013; Yuan et al., 2013)

The social environmental theory incorporates the synergistic interactions between genotype, phenotype, and the environment (Coll, 2004). In the context of the study, displacement of physical activity by television viewing was expected to alter energy balance, switch the metabolism from calorie burning to calorie storing and accumulation of adiposity, and increase consumption of fast foods (Borghese et al., 2015). However, the displacement theory of physical activity by television viewing is not sufficient to embrace all the multiple, synergistic interactions between the individual and the systems around him or her. Lee et al. (2015) determined the association between physical activity

levels, sedentary behavior, and BMI in Malaysian youth aged 7 to 12 years. The outcome of the study indicated that, in addition to a lack of physical activity, screen time was independently and positively associated with BMI. Surprisingly, other types of sedentary activity were not. Another study on television viewing and levels of activity among high school female youth (Graham, Schneider, & Cooper, 2012) confirmed that although a relationship existed between the displacement of physical activity and television-associated sedentary life, displacement of active life with sedentary life was not sufficient to explain the influence of the media environment on youth metabolism and psychological consequences (Maibach, 2007). According to Trinh, Wong, and Faulkner (2015), physical activity and sedentary behavior could not explain mental health problems in youth caused by excessive screen time. An extended period of time spent in front of television was linked to poorer mental health, academic outcomes, and lower self-esteem even in youth who were physically active (Trinh et al., 2015). Exposure to screen time may affect the brain and other organs indirectly, by showing advertisements for and consumption of junk food, thus elevating the levels of circulating sugars and tilting the redox state towards prooxidant pathways, potentially triggering oxidative damage to the tissues (Thanan et al., 2015). Time spent on television viewing, instead of reading, may also lower cognitive function, and learning capabilities (Carson et al., 2015a).

The concept of the synergistic interactions between genotype, phenotype, and the environment in the context of CVD has been extensively investigated. Prevalence of CVD is disproportionate across gender and ethnicities (CDC, 2015). Molecular technology approaches have revealed some of the molecular mechanisms underlining

ethnic differences. Ozaki and Tanaka (2015) reported that in a genome-wide study using nearly 100,000 single nucleotide polymorphisms in over 2,000 individuals, myocardial infarction susceptible genes were identified and were differently distributed across different populations. Results were supported by findings from a meta-analysis study on ethnic differences in CVD risk (Hill et al., 2015). According to Hill et al. (2015), indicators of heart modulation may be different between African Americans and European Americans, after the consideration of several covariates including health status, medication use, and subgroup stratification by sex and age. Similar results were observed in an Asian sample population inclusive of Chinese, Whites, Indian, and Malay patients, where prevalence of CVD risk factors (diabetes, blood pressure, dyslipidemia, BMI, smoking) was unevenly distributed across ethnicity (Gijsberts et al., 2015). Ethnicity was independently associated with the severity of coronary artery disease and modified the strength of association between the severity of disease and risk factors such as gender and diabetes (Gijsberts et al., 2015).

A large number of studies on the effect of excessive screen time have focused on the relationship between screen-based activities, environmental and societal determinants (such as sedentary life, neighborhood safety, obesity, family-related factors, parenting rules, and demographics) and CVD risk (Brindova et al., 2014; Herman, Hopman, & Sabiston, 2015; Kunin-Batson et al., 2015). Relevance of the social ecological theory has been primarily to demonstrate an association between excessive screen time, poorer health, sedentary life, mental health, obesity, and CVD disease, and to facilitate policies to educate parents and caregivers, change behaviors and attitudes towards media screens, and to promote physical activity. Addition of a genetic determinant to the analysis of the

potential association between screen time and CVD risk has not been explored in depth, and may provide insights for culturally relevant, targeted initiative, and educational programs.

The social ecological model addresses the problem of the impact of modern technology to our societal environment, where technology and intense work schedule, often for both parents, leave very little free time for families to spend time together and to focus on physical and spiritual health (Henderson & Petic, 1995). Not all parents are aware of the consequences of excessive screen time, or can prevent triggers that lead the child to lead a more inactive life in front of the television (Halnes et al., 2013). Indeed, a study conducted by Beck, Takayama, Badiner, and Halpern-Felsher (2015) demonstrated that Latino parents had limited knowledge of the consequences of television watching on infants and toddlers. These findings highlighted the importance of interventions at the mesosystem, to achieve changes in behavioral determinants of youth addiction of media screen.

Social and cultural factors affect how parents perceive the use media screens in the household. Brindova et al. (2014) explored the relationship between family related factors and screen based activities in excess of two hours per day, among school-aged youth. The study evaluated the effect of age, gender, availability of a TV or computer in the bedroom, parental rules on television watching and computer use, in a cohort of youth age 11 through 15 years. The lack of parental control, and availability of computers and televisions in the bedroom were strong risk factors for excessive use of television and computers. In a similar study by Carson, Stearns and Janssen (2015b) on 738 youth age zero to five, parents with low physical activity or high screen time were more likely to

have youth with low physical activity or high screen time, pointing to the need for interventions targeting both parents and youth. Kunin-Batson et al. (2015) used cross sectional data from 421 youth of age 5 to 10 years enrolled in a behavioral intervention trial to measure the associations between demographic and household factors, and achievement of recommended goals for physical activity (≥ 60 min per day), screen time (≤ 2 hours per day), fruit and vegetable intake (≥ 5 servings per day), and sugary drinks (none). Each household had socio-demographic predictors that were different for the various guidelines, some of which were not under the control of parents.

At the mesosytem level within the social economical theory, a large majority of youth in the USA spends most of their time in early care and education settings, where unhealthy meals and snacks, as well as additional exposure to media screen, occur in place of an education to healthy eating and active life style (Buscemi et al., 2015). According to Côté-Lussier, Fitzpatrick, Séguin, and Barnett (2015), experience of transient or chronic poverty was associated with fear for own safety at school, which in turn was associated with screen time, poorer weight-related behaviors and increased probability of being obese or overweight.

The Society of Behavioral Medicine has started to address childhood health problems such as obesity by modifying state regulations for early care and education settings related to child nutrition, physical activity, and screen time (Buscemi et al., 2015). Emerging educational models such as the *Let's Move!* campaign, (Let's Move!, 2015), based on the social ecological theory and addressing nutrition, physical activity, and screen time in early care and education settings, are used to provide clear guidance for policymakers to identify specific elements of policy and regulations to transform

educational settings into environments for obesity prevention. Here, I investigated whether genetic components may also play a role in the association between excessive screen time and health risk (i.e. CVD diseases), thus providing evidence for targeted initiatives.

Conceptualizing youths' excessive risk of CVD through a model (Figure 1) offers a useful framework to stipulate direct and indirect pathways between the etiology of CVD and the three domains (excessive screen time, social determinants, and genetic determinants). Solid lines represent published pathways, and dashed lines reflect interactions hypothesized in this study. This approach is consistent with ecological systems theory, and emphasizes how multiple parameters affect youth's future. Understanding how genetic background, excessive screen time, social determinants and occurrence of CVD disease are associated will allow for development of more efficacious preventative strategies.

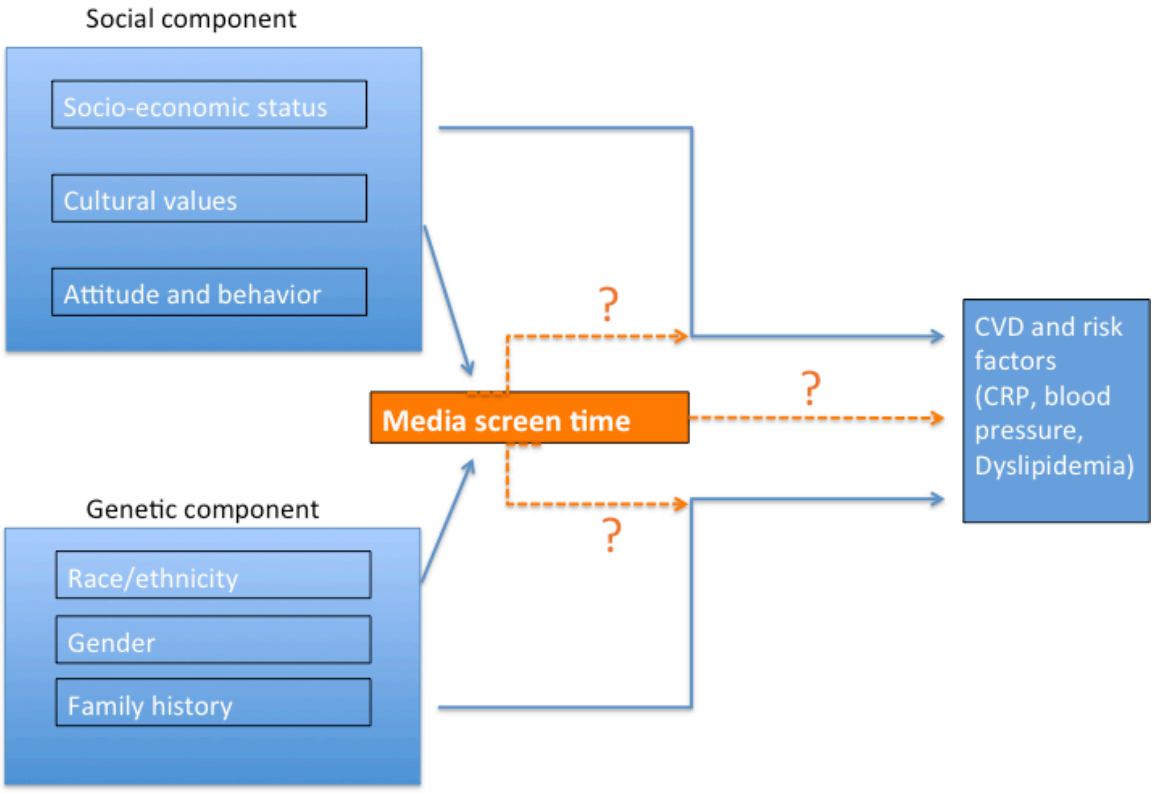

Figure 1. Relationship between excessive screen time, social determinants, and occurrence of CVD disease.

Literature Review Related to Key Variables and/or Concepts/ Rationale for Selection of the Variables

Most youth spend many more hours beyond the recommended two-hour limit in front of the screen (Rideout, Foehr, & Roberts, 2010; Tandon, Zhou, Lozano, & Christakis, 2011). Lack of physical activity, sedentary behavior, and consumption of unhealthy food associated to media screen use have been linked to energy imbalance that shifts the metabolism towards obesogenic trends, increasing the risk of overweight, obesity, metabolic diseases and CVD (Rideout, 2011). Physical inactivity and unhealthy eating are not, however, sufficient to explain all health and social problems associated

with excessive use of media screen. Age, gender and socioeconomic status are associated to media use (Rosen et al., 2014). Genetic background and ethnicity mediate the impact of media use on health (Anderson, Economos & Must, 2008; Herrick, Fakhouri, Carlson & Fulton, 2014; Rideout, 2011).

This literature review covers work on excessive screen time and CVD, risk factors of excessive screen time and CVD, and biomarkers correlated with both excessive screen time and CVD among youth and youth. The choice of variables for this study was based on their association with both CVD and screen time. CRP, hypertension, dyslipidemia, family history, ethnicity, and gender are all known risk factors for CVD, but are also found linked to excessive screen time.

Excessive Screen Time

Media screen time consists of activities performed in front of a screen such as watching television, playing games or watching videos on a computer or a telephone (Medline Plus, 2015). In spite of the American Pediatric Association (APA) recommendations that youth below 2 years of age should not be exposed to any screen time, and youth older than 2 years of age should be exposed to no more than 2 hours of screen time per day, the vast majority of children do not meet the guideline. According to a study conducted with 8950 youth five years and younger, sixty-six percent of them used television, computers, phones and electronic devices for more than 4 hours daily (Tandon, Zhou, Lozano, & Christakis, 2011). Additional exposure may come during day care and home based childcare settings (Christakis & Garrison, 2009; Vanderloo, 2014). Screen time rises to almost 7 hours per day by teenage years (Rideout, et al. 2010). Multitasking increases the time to over 10 hours (Rideout, et al., 2010).

Concerns include potentially severe health consequences and unhealthy life long habits leading to development of obesity and CVD, in addition to mental issues such as depression, low self-esteem and violent behavior (Rideout, 2011). The relationship between excessive screen time and youth is in part mediated by socioeconomic status and parental style; however, because of the central role of screen based media for everything in everyday life, almost the totality of youth is afflicted by this epidemic, regardless of demographics, environmental, or social determinants (Brody, 2015).

Demographics and Socioeconomic Status

In the U.S. socioeconomic status, age, gender, and type of media affect the number of hours that youth spend in front of the screen. On average, the amount of media screen time increases with age (Rideout, 2011; Tandon, et al., 2011). The type of media entertainment and the gender of the user affect the total time spent in front of the screen. A cross-sectional study on youth 12 to 15 years old, using data from the National Health and Nutrition Examination Survey (NHANES) and the NHANES National Youth Fitness Survey, indicated that more than 90 of youth watches TV and uses computers daily and outside the school, in excess of 2 hours per day (Herrick et al., 2014). Girls were more likely than boys to watch TV, while boys were more attracted to videogames (Garcia-Continente et al., 2014; Herrick et al., 2014; Hoyos Cillero & Yago, 2011).

Ethnicity may be another important risk factor for excessive screen time. According to Rideout (2011), 69 of African American and 66 of Hispanic youth had a television in their bedroom, with respect to 28 White youth in the same age group. Similarly, a study on data collected from 2,964 youth age 4 to12 years old in the National Health and Nutrition Examination Surveys 2001–2004, a U.S. nationally representative

cross-sectional study, low active play and high television time were positively correlated with age, gender, race/ethnicity and BMI (Anderson et al., 2008). This observation was later independently confirmed by Herrick, Fakhouri, Carlson & Fulton (2014) using data from the 2012 National Health and Nutrition Examination Survey and the 2012 NHANES National Youth Fitness Survey. Herrick et al (2014) reported that non Hispanic Black were more likely to watch TV more than 2 hours per day than non-Hispanic white and Hispanic. Similarly, maternal education, maternal weight status, outdoor play, having a television in the bedroom, and ethnicity were associated with increased TV viewing time and childhood overweight (Appelhans et al., 2015; Dennison et al., 2002; Myers, Gibbons, Arnup, Volders, & Naughton, 2015).

In the U.S., youth from lower socio-economic status may be disproportionally impacted by the problem of excessive exposure to screen time (Appelhans et al., 2015; Lord et al., 2015). According to Lord et al. (2014), lower income neighborhoods had higher prevalence of negative health behavior, including unhealthy eating and higher screen time. The relationship, however, between TV viewing and sociodemographic variable may not be linear and applicable to all countries (de Jong et al., 2013). Indeed, when path analysis was used to determine direct and indirect associations between the home environment and child weight status, childhood overweight/obesity was found associated to confusion at home, insufficient caregiver limits on screen time, loose rules on bedtime routines, and the presence of a television in children's bedrooms (Appelhans et al., 2015). The positive association with obesity was through numbers of hours of screen time and sleep duration (Appelhans et al., 2015). Interestingly, a study, performed on 5,660 youth age 10 to 18 years and resident in Mexico, indicated that screen time was

higher in youth from families living in urban areas, and with higher socioeconomic status and education; the link between obesity and screen time persisted (Janssen, Medina, Pedroza & Barquera, 2013).

Parental influence and home environment are critical in the relationship between youth and media screen, suggesting that any initiative to modify youth's behavior must include parenting practices at home and home environment (Appelhans et al., 2014; Downing, Hinkley, & Hesketh, 2015). Both amount of screen time and content of program have an impact on youth developmental outcomes, such as victimization and aggressive behavior (Duch, Fisher, Ensari, & Harrington, 2013; Kelishadi, Qorbani, Motlagh, Heshmat, Ardalan, & Jari, 2014). A systematic review covering research literature from 1998 to 2013 indicated that parent's practices and style, perception of consequences of screen time on youth' health, and parents' screen time habits were directly linked to youth's screen time attitudes and habits (Xu, Wen & Rissel, 2015). Similar results were confirmed in an independent study on the association between parental television limits and health behavior among obese youth (Cheng, Koziol, & Taveras, 2015). In this study, multivariable analyses adjusted for child age, sex, race/ethnicity indicated that the likelihood to have a television in the bedroom or to fall asleep while watching TV was linked to parental education and income, and parental limits. Parental limits on screen time, and number of televisions, computers, and videogames' consoles in the household were strong predictors of the amount of time spend engaging in media-related activities (Chaput et al., 2014). Association was stronger when media tools were present in the kid's bedroom (Chaput et al., 2014; Wethington & Sherry, 2013). Youth with several screens in their bedroom spent more time overall in

screen associated activities, with respect to youth without television in the bedroom, and quality of sleep was lower (Chaput et al., 2014). A television in the bedroom increased daily screen time by 25 minutes, and provided 32 higher odds of engaging in screen time for a time longer than the APA's recommended limits (Lo, Waring, Pagoto & Lemon, 2015). Consequences may be more severe for youth with attention deficit hyperactivity disorder, since they already have higher rates of screen time (Hefner, 2013; Lo et al., 2015).

Adverse Health Outcomes

The health issues associated with excessive exposure to media screen are several. Detrimental effects include unhealthy eating, impairment of social and cognitive skills, lower academic achievement, mental and psychological issues, depression, violent behavior, sedentary life, obesity, and CVD (Rideout, 2011). Excessive screen time has been linked to elevated blood pressure, overweight, dyslipidemia, and obesity (Goldfield, et al., 2013; Mark & Janssen, 2008; Mota, et al., 2014; Tremblay, et al., 2011).

In the past 30 years, the prevalence of obesity in youth 12 years or younger has doubled (from 7 to 18), and quadrupled in adolescents (from 5 to 21), raising strong concerns about the risk of CVD development in adulthood (Ogden, et al., 2012). Convincing evidence points to screen time as one of the determinants of obesity (Syvaoja, Tammelin, Ahonen, Kankaanpaa, & Kantomaa, 2014). A through review of the association between screen time, physical activity, and obesity by Syvaoja, et al. (2014) confirmed that television, video games, and computer use were associated with lower physical activity, increased calorie intake, body fat, and higher BMI among U.S., Canadian, and Korean youth. Only 40 of the youth assessed in the 2009-2010 National

Health Examination Survey met both guidelines for physical activity and screen time (Fakhouri, et al., 2013). Sijtsma, Koller, Sauer, and Corpeleijn (2015) used regression path analysis to estimate the direct and indirect effects of television, sleep duration and outdoor play on BMI. Higher screen time was found associated with decreased sleep duration, and consequently with higher BMI. Screen time was not associated with outdoor play time (Sijtsma, et al. 2015). De Jong, Visscher, HiraSing, Heymans, Seidell, and Renders (2013) studied the association between television viewing and computer use with obesity among 4072 children age 4 to 13, in the Netherlands. Use of television for more than 1.5 hour per day increased the risk of being overweight (odds ratio 1.70, CI 1.07 – 2.72), however the results were only borderline statistically significant. Television (TV) watching was linked to number of TV in the household, presence of a TV in the kid's bedroom, and lack of parental rules limiting TV viewing. Interestingly, according to this study, TV viewing and computer use were both associated with lack of sleep, but not with a reduction in physical activity.

In addition to physical problems, excessive screen time may cause symptoms of depression and anxiety, whose severity depends on the amount of time spent in media-screen based activities (Maras, Flament, Murray, Buchholz, Henderson, Obeid, & Goldfield, 2015). Indeed, time spent by college students in online activities, as measured by computer records, could predict depression levels (Kotikalapudi, Chellappan, Montgomery, Wunsch, & Lutzen, 2012).

Cardiovascular Diseases

CVD are a class of diseases that affect the health of the heart and blood vessels (Mendis, Puska, Norrving, & World Health Organization, 2011). These include coronary

artery diseases, stroke, hypertensive and rheumatic heart disease, cardiomyopathy, atrial fibrillation, congenital heart disease, endocarditis, aortic aneurysms, peripheral artery disease, and venous thrombosis (Mendis, et al., 2011). CVD are the most prevalent non-communicable diseases in the world, and account for > 30 of deaths globally, each year (Wong, 2014). The large majority (80) occurs in low-income and middle-income countries (Wong, 2014). The cost exceeds 400 billion dollars per year in health care expenses and loss of productivity (Roger et al., 2011).

More than one third of adults in the U.S. have at least one type of CVD. According to the Centers for Disease Control and Prevention (CDC, 2015), over 2.5 million deaths were caused in the U.S. by CVD in 2013 alone. Prevalence is disproportionate across gender, ethnicities, and locations (Mozaffarian et al., 2015). CVD is the leading cause of death in the U.S. for non-Hispanic whites (23.8), non-Hispanic blacks (23.8), Asian Americans and Pacific Islanders (22.2), and American Indians (18.4) (CDC, 2015b). For Hispanics, and Asian Americans and Pacific Islanders, heart disease is the second caused of death, after cancer (Kochanek, Xu, Murphy, Miniño, & Kung, 2011).

Risk Factors for CVD

A complex network of biological and environmental factors can trigger the onset of CVD (Fryar, Chen, & Li, 2012). The most important behavioral risk factors of CVD are unhealthy diet, obesity, insufficient physical inactivity, and use of tobacco and alcohol, leading to the development of hypertension, dyslipidemia, and underlying inflammation (WHO, 2015a). Epigenetics and modification of biological pathways leading to the development of CVD originate already during childhood, and as early as

the gestational period (Wilson, McNeal & Blackett, 2015). Physical activity, nutrition, family history, genetics, dyslipidemia, diabetes, metabolic syndrome, and obesity can build upon each other and act synergistically (Fryar, et al., 2012). For example, obesity and overweight predispose to hypertension, low physical activity, dyslipidemia, and diabetes; family history and nutrition can predispose to dyslipidemia and metabolic syndrome (Mozaffarian, et al., 2015). Hypertension (blood pressure ≥140/90) and high total cholesterol (particularly LDL, HDL, and triglycerides) are the most important risk factor for premature cardiovascular disease, since the association between them and CVD has been proven to be causative in multiple of clinical trials (Holmes, et al., 2011). Other modifiable risk factors include socioeconomic status, psychological stress, and depression (WHO, 2015). Non-modifiable risk factors include genetics, age, gender, and family history (World Heart Federation, 2015).

Behavioral risk factors developed during childhood have a high chance to get carried from childhood into adulthood (Huang, Prescott, Godfrey, & Davis, 2015). The most prevalent cardiovascular risk factors for youth are overweight, excess body fat, lipid profile, sedentary behavior, and history of CVD in family (Do Prado Junior, Rocha de Faria, Rodrigues de Faria, Castro Franceschini, & Priore, 2015). Therefore, intervention to prevent CVD should start at an early age.

Hypertension. Hypertension is the leading cause of CVD worldwide, with almost one billion people suffering of this condition (WHO, 2015). Hypertension promotes the development of structural and functional defects in the cardiovascular system (Battistoni, Canichella, Pignatelli, Ferrucci, Tocci, & Volpe, 2015). Hypertension accounts for 54 of

all strokes, and 47 of all ischemic diseases globally, and cholesterol accounts for over 30 of all ischemic heart diseases (Arsenault, Boekholdt, & Kastelein, 2011; WHO, 2011).

Although it is found highest among the general adult and elderly population, and in particular among adult African Americans (Battistoni et al., 2015; Mozaffarian et al., 2015), staggering evidence is indicating that hypertension is already present in 5 of U.S. youth, and its prevalence is increasing (Rodriguez-Cruz & Rao, 2015). Unhealthy lifestyle habits (sedentary life, junk food, limited physical activity, and excessive screen time) were responsible for increasing the risk of hypertension already in young youth and teenagers worldwide (Battistoni et al., 2015; Gopinath, Hardy, Kifley, Baur, & Mitchell, 2015). Both lean and overweight youth with hypertension were at risk of CVD (Colangelo, Vu, Szklo, Burke, Sibley, & Liu, 2015); however, according to a study on 10 to19 years old youth, subjects with higher BMI may be at higher risk to develop hypertension, changes in total cholesterol, LDL, triglycerides, insulin, and low HDL when compared to healthy individuals (Do Prado Junior, et al., 2015). Interestingly the diastolic pressure is a predictor of CVD in youth, while the systolic pressure is used for individuals over age 60 years, (Franklin & Wong, 2013).

Lipids and inflammatory biomarkers. Dyslipidemia is a prominent risk factor of CVD, and accounts for more than one third of all cases of coronary heart disease, and more than 20 of cases of ischemic stroke (WHO, 2015b). Prospective studies using statins to reduce LDL and increase HDL have proven the link between dyslipidemia and CVD (Gutierrez, Ramirez, Rundek, & Sacco, 2012). Indeed, a reduction in cholesterol was found to reduce the risk of atherosclerosis by 20 to 40 over a five-year period (Gotto, 2011).

Dyslipidemia responsible for the etiology of CVD is characterized by increase in LDL (>130 mg/fL), triglycerides (>150 mg/dL), both alone or in combination, and by a reduction in HDL (<40 mg/dL) (da Silva, de Almeida-Pititto, & Ferreira 2015). The standard lipid profile, including total cholesterol, LDL, HDL, and triglycerides, in combination with age, hypertension, gender, and family history can be used to predict premature coronary heart diseases in man.

Dyslipidemia and the other traditional risk factors (i.e. obesity, hyper-pressure, poor diet, sedentary life) are not sufficient to explain occurrence of CVD in individuals who are apparently healthy. Inflammation can exacerbate the role of dyslipidemia in the development of atherosclerosis by altering lipid metabolism and oxidative status. CRP, a marker of systemic inflammation, is positively correlated with the severity of risk for CVD. It is found elevated in adult and youth at increased metabolic risk and subsequent cardiovascular risk (Shrivastavaa, Singhb, Raizadac, & Singhd, 2015). CRP is hypothesized to mediate formation of atherosclerosis and coronary heart disease by stimulating the lipids uptake by the immune system, the release of pro-inflammatory cytokines, and the inflammation of the endothelium. Inflammation is also expected to play a major role in the rupture of atherosclerotic plaques and the occurrence of stroke. Atherosclerosis is characterized by endothelial dysfunction, vascular inflammation, and the accumulation of lipids and cellular debris within the intima of the walls of arteries (Upadhyay, 2015). While it is very rare that youth suffer of heart attack from the formation of atherosclerotic plaques, atherosclerosis originates already in childhood and has been linked to dyslipidemic patterns, as demonstrated by pathology and imaging studies (Wilson, McNeal, & Blackett, 2015).

Inflammatory markers and lipids are distributed differently across ethnicities (Adult Treatment Panel III, 2002; Morris & Keith, 2009). African Americans are more likely than other ethnicities to develop hypertension than Whites, and have higher levels of the inflammatory marker CRP (Morimoto et al., 2014, World Heart Federation, 2015). Whites and Asians are more likely to have higher levels of triglycerides and low HDL than African Americans (Frank et al., 2014; World Heart Federation, 2015).

Age, gender, and family history. In addition to ethnicity, gender also affects the risk of developing CVD. According to the World Heart Federation (2015), men were more likely than premenopausal women to develop cardiovascular diseases; the risk of developing heart disease increased by 50 if both parents had a history of stroke and the chances were even higher for women whose mother had a stroke. However, the lower apparent incidence of CVD in women may be a reflection of lower detection rates in women, as opposed to true incidence rates (CDC, 2015c). Black men have the highest death rate, while white women display the lowest (CDC, 2013).

Genetic and Social Determinants of CVD

Both genetics and socioeconomic conditions are linked with the biology of CVD (Havranek et al., 2015). Health disparities of CVD depend not only on genetic predisposition, but also on unfair allocation of economical resources and access to health care, as suggested by a reduction in CVD burden in wealthy countries and increase prevalence in low and middle income countries, particularly in regards to socioeconomic status and gender (Francis et al., 2015). Even within wealthy countries, allocation of resources and heath care services reflect uneven distribution of CVD rates (Havranek et al., 2015). Populations at lower socio-economical status carry a disproportionate burden

of CVD risk factors. Social and economic anxiety is associated to a biological chronic stress response that causes the release of stress hormones, inflammation, endothelial dysfunction, coagulopathies, and metabolic imbalance (Havranek et al., 2015).

Genetic determinants. In addition to social and environmental determinants, genetic factors (i.e. gender, family history, race/ethnicity) are important risk factors for CVD, and may have a direct impact on the development of hypertension, dyslipidemia, and CRP (World Heart Federation, 2015). Family history of CVD and genetics increase the risk of CVD (i.e. stroke, heart failure, and peripheral heart disease). Recently, genetic analysis of loci related to blood lipids and CVD has identified a number of variants associated with abnormal lipid values, suggesting that lipids might mediate the association between gene variants and heart diseases (Holmes, Harrison, Talmud, Hingorani, & Humphries, 2011). The influence of genetic variation may be more evident for dyslipidemia than for other risk factors such has hypertension, diabetes, and metabolic syndrome (Smolkova et al., 2015). New evidence has unveiled some of the molecular mechanisms underlining ethnic differences. Several genome wide association studies performed on thousands of individuals have established the presence of genes that make the individual susceptible of myocardial infarction and mediate thickness of the carotid artery, after adjusting for confounding factors (i.e. age, gender, smoking, blood pressure, lipids, and pharmaceutical drugs) (Ozaki & Tanaka, 2015; Xie et al., 2015). The distribution of these genes is different across different populations (Ozaki & Tanaka, 2015). A meta-analysis study on ethnic differences in CVD risk confirmed that indicators of heart modulation may be different between African American and European American, after correcting for health status and medication use, and stratifying by sex

and age (Hill et al., 2015). Similarly, within an Asian sample population inclusive of Chinese, Whites, Indian and Malay patients, ethnicity was independently associated with the severity of coronary artery disease and prevalence of risk factors (diabetes, blood pressure, dyslipidemia, BMI, and smoking) (Gijsberts et al., 2015). According to Gijsberts et al., (2015), ethnicity increased the strength of the association between severity of CVD and risk factors (i.e. gender and diabetes).

Social determinants. Modifiable risk factors for CVD (i.e. life style, hypertension, and hypercholesterolemia) cluster with ethnicity, culture, and access to health care (Havranek et al., 2015). Access to care is unevenly distributed across all population in the U.S., partly due to the ability to pay for healthcare, partly due to the geographical distribution of health care centers, partly due to people's attitudes towards medical care, or health literacy, or personal and cultural values (Havranek et al., 2015). Social determinants of CVD include SES, race/ethnicity culture, language, access to care, and residential environment (Havranek et al., 2015). SES, inclusive of education, income, and occupation are associated with higher prevalence of cardiovascular events and mortality, due in part to low health literacy, numeracy, stress for job loss or fear of loosing a job, anxiety, anger, or emotional state (Berkman, Sheridan, Donahue, Halpern, & Crotty, 2011).

Poor socioeconomic conditions during childhood predispose the individual to risk factors (i.e. physical activity, BMI, blood pressure, dyslipidemia, smoking, and alcohol consumption) that are carried over to adulthood and establish patterns of disease outcome (Harper, Lynch & Smith, 2011). Racial/ethnic disparities have a genetic component, but may be aggravated by clinicians' attitudes, lower trust between clinicians and minority

patients, mistrust and underuse of available services, perceived bias, and lower satisfaction (Havranek et al., 2015). Associated to the concept of ethnicity, language and cultural barriers have shown to be important determinants for CVD risk. Language barriers were associated with reduced medical care services, while cultural beliefs about the cause of diseases or maintenance of traditions represented major obstacles to proper medical treatment.

Biomarkers Found Increased in Both CVD and After Exposure to Excessive Screen Time

The same risk factors associated with genetic predisposition to CVD (hypertension, dyslipidemia, inflammatory markers, obesity, and physical inactivity) are increased among youth when the child spends long hours in front of media (Hardy, et al., 2010; Stamatakis, et al., 2011). CRP, dyslipidemia, and high blood pressure are increased in youth watching more than 4 hours per day (Pinto Pereira, et al., 2012). Researchers found that television viewing and total screen time among youth were positively associated with adiposity, triglycerides, metabolic syndrome z-score, and high systolic blood pressure (Berentzen et al., 2014; Mota, et al., 2014). Individuals watching more than 2 hours of screen time had higher BMI with respect to age-matched controls who had lower viewing time (Berentzen et al., 2014). The impact of screen time was particularly severe in youth in the 50^{th} to 90^{th} BMI percentile range (Mitchell, Rodriguez, Schmitz, & Audrain-McGovern, 2013). Among pre-school youth 3 to 6 years old, long hours spent in front of the screen and lack of sleep were associated with higher risk for high systolic blood pressure and overweight (Mota, et al., 2014).

The relationship between media screen time, sedentary life, lack of sleep, and obesity suggests an association of screen time with CVD (Danielsen et al., 2011; Grøntved et al., 2014; Hardy, et al., 2010; Sandercock, & Ogunleye, 2012; Stamatakis, et al., 2011). However, the evidence on the link between television watching and cardiovascular risk factors is controversial. In youth with type2 diabetes, increased television watching was associated with increased LDL and triglycerides (Li et al., 2015). However, association between screen time and dyslipidemia may be mediated by adiposity and not be a direct consequence of screen viewing, as suggested by Berentzen et al. (2014). According to Berentzen (2014), there was no direct correlation between cardio-metabolic markers (total cholesterol/HDLC ratio and blood pressure), and screen time; however the authors used a very homogeneous population in terms of ethnicity, which may have biased the results. When a multiethnic Asian population was studied, longer television viewing was linked with higher systolic blood pressure, total cholesterol, triglycerides, and CRP, after adjustment for potential socio-demographic and lifestyle confounders (dietary factors and body mass index) (Nang, Salim, Wu, Tai, Lee, & Van Dam, 2013). Among the various types of media screen time, television viewing but not electronic video games was relevant to the association with cardiovascular risk (Nang et al., 2013; Stamatakis et al., 2013).

Limitations of Existing Research

Although a role may be inferred for excessive screen time on the mediation between genetic background and risk of CVD from partial information from several publications, I did not encounter a single study that focused on the relationship between all these three variables among U.S. youth. Previous studies were limited by the

composition of the study population, which either did not include heterogeneity of ethnicities, or did not use all the variables that I used in the current study (ethnicity, gender, and screen time) to predict risk factors of CVD (dyslipidemia, hypertension, and inflammation).

Summary and Conclusions

Biological, genetics and socioeconomic conditions are linked to the biology of CVD (Havranek et al., 2015). Individuals at socioeconomical disadvantage and minorities, populations already at higher risk for chronic diseases and lower quality of care, have higher propensity to spend longer hours in front of a media screen and to have a television in the bedroom. The combination of biological, genetic, and behavioral risk factors in the context of excessive media time remains to be investigated.

In this study I assessed the association between media screen time and the risk factors of CVD (CRP, lipids, and blood pressure), in relationship with genetic background (ethnicity, and gender). This study revealed previously unknown associations between levels of risk factors and genetic background in youth exposed to excessive time of media screen viewing. Understanding whether excessive media screen time can enhance genetic predisposition to risk factors is important for development of ethnic and cultural relevant policies to reduce the risk of CVD.

I collected numerical data on United States youth's demographics, blood pressure, lipids, and CRP from the National Health and Nutrition Examination Survey, and applied statistics (logistic binary regression analysis) to determine the relationship between variables. The approach for the study was quantitative, consistent with measuring

relationships between parameters and uncovering novel patterns. In Chapter 3, I present a detailed description of the study methodology.

Chapter 3: Research Method

Introduction

The purpose of this study was to determine the effect of genetic background combined with excessive media screen time on markers of cardiovascular risk in youth aged 0 to 20 years. The research questions that I answered are as follows:

1. RQ1. For U.S. youth 0 to 20 years old, what is the relationship between media screen time, genetic background (gender, race/ethnicity), and CRP?

   Null hypothesis. There is no relationship between media screen time, genetic background (gender, race/ethnicity), and CRP in U.S. youth aged 0 to 20 years old.

   Alternative hypothesis. There is a relationship between media screen time, genetic background (gender, race/ethnicity), and CRP in U.S. youth aged 0 to 20 years old.

2. RQ2. For U.S. youth 0 to 20 years old, what is the relationship between media screen time, genetic background (gender, race/ethnicity), and lipids (HDL, LDL, and triglycerides)?

   Null hypothesis. There is no relationship between media screen time, genetic background (gender, race/ethnicity), and lipids in U.S. youth aged 0 to 20 years old.

   Alternative hypothesis. There is a relationship between media screen time, genetic background (gender, race/ethnicity), and lipids, in U.S. youth aged 0 to 20 years old.

3. RQ3. For U.S. youth 0 to 20 years old, what is the relationship between media screen time, genetic background (gender, race/ethnicity), and blood pressure (diastolic and systolic)?

   Null hypothesis. There is no relationship between media screen time, genetic background (gender, race/ethnicity), and blood pressure in U.S.youth aged 0 to 20 years old.

   Alternative hypothesis. There is a relationship between media screen time, genetic background (gender, race/ethnicity), and blood pressure in U.S. youth aged 0 to 20 years old.

In this chapter, I describe the research context, the instrument, and the analytical procedures. Included in the chapter is the description of the target population, sampling strategy, instrumentation, variables and variable operationalization, analysis strategy, and threats to validity.

Research Design and Rationale

The research design was a quantitative, cross sectional study using archival data from the NANHES (CDC, 2015d). Data collected were examined through descriptive statistics and an inferential analysis of variables. The approach for this quantitative study was the correlational research design (Campbell & Stanley, 1963). Quantitative studies are consistent with measuring relationships between parameters and uncovering novel patterns and are therefore suitable to assess the correlation between genetic backgrounds, exposure to media screen, and risk of CVD (Campbell & Stanley, 1963). Quantitative methods are based on statistical analysis.

Study Variables

To determine the relationship between excessive screen time and genetic background in increasing the risk of CVD, I analyzed whether exposure to media screen and genetic background (gender, race/ethnicity) could predict risk factors of CVD (CRP, lipids, and blood pressure). The independent variables (IV) were media screen and genetic background. The dependent variables (DV) were risk factors of CVD.

Independent variables. The independent variables are listed below. They are defined on the NHANES webpage (CDC, 2014).

Media screen time. It was defined as "Hours watch TV or videos past 30 days" (PAD590), "Hours use computer past 30 days" (PAD600), "Total media screen time TSC" (PAD590 + PAD600), and "Total media screen time-APA".

Gender. Boys and girls (RIAGENDR)

Ethnicity. The variable was reported for both boys and girls and included Mexican American, other Hispanic, non-Hispanic White, non-Hispanic Black, and other non-Hispanic race including non-Hispanic multiracial (RIDRETH1).

Dependent variables. All the dependent variables are listed below. They are defined on the NHANES NHANES webpage (CDC, 2014).

CRP. CRP was reported in mg/dL (LBXCRP). CRP is the most commonly used measure of the acute-phase response to inflammation. Inflammation may indicate an increased risk of heart disease.

Lipids. The lipids analyzed in this study were high-density lipoprotein cholesterol (LBDHDD), low-density lipoprotein cholesterol (LBDLDL), and triglycerides (LBXTR). High cholesterol poses individuals at a higher risk for developing heart disease as adults.

Healthy blood levels in youth are considered LDL < 130 mg/dL, HDL > 35 mg/dL, total cholesterol < 170 mg/dL, triglycerides < 150 mg/dL (University of Rochester Medical Center, 2015). For this study, lipids were examined in individuals who fasted at least 8.5 hours or more but less than 24 hours. Specimen were processed *in situ;* plasma was isolated, frozen at -20° C, and shipped to the University of Minnesota for analysis.

Blood pressure. Blood pressure was used for hypertension screening. Blood pressure was taken after study subjects rest for 5 minutes. Three consecutive blood pressure readings (systolic and diastolic) were obtained; one additional reading was done if one of the first three readings failed. Blood pressure was reported as systolic (BPXSY1, ….BPXSY4) and diastolic (BPXDI1, ….BPXDI4).

Research Design

The research design was a quantitative, cross sectional study using archival data from the NANHES (CDC, 2015d). The correlational design included both prediction as well as assessment of relationship, in the absence of variable manipulation (Campbell & Stanley, 1963). This type of study does not make inferences about the cause-effect relationship, as experimental designs do; therefore, the possibility that a different variable affects the relationship among variables cannot be excluded. However, correlational studies are ideal for obtaining preliminary data in support of full experimental studies investigating cause-effect relationships. Correlational studies are required in cases where variables of interest cannot be manipulated, such as age, gender, and ethnic background, as in the case of the current study (Campbell & Stanley, 1963). Correlation designs must be used in cases where it is unfeasible or unethical to manipulate the variables. Here, it would have been unfeasible and unethical to set up an experiment where people were

forced to spend a large part of their life in front of the media screen to then assess whether this acquired behavior increases the risk of CVD. Because of the large number of studies indicating that excessive screen time causes physical and mental harm, it would be against the principle of equipoise to assign a study subject to that arm of the study, as there is sufficient evidence that a reduction in the number of hours spent in front of the media screen is a beneficial treatment. Finally, correlation designs must be used in combination with surveys, unless the survey has built in a way to manipulate the variables. Since this study was based on a preexisting survey, the information had already been collected and cannot be manipulated.

Design choice consistent with research study. The current study was descriptive (cross-sectional study) and used the correlational design to examine the extent to which excessive screen time and genetic background relate to CVD risk factors, in order to predict the impact on the health of behavioral determinants combined with genetic factors.

Cross sectional designs consist in the study of a human condition at one point in time, in the absence of any intervention (Frankfort-Nachmias & Nachmias, 2008). They are suitable for understanding a naturally occurring condition (i.e., health status) of a particular group as well as for studying situations where an intervention would not be ethical (Campbell & Stanley, 1963). Cross sectional studies have several limitations: Samples cannot be chosen randomly, the results have limited generalizability, the independent variable(s) cannot be manipulated, and the cause-effect relationship among variables cannot be established (Campbell & Stanley, 1963). In addition, cross sectional studies are subjected to researcher interpretation and confounders and may therefore

result in misleading conclusions; they are, however, useful to establish associations (or lack of) between variables in a relatively short period of time and at low cost.

Time and resource constraints. A cross sectional study, using secondary and publicly available data, is the fastest and cheapest method to obtain preliminary results and formulate data driven hypotheses. Data are downloadable from the NHANES website at no cost to the user.

Methodology

Population

The study population consisted of participants from the 2009-2010 NHANES (CDC, 2015). NHANES consists of a series of surveys designed to measure the health and nutritional status of adults and youth in the U.S. Surveys combine both in person interviews and physical examinations. The current study was limited to youth age 0 to 20 years, for whom demographic data (age, gender, ethnicity), laboratory data (lipids, CRP, blood pressure), and behavioral data (media screen time) were known (see Table 1).

Sampling and Sampling Procedures

Sampling is a technique used to choose study participants representative of the entire population. In order for the study to be generalizable, sampling must be both meaningful and of appropriate size.

I used archived data from the 2009-2010 NHANES survey (CDC, 2015d). The survey assesses the health status and dietary habits of youth and adults in the United States and records changes over time. NHANES was designed by the National Center for Health Statistics, one of the institutes within the U.S. Centers for Disease Control and Prevention, and is responsible for collecting vital records and producing health statistics

for the nation. It is composed of health questionnaires and physiological and laboratory parameters. The survey includes demographic, health-related, and dietary questions, physical examination, and laboratory data (CDC, 2015d).

NHANES represents the most complete health survey of the U.S. population. The U.S. population in 2015 surpassed 320 million (Unites States Census Bureau, 2015a). According to the U.S. Census Bureau, in 2014, there were 73.6 million children. Of these, 25 million were in the 12 to 17 year age range, and the remaining in the 0 to 12 year age range (ChildStats.gov, 2015). In terms of ethnicity, most children are White (62.2), followed by Hispanics (17.4), Blacks (12.4), and Asian (5.2; Statista, 2015). American Indian and Alaska Native (0.7), Native Hawaiian (0.2), and two or more races (2) represent the smallest group (Statista, 2015). In 2015, males aged 0 to 13 were more than females (29,038 versus 27,802); males aged 14 to 17 were more than females (8,589 versus 8.207; United States Census Bureau, 2015b).

I used data obtained from the NHANES dataset and did not recruit any study subject or collect any primary data. Under these conditions, the sampling of this research study is purposeful convenience sampling (Frankfort-Nachmias & Nachmias, 2008), where data are selected among those available in the NHANES dataset that meet inclusion criteria (i.e., age, gender, available information on lipids, CRP, blood pressure, and screen time). Data from NHANES have been previously used to determine the association between media screen viewing and health parameters (Anderson et al., 2008; Herrick et al., 2014); however, those studies did not include the impact of gender and ethnicity on health outcomes.

Archival data. The NHANES survey was built using a probability based, four stage cluster-sampling strategy (CDC, 2013b). Cluster sampling is used in large sample studies to warrant a fair representation of all individuals in the population. The primary sampling stage was the single counties; the second stage consisted of segments within the primary single units within the selected counties; the third stage was represented by random households, and the fourth stage was individuals among the residents in the households. Finally, sample weights for underrepresented age, gender, and ethnicities were integrated in selection subdomains, and individuals were randomly drawn (CDC, 2013b). NHANES uses disproportionate stratification for people below federal poverty level, Hispanic, non-Hispanic black, and Asian, to guarantee inclusion of populations at risk (CDC, 2014).

Instrumentation and Operationalization of Constructs

Instrumentation. The data required for this study were collected by NHANES using a demographic questionnaire (age, gender, ethnicity), laboratory procedures (lipids, CRP, blood pressure), and physical activity questionnaire (media screen time). All data were collected at individual level. Questions for the demographic and physical activity questionnaire were asked in the home by trained interviewers, with the aid of a Computer-Assisted Personal Interviewing (CAPI) system. The CAPI is a software with built in data to edit and consistency checks to reduce the chances of mistakes during data entry, thus increasing the reliability of the questionnaire (National Health and Nutrition Examination Survey, 2011a). The questionnaire was written either in English or Spanish language. For youth aged 16 and less, a substitute person answered the questions on their behalf. The NHANES staff reviewed the information before departing the household, and

for a subset of data, the participants were contacted to verify the information on the survey for quality assurance and quality control.

Technicians and physicians working on laboratory procedures were professionals in medical technology, extensively trained on the procedure according to written manuals (National Health and Nutrition Examination Survey, 2011b). Scripts were prepared in English and Spanish to describe the laboratory procedure to participants. Individuals were excluded from blood samples-based analysis if they were affected by hemophilia, had received chemotherapy in the past 4 weeks, had minor medical conditions affecting the arms, or were allergic to cleansing reagents (National Health and Nutrition Examination Survey, 2011c). Samples were processed at the site of collection; sample analysis was done off site, in U.S. laboratories. All laboratories participating in NHANES data analysis had to abide to strict quality control protocols developed by NCHS.

Operationalization. Operationalization was done according to Table 1.

Media screen time. Media screen time is operationalized as categorical variable. PAD590 (0 = less than 1 hour, 1 = 1hour, 2 = 2 hours, … 5 ≥ 5 hours, 6 = none, 77 = refused, 99 = don't know). PAD600 (0 = less than 1 hour, 1 = 1hour, 2 = 2 hours, … 5 ≥ 5 hours, 6 = none, 77 = refused, 99 = don't know). I generated TSC by adding PAD590 to PAD600 and operationalized this variable in alignment with the previous two (0 = less than 1 hour, 1 = 1hour, 2 = 2 hours, … 5 ≥ 5 hours, 6 = none, 77 = refused, 99 = don't know).

In addition, I operationalized all three variables to include the APA guidelines of two hours or less screen time per day. Therefore, I defined PAD590APA as 0 (2 hours per day or less: 0 + 1 + 2 + 6) or 1 (more than 2 hours per day: 3 + 4 + 5); similarly, I

defined PAD600APA as 0 (2 hours per day or less: 0 + 1 + 2 + 6) or 1 (more than 2 hours per day: 3 + 4 +5); and I defined TSCAPA as 0 (2 hours per day or less = 0 + 1 + 2 + 6) or 1 (more than 2 hours per day: 3 + 4 + 5).

Gender. Gender is operationalized as categorical (1= male; 2 = female)

Ethnicity. Ethnicity/race is operationalized as categorical (1 = Mexican American, 2 = Other Hispanic, 3 = Non-Hispanic White, 4 = Non-Hispanic Black, and 5 = Other race, including Multi-Racial)

C-reactive protein. CRP is reported for both males and females, age 3 and over, on a continuous scale, ranging from 0.01 to 18.01 mg/dL. I operationalized CRP as 0 (normal) and 1 (at risk), as described in Chapter 4 and in Table I.

Lipids. HDL is reported for both males and females, age 6 and over, on a continuous scale, ranging from 0.01 to 18.01 mg/dL. Triglycerides are reported for both males and females, age 12 and over, on a continuous scale, ranging from 18 to 2,742 mg/dL. LDL is reported for both males and females, age 12 and over, on a continuous scale, ranging from 13 to 266 mg/dL. I operationalized lipids as 0 (normal) and 1 (at risk), as described in Chapter 4 and in Table I.

Blood pressure. Blood pressure is reported for all males and females, age 8 and older, except for those participants who were excluded from the study if the blood pressure cuff did not fit on the arm. Blood pressure is reported separately for systolic and diastolic blood pressure. Unit of measure are mm Hg. Each individual has up to 4 readings, reported on a continuous scale; the range for individual readings of systolic pressure is 70 -232 mm Hg, for diastolic pressure is 0-134 mm Hg. For each study subject, I calculated the average of all individual readings available (BPXDI1,

….BPXDI4, and BPXSY1, ….BPXSY4). I then operationalized both BPXDI and BPXSY as 0 (normal) and 1 (at risk), as described in Chapter 4 and in Table 1

able 1

Operationalization of Variables for a Correlation Study Among U.S. Youth

Variable	**Description**	**IV**	**DV**	**Coding**	**Level**	**Operationalization**
Media screen time	Hours watch TV or videos past 30 days	X		PAD590	categorical	0 = less than 1 hour, 1 = 1hour, 2 = 2 hours, … 5 ≥ 5 hours, 6 = none, 77 = refused, 99 = don't know
Media screen time	Hours use computer past 30 days	X		PAD600	categorical	0 = less than 1 hour, 1 = 1hour, 2 = 2 hours, … 5 ≥ 5 hours, 6 = none, 77 = refused, 99 = don't know
Total media screen time	PAD590 + PAD600	X		TSC	categorical	0 = less than 1 hour, 1 = 1hour, 2 = 2 hours, … 5 ≥ 5 hours, 6 = none, 77 = refused, 99 = don't know
Total media screen time_APA	PAD590 categorized according to APA guidelines	X		PAD590APA	categorical	0 (two hours per day or less of TV/video: 0+1+2+ 6) or 1 (more than two hours per day: 3+4+5)
Total media screen time-APA	PAD600 categorized according to APA guidelines	X		PAD600APA	categorical	0 (two hours per day or less of computer: 0+1+2+ 6) or 1 (more than two hours per day: 3+4+5)

table continues

Variable	Description	IV	DV	Coding	Level	Operationalization
Total media screen time-APA	total screen time categorized according to APA guidelines	X		TSCAPA	categorical	0 (two hours per day or less = 0 +1+2+ 6) or 1 (more than two hours per day = 3+4+5)
Gender	Boys and girls	X		RIAGENDR	Nominal	1= male; 2 = female
Ethnicity/race	Mexican American, Other Hispanic, Non-Hispanic White, Non-Hispanic Black, and Other race	X		RIDRETH1	categorical	1 = Mexican American, 2 = Other Hispanic, 3 = Non-Hispanic White, 4 = Non-Hispanic Black, and 5 = Other race, including Multi-Racial
CRP	C-reactive protein (mg/dL)		X	LBXCRP	categorical	0 = normal, 1 = at risk
Lipids	high-density lipoprotein cholesterol		X	LBDHDD	categorical	0 = normal, 1 = at risk
Lipids	low-density lipoprotein cholesterol		X	LBDLDL	categorical	0 = normal, 1 = at risk
Lipids	triglycerides		X	LBXTR	categorical	0 = normal, 1 = at risk
Blood pressure	Blood pressure (systolic)		X	BPXSY1, ….BPXSY4	categorical	0 = normal, 1 = at risk
Blood pressure	Blood pressure (diasystolic)		X	BPXDI1, ….BPXDI4	categorical	0 = normal, 1 = at risk

Data Cleaning and Storage

I cleaned the data using IBM SPSS version 21. Data cleaning is used to eliminate error and redundancy, thus increasing validity and accuracy. The dataset that I used consisted of single files for each of the variables of interest that were merged in a single file. Data cleaning was particularly important after merging the files to ensure consistency of the sets of data, as manipulation may introduce mistakes. I checked the merged file against the single files, and ensured that the correct files have been merged, and the number of study subject was consistent between the single and the merged file. In the merged file, I reviewed the minimum and maximum value and compared it to the expected range from the NHANES database. I checked that the measurement scale of the data was the same as that in the original database; I also performed quality control on 10 of the data from the merged file, to confirm equivalence between the individual files and the merged file. Variables' typos, spelling data, and mislabeled data are common mistakes. I double-checked dummy values (i.e. 77 = refused, 99 = don't know) against the file's description of coding, contradicting data and non-unique identifiers, and I removed blank cases and fix the mistakes. The command sort and select on the SPSS software helped with this portion of data cleaning. I recoded variables (i.e. blood pressure and screen time), and converted the variables into the proper level of measure.

Data Analysis Plan

I analyzed the data using IBM SPSS version 21. After cleaning the data, my strategy consisted of running frequency tables and descriptive statistics for each variable, checking the output to see if variables had the expected range and frequency, and

identifying missing values to ensure that they were truly missing. I checked the frequency of the recoded variables against those of the original variables.

Descriptive. I used frequencies, percentages and descriptive statistics to summarize the data (categorical and continuous, respectively), and to gain a basic knowledge of the samples and the variables in the study.

Inferential analysis. In addition, I determined if the means for the markers of CVD (CRP, lipids, or blood pressure) were different between boys and girls, or among ethnicities. To compare the means of CRP, lipids, or blood pressure (continuous variables) between two groups (boys and girls) I used the Kruskal-Wallis. To compare the median of CRP, lipids, or blood pressure (continuous variables) among several ethnic groups, I used the Mann-Whitney test. Similarly, to determine if the median of CRP, lipids, or blood pressure were different among youth spending 0 to more 5 hours of time with TV or computer, in hourly increments, I used the Mann-Whitney test. If the time categories were only two (2 hours or less, and more than 2 hours), I used the Kruskal-Wallis test.

I also used inferential analysis to determine the relationship between IVs and DVs. My hypothesis was that a relationship exists between media screen time, genetic background (gender, race/ethnicity), and risk factors of CVD in U.S. youth age 0-20. I originally planned to use multiple linear regression. However, the assumption of normality of distribution was not met; therefore I had to switch to a non-parametric test.

I used bivariate analysis to determine if a model with gender, ethnicity, and the interaction term gender*ethnicity, in addition to media screen time, was a better predictor of CRP with respect to a model with media screen time only. The full test models were

compared to the constant models only. I compared R square and p value between the full model and the constant model only. I evaluated how the IV in the full model modified the ability to predict the DV. The Exp(B) indicated the odds ratio for each IV; the p value and CI indicated if the relationship between DV and IV were significant. The same approach was used to evaluate the relationship between amount of screen time with lipids or blood pressure.

Binomial logistic regression is used to predict a dichotomous dependent variable based on one or more continuous or nominal independent variables. The purpose is to determine if variables are associated with each other (*p* value), to estimate the strength of their relationship, and to find the equation that might predict the risk of CVD (cholesterol, LDL, HDL, CRP, blood pressure) from screen time, gender and ethnicity. Six assumptions must be met for a logistic regression analysis to be valid. The dependent variable should consist of 2 categorical, independent (unrelated) groups (i.e., a dichotomous variable). The independent variables (2 or more) should be measured at the continuous or nominal level. There should be independence of observations. Data must not show multicollinearity. There must be a linear relationship between the variables; the sample size must be appropriate for the expected size effect and number of IV used. For binary logistic regression, in order to detect an odd ratio of 1.5, assuming a Type I error (alpha) of 5 and a type II error of 20 (beta), I would need 778 subjects (G*Power, z tests, logistic regression) (Universitat Dusseldorf, 2013).

Threats to validity. Threats to the validity of this study consisted primarily in the type of sampling used, and the lack of control over potential confounders. Convenience sampling in place of random sampling can pose limitations over the generalizability of

findings, since the researcher cannot estimate to which extent the sample is representative of the population at large (Frankfort-Nachmias & Nachmias, 2008). Lack of control of confounders may also bias the results, and care had to be used when interpreting the data analysis. The relationship with known potential confounders was not included at this stage.

Content validity and construct validity of the NHANES survey has been established through multiple research efforts confirming validity of national estimates by comparison to other national surveys (CDC, 2013b). The information collected in the NHANES database has been used to support national programs for the prevention of high cholesterol and hypertension. Empirical validity of the cardiovascular risk factors used in this study (i.e. blood pressure, lipids, CRP) has been well established by a wealth of literature. High levels of total cholesterol, LDL, and triglycerides, and low levels of HDL, are directly responsible for one third of the cases of ischemic heart diseases (World Heart Federation, 2015). CRP levels have been linked to CVD through inflammatory damage to the vasculature (Wu et al., 2015). Furthermore, these biomarkers were directly measured in the blood of study subjects, minimizing the threats to construct validity of the survey. Finally, the NHANES survey has been extensively queried in the past to measure the relationship between screen time, sedentary life, obesity, and race (Fakhouri et al., 2014).

Reliability. Reliability measures how consistent and dependable are the data collected through the survey (Frankfort-Nachmias & Nachmias, 2008). The NHANES has been extensively used to describe the prevalence of screen time among youth of different ages and ethnicity, and by type of media entertainment (Herrick et al., 2014; Fakhouri et al., 2011), and to measure the relationship between television viewing time

and health risks such as obesity, physical activity, high blood pressure, poorer dietary quality, and cardiovascular diseases (Camhi et al., 2013; Fakhouri et al., 2013; Ford, 2012; Janssen et al., 2013; Peart, Velasco Mondragon, Rohm-Young, Bronner, & Hossain 2011; Sisson, Shay, Broyles, & Leyva, 2012; Twarog, Politis, Woods, Boles, & Daniel, 2015). Findings were consistent among each other and with similar studies conducted from other databases (CDC, 2013b). It has been suggested that self-rated health measurements in surveys are only moderately reliable among racial/ethnic minorities and adults with lower education (Zajacova & Dowd, 2011). To mitigate this potential issue and increase reliability of the instrument and fair representation of all U.S. population, the NHANES survey has over-sampled population at risk (National Health and Nutrition Examination Survey, 2014).

Ethical procedures. All data are archived data from the NHANES. The NCHS Research Ethics Review Board approved the protocol (protocol # 2005-06). Individuals' identity had been protected by de-identification of the data. All participants gave their informed consent at the time of the interview. I completed the Research Ethics Review Application and submit the Walden University IRB, to request approval to conduct my analysis of archived data. In the application, I described the proposed research, the potential risks and benefits, procedures to maintain data integrity and confidentiality, the NHANES survey, and a description or research participants, with inclusion and exclusion criteria. Until the IRB approval had been obtained, no dataset were accessed or analyzed.

Summary

The purpose of this study was to determine the effect of genetic background combined with excessive media screen time on markers of cardiovascular risk in youth

aged 0-20 years. The work was a cross sectional, quantitative study that used archived data from the largest health and nutritional survey in the country, the NHANES. Cross sectional studies are considered quasi-experimental studies, as they lack the rigor of randomization and variable manipulation, thus limiting the validity and generalizability of the study. They, however, carry the benefit of providing quickly and at relatively low cost valuable preliminary information of variables association (or lack of), which can be used to justified more through, expensive studies. The sample population analyzed reflects the U.S. population within the limits of the sampling strategy of the NHANES survey, a probability based, 4 stage cluster-sampling strategy with sample weights for underrepresented age, gender and ethnicities.

The tool used to analyze the data were statistical analysis, as appropriate for manipulation of quantitative information. Independent variables (i.e. exposure to media screen, genetic background and risk factors of CVD) were operationalized as categorical variables. The dependent variables (CRP, lipids, and blood pressure) operationalized in categories (i.e. above or below normal range).

Operationalization of total screen time included a variable (TSC APA), which comprises the APA recommendation of 2 hours limit of screen time per day (total screen time more or less than APA recommendations). Additionally, operationalization of total screen time included a variable consisting of total screen time (computer plus television), as opposite to computer time or television time alone.

Statistical analysis consisted in frequencies or percentages and descriptives of population and variables, to gain basic knowledge of the data. Then I applied inferential analysis to assess the association among screen time (IV) and CVD (DV). I used binary

regression using screen time alone first, and then added gender and ethnicity to see if the predictive power increases with respect to the constant model.

The main threats to the validity of the study were the lack of control over potential confounders, the convenience sampling used in place of random sampling when choosing study subjects from the NHANES database, and the inherent limitations of cross-sectional studies. The major threat to reliability was the use of self-reported data for screen time viewing, which may different from objective, physical measure. In Chapter 4, I present a detailed description of the results of my analysis.

Chapter 4: Results

Introduction

The purpose of this study was to determine the effect of genetic background combined with excessive media screen time on markers of cardiovascular risk in U.S. youth aged 0 to 20 years. The hypothesis is that a relationship exists between media screen time, genetic background (gender, race/ethnicity), and risk factors of CVD in United States youth aged 0 to 20.

The research questions addressed the relationship between media screen time, genetic background, and markers of CVD. The IVs were media screen time (hours spent watching TV or videos, hours using the computer, total hours spent in front of the media screen) and genetic background (gender and race/ethnicity). No data were available on the use of smartphones in the NHANES database; therefore, this type of screen activity was not included in the analysis. The DVs were biomarkers of CVD (CRP, lipids, and blood pressure). No covariables were used in this analysis.

Chapter 4 includes a description of the data-collection method, a summary of descriptive statistics, and the quantitative statistical analysis of the data. The nonparametric Kruskal-Wallis and the Mann-Whitney tests were used to determine differences in median among groups since the assumption of normality of distribution was not met. Logistic regression analysis was used to test Null Hypotheses 1 to 3 and to estimate the relationship between media screen time, genetic background, and biomarkers of CVD.

Tables and graphs are included for clarification purposes. The data collected included descriptive statistics of the sample population, distribution of media screen time

by ethnicity and gender, measures of the central tendency of biomarkers of CVD by ethnicity and gender, and differences in the median of biomarkers of CVD according to exposure to screen time. A comparison analysis was performed on the levels of CVD biomarkers across ethnicities or between genders using the Kruskal-Wallis or the Mann-Whitney test, respectively. Similarly, a comparison analysis was performed on levels of CVD biomarkers across the number of hours spent in front of the screen, using the Kruskal-Wallis or the Mann-Whitney test. Data were analyzed using SPSS version 21. Graphs and tables were generated using SPSS version 21 and Excel 2010. Data collection and descriptive statistics follow this introduction. The remainder of Chapter 4 is organized by research question.

Data Collection

After obtaining approval from Walden University's Institutional Review Board, the data were downloaded from the CDC website for use in SPSS21.0. Data for the individual variables were downloaded from the NHANES 2009-2010 website. This was the most recent dataset that had information on all the variables of interest in the study. The individual files were merged in SPSS using the participants' identification number. Descriptive data from the merged file were compared against the individual files for quality control. Data were sorted by individual variables, and the minimum and maximum for each variable was crosschecked against the NHANES codebook. According to NHANES, some of the variables were collected different age ranges, leaving missing cases. Blood pressure (BP) was collected for subjects age 16 and over, HDL for age 6 and over, LDL for age 12 and older, triglycerides for age 12 and older; and CRP for age 3 and over. More than 5 of the individuals had incomplete information

on biomarkers of CVD; hence, the samples vary depending upon the variables analyzed; the sample size for each variable is reported in the descriptive section of Chapter 4. For my analysis, I addressed this issue using pairwise deletion. In addition, I excluded those variables whose value was outside the expected range (i.e., CRP = 0, systolic blood pressure = 0) from the analysis, per NHANES codebook. I also removed from the dataset individuals lacking information on the amount of time spent on media screen time (either television or computer) and individuals older than 20 years to align the data with the research question. There were no individuals who refused to answer or did not know how much time they had spent in front of a media screen in the past 30 days.

Datasets were checked for the presence of outliers and distribution using boxplots and histograms. Data did not follow a normal distribution, even after the removal of outliers. Furthermore, outliers in a biological sample may represent a diseased state and should not be eliminated. Therefore, no outliers were removed from the dataset, and the inferential analysis was done using binary logistic regression.

Descriptive Demographic Characteristics of the Sample

Reported in Table 2 are the baseline descriptive and demographic characteristics of the sample. The frequency of demographic variables was the same for boys and for girls. There was approximately the same number of boys and girls and of similar age. The distribution across the ethnicities was the same for boys and girls.

Table 2

Descriptive Statistics by Ethnicity and Gender of Study Sample Population (N =4,111)

	n		Age mean	Median	Variance	Stdev	Range
Gender							
Boys	2095	51	8.5	8	37.967	6.162	20
Girls	2016	49	8.24	8	37.417	6.117	20
Ethnicity (all)							
Mexican American	1179	28.7	8.06	7	37.695	6.14	20
Other Hispanic	472	11.5	8.15	8	36.352	6.029	20
NonHispanic White	1390	33.8	8.37	8	38.133	6.175	20
NonHispanic Black	788	19.2	9.03	9	37.556	6.128	20
Other race	282	6.9	8.23	7	37.137	6.094	20
Ethnicity (boys)							
Mexican American	609	29.1	8.27	8	38.048	6.168	20
Other Hispanic	244	11.6	8.03	7	36.962	6.08	20
NonHispanic White	708	33.8	8.51	8	38.598	6.213	20
NonHispanic Black	391	18.7	9.15	9	38.711	6.222	20
Other race	143	6.8	8.49	8	33.477	5.786	20
Ethnicity (girls)							
Mexican American	570	28.3	7.84	7	37.287	6.106	20
Other Hispanic	228	11.3	8.27	8	35.829	5.986	20
NonHispanic White	682	33.8	8.23	7	37.666	6.137	20
NonHispanic Black	397	19.7	8.92	9	36.485	6.04	20
Other race	139	6.9	7.96	6	41.027	6.405	20

The population, however, was oversampled for Mexican Americans (26.4) and non-Hispanic Blacks (20.8) and undersampled for non-Hispanic Whites (33.9) to guarantee an inclusion of populations at risk (CDC, 2014). In the United States, most children are White (62.2), followed by Hispanics (17.4), Blacks (12.4), and other races (8; Statista, 2015). In addition, the large amount of missing data represents a potential for lack of external validity.

Baseline Descriptive of Categorical Variables

The frequencies and percentages for measures of screen time viewing (hours watching TV or videos and hours using computers) are reported in Table 3 per ethnicity and regardless of gender (all), as well as per ethnicity and by gender (boys, girls).

Table 3

Distribution by Ethnicity/Race and Gender of Media Screen Time Among the Study Sample Size (N = 4,111)

	<1h		1h		2h		3h		4 h		> 5h		none	
	n	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%
Hours watching TV or videos (all)														
Mexican American	946	80	67	6	89	8	29	2	22	2	19	2	7	1
Other Hispanic	378	80	20	4	39	8	15	3	10	2	9	2	1	0
NonHispanic White	1126	81	77	6	92	7	52	4	18	1	20	1	5	0
NonHispanic Black	628	80	45	6	66	8	26	3	11	1	12	2	0	0
Other race	218	77	13	5	21	7	14	5	9	3	7	2	0	0
Hours watching TV or videos (boys)														
Mexican American	488	80	44	7	42	7	13	2	11	2	7	1	4	1
Other Hispanic	200	82	7	3	21	9	5	2	5	2	5	2	1	0
NonHispanic White	569	80	44	6	40	6	32	5	10	1	9	1	4	1
NonHispanic Black	315	81	20	5	31	8	15	4	4	1	6	2	0	0
Other race	112	78	8	6	11	8	5	3	4	3	3	2	0	0
Hours watching TV or videos (girls)														
Mexican American	458	80	23	4	47	8	16	3	11	2	12	2	3	1
Other Hispanic	178	78	13	6	18	8	10	4	5	2	4	2	0	0
NonHispanic White	558	82	33	5	52	8	20	3	8	1	11	2	1	0
NonHispanic Black	313	79	25	6	35	9	11	3	7	2	6	2	0	0
Other race	106	76	5	4	10	7	9	6	5	4	4	3	0	0

(table continues)

	<1h		1h		2h		3h		4 h		> 5h		none	
	n	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%
Hours using computers (all)														
Mexican American	1000	85	53	4	20	2	7	1	3	0	6	1	90	8
Other Hispanic	399	85	21	4	9	2	7	1	1	0	0	0	35	7
NonHispanic White	1192	86	44	3	22	2	5	0	2	0	4	0	121	9
NonHispanic Black	674	86	40	5	12	2	7	1	1	0	2	0	52	7
Other race	236	84	13	5	2	1	3	1	1	0	0	0	27	10
Hours using computers (boys)														
Mexican American	523	86	26	4	10	2	3	0	3	0	4	1	40	7
Other Hispanic	208	85	12	5	4	2	2	1	0	0	0	0	18	7
NonHispanic White	595	84	21	3	15	2	3	0	1	0	2	0	71	10
NonHispanic Black	333	85	19	5	7	2	6	2	0	0	0	0	26	7
Other race	120	84	7	5	2	1	0	0	1	1	0	0	13	9
Hours using computers (girls)														
Mexican American	477	84	27	5	10	2	4	1	0	0	2	0	50	9
Other Hispanic	191	84	9	4	5	2	5	2	1	0	0	0	17	7
NonHispanic White	597	88	23	3	7	1	2	0	1	0	2	0	50	7
NonHispanic Black	341	86	21	5	5	1	1	0	1	0	2	1	26	7
Other race	116	83	6	4	0	0	3	2	0	0	0	0	14	10

The highest prevalence of children watching TV or videos 2 hours of more per day was between other race (18) and non-Hispanic Blacks (15) (Figure 2). Other Hispanic (4), Mexican American (3), and non-Hispanic Blacks (3) had the highest prevalence of using the computer for two hours or more per day (Figure 2).

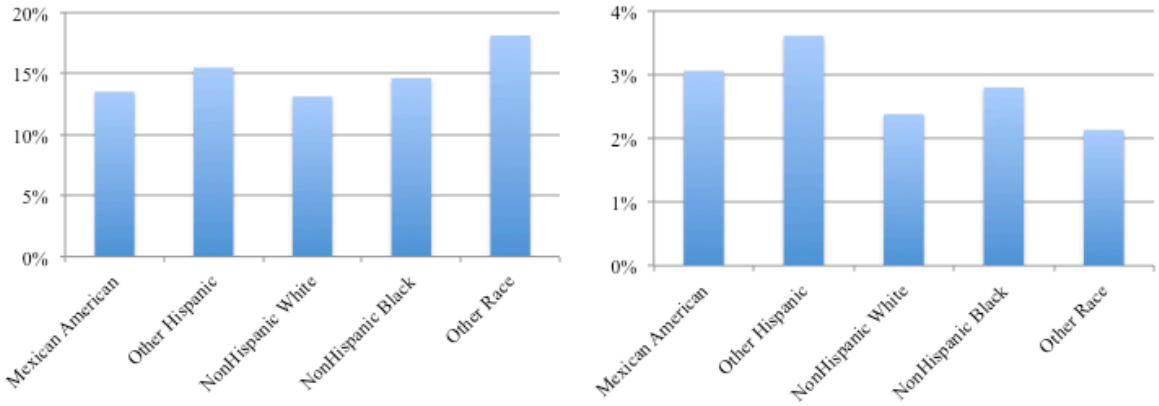

Figure 2. Distribution of individuals by race/ethnicity watching TV or videos ($N = 580$) (left panel) or using computers ($N = 114$) (right panel) for 2 hours or more per day.

Mexican American and other race girls reported watching TV and videos 2 hours or more per day. Other Hispanic (4.8) and Mexican American girls (2.81) had the highest prevalence for using the computer for 2 hours per day or more, followed by non-Hispanic Blacks (2.3), other race (2.2) and Non-Hispanic Whites (1.8; Figure 3 and Table 4).

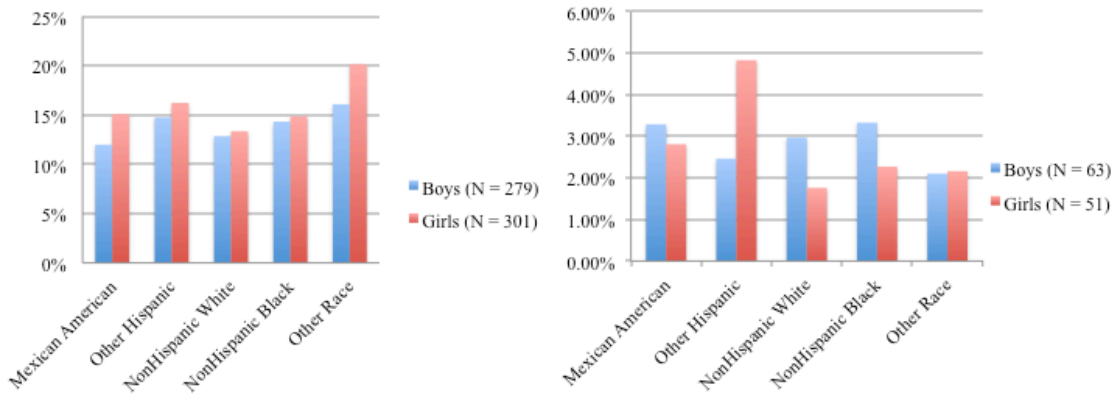

Figure 3. Distribution by race/ethnicity and gender, watching TV or videos (left panel) or using computers (right panel) for 2 hours or more per day.

When measuring time spent in front of a media screen as above or below the 2-hour limit recommended by the American Pediatric Association (APA, n.d.), more Mexican Americans than any other race were above the limit for television viewing, while more non-Hispanic Whites than any other race were above the limit for computer use (Table 4).

Table 4
Total Screen Time According to APA Recommendations: Distribution by Ethnicity/Race and Gender (N = 4111)

	0-2 hours per day		> 2 hours per day	
	n	%	*n*	%
Hours watching TV or videos (all)				
Mexican American	1109	0.94	70	0.06
Other Hispanic	438	0.93	34	0.07
NonHispanic White	1300	0.94	90	0.06
NonHispanic Black	739	0.94	49	0.06
Other race	252	0.89	30	0.11
Hours using computer (all)				
Mexican American	1163	0.99	16	0.01
Other Hispanic	464	0.98	8	0.02
NonHispanic White	1379	0.99	11	0.01
NonHispanic Black	778	0.99	10	0.01
Other race	278	0.99	4	0.01
Total screen time (computer + television) (all)				
Mexican American	1073	0.91	106	0.09
Other Hispanic	422	0.89	50	0.11
NonHispanic White	1265	0.91	125	0.09
NonHispanic Black	714	0.91	74	0.09
Other race	246	0.87	36	0.13
Hours watch TV or videos (boys)				
Mexican American	578	0.95	31	0.05
Other Hispanic	229	0.94	15	0.06
NonHispanic White	657	0.93	51	0.07
NonHispanic Black	366	0.94	25	0.06
Other race	131	0.92	12	0.08
Hours use computer (boys)				
Mexican American	599	0.98	10	0.02
Other Hispanic	242	0.99	2	0.01
Mexican American				
Other Hispanic				

table continues

	0-2 hours per day		> 2 hours per day	
	n	%	n	%
Hours use computer (boys)				
Mexican American	561	0.92	48	0.08
Other Hispanic	219	0.90	25	0.10
NonHispanic White	640	0.90	68	0.10
NonHispanic Black	354	0.91	37	0.09
Other race	128	0.90	15	0.10
Hours watch TV or videos (girls)				
Mexican American	531	0.93	39	0.07
Other Hispanic	209	0.92	19	0.08
NonHispanic White	643	0.94	39	0.06
NonHispanic Black	373	0.94	24	0.06
Other race	121	0.87	18	0.13
Hours use computer (girls)				
Mexican American	564	0.99	6	0.01
Other Hispanic	222	0.97	6	0.03
NonHispanic White	677	0.99	5	0.01
NonHispanic Black	393	0.99	4	0.01
Other race	136	0.98	3	0.02
Total screen time (computer + television) (girls)				
Mexican American	512	0.90	58	0.10
Other Hispanic	203	0.89	25	0.11
NonHispanic White	625	0.92	57	0.08
NonHispanic Black	360	0.91	37	0.09
Other race	118	0.85	21	0.15

Figure 4 is a summary of the distribution of percentage for each nominal category of screen time (television viewing, computer use, television plus computer use), by race as well as by race and by gender.

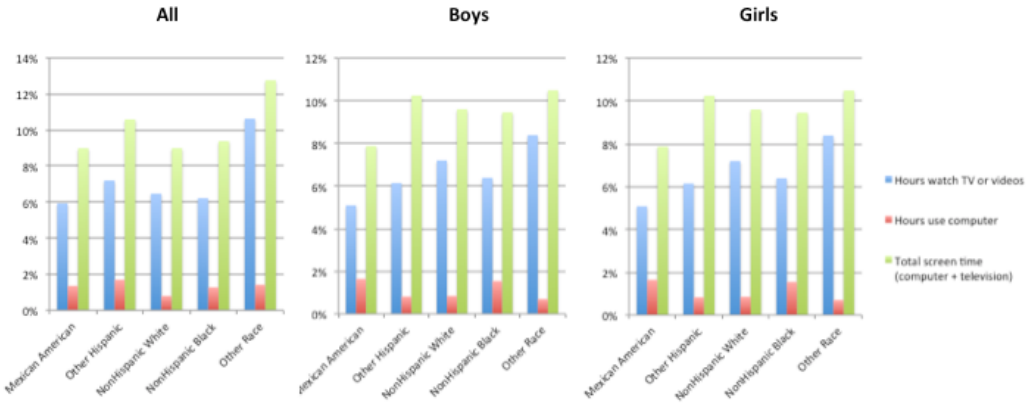

Figure 4. Two or more hours of screen time daily- prevalence by ethnicity/race.

Among the boys, Other Hispanic (10), Non-Hispanic White (10) and Other race (10) had the highest prevalence of total screen time, followed by Non-Hispanic Blacks (9), Mexican American (8). Among the girls, Other race (15), had the highest prevalence of total screen time, followed by Other Hispanic (11), Mexican American (10), Non-Hispanic Blacks (9) and Non-Hispanic White (8).

Univariate Analyses

Descriptive statistics were run for each biomarker of CVD, according to ethnicity. The Kruskal Wallis test and the Mann–Whitney U test were conducted to assess whether significant differences existed between the medians and means of CVD biomarkers among races and genders, respectively. The use of non-parametric test was warranted by the lack of normal distribution of the variables, even after removal of outliers. Results of

the analysis are summarized in the table 5 and represented graphically in Figure 5. Expression levels of CVD biomarkers changed with ethnicity (Figure 4a) and gender (Figure 4b). However the analysis did not yield any statistically significant differences at the 95 confidence level. Triglycerides were borderline significant ($p = 0.055$), suggesting that this result may be possibly significant if measured on a larger scale, warranting further research.

Table 5
Biomarkers of CVD risk: difference in mean, according to screen time measured in hourly increments

	Hours watch TV or videos past 30 days														
0	n	<1	n	1h	n	2h	n	3h	n	4h		n	≥5h	n	*p*
All															
CRP	0.4	12	0.3	2802	0.4	191	0.3	271	0.3	117	0.4	55	0.3	50	0.37
HDL	50	11	53	2637	51	175	52	251	53	107	58	49	58	54	**0.04***
Trygl	100	4	123	1155	116	75	114	109	118	56	107	26	152	24	0.94
LDL	97	4	110	1164	113	75	110	111	101	57	111	16	98	24	0.38
Sys BP	109	12	114	2506	110	165	114	233	114	97	112	51	115	49	0.62
Dia BP	67	12	63	2506	68	165	64	233	60	97	64	51	65	49	0.38
Boys															
CRP	0.2	8	0.3	1431	0.4	108	0.4	122	0.2	59	0.2	27	0.3	21	0.80
HDL	36	8	53	1339	51	95	52	117	55	57	58	29	61	22	0.09
Trygl	104	3	124	615	117	41	114	53	119	27	104	16	154	10	0.86
LDL	107	3	110	619	110	41	108	53	108	27	107	16	87	10	0.71
Sys BP	112	9	114	1279	110	92	115	109	112	52	113	24	118	21	0.34
Dia BP	68	9	62	1279	61	92	64	109	62	52	67	24	67	21	0.09
Girls															
CRP	0.6	4	0.3	1371	0.3	83	0.3	149	0.3	58	0.6	28	0.3	29	0.25
HDL	59	3	53	1298	51	80	53	134	52	50	60	21	57	32	0.44
Trygl		14	122	540	116	34	115	56	118	29	113	10	151	14	0.99
LDL		1	111	545	117	34	113	58	94	30	118	10	106	14	0.14
Sys BP	102	3	114	1221	112	73	113	124	112	45	112	27	113	28	0.94
Dia BP	63	3	63	1221	62	73	63	124	58	45	60	27	63	28	0.63

	Hours used computers past 30 days													
	none	n	<1	n	1h	n	2h	n	3h	n	4h	n	≥5h	n	p
All															
CRP	0.3	282	0.3	2975	0.3	149	0.5	54	0.1	22	0.4	7	0.3	9	0.26
HDL	52	57	53	695	54	33	55	10	56	7	59	3	52	4	0.59
LDL	120	200	123	2265	112	100	149	40	112	24	59	6	120	9	0.13
Trygl	109	198	111	2255	102	100	114	40	126	24	101	6	109	9	0.21
Sys BP	112	252	114	2632	113	134	112	44	74	26	111	3	112	6	0.22
Dia BP	62	252	63	2632	62	134	60	44	38	26	74	3	62	6	0.11

table continues

	none	n	<1	n	1h	n	2h	n	3h	n	4h	n	≥5h	n	p
Boys															
CRP	0.3	148	0.3	1501	0.2	74	0.5	32	0.4	12	0.1	5	0.2	4	0.68
HDL	51	140	53	1404	52	74	58	31	52	11	58	4	64	3	0.36
LDL	124	71	124	640	106	35	92	11	143	4	112	2	53	2	0.25
Trygl	111	71	110	644	96	35	94	11	108	4	126	2	105	2	0.13
Sys BP	112	130	114	1343	113	63	110	22	116	14	99	1	112	3	0.75
Dia BP	63	130	63	1343	61	63	65	22	62	14	55	1	77	3	0.61
Girls															
CRP	0.3	134	0.3	1474	0.3	75	0.4	22	0.5	10	0.2	2	0.5	5	0.15
HDL	54	128	53	1384	55	64	53	24	57	11	49	1	56	5	0.87
LDL	115	54	122	578	118	36	120	14	170	1			71	1	0.64
Trygl	108	56	111	584	108	36	99	14	137	1			94	1	0.75
Sys BP	113	122	114	1289	113	71	115	22	108	12	62	2	110	3	0.45
Dia BP	60	122	63	1289	64	71	65	22	58	12	29	2	70	3	0.10

* significant at the 95% confidence level

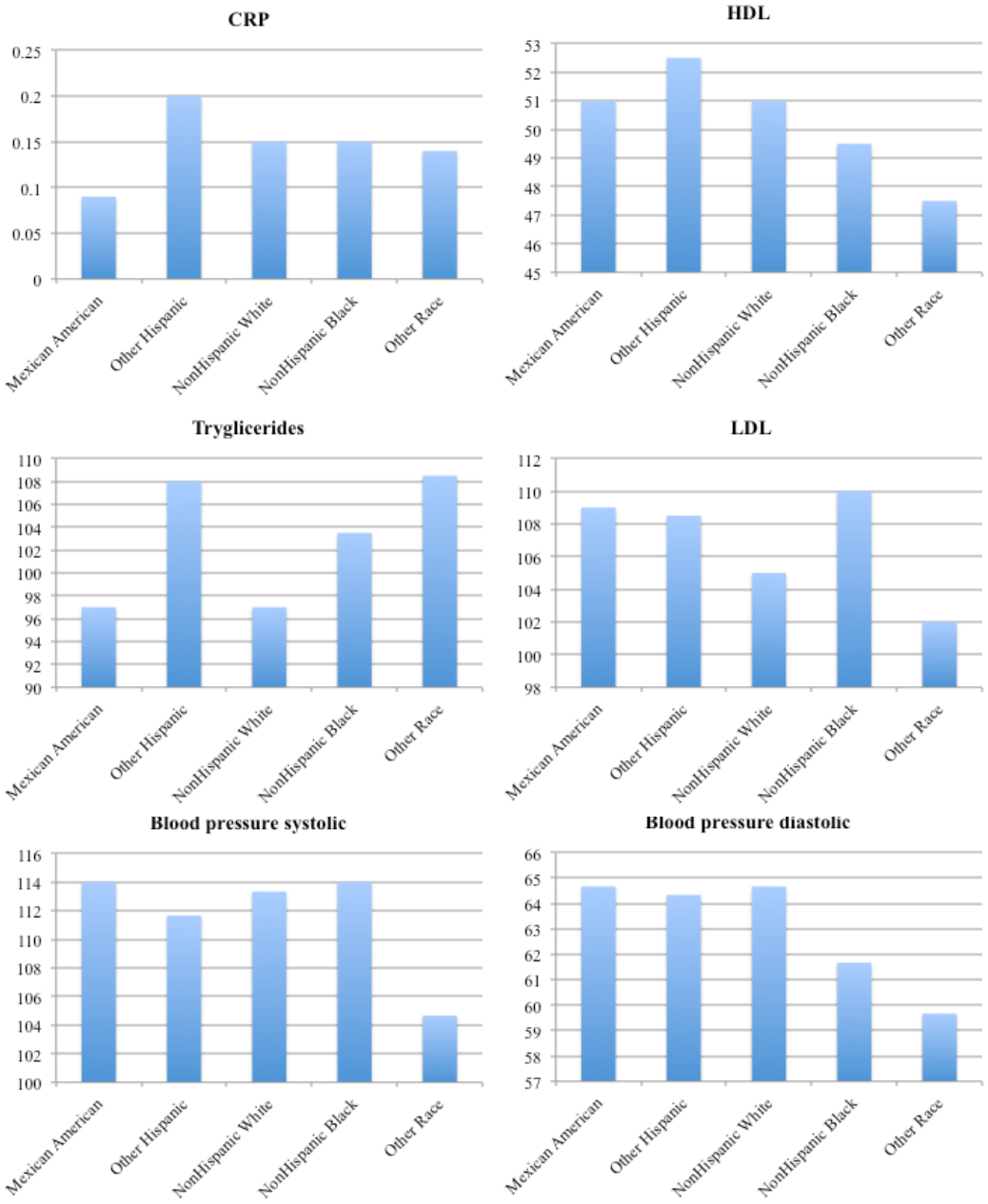

Figure 5a. Median values of biomarkers of CVD by ethnicity.

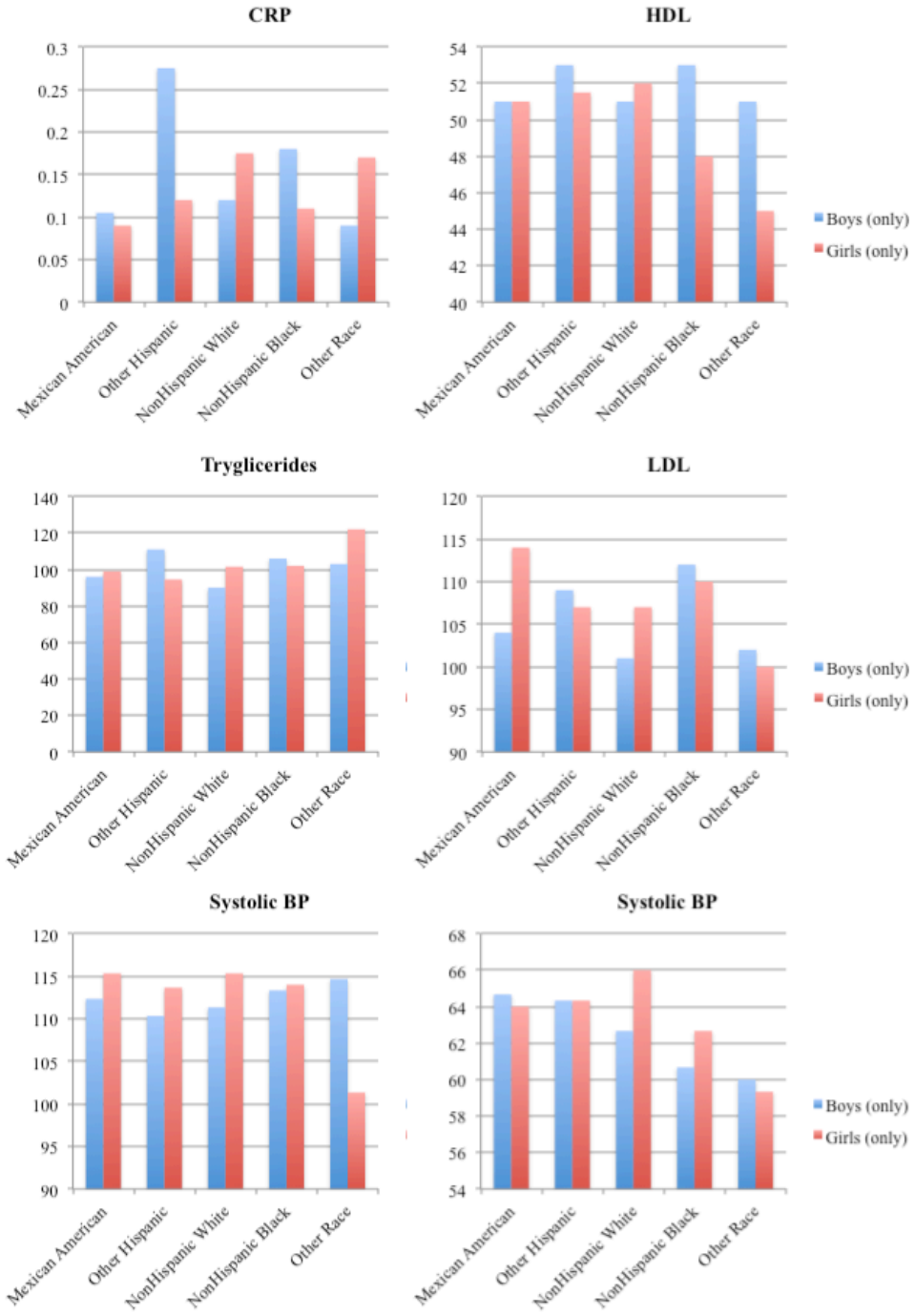

Figure 5b. Median values of biomarkers of CVD by ethnicity and gender.

Expression levels of CVD biomarkers were also assessed in relationship to the amount of time spent in front of a media screen. Table 6 and Figure 6 include summaries of the values of CRP, HDL, triglycerides, LDL and blood pressure in children exposed to TV of computers at increments of 1 hour daily. When evaluating the biomarkers in relationship to time spent in front of the TV, the levels of HDL were statistically different at the 95 confidence level in the sample population as a whole, but not when the sample population was stratified by gender, suggesting that a larger sample size is necessary to reach statistical significance in the sample when stratified by gender.

Table 6
Biomarkers of CVD risk: difference in mean, according to screen time measured in hourly increments

	Hours watch TV or videos past 30 days														
	none	n	<1	n	1h	n	2h	n	3h	n	4h	n	≥5h	n	*p*
All															
CRP	0.4	12	0.3	2802	0.4	191	0.3	271	0.3	117	0.4	55	0.3	50	0.37
HDL	50	11	53	2637	51	175	52	251	53	107	58	49	59	54	**0.04***
Trigl	100	4	123	1155	116	75	114	109	118	56	107	26	152	24	0.94
LDL	97	4	110	1164	113	75	111	111	101	57	111	16	98	24	0.38
Sys BP	109	12	114	2506	110	165	114	233	114	97	112	51	115	49	0.62
Dia BP	67	12	63	2506	68	165	64	233	60	97	64	51	65	49	0.38
Boys															
CRP	0.2	8	0.3	1431	0.4	108	0.4	122	0.2	59	0.2	27	0.3	21	0.80
HDL	36	8	53	1339	51	95	52	117	55	57	58	29	61	22	0.09
Trigl	104	3	124	615	117	41	114	53	119	27	104	16	154	10	0.86
LDL	107	3	110	619	110	41	108	53	108	27	107	16	87	10	0.71
Sys BP	112	9	114	1279	110	92	115	109	112	52	113	24	118	21	0.34
Dia BP	68	9	62	1279	61	92	64	109	62	52	67	24	67	21	0.09
Girls															
CRP	1	4	0	1371	0	83	0	149	0	58	1	28	0	29	0.25
HDL	59	3	53	1298	51	80	53	134	52	50	60	21	57	32	0.44
Trigl		14	122	540	116	34	115	56	118	29	113	10	151	14	0.99
LDL		1	111	545	117	34	113	58	94	30	118	10	106	14	0.14
Sys BP	102	3	114	1221	112	73	113	124	112	45	112	27	113	28	0.94
Dia BP	63	3	63	1221	62	73	63	124	58	45	60	27	63	28	0.63
	Hours used computers past 30 days														
	none	n	<1	n	1h	n	2h	n	3h	n	4h	n	≥5h	n	p
All															
CRP	0.3	282	0.3	2975	0.3	149	0.5	54	0.1	22	0.4	7	0.3	9	0.26
HDL	52	57	53	695	54	33	55	10	56	7	59	3	52	4	0.59
LDL	120	200	123	2265	112	100	149	40	112	24	59	6	120	9	0.13

table continues

	Hours used computers past 30 days														
	none	n	<1	n	1h	n	2h	n	3h	n	4h	n	≥5h	n	p
Trigl	109	198	111	2255	102	100	114	40	126	24	101	6	109	9	0.21
Sys BP	112	252	114	2632	113	134	112	44	74	26	111	3	112	6	0.22
Dia BP	62	252	63	2632	62	134	60	44	38	26	74	3	62	6	0.11
Boys															
CRP	0.3	148	0.3	1501	0.2	74	0.5	32	0.4	12	0.1	5	0.2	4	0.68
HDL	51	140	53	1404	52	74	58	31	52	11	58	4	64	3	0.36
LDL	124	71	124	640	106	35	92	11	143	4	112	2	53	2	0.25
Trigl	111	71	110	644	96	35	94	11	108	4	126	2	105	2	0.13
Sys BP	112	130	114	1343	113	63	110	22	116	14	99	1	112	3	0.75
Dia BP	63	130	63	1343	61	63	65	22	62	14	55	1	77	3	0.61
Girls															
CRP	0.3	134	0.3	1474	0.3	75	0.4	22	0.5	10	0.2	2	0.5	5	0.15
HDL	54	128	53	1384	55	64	53	24	57	11	49	1	56	5	0.87
LDL	115	54	122	578	118	36	120	14	170	1			71	1	0.64
Trigl	108	56	111	584	108	36	99	14	137	1			94	1	0.75
Sys BP	113	122	114	1289	113	71	115	22	108	12	62	2	110	3	0.45
Dia BP	60	122	63	1289	64	71	65	22	58	12	29	2	70	3	0.10

*significant at the 95% confidence level

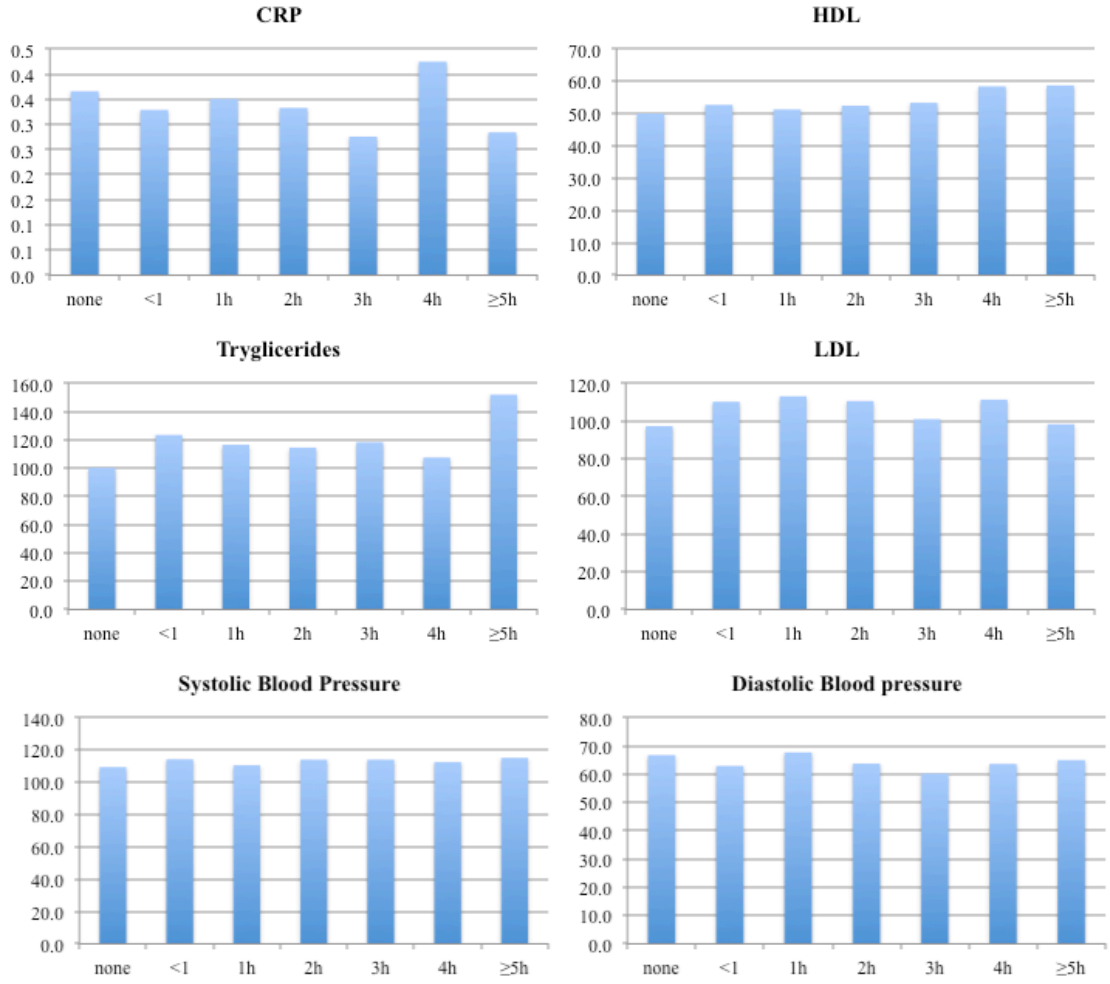

Figure 6a. Median values of CRP, HDL, triglycerides, LDL and blood pressure in children exposed to TV at increments of 1 hour daily.

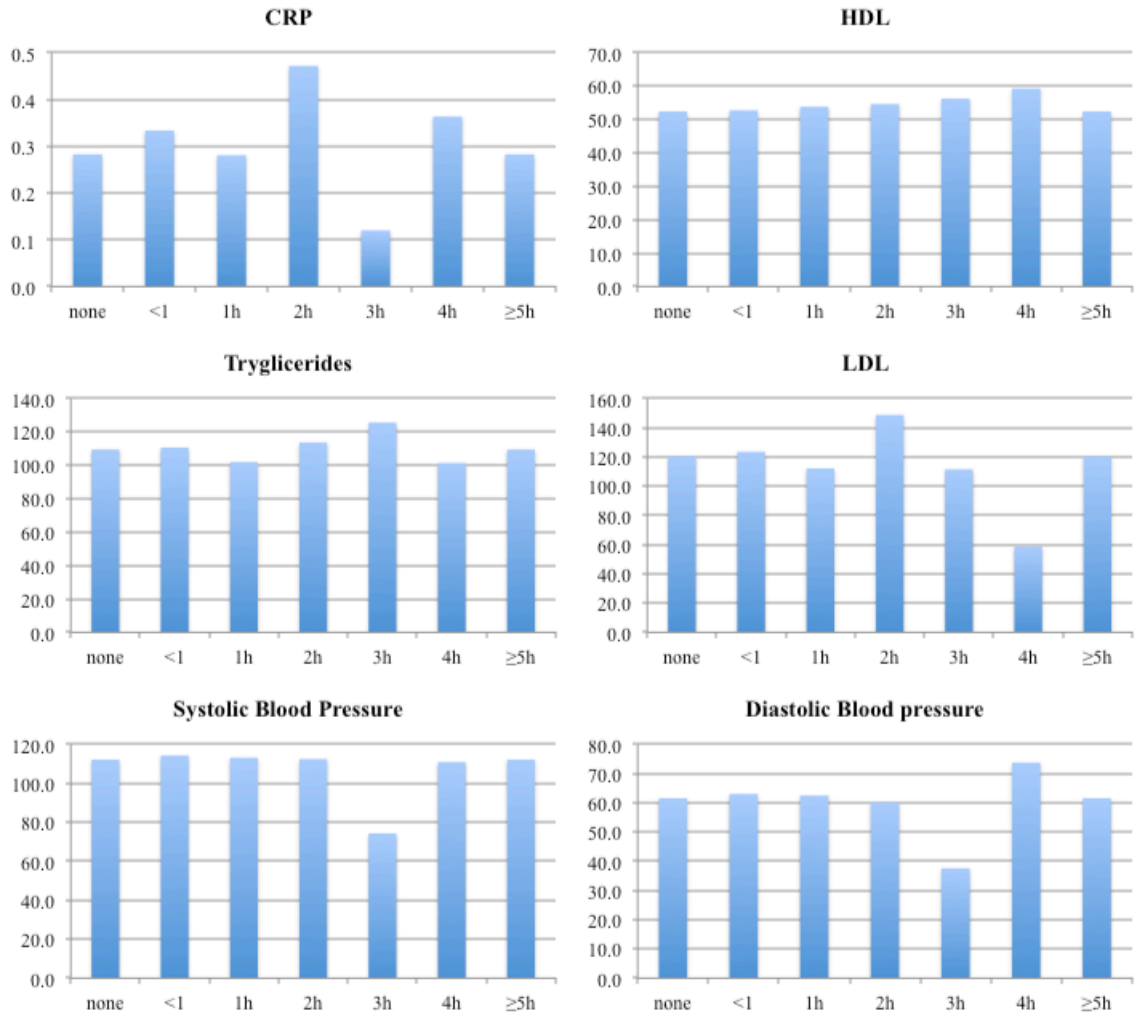

Figure 6b. Median values of CRP, HDL, triglycerides, LDL and blood pressure in children exposed to computers at increments of 1 hour daily.

Next, the sample population was divided into only two categories, based on APA recommended screen time limits, and differences in biomarkers were assessed using the non-parametric Mann-Whitney test. HDL was significantly higher in children watching TV ($p = 0.005$) for more than 2 hours per day; LDL was significantly higher in children using computers ($p=0.043$) for more than 2 hours per day (Table 7). Difference remained

significant for LDL when only boys were analyzed. CRP was found significantly higher in girls using computers for more than 2 hours per day ($p = 0.006$).

Table 7
Biomarkers of CVD risk: difference in mean, according to APA recommended screen time limits

	Hours watching TV or videos					Hours using computer				
	2h or less	n	more than 2h	n	p	2h or less	n	more than 2h	*n*	p
All										
CRP	0.33	3276	0.31	222	0.7	0.33	3460	0.38	38	0.933
HDL	52.6	3074	55.9	210	**0.005***	52.8	3249	56	35	0.508
Trigl	122	1343	123	106	0.84	122	1439	114	10	0.128
LDL	110	1354	103	107	0.07	110	1451	112	10	**0.043***
Sys BP	114	2916	113	197	0.79	114	3078	109	35	0.529
Dia BP	63	2916	62	197	0.97	63	3078	60	35	0.543
Boys										
CRP	0.33	1669	0.23	107	0.99	0.32	1755	0.3	21	0.96
HDL	52.6	1559	56.7	108	0.39	52.8	1649	55.3	18	0.944
Trigl	123	712	121	53	0.81	123	757	112	8	0.146
LDL	109	716	104	53	0.95	109	761	111	8	**0.009***
Sys BP	114	1489	114	97	0.07	114	1568	114	18	0.709
Dia BP	62	1489	65	97	0.23	63	1568	64	18	0.783
Girls										
CRP	0.33	1607	0.34	115	0.58	0.33	1705	0.48	17	**0.006***
HDL	52.6	1515	55	102	0.16	52.7	1600	56.5	17	0.346
Trigl	121	631	125.5	53	0.82	121.6	682	120.5	2	0.848
LDL	111	638	101.6	54	0.20	110.5	690	115.5	2	0.766
Sys BP	114	1427	112.2	100	0.97	114	1510	103	17	0.242
Dia BP	63	1427	60	100	0.10	63	1510	57	17	0.152

*significant at the 95 confidence level

Results

All Research Questions were analyzed using binary logistic regression, which does not require assumption of normal distribution of data. Independent variables were dichotomized as 0 (normal range) and 1 (at risk), according to values published in the medical literature. Table 8 below summarizes the process of variable dichotomization.

Table 8

Dichotomization of Independent Variables

	Normal range	Coding	Values above normal/ideal range (at risk)	Coding	Reference
CRP	<1 mg/L	0	>1 mg/L	1	http://emedicine.medscape.com/article/2086909-overview
HDL	≥60 g/dL	0	<60 mg/dL	1	http://www.cholesterolmenu.com/cholesterol-levels-chart/
Triglycerides	≤150 mg/dL	0	>150 mg/dL	1	
LDL	≤100 mg/dL	0	>100 mg/dL	1	
Systolic blood pressure	≤120 mmHg	0	>120 mmHg	1	http://www.nhlbi.nih.gov/health-pro/guidelines/current/hypertension-pediatric-jnc-4/blood-pressure-tables
Diastolic blood pressure	≤80 mmHg	0	>150 mmHg	1	

Once the variables were dichotomized, the percentage of subjects within and above normal levels was calculated for each variable, by ethnicity and gender. For all variables, there was no major difference between boy and girls (Table 8). On the other hand, distribution of values varied across ethnicities. A larger percentage of individuals with triglycerides above normal/ideal values were observed in non-Hispanic Blacks and

Other race. More non Hispanic Whites and non Hispanic Blacks had LDL levels above the normal/ideal range, with respect to all other races (Table 9).

Table 9
Proportion by ethnicity/race and by age of subjects with normal and above/below normal values (at risk)

		Normal/ideal range	At risk
CRP	Mexican American	93	7
	other Hispanic	92	8
	non Hispanic White	92	8
	non Hispanic Black	93	7
	Other race	93	7
	Boys	93	7
	Girls	92	8
HDL	Mexican American	29	71
	other Hispanic	30	70
	non Hispanic White	29	71
	non Hispanic Black	28	72
	Other race	28	72
	Boys	29	71
	Girls	29	71
Triglycerides	Mexican American	80	20
	other Hispanic	77	23
	non Hispanic White	76	24
	non Hispanic Black	72	28
	Other race	63	37
	Boys	76	24
	Girls	76	24
LDL	Mexican American	44	56
	other Hispanic	43	57
	non Hispanic White	41	59
	non Hispanic Black	39	61
	Other race	46	54
	Boys	43	57
	Girls	42	58

(*table continues*)

		Normal/ideal range	At risk
Systolic blood pressure	Mexican American	65	35
	other Hispanic	69	31
	non Hispanic White	64	36
	non Hispanic Black	66	34
	Other race	67	33
	Boys	66	34
	Girls	65	35
Diastolic blood pressure	Mexican American	88	12
	other Hispanic	89	11
	non Hispanic White	88	12
	non Hispanic Black	88	12
	Other race	90	10
	Boys	88	12
	Girls	88	12

When running a logistic regression analysis three key assumptions must be met: linearity, independence of error, and multicollinearity. The assumption of independent errors is only necessary when data is clustered hierarchically and therefore does not apply to this analysis.

The assumption of linearity in logistic regression requires that any explanatory variables have a linear relationship with the logit of the outcome variable. The assumption was assessed by looking at the model fit statistics and pseudo R^2 (Hosmer and Lemeshow test). The goodness of fit suggested that the models were a good fit to the data, given that $p > .05$ for all cases tested.

The assumption of multicollinearity requires that predictor variables should not be highly correlated with each other. Usually values of $r = .8$ or more are cause for concern. The Variance Inflation Factor (VIF) and tolerance statistics can be used to help verify that multicollinearity is not a problem. In this case, none of the predictor variables was significantly correlated with each other.

The findings of the analysis suggest that screen time had a significant relationship with some of the CVD risk factors, and that gender and ethnicity/race contributed to the significance of the model in some instances. Table 10 provides an overview of the result of the analysis for all the three research questions. Model 1 is the model with just the independent variable (CRP, or HDL, or triglycerides, or LDL, or systolic blood pressure or diastolic blood pressure) and only one dependent variable describing media screen time (PAD590, or PAD600, or TST, or APA PAD590, or APA PAD600 or APA PAD TST). Model 2 includes also gender, ethnicity and the interaction terms of gender*media screen time, as well as ethnicity*media screen time. In the Chapter sections below, only

the full models that showed statistically significant (or borderline significant) associations are reported. All research questions were answered with binary logistic regression analysis, using the “Enter” function, since I had no preliminary data to use for a hierarchical regression analysis.

Table 10

Summary of p Values of Binary Logistic Regression Models

	DV	CRP	HDL	Trigl	LDL	BP Sys	BP Dias
IV							
PAD590	Model1	p = .802	**p = .023***	p = 0.296	p = 0.496	p = 0.311	p = 0.412
	Model2	**p = .063**	p = .389	p = 0.516	p = 0.762	p = 0.897	p = 0.203
PAD600	Model1	p = .094	p = .702	**p = .065**	p = .454	p = .572	p = .224
	Model2	p = .198	p = .697	p = .088	p = .321	p = .68	p = .819
PAD590APA	Model1	p = .3	**p = .018***	p=.588	p = .330	p = .328	p = .346
	Model2	p = .329	p = .116	p = .493	p = .588	p = .588	p = .429
PAD600APA	Model1	p = .512	p = .291	p = .269	p = .886	p = .988	p = .954
	Model2	p = .163	p = .535	**p = .035***	p = .335	p = .850	p = .853
TST	Model1	p = .803	**p = .032***	p = .105	p = .451	p = .676	p = .195
	Model2	**p = .014***	p = .568	p = .705	p = .637	p = .801	**p = .063**
APA TST	Model1	p = .907	**p = .018***	p = .352	p = 0.315	p = .769	**p = .020***
	Model2	p = .772	p = .403	p = .134	p = 0.566	p = .743	p = .082

Note. * Significant at the 95 confidence level

Research Question 1

For U.S. youth 0 to 20 years old, what is the relationship between media screen time, genetic background (gender, race/ethnicity), and CRP?

Null hypothesis. There is no relationship between media screen time, genetic background (gender, race/ethnicity), and CRP in U.S. youth aged 0 to 20 years old.

Alternative hypothesis. There is a relationship between media screen time, genetic background (gender, race/ethnicity), and CRP in U.S. youth aged 0 to 20 years old.

The binary logistic regression analysis was conducted to predict levels of CRP for U.S. youth years, using either media screen time alone as predictor (Model 1), or screen time, gender, and race/ethnicity, as well as the interaction terms screen time*gender and screen time*ethnicity (Model 2). Only in case of PAD590 and TST, the models significantly predicted CRP.

In case of PAD590, a test of the full Model 1 against a constant only model was not statistically significant (χ^2 (6) = 3.055, p = .802). However, when gender, race/ethnicity, and the interaction terms screen time*gender and screen time*ethnicity were added to the model, test of the full Model 1 against a constant only model was borderline statistically significant (χ^2 (39) = 53.301, p = .063). The coefficient of determination Nagelkerke R^2 of .036 indicated explains approximately 3.6 of the variance in instances of CRP higher than normal/ideal levels. Prediction success was 99.9 for CRP within normal/ideal levels, and .8 for CRP above normal/ideal levels. Upon examination of the individual variables, the Wald criterion demonstrated that only the interaction factor PAD590(2)*RIDRETH1(2) made a borderline significant contribution to the

prediction; in other words, the regression analysis suggested that among youth watching TV for 1 hour per day (PAD590(2)), other Hispanic youth (RIDRETH1(2)) was 3.005 times more likely to have above normal/ideal levels with respect to Mexican American (RIDRETH1(1)) (p = .055). Similarly, in case of TST, a test of the full Model 1 against a constant only model was not statistically significant (χ^2 (6) = 3.049, p = .803). However, when gender, race/ethnicity, and the interaction terms screen time*gender and screen time*ethnicity were added to the model, the test of the full Model 1 against a constant only model was borderline statistically significant (χ^2 (39) = 60.772, p = .014). The coefficient of determination Nagelkerke R^2 of .041 indicated explains approximately 4.1 of the variance in instances of CRP higher than normal/ideal levels. Prediction success was 100 for CRP within normal/ideal levels, and 0 for CRP above normal/ideal levels. Upon examination of the individual variables, the Wald criterion demonstrated that only the interaction factor RIDRETH1(4)*TST(5) and RIAGENDR(1)*TST(3) made a significant contribution to the prediction; in other words, the regression analysis suggested that among non Hispanic Blacks (RIDRETH1(4)), youth watching total media screen for 5 hours or more were 9.618 times more likely to have above normal/ideal levels of CRP with respect to non-Hispanic Blacks watching no media screen (p = .028). Furthermore, among boys (RIAGENDR(1)), those watching total media screen for 3 hours (TST(3), were 10.330 times more likely to have above normal/ideal levels of CRP with respect to those watching no media screen (p = .033).

Research Question 2

For U.S. youth 0 to 20 years old, what is the relationship between media screen time, genetic background (gender, race/ethnicity), and lipids (HDL, LDL, and triglycerides)?

Null hypothesis. There is no relationship between media screen time, genetic background (gender, race/ethnicity), and lipids in U.S. youth aged 0 to 20 years old.

Alternative hypothesis. There is a relationship between media screen time, genetic background (gender, race/ethnicity), and lipids, in U.S. youth aged 0 to 20 years old.

The binary logistic regression analysis was conducted to predict levels of HDL, or triglycerides or LDL for U.S. youth 0 to 20 years old, using either media screen time alone as predictor (Model 1), or screen time, gender, race/ethnicity, as well as the interaction terms screen time*gender and screen time*ethnicity (Model 2). Only Model 1 for PAD590, PAD590APA, TST and APA TST significantly predicted HDL. Model 2 for PAD600APA was statistically significant when predicting triglycerides. LDL could not be predicted from any of the dependent variables.

HDL. In case of PAD590, a test of the full Model 1 against a constant only model was statistically significant (χ^2 (6) = 14.694, p = .023). Prediction success was 2.6 for HDL within normal/ideal levels, and 99 for HDL below normal/ideal levels. Upon examination of the individual variables, the Wald criterion demonstrated that only the PAD590(4) made a significant contribution to the prediction; in other words, the regression analysis suggested that youth watching TV for 4 hour per day (PAD590(4), were 0.38 times less likely to have below normal/ideal values of HDL (p = .001). The

coefficient of determination Nagelkerke R^2 of .006 explains less than 1 of the variance in instances of HDL lower than normal/ideal levels. When gender, race/ethnicity, and the interaction terms screen time*gender and screen time*ethnicity were added to the model, the test of the full Model 2 against a constant only model was not statistically significant (χ^2 (39) = 40.858, p = .389).

In case of PAD590APA, a test of the full Model 1 against a constant only model was statistically significant (χ^2 (1) = 5.576, p = .018). However, the coefficient of determination Nagelkerke R^2 of .002 indicated that the model explains only 0.2 of the variance in instances of HDL lower than normal/ideal levels. Prediction success was 0 for HDL within normal/ideal levels, and 100 for HDL below normal/ideal levels. When gender, race/ethnicity, and the interaction terms screen time*gender and screen time*ethnicity were added to the model, the test of the full Model 2 against a constant only model was not statistically significant (χ^2 (11) = 10.416, p = .493).

In case of TST, a test of the full Model 1 against a constant only model was statistically significant (χ^2 (6) = 13.826, p = .032). The coefficient of determination Nagelkerke R^2 of .006 indicated that the model explains less than 1 of the variance in instances of HDL lower than normal/ideal levels. Upon examination of the individual variables, the Wald criterion demonstrated that only the TST(5) made a significant contribution to the prediction; in other words, the regression analysis suggested that youth spending 5 hours or more per day in front of the screen (TST(5)), were 0.53 times less likely to have below normal values of HDL (p = .002). Prediction success was 0 for HDL within normal/ideal levels, and 100 for HDL below normal/ideal levels. When

gender, race/ethnicity, and the interaction terms screen time*gender and screen time*ethnicity were added to the model, the test of the full Model 2 against a constant only model was not statistically significant (χ^2 (39) = 36.859, p = .568).

When TST was expressed according to APA specifications (more or less than 2 hours daily), a test of the full Model 1 against a constant only model was statistically significant (χ^2 (1) = 5.401, p = .018). The coefficient of determination Nagelkerke R^2 of .002 indicated that the model explains approximately only 0.2 of the variance in instances of HDL lower than normal/ideal levels. Upon examination of the individual variables, the Wald criterion indicated that youth spending more than two hours per day in front of the screen were 0.741 times less likely to have below normal values of HDL (p = .001) with respect to youth exposed to less than two hours per day. Prediction success was 0 for HDL within normal/ideal levels, and 100 for HDL below normal/ideal levels. When gender, race/ethnicity, and the interaction terms screen time*gender and screen time*ethnicity were added to the model, the test of the full Model 2 against a constant only model was not statistically significant (χ^2 (11) = 11.496, p = .403).

Triglycerides. In case of PAD600, a test of the full Model 1 against a constant only model was only borderline statistically significant (χ^2 (6) = 11.875, p = .065). The coefficient of determination Nagelkerke R^2 of .012 indicated explains approximately 1 of the variance in instances of triglycerides higher than normal/ideal levels. Prediction success was 99.7 for triglycerides within normal/ideal levels, and 1.1 for triglycerides above normal/ideal levels. When gender, race/ethnicity, and the interaction terms screen time*gender and screen time*ethnicity were added to the model, the test of the full

Model 2 against a constant only model was not statistically significant (χ^2 (29) = 39.772, p = .088).

When PAD600 was expressed according to APA specifications (more or less than 2 hours daily), a test of the full Model 1 against a constant only model was not statistically significant (χ^2 (1) = 1.221, p = .269). However, when gender, race/ethnicity, and the interaction terms screen time*gender and screen time*ethnicity were added to the model, the test of the full Model 2 against a constant only model was statistically significant (χ^2 (10) = 19.398, p = .035). The coefficient of determination Nagelkerke R^2 of .02 indicated that 2 of the variance was explained, in instances of triglycerides higher than normal/ideal levels. Prediction success was 99.9 for triglycerides within normal/ideal levels, and 0.6 for triglycerides above normal/ideal levels.

Upon examination of the individual variables, the Wald criterion demonstrated that RIDRETH1(3) and RIDRETH1(4) made a significant contribution to the prediction; in other words, the regression analysis suggested that non Hispanic Blacks (p = .012) and Other race (p = .001) were, respectively, 1.583 and 2.287 times more likely to have above normal/ideal levels of triglycerides with respect to the reference category Mexican American.

Research Question 3

For U.S. youth 0 to 20 years old, what is the relationship between media screen time, genetic background (gender, race/ethnicity), and blood pressure (diastolic and systolic)?

Null hypothesis. There is no relationship between media screen time, genetic background (gender, race/ethnicity), and blood pressure, in U.S. youth age 0 to 20 years old.

Alternative hypothesis. There is a relationship between media screen time, genetic background (gender, race/ethnicity), and blood pressure, in U.S. youth age 0 to 20 years old.

Only diastolic, but not systolic, blood pressure could be predicted by a binary logistic model. In case of TST, a test of the full Model 1 against a constant only model was not statistically significant (χ^2 (7) = 9.886, p = .195). When gender, race/ethnicity, and the interaction terms screen time*gender and screen time*ethnicity were added to the model, the test of the full Model 2 against a constant only model only borderline statistically significant (χ^2 (44) = 59.2, p = .063). The coefficient of determination Nagelkerke R^2 of .037 indicated explains approximately 3.7 of the variance in instances of diastolic pressure higher than normal/ideal levels. Prediction success was 100 for diastolic pressure within normal/ideal levels, and .3 for diastolic pressure above normal/ideal levels. Upon examination of the individual variables, the Wald criterion demonstrated that only TST(5) and the interaction factor RIDRETH1(4)*TST(3) made a significant contribution to the prediction; in other words, the regression analysis suggested that among youth watching total screen time for five or more hours per day (TST(5) were 5.384 times more likely to have diastolic blood pressure above normal/ideal values with respect to youth not exposed to any screen time (p = .007). Furthermore, among Non Hispanic Black, youth watching total screen time for three

hours daily were 6.242 times more likely to have diastolic blood pressure above normal/ideal values with respect to youth not exposed to any screen time ($p = .026$).

When TST was expressed according to APA specifications (more or less than 2 hours daily), a test of the full Model 1 against a constant only model was statistically significant (χ^2 (1) = 5.376, $p = .020$). The coefficient of determination Nagelkerke R^2 of .003 indicated that 0.3 of the variance was explained, in instances of diastolic blood pressure higher than normal/ideal levels. However, when gender, race/ethnicity, and the interaction terms screen time*gender and screen time*ethnicity were added to the model, the test of the full Model 2 against a constant only model was no longer statistically significant (χ^2 (11) = 17.991, $p = .082$).

Summary

Analysis of the data indicates that the frequency of demographic variables was the same for boys and for girls, but the population was oversampled for Mexican Americans and non-Hispanic Blacks, and under-sampled for non-Hispanic Whites. The distribution of study subjects across number of screen time watched was uneven, and highly skewed towards lower exposure time. Preference for media type (television or computer) was different across ethnicities and gender. CVD biomarkers were expressed at different levels across ethnicity and gender, although not a statistically significant level. A positive trend was observed for some of the CVD biomarkers with increasing exposure to media screen (HDL, triglycerides, LDL, blood pressure), but increase in values was minor and variability high. When the percentage of subjects within and above normal levels of CVD

risk factors was calculated for each variable, distribution of values varied across ethnicities.

Results from binary logistic regression analysis indicated that exposure to screen time, alone or in combination with gender and race/ethnicity, increased the risk of having at risk levels for some of the CVD risk factors, depending upon the type of media exposure and ethnicity. The models however had a very low percentage of success in correctly predicting classification to risk category groups.

Chapter 5 includes a discussion and analysis of the findings of the analyses summarized above, the limitations of the study, and the impact on social change. Recommendations on further research on the relationship between exposure to media screen and cardiovascular risk factors are proposed.

Chapter 5: Discussion, Recommendations, and Conclusions

Introduction

Spending time in front of a media screen has become the most prevalent activity of U.S. youth, second only to sleep (Rideout et al., 2010) and can increase risk of CVD by altering metabolic homeostasis and leading to hormonal imbalance, obesity, dyslipidemia, and hypertension (Rosen et al., 2014). Besides displacement of physical activity by a media screen-associated sedentary life, factors yet unknown have been suggested as critical in the development of CVD. The purpose of this descriptive, quantitative, correlational study, based on a cross-sectional analysis of archived data from the 2009 – 2010 NHANES for United States youth 0 to 20 years old, was to determine the effect of genetic background combined with excessive media screen time on markers of cardiovascular risk in United States youth aged 0 to 20 years.

Results indicated that CVD biomarkers are expressed at different levels across ethnicity and gender and that exposure to screen time, alone or in combination with gender and race/ethnicity, increased the risk of having above normal levels for some of the CVD risk factors, depending upon the type of media exposure and ethnicity. In the case of CRP and triglycerides, inclusion in the model of gender, race/ethnicity, and the interaction terms screen time*gender and screen time*ethnicity were better predictors than screen time alone. The models were, however, poor predictors of the classification to risk category groups.

Interpretation of the Findings

The prevalence of CVD risk factors varies across gender, ethnicities, and the environment (CDC, 2015b). The results of this study confirmed differences across ethnicities and accentuated risk for other race, non-Hispanic Whites, and non-Hispanic Blacks; however, the sample size was not large enough to reach statistical significance at the 95 confidence level. Among U.S. youth aged 0 to 20 years old, CRP was highest between other Hispanic and other race, and lowest among Mexican Americans. HDL was lowest among non-Hispanic Blacks and other race, while triglycerides were highest among other race followed by other Hispanic and non-Hispanic Whites. Non-Hispanic Whites, non-Hispanic Blacks, and Mexican Americans had the highest systolic blood pressure. Results are in alignment with previously published data indicating that Whites and Asians are more likely to have higher levels of triglycerides and low HDL than non-Hispanic Blacks (Frank et al., 2014; Morimoto et al., 2014, Mozaffarian et al., 2015; World Heart Federation, 2015). Non-Hispanic Blacks have been reported as the population most at risk to develop hypertension and have higher levels of the inflammatory marker CRP, which was not confirmed in the current study. A major difference between this study and published literature is the age range of the study population, youth in the former and adults in the latter studies, suggesting that age may be an important factor in the development of CVD risk factors among non-Hispanic Blacks.

Besides CVD risk factors, prevalence of media screen time viewing is also associated with ethnicity and gender (Anderson et al., 2008; Herrick et al., 2014;

Gijsberts et al., 2015; Rideout, 2011). In alignment with published evidence (Garcia-Continente et al., 2014; Herrick et al., 2014; Hoyos Cillero & Yago, 2011), my results indicate that across all ethnicities, girls were more likely than boys to watch TV for 2 hours or more per day and boys were generally more attracted to videogames than girls. Ethnicity has been suggested as another important risk factor for excessive screen time (Carlson & Fulton, 2014; Herrick et al., 2014; Rideout, 2011). The data from the 2009-2010 NHANES survey analyzed in this study confirm that distribution of total screen time varies according to ethnicities and indicate that other tace and other Hispanics are more likely to watch TV more than 2 hours per day than any other race; this finding is particularly interesting in view of the increased levels of CRP in these two races (Other race and other Hispanics) compared to all others. Results by others using the 2012 NHANES survey (Herrick et al., 2014) suggested that the trend was highest for non-Hispanic Blacks, although a direct comparison between the two studies cannot be done, since the latter study was limited to children 12 to 15 years old and included only Hispanics, non-Hispanic Blacks, and non-Hispanic Whites. Interestingly, the percentage of individuals (regardless of age) reporting TV watching and computer use for more than 2 hours daily was much larger in the 2012 NHANES survey, indicating a rapid increase over time of media screen use and/or reporting, possibly linked to social acceptability of this behavior in more current times.

An abnormal lipid profile, a marker of inflammation and hypertension, is found associated with prolonged exposure to excessive media screen time (Hardy, et al., 2010; Goldfield et al., 2013; Mark & Janssen, 2008; Mota, et al., 2014; Stamatakis, et al., 2011;

Tremblay et al., 2011). Hypertension, dyslipidemia, and inflammatory markers (CRP) are increased among youth when the child spends long hours in front of media (Hardy, et al., 2010; Pinto Pereira, et al., 2012; Stamatakis, et al., 2011). On the other hand, according to Berentzen (2014), there was no direct correlation between cardio-metabolic markers and screen time. The results of the current study suggest that some biomarkers (i.e., CRP, triglycerides, and systolic blood pressure) are occasionally increased in youth exposed to screen time with respect to no exposure; however, the difference is small and the trends are often inconsistent, warranting further investigation. The only statistically significant difference in the distribution of CVD risk factors across time categories of screen time viewing is represented by HDL, which shows a steady increase in levels with increasing in screen time viewing. According to clinical guidelines, HDL levels are inversely correlated with CVD risk, posing individuals with low HDL cholesterol at higher risk for cardiovascular disease (Adult Treatment panel III, 2002). The findings of the analysis proposed here are puzzling and suggest that the correlation between exposure to screen time and risk factors of CVD is likely masked. Indeed, sociodemographic, lifestyle, and diabetic status have been indicated as confounders of the correlation between cardio-metabolic markers and screen time (Li et al., 2015, Nang et al., 2013).

Evaluation of cardio-metabolic risk factors in relationship to screen time suggests that the relationship between ethnicity, gender, and type/duration of exposure to media screen may indeed be important for the etiology of some biomarkers (i.e., CRP, triglycerides, diastolic blood pressure), but the prediction models in this study are still inefficient in predicting a clinically relevant number of cases.

According to the social ecological theory, etiology of diseases results from a synergistic interaction between genotype, phenotype, and the environment (Bronfenbrenner & Ceci, 1994). Under these premises, the social ecological theory well explains the ethnic differences in CVD risk and severity (Gijsberts et al., 2015, Hill et al., 2015) and proposes a link excessive screen time and social and genetic determinants. My results indicate that gender and ethnicity play a role in moderating the exposure to media screen time as well as the likelihood of developing CVD risk factors. Other Hispanics youth are more likely to have above normal levels of CRP with respect to Mexican Americans exposed to the same amount of TV. Non-Hispanic Blacks may be more sensitive to time-dependent exposure to total screen time than other races; similarly, among boys, time-dependent exposure to total media screen impacts levels of CRP. Non-Hispanic Blacks and otherRace exposed to computers for more than the recommended APA limits (2 hours daily) are more likely to have above normal/ideal levels of triglycerides with respect to Mexican Americans. Exposure to total screen time for more than 5 hours daily increased the risk of high diastolic blood pressure; being non-Hispanic Black increased the risk of developing high diastolic blood pressure already at exposure of 3 hours daily. Diastolic pressure, but not systolic pressure, is a predictor of CVD in youth (Franklin & Wong, 2013).

Limitation of the Study

The study has several limitations that limit its validity and generalizability. First, cross-sectional studies do not lend themselves to the determination of cause and effects; however, this study design was the most suitable for the collection of preliminary

information to support additional research. In addition, the number of individuals varied considerably across media screen time category and, in case of long exposure, was extremely limited. Moreover, the amount of screen time watched is likely to have been underreported. For these reasons, estimates of parameters tested may be inaccurate. Furthermore, evaluation of potential confounders (i.e., diabetes, obesity, lifestyle, and family history of CVD) was not a part of this study. Each of these potential confounders may provide alternative explanations of the relationship between observed levels of CVD risk factors and media screen time.

The dataset from the NHANES database for CVD risk factors was not complete for all ages included in the study, resulting in more than 5 of missing cases. The reason why data were missing is not known. This severely limits the generalizability of the study, reduces the power of the analysis, and warrants further additional studies to confirm the results presented here. Convenience sampling was used in place of random sampling, thus posing limitations to the generalizability of the conclusions. Furthermore, the oversampling of racial/ethnic minorities and adults with lower education to mitigate the low reliability of self-reported health measurement (Zajacova & Dowd, 2011) to ensure representativeness of the population at risk (National Health and Nutrition Examination Survey, 2014) has resulted in the composition of the sample population that does not faithfully reflect the demographics of the true population. This further limits the generalizability of the study. Therefore, results should be interpreted with caution.

Recommendations

In this study, I suggested that the risk of CVD induced by exposure to media screen time may be moderated by ethnicity, gender, and type/duration of exposure. The implications of the study support the general notion that the etiology of diseases may be gender and race/ethnicity dependent and is moderated by life-style factors. Results, although interesting, are severely limited by the considerable amount of missing data in the dataset, the largely uneven distribution of subjects across media screen time categories, and the unfaithful representation of the real population.

The findings of this study suggest that the risk of CVD associated to media screen time exposure is mediated by different factors, depending upon race/ethnicity and genders. Therefore, different types of intervention are necessary to reduce the risk of CVD, depending upon the target population. Preventative interventions should address not only the amount but also the type of media screen time and should be culturally relevant.

Knowledge of the molecular mechanisms underlying disease development in different races/ethnicities must be further investigated to allow for proper pharmacological intervention. Further research is necessary to better characterize the interplay between molecular determinants of CVD, genetic attributes (gender, ethnicity, age) and lifestyle. Another cross-sectional, observational study is recommended that would investigate the current pattern of media screen time viewing, including cell phone use, and with sufficient power to detect the differences in the expression of risk factors as observed in this study. A collection of a primary dataset for the specific purpose of

addressing the research questions is recommended, with an emphasis on the relationship between screen time and CRP, triglycerides and diastolic blood pressure, and inclusion of information on obesity, diabetes, and lifestyle. The study population should be sampled using stratified random selection. This would ensure that each stratum (i.e., screen time category, gender, ethnicity) is represented evenly. Results from this study would better identify if a particular gender or ethnicity has more propensity to develop CVD in response to exposure to media screen time and through what type of molecular mechanism. Additional studies would then focus on the relationship between comorbidities (i.e., obesity, diabetes) or lifestyle and media screen time in the categories most at risk, identified in the previous study.

To overcome some important limitations of the current study, future research in this area is needed to develop new surveys on exposure to media screens based on a properly selected study population and objective measurement of the time that individuals spend in front of television, cell phones, computers, and movies, instead of self-reported measurements. Engagement with policy makers is required to bring more attention to this health issue and to advocate for funding to support further studies on this topic. Indeed, the conclusions of my preliminary work, although not yet generalizable, support a large body of published evidence indicating that excessive and uncontrolled exposure to electronic media screen has pernicious effects on the health of youth, with negative consequences on the quality of life as adults.

Implications

The implications for positive social change that emerge from this study are that interventions are urgently needed to regulate exposure to media screen time since excessive use of media screen increases risk factors for CVD in those populations who are already at higher risk of poor health. In the context of increased risk of CVD from excessive exposure to media screen, understanding the reasons behind different choices of media type between boys and girls, or the molecular mechanisms at the basis of the different sensitivity observed across races, will help public health officials and practitioners to provide culturally relevant guidance, targeting the individual, the parents, and the community. Interventions that limit exposure total screen time will reduce the risk of increased blood pressure among all races. However, culturally relevant intervention should be designed for non-Hispanic Blacks, other Hispanics, and other Race, ethnicities with the highest propensity for increased blood pressure, CRP, and triglycerides, and who also spend the largest amount of time in front of the media screen.

Conclusions

My findings suggest that traditional interventions to reduce the risk of CVD may not sufficient, as not enough is known about the causes of inter race differences in risk factor's sensitivity, and the motivation behind excessive exposure to screen time varies among population groups and genders. A deep understanding of what drives groups to excessive TV viewing and computer gaming is necessary to create effective preventative campaigns.

References

Adult Treatment panel III (2002). Third report of the national cholesterol education program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report. *Circulation, 106*(25), 3143-3421, PubMed.

Anderson, S. E., Economos, C. D., & Musk A. (2008). Active play and screen time in U.S. children aged 4 to 11 years in relation to socio-demographic and weight status characteristics: A nationally representative cross-sectional analysis. *BMC Public Health, 8*, 366. doi:10.1186/1471-2458-8-366.

APA. (n.d.). *Media and children*. Retrieved from https://www.aap.org/en-us/advocacy-and-policy/aap-health-initiatives/pages/media-and-children.aspx

Appelhans B. M., Fitzpatrick, S. L., Li, H., Cail, V., Waring, M. E., Schneider, K. L., … & Pagoto, S. L. (2014). The home environment and childhood obesity in low-income households: Indirect effects via sleep duration and screen time. *BMC Public Health, 9*(14), 1160. doi:10.1186/1471-2458-14-1160.

Arsenault, B. J., Boekholdt, S. M., & Kastelein, J. J. P. (2011). Lipid parameters for measuring risk of cardiovascular disease. *Nature Review Cardiology, 8*(4), 197-206. doi:10.1038/nrcardio.2010.223

Atkins, M. S., Rusch, D., Mehta, T. G., & Lakind, D. (2015). Future directions for dissemination and implementation science: Aligning ecological theory and public health to close the research to practice gap. *Journal Clinical Child and Adolescent Psychology*, *9*, 1-12. doi:10.1080/15374416.2015.1050724

Battistoni, A., Canichella, F., Pignatelli, G., Ferrucci, A., Tocci, G., & Volpe, M. (2015). Hypertension in young people: Epidemiology, diagnostic assessment and therapeutic approach. *High blood press cardiovascular prevention. 22*(4), 381-8. doi:10.1007/s40292-015-0114-3.

Beck, A. L., Takayama, J., Badiner, N., & Halpern-Felsher, B. (2015). Latino parents' beliefs about television viewing by infants and toddlers. *Journal Health Care Poor Underserved, 26*(2), 463-74. doi:10.1353/hpu.2015.0037.

Berentzen, N. E., Smit, H. A., van Rossem, L., Gehring, U., Kerkhof, M., Postma, D. S…& Wijga, A. H. (2014). Screen time, adiposity and cardiometabolic markers: Mediation by physical activity, not snacking, among 11-year-old children. *International Journal Obesity (London), 38*(10), 1317-23. doi:10.1038/ijo.2014.110.

Berkman, N. D., Sheridan, S. L., Donahue, K. E., Halpern, D. J., & Crotty, K. (2011). Low health literacy and health outcomes: An updated systematic review. *Annals Internal Medicine, 155*, 97–107. doi:10.7326/0003-4819-155-2-201107190-00005.

Borghese, M. M., Tremblay, M. S., Leduc, G., Boyer, C., Bélanger, P., LeBlanc, A. G., … & Chaput, J. P. (2015). Television viewing and food intake during television viewing in normal-weight, overweight and obese 9- to 11-year-old Canadian children: A cross-sectional analysis. *Journal of Nutritional Science, 4*, e8. doi:10.1017/jns.2014.72

Börnhorst, C., Wijnhoven, T. A., Kunešová, M., Yngve, A., Rito, A. I., Lissner, L.... &

Breda, J. (2015). WHO European Childhood Obesity Surveillance Initiative: Associations between sleep duration, screen time and food consumption frequencies. *BMC Public Health, 15*(1), 1-11. doi:10.1186/s12889-015-1793-3.

Branchi, I., & Cirulli, F. (2014). Early experiences: Building up the tools to face the challenges of adult life. *Developmental Psychobiology, 56*(8), 1661-74. doi:10.1002/dev.21235.

Brindova, D., Pavelka, J., Ševčikova, A., Žežula, I., van Dijk, J. P., Reijneveld, S. A., & Geckova, A. M. (2014). How parents can affect excessive spending of time on screen-based activities. *BMC Public Health, 14*, 1261. doi:10.1186/1471-2458-14-1261

Brody, J. E. (2015). *Screen addiction is taking a toll on children*. Retrieved from http://well.blogs.nytimes.com/2015/07/06/screen-addiction-is-taking-a-toll-on-children/

Brofenbrenner, U. (1977). Towards an experimental ecology of human development. *American Psychologist, 32*, 513-531. Pubmed.

Bronfenbrenner, U., & Ceci, S. J. (1994). Nature-nurture reconceptualized in developmental perspective: A bioecological model. *Psychology Reviews, 101*(4), 568-86.

Buscemi, J., Kanwischer, K., Becker, A. B., Ward, D. S., Fitzgibbon, M. L., & on behalf of the Society of Behavioral Medicine Health Policy Committee. (2015). Society of Behavioral Medicine position statement: Early care and education (ECE) policies can impact obesity prevention among preschool-aged children.

Translational Behavioral Medicine, *5*(1), 122–125. doi:10.1007/s13142-014-0297-5.

Camhi, S. M., Waring, M. E., Sisson, S. B., Hayman, L. L., & Must, A. (2013). Physical activity and screen time in metabolically healthy obese phenotypes in adolescents and adults. *Journal of Obesity*, *98*, 4613. doi:10.1155/2013/984613.

Campbell, D. T., & Stanley, J. C. (1963). *Experimental and quasi-experimental designs for research*. Boston: Houghton Mifflin.

Carson, V., Rosu, A., & Janssen, I. (2014). A cross-sectional study of the environment, physical activity, and screen time among young children and their parents. *BMC Public Health, 14*(1), 1-18. doi:10.1186/1471-2458-14-61.

Carson, V., Kuzik, N., Hunter, S., Wiebe, S. A., Spence, J. C., Friedman, A., …& Hinkley, T. (2015). Systematic review of sedentary behavior and cognitive development in early childhood. *Preventive Medicine, 78*, 115-22. doi:10.1016/j.ypmed.2015.07.016.

Carson, V., Stearns, J., & Janssen, I. (2015b). The relationship between parental physical activity and screen time behaviors and the behaviors of their young children. *Pediatric Exercise Science, 27*(3), 390-5. doi:10.1123/pes.2014-0214.

CDC. (2013a). Vital signs: Avoidable deaths from heart disease, stroke, and hypertensive disease—United States, 2001–2010. *MMWR Morbidity Mortality Weekly Reports, 62*, 721–7. PubMed.

CDC. (2013b). *NHANES Selected Bibliography - NHANES 1999-2012*. Retrieved from http://wwwn.cdc.gov/nchs/nhanes/bibliography/

CDC. (2014). *Questionnaires, Datasets, and Related Documentation*. Retrieved from http://www.cdc.gov/nchs/nhanes/nhanes_questionnaires.htm

CDC. (2015a). *About Heart Disease and Stroke.* Retrieved from http://millionhearts.hhs.gov/abouthds/risk-factors.html

CDC. (2015b). *Heart Disease Facts and Statistics.* Retrieved from http://www.cdc.gov/heartdisease/statistics.htm

CDC. (2015c). *Women and Heart Disease Fact Sheet.* Retrieved from http://www.cdc.gov/dhdsp/data_statistics/fact_sheets/fs_women_heart.htm

Chang, J., Guy, M. C., Rosales, C., de Zapien, J. G., Staten, L. K., Fernandez, M. L., & Carvajal, S. C. (2013). Investigating social ecological contributors to diabetes within Hispanics in an underserved U.S.-Mexico border community. *International Journal of Environmental Research and Public Health, 10*(8), 3217–3232. doi:10.3390/ijerph10083217.

Chaput, J. P., Leduc, G., Boyer, C., Bélanger, P., LeBlanc, A. G., Borghese, M. M., & Tremblay, M. S. (2014). Electronic screens in children's bedrooms and adiposity, physical activity and sleep: Do the number and type of electronic devices matter? *Canadian Journal Public Health, 105*(4), e273-9. PubMed.

Cheng, J. K., Koziol, R. L., & Taveras, E. M. (2015). Parental guidance advised: Associations between parental television limits and health behaviors among obese children. *Academy Pediatrics, 15*(2), 204-9. doi:10.1016/j.acap.2014.08.007

ChildStats.gov. (2015). *Demographic Background*. Retrieved from http://www.childstats.gov/americaschildren/demo1.asp

Chinapaw, M. J., Proper, K. I., Brug, J., van Mechelen, W., & Singh, A. S. (2011). Relationship between young peoples' sedentary behaviour and biomedical health indicators: A systematic review of prospective studies. Obesity Review, 12(7), e621-32. doi:10.1111/j.1467-789X.2011.00865.x.

Christakis, D. A., & Garrison, M. M. (2009). Preschool-aged children's television viewing in childcare settings. *Pediatrics, 124*(6), 1627-1632. doi:10.1542/peds.2009-0862.

Colangelo, L. A., Vu, T. H., Szklo, M., Burke, G. L., Sibley, C., & Liu, K. (2015). Is the association of hypertension with cardiovascular events stronger among the lean and normal weight than among the overweight and obese? The multi-ethnic study of atherosclerosis. *Hypertension, 66*(2), 286-93. Pubmed.

Coll, C. (2004). *Nature and nurture the complex interplay of genetic and environmental influences on human behavior and development.* Mahwah, N.J.: Lawrence Erlbaum

Corona, E., Chen, R., Sikora, M., Morgan, A. A., Patel, C. J., … & Ramesh, A. (2013). Analysis of the genetic basis of disease in the context of worldwide human relationships and migration. *PLoS Genet 9*(5), e1003447. doi:10.1371/journal.pgen.1003447.

Côté-Lussier, C., Fitzpatrick, C., Séguin, L., & Barnett, T. A. (2015). Poor, unsafe, and overweight: The role of feeling unsafe at school in mediating the association among poverty exposure, youth screen time, physical activity, and weight status. *American Journal Epidemiology, 182*(1), 67-79. doi:10.1093/aje/kwv005.

Danielsen, Y. S., Júlíusson, P. B., Nordhus, I. H., Kleiven, M., Meltzer, H. M., Olsson, S.J., & Pallesen, S. (2011). The relationship between life-style and cardio-metabolic risk indicators in children: The importance of screen time. *Acta Paediatrica, 100*(2), 253-9. doi:10.1111/j.1651-2227.2010.02098.x

da Silva, I. T., de Almeida-Pititto, B., & Ferreira, S. R. (2015). Reassessing lipid metabolism and its potentialities in the prediction of cardiovascular risk. *Archives Endocrinology Metabolism, 59*(2), 171-80. doi:10.1590/2359-3997000000031

de Jong, E., Visscher, T. L. S., HiraSing, R. A., Heymans, M. W., Seidell, J. C., & Renders, C. M. (2013). Association between TV viewing, computer use and overweight, determinants and competing activities of screen time in 4- to 13-year-old children. *International Journal of Obesity, 37,* 47–53. doi:10.1038/ijo.2011.244.

Dennison, B. A., Erb, T. A., & Jenkins, P. L. (2002). Television viewing and television in bedroom associated with overweight risk among low-income preschool children. *Pediatrics, 109*, 1028-1035. Pubmed.

Do Prado Junior, P. P., Rocha de Faria, F., Rodrigues de Faria, E., Castro Franceschini, S. D., & Priore, S.E. (2015). Cardiovascular risk and associated factors in adolescents. *Nutrition Hospitalaria, 32*(n02), 897-904. doi:10.3305/nh.2015.32.2.8824.

Dong, G., Yanbo, H., & Xiao, L. (2013). Reward/punishment sensitivities among internet addicts: implications for their addictive behaviors. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 46*, 139–145.

doi:10.1016/j.pnpbp.2013.07.007

Downing, K. L., Hinkley, T., & Hesketh, K. D. (2015). Associations of parental rules and socioeconomic position with preschool children's sedentary behavior and screen time. *Journal Physical Activity Health, 12*(4), 515-21. doi:10.1123/jpah.2013-0427

Duch, H., Fisher, E. M., Ensari, I., & Harrington, A. (2013). Screen time use in children under 3 years old: A systematic review of correlates. *The International Journal of Behavioral Nutrition and Physical Activity*, *10*, 102. doi:10.1186/1479-5868-10-102.

Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & van Ijzendoorn, M.H. (2011). Differential susceptibility to the environment: an evolutionary-neurodevelopmental theory. *Development and Psychopathology, 23*(1), 7-28. doi:10.1017/S0954579410000611.

Fakhouri, T. H., Hughes, J. P., Brody, D. J., Kit, B. K., & Ogden C.L. (2013). Physical activity and screen-time viewing among elementary school-aged youth in the United States from 2009 to 2010. *JAMA Pediatrics, 167*(3), 223-9. doi:10.1001/2013.jamapediatrics.122.

Faienza, M. F., Acquafredda, A., Tesse, R., Luce, V., Ventura, A., Maggialetti, N., ... & Cavallo, L. (2013). Risk factors for subclinical atherosclerosis in diabetic and obese children. *International journal of medical sciences*, *10*(3), 338. doi:10.7150/ijms.5181.

Fakhouri, T. H. I., Hughes, J. P., Burt, V. L., Song, M. K., Fulton, J. E., & Ogden, C. L. (2014). Physical Activity in U.S. Youth Aged 12–15 Years, 2012. *NCHS Data Brief No. 141*, 1-8. Pubmed.

Flint, A. J. (2010). Excess weight and the risk of incident coronary heart disease among men and women. *Obesity (Silver Spring), 18,* 377–383. doi:10.1038/oby.2009.223

Ford, E. S. (2012). Combined television viewing and computer use and mortality from all-causes and diseases of the circulatory system among adults in the United States. *BMC Public Health, 12*, 70. doi:10.1186/1471-2458-12-70.

Francis, D. K., Bennett, N. R., Ferguson, T. S., Hennis, A. J., Wilks, R. J., ….& Harris, E. N. (2015). Disparities in cardiovascular disease among Caribbean populations: a systematic literature review. *BMC Public Health*, *15*, 828. doi:10.1186/s12889-015-2166-7

Frank, A. T. H., Zhao, B., Jose, P. O., Azar, K. M. J., Fortmann, S. P., & Palaniappan, L. P. (2014). Racial/ethnic differences in dyslipidemia patterns. *Circulation, 129*(5), 570–579. doi:10.1161/CIRCULATIONAHA.113.005757

Frankfort-Nachmias, C., & Nachmias, D. (2008). *Research methods in the social sciences* (7th ed.). New York: Worth.

Franklin S. S, & Wong, N. D. (2013). Hypertension and cardiovascular disease: contributions of the Framingham heart study. *Global Heart, 8*(1), 49-57. doi:10.1016/j.gheart.2012.12.004.

Fryar, C. D., Chen, T., & Li, X. (2012). Prevalence of uncontrolled risk factors for cardiovascular disease: United States, 1999–2010. *NCHS Data Brief, 103,* 1-8. Pubmed.

Fuchun, L., Zhou, Y., Du, Y., Qin, L., Zhao, Z., Xu, J., & Lei, H. (2012). Abnormal white matter integrity in adolescents with internet addiction disorder: A tract-based spatial statistics study. *PloS One, 7*(1), e30253. doi:10.1371/journal.pone.0030253

Garcia-Continente, X., Pérez-Giménez A., Espelt, A., & Nebot Adell, M. (2014). Factors associated with media use among adolescents: A multilevel approach. *European Journal Public Health, 24*(1), 5-10. doi:10.1093/eurpub/ckt013.

Gijsberts, C. M., Seneviratna, A., de Carvalho, L. P., den Ruijter, H. M., Vidanapthirana, P., Sorokin, V., … & Chan, M. Y. (2015). Ethnicity modifies associations between cardiovascular risk factors and disease severity in parallel Dutch and Singapore coronary cohorts. *PLoS ONE, 10*(7), e0132278. doi:10.1371/journal.pone.0132278.

Gilliam, M. B., & Schwebel, D. C. (2013). Physical activity in child and adolescent cancer survivors: a review. *Health Psychology Review*, *7*(1), 92–110. Pubmed.

Goldfield G. S., Saunders, T. J., Kenny, G. P., Hadjiyannakis, S., Phillips, P., & Alberga, A. S. (2013). Screen viewing and diabetes risk factors in overweight and obese adolescents. *American Journal Preventive Medicine, 44*(4 Suppl 4), S364–70. doi:10.1016/j.amepre.2012.11.040.

Golden, S. D., & Earp, J. L. (2012). Social ecological approaches to individuals and their

contexts: twenty years of health education & behavior health promotion interventions. *Health Education and Behavior, 39*(3), 364-372. doi:10.1177/1090198111418634

Gopinath, B., Hardy, L. L., Kifley, A., Baur, L. A., & Mitchell, P. (2014). Activity behaviors in schoolchildren and subsequent 5-yr change in blood pressure. *Medicine & Science in Sports & Exercise, 46*(4), 724-9. doi:10.1249/MSS.0000000000000166

Gotto, A. M. (2011). Jeremiah Metzger Lecture: cholesterol, inflammation and atherosclerotic cardiovascular disease: is it all LDL? *Transactions of the American Clinical and Climatological Association*, *122*, 256–289. PubMed.

Graham, D. J., Schneider, M., and Cooper, D. M. (2012). Television viewing: moderator or mediator of an adolescent physical activity intervention? *American Journal Health Promotion, 23*(2), 88–91. doi:10.4278/ajhp.07053050

Grøntved, A., Ried-Larsen, M., Møller, N. C., Kristensen, P. L., Wedderkopp, N., Froberg, K., Hu, F. B., Ekelund, U., & Andersen, L. B. (2014). Youth screen-time behaviour is associated with cardiovascular risk in young adulthood: The European Youth Heart Study. *European Journal of Preventive Cardiology, 21*(1), 49-56. doi:10.1177/2047487312454760

Gutierrez, J., Ramirez, G., Rundek, T., & Sacco, R. L. (2012). Statin therapy in the prevention of recurrent cardiovascular events: A sex-based meta-analysis. *Archives Internal Medicine, 172(12),* 909-19. doi:10.1001/archinternmed.2012.2145.

Hardy, L. L., Denney-Wilson, E., Thrift, A. P., Okely, A. D., & Baur, L. A. (2010). Screen time and metabolic risk factors among adolescents. *Archives Pediatric Adolescent Medicine, 164*(7), 643-649. doi:10.1001/archpediatrics.2010.88.

Haines, J., O'Brien, A., McDonald, J., Goldman, R. E., Evans-Schmidt, M., Price, S., Sherry, B. L., & Taveras, E. (2013). Television viewing and televisions in bedrooms: Perceptions of racial/ethnic minority parents of young children. *Journal Of Child & Family Studies, 22*(6), 749-756. doi:10.1007/s10826-012-9629-6.

Hardy, L. L., Denney-Wilson, E., Thrift, A. P., Okely, A. D., & Baur, L. A. (2010). Screen time and metabolic risk factors among adolescents. *Archives Pediatric Adolescent Medicine, 164*(7), 643-649. doi:10.1001/archpediatrics.2010.8.

Harper, S., Lynch, J., & Smith, G. D. (2011). Social determinants and the decline of cardiovascular diseases: Understanding the links. *Annual Review Public Health, 32*, 39–69. doi:10.1146/annurev-publhealth-031210-101234.

Havranek, E. P., Mujahid, M. S., Barr, D. A., Blair, I. V., Cohen, M. S., Cruz-Flores, S., Davey-Smith, G., & Dennison-Himmelfarb, C. R. (2015). Social determinants of risk and outcomes for cardiovascular disease: A scientific statement from the American Heart Association. *Circulation, 132*(9), 873-98. doi:10.1161/CIR.0000000000000228.

Henderson, Z. P. & Petic, Z. (1995). Renewing our social fabric. *Human Ecology Forum, 23*(1), 16-19. Questia.

Herman, K. M., Hopman, W. M., & Sabiston, C. M. (2015). Physical activity, screen

time and self-rated health and mental health in Canadian adolescents. *Preventative Medicine, 73*, 112-6. doi:10.1016/j.ypmed.2015.01.030

Herrick, K. A., Fakhouri, T. H., Carlson, S. A., & Fulton, J. E. (2014). TV watching and computer use in U.S. youth aged 12-15, 2012. *NCHS Data Brief,* 157, 1-8. PubMed.

Hill, L. K., Hu, D. D., Koenig, J., Sollers, J. J., Kapaku, G., Wang, X., Snieder, H., & Thayer, J. F. (2015). Ethnic differences in resting heart rate variability: A systematic review and meta-analysis. *Psychosomatic Medicine, 77*(1), 16-25. doi:10.1097/PSY.0000000000000133.

Hefner, H. L. (2013). *Risks of television viewing among children with and without the ADHD diagnosis*. Retrieved from http://search.proquest.com/docview/1418292614?accountid=14872.

Holmes, M. V., Harrison, S., Talmud, P. J., Hingorani A. D., & Humphries, S. E. (2011). Utility of genetic determinants of lipids and cardiovascular events in assessing risk. *Nature Review Cardiology, 8*, 207–221. doi:10.1038/nrcardio.2011.6

Holmes, M. V., Asselbergs, F. W., Palmer, T. M., Drenos, F., & Lanktree, M. B. … & Casas, J. (2014). Mendelian randomization of blood lipids for coronary heart disease. *European Heart Journal.* doi:10.1093/eurheartj/eht571

Hong, J. S., Kral, M. J., Espelage, D. L., & Allen-Meares, P. (2012). The social ecology of adolescent-initiated parent abuse: A review of the literature. *Child Psychiatry and Human Development, 43*(3), 431-54. doi:10.1007/s10578-011-0273-y.

Hong, S. B., Kim, J. W., Choi, E. J., Kim, H. J., Suh, J. E., Kim, C. J., & Klauser, P. (2013). Reduced orbitofrontal cortical thickness in male adolescents with internet addiction. *Behavioral and Brain Functions, 9*(1), 11. doi:10.1186/1744-9081-9-11.

Hoyos Cillero, I., & Jago, R. (2011). Socio-demographic and home environment predictors of screen viewing among Spanish school children. *Journal Public Health (Oxf), 33*, 392 – 402. doi:10.1093/pubmed/fdq087.

Holmes, M. V., Harrison, S., Talmud, P. J., Hingorani A. D., & Humphries, S. E. (2011). Utility of genetic determinants of lipids and cardiovascular events in assessing risk. *Nature Review Cardiology, 8*, 207–221. doi:10.1038/nrcardio.2011.6.

Hummer, T. A., Kronenberger, W. G., Wang, Y., Anderson, C. C., & Mathews, V. P. (2014). Association of television violence exposure with executive functioning and white matter volume in young adult males. *Brain and Cognition, 88,* 26-34. doi:10.1016/j.bandc.2014.04.010.

Huang, R. C., Prescott, S. L., Godfrey, K. M., & Davis, E. A. (2015). Assessment of cardiometabolic risk in children in population studies: Underpinning developmental origins of health and disease mother–offspring cohort studies. *Journal of Nutritional Science, 4*, e12. doi:10.1017/jns.2014.69.

Hummer, T. A., Kronenberger, W. G., Wang, Y., Anderson, C. C., Mathews, V. P. (2014). Association of television violence exposure with executive functioning and white matter volume in young adult males. *Brain Cognition, 88,* 26-34. doi:10.1016/j.bandc.2014.04.010.

Jackson, C. H., Richardson, S., & Best, N. G. (2008). Studying place effects on health by synthesising individual and area-level outcomes. *Social Science Medicine, 67*(12), 1995-2006. doi:10.1016/j.socscimed.2008.09.041.

Janssen, I., Medina, C., Pedroza, A., & Barquera, S. (2013). Screen time in Mexican children: Findings from the 2012 National Health and Nutrition Survey (ENSANUT 2012). *Salud Publica Mexicana, 55*(5), 484-91. PubMed.

Kelishadi, R., Qorbani, M., Motlagh, M. E., Heshmat, R., Ardalan, G., & Jari, M. (2014). Relationship between leisure time screen activity and aggressive and violent behaviour in Iranian children and adolescents: The CASPIAN-IV Study. *Paediatric International Children Health, 35*(4), 305-11. doi:10.1080/20469047.2015.1109221.

Kochanek, K. D., Xu, J. Q., Murphy, S. L., Miniño, A. M., & Kung, H. C. (2011). Deaths: Final data for 2009. *National Vital Statistic Reports, 60*(3), 1-51. PubMed.

Kotikalapudi, R., Chellappan, S., Montgomery, F., Wunsch, D., & Lutzen, K. (2012). Associating internet use with depressive behavior among college students. *IEEE Technology and Society Magazine, Winter 2012,* 73-80. doi:10.1109/MTS.2012.2225462

Krieger, N. (2011). *Epidemiology and the people's health: Theory and context.* New York, NY: Oxford University Press.

Kronish, I. M, Carson, A. P., Davidson, K. W., Muntner, P., & Safford, M. M. (2012). Depressive symptoms and cardiovascular health by the American Heart

Association's definition in the reasons for geographic and racial differences in stroke (REGARDS) study. *PLoS One, 7*, e52771. doi:10.1371/journal.pone.0052771

Kunin-Batson, A. S., Seburg, E. M., Crain, A. L., Jaka, M. M., Langer, S. L., Levy, R. L., & Sherwood, N. E. (2015). Household factors, family behavior patterns, and adherence to dietary and physical activity guidelines among children at risk for obesity. *Journal Nutrition Education Behavior, 47*(3), 206-15. doi:10.1016/j.jneb.2015.01.002.

Lee, S. T., Wong, J. E., Nik Shanita, S., Ismail, M. N., Deurenberg, P., & Poh, B. K. (2015). Daily physical activity and screen time, but not other sedentary activities, are associated with measures of obesity during childhood. *International Journal of Environmental Research and Public Health, 12*(1), 146–161. doi:10.3390/ijerph120100146

Let's Move! (2015). *America's move to raise a healthier generation of kids.* Retrieved from http://www.letsmove.gov/

Lo, C. B., Waring, M. E., Pagoto, S. L., & Lemon, S. C. (2015). A television in the bedroom is associated with higher weekday screen time among youth with ADHD. *Preventive Medicine Reports, 2,* 1-3. PubMed.

Lo, C. L., Zhou, F. C. (2014). Environmental alterations of epigenetics prior to the birth. *International Review Neurobiology, 115*, 1-49. doi:10.1016/B978-0-12-801311-3.00001-9.

Loomis, T. A., Viola, D. (2012). Obesity, asthma, and allergies: The inflammatory connection. A case study. *ICAN Infant Child & Adolescent Nutrition, 4*(2), 106-110. doi:10.1177/1941406411436212.

Lord, S., Manlhiot, C., Tyrrell, P. N., Dobbin, S., Gibson, D., Chahal, N., Stearne, K., Fisher, A., & McCrindle, B. W. (2015). Lower socioeconomic status, adiposity and negative health behaviors in youth: A cross-sectional observational study. *BMJ Open, 5*(5), e008291. doi:10.1136/bmjopen-2015-008291

Maibach, E. (2007). The influence of the media environment on physical activity: Looking for the big picture. *American Journal Health Promotion, 21*(4S), 353–362. PubMed.

Maras, D., Flament, M. F., Murray, M., Buchholz, A., Henderson, K. A., Obeid, N., & Goldfield, G. S. (2015). Screen time is associated with depression and anxiety in Canadian youth. *Preventive Medicine, 73*, 133-8. doi:10.1016/j.ypmed.2015.01.029.

Mark, A. E., Janssen, I. (2008). Relationship between screen time and metabolic syndrome in adolescents. *Journal Public Health (Oxf) 30*(2), 153–60. PubMed.

Martin, K. (2011). *Electronic overload: the impact of excessive screen use on child and adolescent health and wellbeing.* Perth, Western Australia: Department of Sport and Recreation.

Martínez-Vizcaino, V., Mota, J., Solera-Martínez, M., Notario-Pacheco, B., Arias-Palencia, N., García-Prieto, J. C., González-García, A., Álvarez-Bueno, C., Sánchez-López, M., & MOVI-KIDS group. (2015). Rationale and methods of a

randomised cross-over cluster trial to assess the effectiveness of MOVI-KIDS on preventing obesity in preschoolers. *BMC Public Health, 15*(176). doi:10.1186/s12889-015-1512-0.

Matthews, C. E., Cohen, S. S., Fowke, J. H., Han, X., Xiao, Q., Buchowski, M. S., Hargreaves, M. K., Signorello, L. B., & Blot, W. J. (2014). Physical activity, sedentary behavior, and cause-specific mortality in black and white adults in the southern community cohort study. *American Journal Epidemiology, 180*(4), 394-405. doi:10.1093/aje/kwu142.

McCullough, M. E., Pedersen, E. J., Schroder, J. M., Tabak, B. A., & Carver, C. S. (2013). Harsh childhood environmental characteristics predict exploitation and retaliation in humans. *Proceedings Biological Sciences, 280*(1750), 20122104. doi:10.1098/rspb.2012.2104.

Medline Plus. (2015). *Screen time and children.* Retrieved from http://www.nlm.nih.gov/medlineplus/ency/patientinstructions/000355.htm

Mendis, S., Puska, P., Norrving, B., & World Health Organization (2011). *Global Atlas on Cardiovascular Disease Prevention and Control*. World Health Organization in collaboration with the World Heart Federation and the World Stroke Organization. pp. 3–18.

Mitchell, J. A., Rodriguez, D., Schmitz, K. H., & Audrain-McGovern, J. (2013). Greater screen time is associated with adolescent obesity: A longitudinal study of the BMI distribution from ages 14 to 18. *Obesity (Silver Spring) 21*(3), 572–575. doi:10.1002/oby.20157.

Morimoto, Y., Conroy, S. M., Ollberding, N. J., Kim, Y., Lim, U., Cooney, R.V., ….. & Maskarinec, G. (2014). Ethnic differences in serum adipokine and C-reactive protein levels: The multiethnic cohort. *International Journal Obesity (Lond), 38*(11), 1416-1422. doi:10.1038/ijo.2014.25.

Morris, A., & Keith, C. F. (2009). Hyperlipidemia in racial/ethnic minorities: differences in lipid profiles and the impact of statin therapy. *Clinical Lipidology, 4*(6), 741-754. doi:10.2217/clp.09.70.

Mota, J., Silva-Santos, S., Barros, M., Farias Júnior, J. C., Vale, S. (2014). Influence of different behavioral factors and obesity status on systolic blood pressure among pre-schoolchildren. *Annals Human Biology, 41*(6), 506-10. doi:10.3109/03014460.2014.909884.

Mozaffarian, D., Benjamin, E. J., & Go, A. S. (2015). Heart disease and stroke statistics—2015 update: A report from the American Heart Association. *Circulation, 131*, e29–322. doi:10.1161/CIR.0000000000000152.

Myers, J., Gibbons, K., Arnup, S., Volders, E., & Naughton, G. (2015). Early childhood nutrition, active outdoor play and sources of information for families living in highly socially disadvantaged locations. *Journal Paediatric Children Health, 51*(3), 287-93. doi:10.1111/jpc.12713.

Nang, E. E., Salim, A., Wu, Y., Tai, E. S., Lee, J., & Van Dam, R. M. (2013). Television screen time, but not computer use and reading time, is associated with cardio-metabolic biomarkers in a multiethnic Asian population: A cross-sectional study.

International Journal Behavioral Nutrition Physics Acta, 30(10), 70-75. doi:10.1186/1479-5868-10-70.

National Center for Health Statistics. (2012). *Health, United States, 2011: with special features on socioeconomic status and health*. Hyattsville, MD; U.S. Department of Health and Human Services; 2012.

NHANES. (2011a). *2009-2010 Data Documentation, Codebook, and Frequencies. Demographic Variables and Sample Weights (DEMO_F).* Retrieved from http://wwwn.cdc.gov/Nchs/Nhanes/2009-2010/DEMO_F.htm#Analytic_Notes

NHANES. (2011b). *2009-2010 Data Documentation, Codebook, and Frequencies. Physical Activity (PAQ_F).* Retrieved from http://wwwn.cdc.gov/Nchs/Nhanes/2009-2010/DEMO_F.htm#Analytic_Notes

NHANES. (2011c). *2009-2010 Data Documentation, Codebook, and Frequencies C-Reactive Protein (CRP) (CRP_F).* Retrieved from http://wwwn.cdc.gov/Nchs/Nhanes/2009-2010/CRP_F.htm

Ogden, C. L., Carroll, M. D., Kit, B. K., Flegal, K. M. (2014). Prevalence of childhood and adult obesity in the United States, 2011-2012. *Journal of the American Medical Association, 311*(8), 806-814. doi:10.1001/jama.2014.732.

Ohira, T., Diez, Roux, A. V., Polak, J. F., Homma, S., Iso, H., & Wasserman, B. A. (2012). Associations of anger, anxiety, and depressive symptoms with carotid arterial wall thickness: The multi-ethnic study of atherosclerosis. *Psychosomatic Medicine, 74*, 517–525. doi:10.1097/PSY.0b013e31824f6267.

Ozaki, K., Tanaka, T. (2015) Molecular genetics of coronary artery disease. *Journal Human Genetics*, *6*(1), 71-77. doi:10.1038/jhg.2015.70.

Parkes, A., Sweeting, H., Wight, D., & Henderson, M. (2013). Do television and electronic games predict children's psychosocial adjustment? Longitudinal research using the UK millennium cohort study. *Archives of Disease in Childhood, 98*(5), 341–348. doi:10.1136/archdischild-2011-301508.

Peart, T., Velasco Mondragon, H. E., Rohm-Young, D., Bronner, Y., & Hossain, M. B. (2011). Weight status in U.S. youth: The role of activity, diet, and sedentary behaviors. *American Journal Health Behavior, 35*(6), 756-64. PubMed.

Peiro-Velert, C., Valencia-Peris, A., Gonzalez, L. M., Garcia-Masso, X., & Serra-Ano, P. (2014). Screen media usage, sleep time and academic performance in adolescents: clustering a self-organizing maps analysis. *PLoS ONE 9*(6), e99478. doi:10.1371/journal.pone.0099478.

Pinto Pereira, S.M., Ki, M, & Power, C. (2012) Sedentary behavior and biomarkers for cardiovascular disease and diabetes in mid-life: The role of television viewing and sitting at work. *PLoS ONE 7*(2): e31132. doi:10.1371/journal.pone.0031132.

Richardson, S., Shaffer, J. A., Falzon, L., Krupka, D., Davidson, K. W., & Edmondson, D. (2012). Meta-analysis of perceived stress and its association with incident coronary heart disease. *American Journal Cardiology, 110*, 1711–1716. doi:10.1016/j.amjcard.2012.08.004.

Rideout, V. J., Foehr, U. G., & Roberts, D. F. (2010). *Generation M2: Media in the Lives of 8- to 18-Year-Olds*. Menlo Park, CA: Kaiser Family Foundation, 2.

Rideout, V. (2011). *Zero to eight: Children's media use in America*. San Francisco, CA: Commonsense Media.

Rodriguez-Cruz, E., Rao, P. S. (2015). *Pediatric hypertension.* Retrieved from http://emedicine.medscape.com/article/889877-overview#a5.

Roger, V. L., Go, A. S. Lloyd-Jones, D. M. Adams, R. J. Berry, J. D., & Brown, T. M. (2011). Heart disease and stroke statistics—2011 update: A report from the American Heart Association. *Circulation, 123*(4), e18–e209. doi:10.1161/CIR.0b013e3182009701.

Rosen, L. D., Lim, A. F., Felt, J., Carrier, L. M., Cheever, N.A., Lara-Ruiz, J. M., Mendoza, J. S., & Rokkum, J. (2014). Media and technology use predicts ill-being among children, preteens and teenagers independent of the negative health impacts of exercise and eating habits. *Computers Human Behavior, 35*, 364–375. PubMed.

Sandercock, G. R., & Ogunleye, A. A. (2012). Screen time and passive school travel as independent predictors of cardiorespiratory fitness in youth. *Preventative Medicine, 54*(5), 319-22. doi:10.1016/j.ypmed.2012.03.007.

Shrivastavaa, A. K., Singhb, H. V., Raizadac, A., & Singhd, S. K. (2015). C-reactive protein, inflammation and coronary heart disease. *The Egyptian Heart Journal, 67*(2), 89–97. doi:10.1016/j.ehj.2014.11.005.

Sijtsma, A., Koller, M., Sauer, P.J ., & Corpeleijn, E. (2015). Television, sleep, outdoor play and BMI in young children: The GECKO Drenthe cohort. *European Journal Pediatrics, 174*(5), 631-9. doi:10.1007/s00431-014-2443-y.

Sisson, S. B., Shay, C. M., Broyles, S. T., Leyva, M. (2012). Television-viewing time and dietary quality among U.S. youth and adults. *American Journal Preventive Medicine, 43*(2), 196-200. doi:10.1016/j.amepre.2012.04.016.

Stamatakis, E., Hamer, M., & Dunstan, D. W. (2011). Screen-based entertainment time, all-cause mortality, and cardiovascular events: Population-based study with ongoing mortality and hospital events follow-up. *Journal American College Cardiology, 57*(3), 292-299. doi:10.1016/j.jacc.2010.05.065.

Suchert, V., Hanewinkel, R., & Isensee, B. (2015). Sedentary behavior and indicators of mental health in school-aged children and adolescents: a systematic review. *Preventive Medicine, 76*, 48-57. doi:10.1016/j.ypmed.2015.03.026.

Syväoja, H. J., Tammelin, T. H., Ahonen, T., Kankaanpää, A., & Kantomaa, M. T. (2014). The associations of objectively measured physical activity and sedentary time with cognitive functions in school-aged children. PLoS ONE, 9(7), e103559. doi:10.1371/journal.pone.0103559.

Takeuchi, H., Taki, Y., Hashizume, H., Asano, K., Asano, M., Sassa, Y., Yokota, S., Kotozaki, Y., Nouchi, R., & Kawashima, R. (2013). The impact of television viewing on brain structures: cross-sectional and longitudinal analyses. *Cerebral Cortex, 25*(5), 1188-97. doi:10.1093/cercor/bht315.

Tandon, P. S., Zhou, C., Sallis, J. F., Cain, K. L., Frank, L. D., & Saelens, B. E. (2012). Home environment relationships with children's physical activity, sedentary time, and screen time by socioeconomic status. *The International Journal of Behavioral Nutrition and Physical Activity, 9,* 88. doi:10.1186/1479-5868-9-88.

Thanan, R., Oikawa, S., Hiraku, Y., Ohnishi, S., Ma, N., Pinlaor, S., …& Murata, M. (2015). Oxidative stress and its significant roles in neurodegenerative diseases and cancer. *International Journal of Molecular Sciences*, *16*(1), 193–217. doi:10.3390/ijms16010193.

Tremblay, M. S., LeBlanc, A. G., Kho, M. E., Saunders, T. J., Larouche, R., & Colley, R. C. (2011). Systematic review of sedentary behavior and health indicators in school-aged children and youth. *International Journal Behavioral Nutritional Physical Activity 8*, 98. doi:10.1186/1479-5868-8-98.

Trinh, L., Wong, B., Faulkner, G. E. (2015). The independent and interactive associations of screen time and physical activity on mental health, school connectedness and academic achievement among a population-based sample of youth. *Journal of Canadian Academy of Child Adolescent Psychiatry, 24*(1), 17-24. PubMed.

Twarog, J. P., Politis, M. D., Woods, E. L., Boles, M. K., Daniel, L. M. (2015). Daily television viewing time and associated risk of obesity among U.S. preschool aged youth: An analysis of NHANES 2009-2012. Obesity Research & Clinical Practice, 9(6), 636-8. doi:http://dx.doi.org/10.1016/j.orcp.2015.09.004.

Ungar, M., Ghazinour, M., & Richter, J. (2013). Annual research review: What is resilience within the social ecology of human development? *Journal of Child Psychology and Psychiatry, 54*(4), 348-66. doi:10.1111/jcpp.12025.

Unites States Census Bureau. (2015a). *U.S. and World Population Clock.* Retrieved from http://www.census.gov/popclock/

United States Census Bureau. (2015b). *Population Projections*. Retrieved from https://www.census.gov/population/projections/data/national/2014/summarytables.html

Universitat Dusseldorf. (2013). *G*Power: Statistical Power Analyses for Windows and Mac.* Retrieved from http://www.gpower.hhu.de/en.html

University of Rochester Medical Center. (2015). *Cholesterol, LDL, HDL, and Triglycerides in Children and Adolescents.* Retrieved from https://www.urmc.rochester.edu/encyclopedia/content.aspx?ContentTypeID=90&ContentID=P01593

Upadhyay, R. K. (2015). Emerging risk biomarkers in cardiovascular diseases and disorders. *Journal of Lipids,* 971453.

Väistö, J., Eloranta, A. M., Viitasalo, A., Tompuri, T., Lintu, N., Karjalainen, P., …… & Lakka, T. A. (2014). Physical activity and sedentary behaviour in relation to cardiometabolic risk in children: Cross-sectional findings from the physical activity and nutrition in children (PANIC) study. *International Journal of Behavioral Nutrition and Physical Activity, 11*(55), 1-10. doi:10.1186/1479-5868-11-55.

Van Cleve, J., & Akçay E. (2014). Pathways to social evolution: Reciprocity, relatedness, and synergy. *Evolution, 68*(8), 2245-58. doi:10.1111/evo.12438.

Vanderloo, L. M. (2014). Screen-viewing among preschoolers in childcare: a systematic review. *BMC Pediatrics, 14*, 205. doi:10.1186/1471-2431-14-205.

Vives, A., Amable, M., Ferrer, M., Moncada, S., Llorens, C., Muntaner, C., Benavides, F.

G., & Benach, J. (2013). Employment precariousness and poor mental health: evidence from Spain on a new social determinant of health. *Journal Environmental Public Health, 2013*, 978656. doi:http://dx.doi.org/10.1155/2013/978656.

Wells, J. C. (2012). Ethnic variability in adiposity, thrifty phenotypes and cardiometabolic risk: Addressing the full range of ethnicity, including those of mixed ethnicity. *Obesity Review, 13*(Suppl 2), 14-29. doi:10.1111/j.1467-789X.2012.01034.x.

Weng, Chuan-Bo, Ruo-Bing Qian, Xian-Ming Fu, Bin Lin, Xiao-Peng Han, Chao-Shi Niu, & Ye-Han Wang. (2014). Gray matter and white matter abnormalities in online game addiction. *European Journal of Radiology, 82*(8), 1308–1312. doi:10.1016/j.ejrad.2013.01.031

Wethington H, Pan L, & Sherry B. (2013). The association of screen time, television in the bedroom, and obesity among school-aged youth: 2007 national survey of children's health. *Journal School Health, 83*, 573-581. doi:10.1111/josh.12067.

WHO. (2011). *The Atlas of Heart Disease and Stroke. Part 2 Risk factors.* Retrieved from http://www.who.int/cardiovascular_diseases/resources/atlas/en/

WHO. (2015). *Hypertension.* Retrieved from http://www.world-heart-federation.org/cardiovascular-health/cardiovascular-disease-risk-factors/hypertension/

WHO. (2015a). *Cardiovascular Diseases (CVDs).* Retrieved from http://www.who.int/mediacentre/factsheets/fs317/en/

WHO. (2015b). *Raised Cholesterol*. Retrieved from http://www.who.int/gho/ncd/risk_factors/cholesterol_text/en/

Wijga, A. H., Scholtens, S., Bemelmans, W. J., Kerkhof, M., Koppelman, G. H., & Brunekreef, B. (2010). Diet, screen time, physical activity, and childhood overweight in the general population and in high-risk subgroups: Prospective analyses in the PIAMA birth cohort. *Journal Obesity, 2010*(42396), 9. doi:http://dx.doi.org/10.1155/2010/423296.

Wilson, D. P., McNeal, C., & Blackett, P. (2015). Pediatric dyslipidemia: Recommendations for clinical management. *Southern Medical Journal, 108*(1), 7-14. doi:10.14423/SMJ.0000000000000219.

Wong, N.D. (2014). Epidemiological studies of CHD and the evolution of preventive cardiology. *Nature Reviews Cardiology 11*, 276–289. doi:10.1038/nrcardio.2014.26.

World Heart Federation. (2015). *Cardiovascular Disease Factors.* Retrieved from http://www.world-heart-federation.org/press/fact-sheets/cardiovascular-disease-risk-factors/

Wu, Y., Potempa, L., & El Kebir, D. (2015). C-reactive protein and inflammation: conformational changes affect function. *Biological Chemistry, 396*(11), 1181-1197. doi:10.1515/hsz-2015-0149.

Xie, G., Myint, P. K., Voora, D., Laskowitz, D. T., Shi, P., Ren, F., Wang, H., Yang, Y., Huo, Y., Gao, W., & Wu, Y. (2015). Genome wide association study on progression of carotid artery intima media thickness over 10 years in a Chinese

cohort. *Atherosclerosis, 243*(1), 30-37. doi:10.1016/j.atherosclerosis.2015.08.034.

Xu, H., Wen, L. M., Rissel, C. (2015). Associations of parental influences with physical activity and screen time among young children: a systematic review. *Journal Obesity, 2015*:546925. doi:10.1155/2015/546925

Yuan, Kai, Ping Cheng, Tao Dong, Yanzhi Bi, Lihong Xing, Dahua Yu, Limei Zhao,…..& Tian, J. (2013). Cortical thickness abnormalities in late adolescence with online gaming addiction. *PLoS ONE, 8*(1), e53055. doi:http://dx.doi.org/10.1371/journal.pone.0053055.

Yin, Z., Moore, J. B., Johnson, M. H., Vernon, M. M., Grimstvedt, M., & Gutin, B. (2012). Micro- and macro-level correlates of adiposity in children. *Journal Public Health Management and Practice, 18*(5), 445-455. PubMed.

Zajacova, A., & Dowd, J.B. (2011). Reliability of self-rated health in US adults. *American Journal of Epidemiology.* Retrieved from http://aje.oxfordjournals.org/content/early/2011/09/02/aje.kwr204.full.pdf