

**CROSS-LAGGED ANALYSES OF TELEVISION VIEWING AND
CARDIOMETABOLIC RISK:
THE CORONARY ARTERY RISK DEVELOPMENT IN YOUNG ADULTS STUDY**

by

Chung-Yu Chen

B.S., National Sun Yat-Sen University, Taiwan, 2003

M.S., Chang Gung University, Taiwan, 2005

Submitted to the Graduate Faculty of
Graduate School of Public Health in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy

University of Pittsburgh

2012

UNIVERSITY OF PITTSBURGH
GRADUATE SCHOOL OF PUBLIC HEALTH

This dissertation was presented

by

Chung-Yu Chen

It was defended on

June 6, 2012

and approved by

Committee Chair

Anthony Fabio, PhD, MPH
Assistant Professor
Department of Epidemiology
Graduate School of Public Health
University of Pittsburgh

Committee Members

Tammy Chung, PhD
Associate Professor
Departments of Psychiatry and Epidemiology
School of Medicine, Graduate School of Public Health
University of Pittsburgh

Kevin H. Kim, PhD
Associate Professor
Department of Psychology
School of Education
University of Pittsburgh

Mark A. Pereira, PhD, MPH
Associate Professor
Division of Epidemiology and Community Health
School of Public Health
University of Minnesota

Janice C. Zgibor, RPh, PhD
Assistant Professor
Department of Epidemiology
Graduate School of Public Health
University of Pittsburgh

Copyright © by Chung-Yu Chen

2012

**CROSS-LAGGED ANALYSES OF TELEVISION VIEWING AND
CARDIOMETABOLIC RISK:
THE CORONARY ARTERY RISK DEVELOPMENT IN YOUNG ADULTS STUDY**

Chung-Yu Chen, PhD

University of Pittsburgh, 2012

ABSTRACT

Background: Little research has been done on the prospective associations between television viewing and cardiometabolic risk factor profiles and obesity in young adults. Additionally, individuals with high levels of hostility may be more susceptible to the influence of television viewing on cardiometabolic risk factor profiles.

Objective: This study aimed to examine whether TV viewing is predictive of adverse cardiometabolic risk factor profiles (waist circumference, HOMA insulin resistance, fasting triglycerides, HDL-cholesterol, systolic blood pressure, and clustered cardiometabolic risk score) from young to middle adulthood in the U.S. adults. We also examined whether hostility personality trait modifies the association between TV viewing and cardiometabolic risk factor profiles.

Methods: There were a total of 3,269 participants in a prospective study of Black and White adults aged 23 to 35 at examination year 5 in 1990/1. Cross-lagged panel models were analyzed at three 5-year time periods to test whether TV viewing predicts adverse cardiometabolic risk factor profiles.

Results: Individuals who watched more TV were more likely to report adverse cardiometabolic risk factor profiles in the following 5 years. The prospective association between TV viewing and obesity was observed in young adulthood but not in middle adulthood. The cross-lagged

effects of TV viewing on clustered cardiometabolic risk score were significant in the high hostility group but not in the low hostility group.

Conclusion: These findings suggest that TV viewing is positively associated with adverse cardiometabolic risk factor profiles and that this association is modified by hostility.

Public Health Significance: TV viewing and cardiometabolic risk are highly prevalent in our society. As such, even a modest association between the exposure and outcomes could have high public health significance. Our data suggest that reduction in TV viewing time, especially for people who reported high viewing time and with a propensity towards a hostile disposition, could reduce cardiometabolic risk.

TABLE OF CONTENTS

ACKNOWLEDGMENT	XIV
1.0 INTRODUCTION.....	1
1.1 SPECIFIC AIM	4
1.1.1 Project#1	5
1.1.2 Project#2	5
1.1.3 Project#3	5
1.2 LITERATURE REVIEW	6
1.2.1 Television viewing.....	6
1.2.1.1 Households with TV sets.....	6
1.2.1.2 TV viewing trends	6
1.2.1.3 TV contents	7
1.2.2 Cardiovascular disease (CVD).....	8
1.2.2.1 Definition.....	8
1.2.2.2 Prevalence of CVD	8
1.2.2.3 CVD mortality	9
1.2.2.4 Cardiometabolic risk	9
1.2.3 Overweight and obesity	10
1.2.3.1 Definition.....	10

1.2.3.2	Prevalence	10
1.2.3.3	Relation of TV viewing to obesity	11
1.2.3.4	Cross-sectional studies	11
1.2.3.5	Prospective cohort studies	17
1.2.3.6	Summary	20
1.2.3.7	Potential mechanisms linking TV viewing and obesity	21
1.2.3.8	Relation of obesity to CVD	22
1.2.4	High cholesterol.....	23
1.2.4.1	Definition.....	23
1.2.4.2	Prevalence	24
1.2.4.3	Relation of TV viewing to high cholesterol.....	24
1.2.4.4	Cross-sectional studies.....	24
1.2.4.5	Prospective cohort studies	29
1.2.4.6	Summary	30
1.2.4.7	Relation of high cholesterol to CVD.....	31
1.2.5	High blood pressure.....	32
1.2.5.1	Definition.....	32
1.2.5.2	Prevalence	32
1.2.5.3	Relation of TV viewing to high blood pressure	32
1.2.5.4	Cross-sectional studies.....	33
1.2.5.5	Prospective cohort studies	34
1.2.5.6	Summary	34
1.2.5.7	Relation of high blood pressure to CVD	35

1.2.6	Hostility.....	35
1.2.6.1	Definition.....	35
1.2.6.2	Relation of TV viewing with hostility.....	36
1.2.6.3	Relation of hostility to CVD.....	37
1.2.6.4	Potential mechanisms linking hostility and CVD.....	38
2.0	PROJECT#1: LONGITUDINAL ASSOCIATION BETWEEN TELEVISION VIEWING AND CARDIOMETABOLIC RISK: CROSS-LAGGED ANALYSES OF TV VIEWING AND CARDIOMETABOLIC RISK.....	40
2.1	ABSTRACT.....	41
2.2	INTRODUCTION.....	42
2.3	MATERIALS AND METHODS.....	44
2.4	RESULTS.....	49
2.4.1	Participant characteristics.....	49
2.4.2	Distribution of TV viewing, cardiometabolic risk variables, and physical activity score by year.....	50
2.4.3	Correlations among TV viewing and cardiometabolic risk variables....	50
2.4.4	Cross-lagged panel model between TV viewing and clustered cardiometabolic risk score.....	51
2.4.5	Cross-lagged effects of TV viewing on five cardiometabolic risk variables.....	52
2.5	DISCUSSION.....	52
2.6	TABLES AND FIGURES.....	56

3.0	PROJECT#2: PROSPECTIVE CROSS-LAGGED ANALYSES OF TELEVISION VIEWING AND OBESITY IN THE CORONARY ARTERY RISK DEVELOPMENT IN YOUNG ADULTS (CARDIA) STUDY	61
3.1	ABSTRACT.....	62
3.2	INTRODUCTION	63
3.3	MATERIALS AND METHODS.....	65
3.4	RESULTS	69
3.4.1	Participant characteristics	69
3.4.2	Means of TV viewing, BMI, WST, and physical activity score by year.	69
3.4.3	Correlations among TV viewing and BMI and WST	70
3.4.4	Cross-lagged panel model between TV viewing and BMI and WST	70
3.5	DISCUSSION.....	71
3.6	TABLES AND FIGURES.....	75
4.0	PROJECT#3: THE INFLUENCE OF HOSTILE PERSONALITY TRAIT ON THE RELATIONSHIP BETWEEN TELEVISION VIEWING AND CARDIOMETABOLIC RISK.....	79
4.1	ABSTRACT.....	80
4.2	INTRODUCTION	81
4.3	MATERIALS AND METHODS.....	83
4.4	RESULTS	88
4.4.1	Participant characteristics by hostility	88
4.4.2	Distribution of TV viewing, cardiometabolic risk variables, and physical activity score by hostility at each examination year	89

4.4.3	Correlations among TV viewing and cardiometabolic risk variables by hostility	89
4.4.4	Cross-lagged panel model between TV viewing and clustered cardiometabolic risk score by hostility.....	89
4.4.5	Cross-lagged effects of TV viewing on five cardiometabolic risk variables by hostility	91
4.5	DISCUSSION.....	91
4.6	TABLES AND FIGURES.....	95
5.0	DISCUSSION AND CONCLUSION	101
5.1	SUMMARY	101
5.2	PROSPECTIVE ASSOCIATION BETWEEN TV VIEWING AND CARDIOMETABOLIC RISK	102
5.3	PROSPECTIVE ASSOCIATION BETWEEN TV VIEWING AND OBESITY	102
5.4	THE IMPACT OF HOSTILITY ON THE ASSOCIATION BETWEEN TV VIEWING AND CARDIOMETABOLIC RISK.....	103
5.5	PUBLIC HEALTH SIGNIFICANCE.....	103
5.6	LIMITATIONS OF THIS STUDY	104
5.7	FUTURE STUDIES.....	105
	APPENDIX.....	106
	SUPPLEMENTAL TABLES AND FIGURES	106
	BIBLIOGRAPHY	117

LIST OF TABLES

Table 2-1. Demographic and behavioral distribution of participants by TV viewing time at baseline (year 5).....	56
Table 2-2. Distribution of TV viewing, cardiometabolic risk variables, and physical activity by year.....	57
Table 2-3. Correlations between TV viewing and cardiometabolic risk variables.....	58
Table 2-4. Cross-lagged effects from TV viewing to five cardiometabolic risk variables respectively	60
Table 3-1. Demographic and behavioral distribution of participants by TV viewing time at baseline (year 5).....	75
Table 3-2. Means of TV viewing, body mass index, waist circumference, and physical activity score by year	76
Table 3-3. Correlations between TV viewing and BMI and waist circumference	77
Table 4-1. Demographic and behavioral distribution of participants by levels of hostility at baseline (year 5).....	95
Table 4-2. Distribution of TV viewing, cardiometabolic risk variables, and physical activity by year in the low hostility and high hostility groups.....	96
Table 4-3. Correlations between TV viewing and cardiometabolic risk variables by levels of hostility	97

Table 4-4. Chi-square tests for difference testing between the low and high hostility groups.....	99
Table 4-5. Cross-lagged effects from TV viewing to five cardiometabolic risk variables by levels of hostility	100
Table A-1. Coefficients and <i>p</i> values for individual path in the cross-lagged panel model of TV viewing and cardiometabolic risk for the whole population (N = 3,269).....	106
Table A-2. Coefficients and <i>p</i> values for individual path in the cross-lagged panel model of TV viewing and BMI for the whole population (N = 3,269)	109
Table A-3. Coefficients and <i>p</i> values for individual path in the cross-lagged panel model of TV viewing and waist circumference (WST) for the whole population (N = 3,269)	111
Table A-4. Coefficients and <i>p</i> values for individual path in the cross-lagged panel model of TV viewing and cardiometabolic risk for the low hostility group (N = 1,547)	113
Table A-5. Coefficients and <i>p</i> values for individual path in the cross-lagged panel model of TV viewing and cardiometabolic risk for the high hostility group (N = 1,722)	115

LIST OF FIGURES

Figure 2-1. A cross-lagged panel model showing that TV viewing predicts increases in clustered cardiometabolic risk score independent of covariates. Regression weights are standardized. R^2 represents the estimated proportion of the assumed underlying continuous variable explained by the model.....	59
Figure 3-1. A cross-lagged panel model showing the effects of duration of TV viewing on BMI. Regression weights are standardized. R^2 represents the estimated proportion of the assumed underlying continuous variable explained by the model.	78
Figure 3-2. A cross-lagged panel model showing the effects of duration of TV viewing on waist circumference.....	78
Figure 4-1. Two cross-lagged panel models showing the associations between duration of TV viewing and clustered cardiometabolic risk score are stronger for people with high hostility relative to those with low levels of hostility. Regression weights are standardized. R^2 represents the estimated proportion of the assumed underlying continuous variable explained by the model.	98

ACKNOWLEDGMENT

Thank you, Lord, I had several supportive doctoral committee members, Prof. Tammy Chung, Prof. Kevin H. Kim, Prof. Mark A. Pereira, and Prof. Janice C. Zgibor. I have received so much inspiration and encouragement over their guidance. I would like to thank them for advising me to complete my doctoral dissertation and receive my doctoral degree. Especially, thank you, Lord, for giving me a wise, remarkable, and caring advisor, Prof. Anthony Fabio. He provided me an extremely professional and comfortable research environment during my doctoral study. He also offered his advice and assistance whenever I needed them. Thank you, Lord, my family has always been supporting my dream. Thank you, Lord, many friends have enriched my life. I have been so blessed. Praise the Lord! The Lord Jesus takes care of my needs always. I am being here to present my dissertation work to my readers and to my all-inclusive God.

1.0 INTRODUCTION

Television (TV) viewing is a global social/environmental exposure that adversely affects many areas of public health. Time engaged in TV viewing has continued to increase. U.S. Data from 2000 indicate that people spent 4.1 hours watching TV per day.¹ This grew to 5.3 hours per day in 2010.² The estimated number of households with TV sets grew nearly twenty times from 1970 (58,500) to 2011 (115,9000), when the percent penetration reached 98.9%.³ In addition to the proliferation of TV sets and increased individual TV exposure time, the breath of TV program options and the number of advertisements on TV has increased over the past several decades in profound ways. In 1972, before cable TV became broadly accessible, 31% of all households could watch nine or more TV channels.⁴ By 1986, the number of channels per household has since had increased exponentially due to the proliferation and expansion of cable TV and satellite services.⁴

However, there may be a correlation between the dramatic increases in TV viewing and the growing pervasiveness of some diseases. One such disease is cardiovascular disease (CVD), one of the most pressing public health burdens in terms of cost and incidence. CVD is the leading cause of mobility and mortality in the U.S. and worldwide.⁵⁻⁶ There are 81 million American adults living with one or more types of CVD and 53% are estimated to be under age 60.⁵ Heart disease and stroke rank as the first and third causes of death in the U.S.⁷ In 2006, 34.3% of all 2,426,264 deaths were caused by CVD.⁷ Of these, 33% of deaths due to CVD were

people younger than 65 years old. There are many predictive factors leading to CVD such as cardiometabolic risk (e.g., abdominal obesity, high blood pressure, high cholesterol, and insulin resistance). These disorders evolve early in life and carry a high public health burden.

Prolonged sedentary behaviors, especially TV viewing, are thought to be one of the risk factors underlying the development of cardiometabolic risk. Positive associations between excessive TV viewing and cardiometabolic risk are consistently observed for adults in many populations including American⁸, French⁹, Australian¹⁰⁻¹², British¹³, and Flemish¹⁴. These studies also found deleterious effect of TV viewing on individual cardiometabolic risk variables such as obesity, hypertension, dyslipidaemia, and increased plasma glucose.

Besides a sedentary lifestyle, exposure to abundant images and messages included in TV programming and advertisements may promote behaviors that increase the risk of chronic disease, including unhealthy food choices, cigarette smoking, and excessive alcohol intake. Studies indicate that fast-food (23%), sweets (22%), beverage (17%), cereal (11%), and snacks (22%) made up the majority of advertisements among food-related advertisings.¹⁵ While young people watch their favorite sport programs, large numbers of alcohol advertisings are televised to them, especially beer.¹⁶⁻¹⁷ In addition, the entertainment industry may promote smoking and alcohol use because role models are usually portrayed engaging in these harmful habits.¹⁸ Mechanisms between TV viewing and cardiometabolic risk are not fully understood yet- but a decrease in overall energy expenditure mixed with an increase in total energy intake from overconsumption during TV viewing and influence of TV food advertisements are two potential mechanisms to explore.¹⁹

Hostility is also thought to be a determinant of CVD in many populations²⁰⁻²⁷ by increasing blood pressure, heart rate, and stress-related hormones in response to potential

stressors.²⁸ Studies report that violent TV programs can increase viewers' responses such as anger and contempt.²⁹ Experimental studies suggest that children who watched a violent program were more likely to behave in a hostile way during social interaction.³⁰⁻³² While watching TV, hostile people may experience stronger negative emotions and aggressive behaviors relative to calm counterparts since hostile people have a propensity to show malicious intent and feel anger towards others and a tendency to respond to frustrating situations with anger or irritation.²⁸ One cross-sectional study found a positive association between TV viewing and hostility, suggesting that there may be a mutual influence between them.¹⁹ People with high hostility may spend more time watching TV than those with low hostility. Given to the susceptibility of TV-induced negative emotions and tendency to prolonged TV exposure, hostile people may be more likely to develop CVD than agreeable people. Furthermore, hostile people are more likely to have unfavorable health behaviors including smoking, drinking, unhealthy eating, and physical inactivity, which in turn may be exaggerated further by TV viewing.²⁸ Rooted in these findings and theories, those with high hostility may show a stronger association between TV viewing and cardiometabolic risk compared with those with low levels of hostility.

We hypothesize that there may be a prospective effect of TV viewing on cardiometabolic risk and the hostile personality trait may be an important effect modifier for this association. To our knowledge, no studies have examined the prospective association between TV viewing and clustered cardiometabolic risk in the U.S. population. Also, the hypothesis that the direct association between TV viewing and cardiometabolic risk may be modified by a propensity towards a hostile disposition have yet to be examined. In order to test hypotheses from this theoretical framework, we identified a rich longitudinal dataset with multiple assessments of the exposure (TV), outcomes, and the various demographic and lifestyle factors that would be

examined as confounders. The Coronary Artery Risk Development in Young Adults (CARDIA) Study is a 25-year multi-center prospective study of CVD risk evolution in Black and White young adults (18 – 30 years of age at baseline in 1985/6) in four U.S. metropolitan regions. We used this dataset to evaluate the relationship between TV viewing and cardiometabolic risk. Additionally, we examined whether this relationship is modified by hostility.

1.1 SPECIFIC AIM

An understanding of the interrelationships between TV viewing and cardiometabolic risk and hostility through this study will provide insight into important potential mechanisms leading to CVD. We aimed to analyze the prospective CARDIA study data sets to test our hypotheses on TV viewing and cardiometabolic risk and hostile personality trait. This research used cross-lagged panel models to analyze the cross-lagged effects between TV viewing and cardiometabolic risk. The research encompasses three discrete, but related projects:

1.1.1 Project#1

Specific Aim 1. Describe the prospective associations between TV viewing and cardiometabolic risk.

Hypothesis 1.1 Duration of TV viewing will be positively associated with cardiometabolic risk.

1.1.2 Project#2

Specific Aim 2. Describe the prospective associations between TV viewing and obesity including body mass index (BMI) and waist circumference.

Hypothesis 2.1 Duration of TV viewing will be positively associated with obesity.

1.1.3 Project#3

Specific Aim 3. Evaluate whether hostility modifies the association between TV viewing and cardiometabolic risk.

Hypothesis 3.1 The association between duration of TV viewing and cardiometabolic risk will be stronger for those with high hostility relative to those with low levels of hostility.

1.2 LITERATURE REVIEW

1.2.1 Television viewing

1.2.1.1 Households with TV sets

U.S. households with TV sets have dramatically increased since 1950. During the period, the prevalence was only 9%. However, within 5 years it jumped to 64.5%. By 1970 it was up to 95.3%, and from there it increased to 98.9% in 2011. The estimated number of households with TV sets increased nearly thirty fold from 1950 (3.8 million) to 2011 (115.9 million) when the percent prevalence reached 98.9%.³

1.2.1.2 TV viewing trends

Time spent in TV viewing has been greatly increasing in recent years. The U.S. Census Bureau reported that in 2000 an individual (12 and older) spent an average of 4.1 hours watching TV per day.¹ This grew to 4.7 hours per day in 2009. The 2010 Nielson Three Screen Report showed that the average American (over age 2) spent 5.3 hours viewing TV per day, a 1.3% increase from 2009.²

According to the Bureau of labor statistics of 2009, time spent watching TV varies by gender, age, race, employment status, salary, and education (data refer to persons 15 years and over).³³ Men watch TV 3.1 hours per day compared with 2.56 hours for women. Persons 15-19 years and over 55 years watch TV around 3 hours per day, while persons 20-54 years watch TV 2.5 per day. The average hours for TV viewing were 3.7 hours per day for Blacks and 2.7 hours per day for Whites.

1.2.1.3 TV contents

Myriad messages, scenes, and products on TV programming and advertisements may contribute to harmful health behaviors, including unhealthy food choices, smoking, alcohol use, aggression, and violence. The market tends to target adolescents as direct consumers given that adolescents are much more likely than children to have and spend their own money. An estimate of food advertising was around 20% of nonprogram content exposure to adolescents.¹⁵ The number of food advertisements per day increased with age (11.5, 13.1, and 13.6 for audiences aged 2-5 years, 6-11 years, and 12-17 year, respectively).³⁴ Among food-related advertisings, fast-food (23%), sweets (22%), beverage (17%), cereal (11%), and snacks (22%) made up the majority of advertisements.¹⁵ Several studies have reported more fast-food and sweets commercials geared towards Blacks.^{15, 34-36} While young people watch their favorite sport programs, large numbers of alcohol advertisings are televised to them, especially beer.¹⁶⁻¹⁷ In addition, the entertainment industry may influence smoking and alcohol use because role models are usually portrayed engaging in these harmful habits.¹⁸ Roberts et al, found that smoking and alcohol use behaviors were prevalent elements of music videos, of which rap music videos had the highest portrayal of smoking.³⁷ Violence is also a popular element in music videos. The highest percentage of violence is associated with rap music videos.³⁸ Anderson found that large numbers of violent commercials from promotions of television programs and big-screen movies showed during the family-oriented programming.³⁹ Robert and colleagues also observed that during major sporting events, 49% of commercial breaks included at least one commercial portraying unsafe or violent behavior.⁴⁰ Dubious lifestyle habits are ubiquitous in televised programming and advertisements even during family-oriented programming. This environment makes it difficult for parents to protect their children from unhealthy ideas. Therefore, the American Academy of Pediatrics

discouraged TV exposure to young children, especially the banning of TV viewing under the age of 2.⁴¹

1.2.2 Cardiovascular disease (CVD)

1.2.2.1 Definition

Cardiovascular disease is not just a single disorder. It includes a number of over 60 different disorders affecting the heart and blood vessels, which consist of coronary heart disease, cerebrovascular disease (stroke), peripheral arterial disease, rheumatic heart disease, congenital heart disease, deep vein thrombosis and pulmonary embolism.⁶ Heart disease can represent a broad range of diseases of heart and blood vessels, but heart disease is more often used to specifically describe coronary artery disease or coronary heart disease.⁴²

1.2.2.2 Prevalence of CVD

In 2006, 81 million Americans adults lived with one or more types of CVD. Of these, 53% are estimated to be under age 60. CVD is also a health threat to young people now. An estimated 39 million (37.9%) males and 42.1 million (35.7%) females live with CVD. The prevalence of CVD is higher among Blacks than Whites for both men and women. The prevalence increases as people age, over 70% for those 60+, over 39% for those 40-59, and fewer than 15% for those 20-39.⁵ The prevalence of CVD does not differ much by gender except for people aged 20-39, where males had greater prevalence of CVD than females.

1.2.2.3 CVD mortality

CVD is the leading cause of death not only in the United States⁷ but also worldwide.⁶ According to National vital statistics reports, heart disease and stroke rank as the first and third causes of death in the U.S. Across various race and ethnic groups, heart disease and stroke are still the top killers of older persons.⁷ Since 1900, there has been a dramatic increase in the number of CVD deaths.⁵ 34.3% of all 2,426,264 deaths was due to CVD in 2006.⁷ Of these CVD deaths, more than 151,000 Americans were younger than 65 years old. That is, about 33% of deaths due to CVD were premature.

1.2.2.4 Cardiometabolic risk

Cardiometabolic risk refers to a set of intermediate risk factors contributing to the development of CVD such as central obesity, insulin resistance, hyperglycemia, dyslipoproteinemia, and hypertension.⁴³ These medical disorders can occur individually, but generally occur in cluster. Age, race, sex, and family history are major biological risk factors contributing to the cardiometabolic risk. Physical inactivity and smoking are key behavioral risk factors. According to the statistics of World Health Organization (WHO), around 80% of coronary heart disease and cerebrovascular disease are associated with these behavioral risk factors.⁶ Some intermediate risk factors mentioned above are also observed in type 2 diabetes, including obesity, insulin resistance, dyslipoproteinemia, and hypertension.⁴³ Hence, prevention and intervention for cardiometabolic risk are beneficial for reduction in both of cardiovascular disease and type 2 diabetes.

1.2.3 Overweight and obesity

1.2.3.1 Definition

Obesity is an increasing public health threat because it is a common risk for chronic diseases. The formula for body mass index (BMI) is $\text{weight (kg)} / [\text{height (m)}]^2$.² For adults, individuals who have a BMI between 25 and 29.9 are considered overweight; those who have a BMI of 30 or higher are considered obese.⁴⁴ BMI has been considered a reliable indirect indicator of body fatness. Another measure of obesity is defined as a waist circumference (WC) ≥ 102 cm in men and ≥ 88 cm in women.⁴⁵ Waist circumference can represent the magnitude of abdominal obesity, which is important for predicting chronic diseases.

1.2.3.2 Prevalence

The trend of becoming overweight and obese is increasing in children and adults over time. Based on the 2000 CDC growth chart, there were around 10 million American children and adolescents aged 6 -19 who are overweight. Based on the 95th percentile or higher of BMI-for-age values of the CDC growth charts, in children aged 6-11 years, the prevalence of overweight increased from 4% in 1971-74 to 17% in 2003-06. In adolescents aged 12-19 years, the prevalence of overweight increased from 6.1% to 17.6%. Seventy percent of overweight adolescents will become overweight adults, and if one or both parents are overweight or obese, this percentage increases to 80%.⁵ The 2006 prevalence of overweight or obesity among adults 20 years of age and over was 66.3 percent (an estimated 144,100,000 people). In 2008, 26.6% of adults were obese. Among adults, the CDC observed that from 1999-2008, obesity and abdominal obesity significantly increased in men and abdominal obesity increased in women.⁴⁶

More concerning is the WHO estimates that by 2015, there will be 2.3 billion overweight or obese people worldwide. Of those, more than 700 million will be obese.⁵

1.2.3.3 Relation of TV viewing to obesity

Over the past three decades, TV viewing has been proposed as a determinant of obesity in numerous studies. The increasing trends of TV viewing and obesity also support the hypothesis that TV viewing plays a role in the obesity epidemic. The following literature review discusses the association and potential mechanisms of TV viewing to obesity in different types of epidemiological studies.

1.2.3.4 Cross-sectional studies

Dietz and colleague⁴⁷ conducted the first study to investigate the association of TV viewing and obesity. This study included data from cycles II (in 1963-1965) and III (in 1966-1970) of the National Health Examination Survey. There were 6,965 children aged 6 to 11 years in cycle II and 6,671 children aged 12 to 17 years in cycle III. Two cross-sectional samples and one prospective sample were examined in this study. To define obesity, triceps skinfolds were measured by pediatricians, specially trained nurses, and technicians. A triceps skinfold equal to or greater than the 85th percentile represented obesity and a triceps skinfold equal to or greater than the 95th percentile represented superobesity. The numbers of hours of TV watching per day were assessed via two kinds of sources: parental reports in cycle II and self-reports in cycle III.

The authors found significant associations between obesity, superobesity, and time spent TV viewing in both cross-sectional and prospective samples. Each hourly increment of TV viewing was associated with a 1.2 to 2.9% high prevalence of obesity. The prevalence of superobesity increased 0.6 to 1.4% for each additional hour of TV viewing. These positive

relationships were attenuated slightly but still remained significant after controlling parents' education, age, income, number of children, birth order, race, and condition restricting activity.

The Dietz study demonstrated a positive relationship of TV viewing to obesity in children and adolescents, in both cross-sectional and prospective studies. This article is the first study to mention two potential mechanisms between TV viewing and obesity: 1) reduced energy expenditure due to less energy needs; 2) increased energy intake due to snacking while watching TV and influence of food advertising and food references in TV programs.

Nevertheless, the coefficients estimates in this study were significant but small. That might be partly explained by the low TV set penetration and small number of TV channels and programs in 1960s. The television influence might be much lower in 1960s compared with nowadays.

Sidney et al.¹⁹ examined cross-sectional associations between self-reported duration of TV viewing and cardiovascular risk factors among 4,280 Blacks and Whites aged 23-35 years. This study used data from the year-5 follow-up examination (1990/1991) of the Cardiovascular Risk Development in Young Adults (CARDIA) study. The CARDIA study included four geographic locations: Birmingham, Alabama; Chicago, Illinois; Minneapolis, Minnesota; and Oakland, California. Participants were categorized into heavy TV viewers (≥ 4 h/day) and light TV viewers (0 to 1 h/day). The definition of obesity was a BMI equal to or greater than 27.8 kg/m² for men and 27.3 kg/m² for women. Data suggested that compared to light TV viewers, heavy TV viewers had higher odds of being obese, with race-gender stratified odds ratios varying from 1.5 (95% CI= 1.1-2.2) in Black women to 2.3 (1.4-3.9) in White women.

This study suggested a positive association between prolonged TV viewing and obesity prevalence among young adults. The model was adjusted for age, education, physical activity, smoking, alcohol use, and examination center.

In a Spanish study, Vioque et al.⁴⁸ examined the association of TV watching and physical activity with obesity among Mediterranean population. The Health and Nutrition Survey randomly selected 814 males and 958 females aged 15 years and older in 1994. Obesity was defined as BMI at least 30 kg/m². Participants were asked “How many hours per week do you usually spend watching TV?” Time spent watching TV was also categorized as: ≤ 1 h/day, 2 h/day, 3 h/day and ≥ 4 h/day.

The authors reported that obese people spent more time watching TV daily (mean \pm s.d.: 3.6 \pm 1.5 h/day) compared with non-obese ones (3.0 \pm 1.4 h/day). In multivariate analysis, obesity was more prevalent in people watching TV ≥ 4 h/day than people watching ≤ 1 h/day (Odds ratio: 2.38; 95% CI: 1.54-3.69). A significant dose-response was observed between TV watching and obesity in which the prevalence of obesity increased by 30% for each additional hour of TV watched.

Jakes et al.⁴⁹ examined the relation of TV viewing and recreational activity with obesity and CVD biomarkers among 14,189 adults, aged 45 to 74 years, living in Norfolk, UK. TV viewing was reported in four categories: < 2 , 2-2.9, 3-3.9, and > 4 h/day. Participants were asked for the frequency and time of physical activity per week. The MET.hr/week of vigorous recreational activity was ≥ 5 . BMI, waist to hip ratio (WHR) and percent body fat were makers of obesity.

All age-adjusted means of obesity markers (BMI, waist, hip, WHR, body fat) significantly increased with the amount of TV viewing for both genders. After controlling for

age, alcohol consumption, smoking, treatment for hypertension, vigorous and total physical activity, these significant relationships were still observed. The authors also investigated the combined influence of TV viewing and vigorous activity on BMI. The adjusted mean BMI of men who watched TV more than 4 h/day and did not do vigorous activity weekly increased 1.44 kg/m² compared with those who watched TV less than 2 h/day and did at least one hour of vigorous activity weekly. The equivalent comparison in women was 1.92 kg/m².

This study suggested that time spent TV viewing was associated with obesity. Moreover, the relative contribution of TV viewing and vigorous activity in predicting obesity was observed. Owing to the use of different markers of obesity, this study provided more evidence about the association of TV viewing and obesity.

Parsons et al.⁵⁰ analyzed the cross-sectional association of physical activity, TV viewing and BMI at six ages from childhood to adulthood in the 1958 British birth cohort before further longitudinal analysis. A TV viewing frequency was recorded at ages 11, 16 and 23 years old. BMI (kg/m²) was measured by trained medical personnel at 11 and 16 years but was obtained from self-report at 23 years. At ages 11 and 16, TV viewing frequency was coded as often, sometimes or never/hardly ever. On the other hand, participants at 23 years were asked for TV viewing time (≥ 5 times a week, 3-4 times a week, and \leq twice a week).

The study found a significant positive association between BMI and TV viewing frequency at 11 years in females and at 23 years in both genders. In contrast, BMI was not associated with TV viewing frequency at 11 years in males and at 16 years in both genders. The authors reported that the association still remained significant after adjusting for covariates. However, they did not describe clearly which confounders were included in models. This study derived from a birth cohort provided several cross-sectional analyses at different ages. However,

owing to different categories of TV viewing frequency at 11, 16, and 23 years, it is difficult to compare a change of TV viewing habits over time. The impact of TV viewing to obesity might be less at 11 (1969) and 16 (1975) years than at 23 (1981) years because the prevalence of TV sets and TV advertising was less in 1969 and 1975 than 1981. In addition, duration of TV viewing may show stronger association than frequency, which might provide another explanation for the null relationship at 16 years.

Bowman et al.⁵¹ used a nationally representative population to investigate the links among TV viewing, energy intake, obesity, and health status in adults aged 20 years or older. There were 9,157 participants recruited from the U.S. Department of Agriculture's Continuing Survey of Food Intakes by Individuals 1994-1996. Self-reported data included 24-hour dietary recall, height and weight (BMI), and the daily hours of TV viewing. TV viewing data was also coded as < 1, 1-2, and > 2 h/day. Overweight was defined as $BMI \geq 25 \text{ kg/m}^2$ and obese was $BMI \geq 30 \text{ kg/m}^2$.

Prolonged TV viewing per day was significantly associated with increased BMI in both men and women. People who watched TV more than 2 hours per day had higher BMI and also had a higher percentage of being overweight and obese compared with those who watched less than 1 hour. Among age groups, adults aged 66 or older had the highest percentage of watching TV more than 2 hours daily. Of racial groups, Blacks were more likely to watch TV greater than 2 hours a day. Education and income levels had inverse relationships with the amount of TV viewing. More unemployed adults including retired people watched more than 2 hours of television everyday than those who were employed. There was a positive correlation between prolonged TV viewing and adverse health status including diabetes, hypertension, heart disease, and high bold cholesterol. Consistently, within the above socioeconomic and demographic

characteristics and health status, the likelihood of overweight or obesity increased with the amount of time spent watching TV. Additionally, people who watched more than 2 hours of TV daily obtained higher total energy intake from supper and snacks. This study provided a broad perspective for TV viewing-obesity hypothesis by analyzing this association within various socioeconomic and demographic characteristics. Therefore, more confounding factors should be carefully considered for relation of TV viewing and obesity in an adult population.

Cleland et al.⁵² tried to understand which behavioral pathways determine the influence of TV viewing on obesity, whereby food consumption during viewing time and reduced leisure-time physical activity were tested as mediators of this association.

They enrolled 2,001 Australian adults aged 26-36 years from 2004 to 2006. Waist circumference (WC) was measured by trained technicians who followed a standardized protocol. Based on World Health Organization cutoffs, the definition of moderate abdominal obesity was waist circumference between 94 and 101.9 cm in men and between 80 and 87.9 cm in women. The definition of severe abdominal obesity was waist circumference ≥ 102 cm for men and ≥ 88 cm for women. Average daily TV viewing time was calculated by summing TV viewing time on weekdays and weekend and dividing it by 7. Frequency of food and beverage consumption during TV viewing time was categorized into four groups: never, 1-2, 3-4, and ≥ 5 times/wk. Participants were asked to report time spent on leisure-time physical activity during the past week. Leisure-time physical activity was divided into four groups based on quartile splits.

A higher prevalence of severe abdominal obesity was seen in women who watched TV > 3 h/day compared with those who watched ≤ 1 h/day [prevalence ratio (PR): 1.89; 95% CI: 1.32-2.71]. Men who watched TV > 3 h/day had a higher prevalence of moderate abdominal obesity than those who watched ≤ 1 h/day (PR: 2.16; 95% CI: 1.37-3.41). This association was

attenuated after controlling for food and beverage consumption but not controlling for leisure-time physical activity (PR: 1.48; 95% CI: 1.01-2.17 for women; PR: 1.73; 95% CI: 1.06-2.83 for men). This study found not only the association between TV viewing and obesity but also the potential mediator which could explain the association. Increased food and beverage consumption seemed to be a more important mediator relative to physical inactivity.

1.2.3.5 Prospective cohort studies

Hu *et al.*⁵³ used a prospective study to explore the influence of television watching and other sedentary behaviors on risk of obesity and type 2 diabetes mellitus in women participating in the Nurses' Health Study. Baseline mean age was about 60. 50,277 women were followed up from 1992 to 1998. For the obesity analysis, women with BMI > 30 were excluded at baseline. Average weekly time spent watching TV or VCR was categorized into 5 groups: 0-1, 2-5, 6-20, 21-40, and > 40 h/week. Incidence of obesity was defined while participants who were non-obese (BMI < 30) at baseline became obese (BMI ≥ 30) at the end of 6-year follow-up.

At baseline, women who watched more TV were more likely to have high BMI. A prospective analysis also reported that TV watching was positively associated with risk of obesity. In multivariate analysis, the age-adjusted relative risks (RRs) were 1.0, 1.23, 1.42, 1.68, and 2.00 for respective categories of TV watching. Further adjustment for age, smoking, alcohol consumption, and exercise level did not attenuate the RRs greatly. Neither did further adjustment for dietary factors and other covariates. A significant dose-response was observed between TV watching and obesity in which the risk of obesity increased by 23% (95% CI: 17-30%) for each additional 2-h/day of TV watched. Moreover, TV watching predicted the highest risk of obesity compared with other sedentary activities including sitting at work, reading, mealtime, and at desk. The study reported an independent association between TV watching and obesity risk

regardless of exercise level and diet consumption. This result may be explained by a lower energy expenditure TV watching causes compared with other sedentary behaviors.

In a 1970 British Birth Cohort Study, Viner et al.⁵⁴ investigated the relation of duration, timing, and content of TV viewing in childhood to BMI in adulthood. Data included weekday and weekend TV viewing duration, type of programs, and maternal attitudes toward TV at age 5 years, TV viewing frequency at 10 years, and BMI at 10 and 30 years. BMI \geq 30 kg/m² represented obesity.

Children who watched more TV during weekdays and weekends were more likely to have higher subsequent BMI at 10 and 30 years of age. TV viewing habits at 5 years were positively associated with frequency of TV viewing at 10 years. On the other hand, negative maternal attitude toward TV at 5 years caused a lower frequency of TV viewing at 10 years. In multivariate regression, the risk of obesity increased 7% with each additional hour of weekend TV viewing (odds ratio: 1.07, 95% CI: 1.01, 1.13). However, weekday TV viewing, type of programs, maternal attitude toward TV at 5 years, and TV viewing frequency at 10 years were not independent risk factors of BMI at 30 years.

The findings suggested a long-term effect of weekend but not weekday TV viewing in early childhood on increased BMI in adulthood. The different results between weekday and weekend TV viewing might indicate that timing of TV viewing could be an important risk factor of obesity due to the difference among content of programs, food advertising, level of parental supervision, and energy consumption during weekday versus weekend. However, the lack of follow-up data between age 10 and 30 years is a limitation.

Parsons et al.⁵⁵ conducted a prospective study in the 1958 British birth cohort previously mentioned. The aim here was to estimate whether TV viewing frequency in childhood (11 and 16

years) and young adulthood (23 years) was associated with increased BMI (16, 23, 33, and 45 years) through to adulthood and waist-hip ratio (45 years) in middle adulthood. The findings were that watching TV often at 16 years of age predicted an increase in BMI from age 16 to age 45 in men (0.011 kg/m² every year, 95% CI: 0.003-0.019) and women (0.013 kg/m² every year, 95% CI: 0.003-0.023). Women, but not men, who watched TV more frequently at 11, 16, or 23 years had an increase in BMI. Furthermore, waist-hip ratio at 45 years was 0.01 higher for participants aged 23 years watching TV \geq 5 times per week than those watching less often. Waist-hip ratio was 0.03-0.04 higher for participants aged 45 years watching TV \geq 4 h/day compared with those watching < 1 h/day. The research suggested that frequency of TV viewing influenced BMI gains from adolescence to adulthood. Moreover, frequency and duration of TV viewing affected central adiposity of adults.

Wijndaele et al.¹² conducted a prospective analysis to explore the association between increased TV viewing time and adverse cardiometabolic risk changes among 3,846 Australian adults measured in 1999-2000 (baseline) and 2004-2005 (follow-up). Duplicate waist circumference was measured by trained personnel. Change in TV viewing time was derived from follow-up minus baseline.

In multiple linear regression models, the authors observed that change in TV viewing time was associated with an increment in waist circumference during 5 years of follow-up (unstandardized β coefficients: for men: 0.43 cm, 95% CI: 0.08-0.78 cm; for women: 0.68 cm, 95% CI= 0.30-1.05). This association still remained significant even adjusting for baseline physical activity and diet quality. Conversely, there was no significant association seen between baseline TV viewing and waist circumference. Several possibilities might be considered. First, measurement error could cause misclassification which biases the relation toward the null. Next,

during the 5-year follow-up, 80% of participants changed their TV viewing time (generally, an increase in TV viewing time) so change in TV viewing time could reflect the trend of waist circumference change better than baseline TV viewing. Third, the effect of TV viewing on obesity could be primarily short term (< 5 years) so baseline TV viewing might not predict obesity 5 years later.

1.2.3.6 Summary

Across age, race, gender, and countries, most of the evidence supports a positive association between TV viewing and obesity, although some data show opposite relationships. The increases in obesity risk varied by the covariates controlled and by the way in which TV viewing and obesity were measured and categorized.

A meta-analysis⁵⁶ examined the relationship between TV viewing and body fatness among children and youth by reviewing 52 independent samples from 30 studies (N=44,707) with different designs. The effect size (Pearson r) of this relationship was 0.084 (fully corrected). Among different body fatness measures, BMI showed a significant effect size (0.087). The authors indicated that this positive relationship between TV viewing and obesity was statistically significant, but it was too small to represent meaningful clinical evidence. However, small effects may deeply influence health status of populations and individuals if they accumulate.

If the relation of TV viewing to obesity is real, then it could have profound effects in the area of obesity prevention. Reducing prolonged TV viewing could provide an efficient way for decreasing the chances of developing obesity. Limiting unhealthy food advertisements and TV programs encouraging harmful lifestyle would be other solutions. For children, parental supervision could be very crucial for improving their eating pattern. The reviewed literature suggests that TV viewing frequency and duration increases the risk of obesity among adults.

1.2.3.7 Potential mechanisms linking TV viewing and obesity

Three potential theories of the influence of TV viewing on obesity include reduced physical activity, lower resting metabolic rate, and increased energy intake. People watching more TV have a higher prevalence of physical inactivity.^{19, 57} However, some studies found that watching TV displaces physical activity during leisure time⁵⁸⁻⁶⁰, but other studies did not.⁶¹⁻⁶³ On the other hand, data suggests that watching TV causes lower metabolic rate than other sedentary behaviors.⁶⁴ This may explain why compared with other sedentary behaviors such as driving a car, reading, and writing, TV viewing was most strongly related to the risk of obesity.⁵³

The other theory is that TV viewing increases the risk of obesity due to snacking during TV watching time or increased exposure to food advertisements. Among children, the positive effect of TV viewing on high-energy food intake has been consistently observed.⁶⁵⁻⁶⁷ This relationship was also assessed among adults by Cleland et al.⁵² The increased prevalence ratios of obesity among adults with high TV viewing were 2.16 for men and 1.89 for women. After adjusting for food and beverage consumption, the prevalence ratios became attenuated (1.73 for men and 1.48 for women). They suggested that food and beverage consumption during TV watching may be one mediator between TV viewing and abdominal obesity.

The positive relationship between TV viewing and obesity may be mutually influenced. TV viewing may increase obesity risk, while overweight and obese people may tend toward sedentary activities like TV viewing and consumption of high caloric food while watching TV compared to non-overweight people. A Spanish cross-sectional study showed a significant interaction effect of TV viewing and weight status on abdominal obesity.⁶⁸ The overweight group reported the significant positive association between TV viewing and waist circumference but the non-overweight group did not.

1.2.3.8 Relation of obesity to CVD

Obesity has been demonstrated as a determinant of cardiovascular risk in a variety of epidemiological studies. In the Framingham study,⁶⁹ a 26-year follow-up study found that obesity was an independent risk factor of development of coronary heart disease. The subsequent Framingham experience observed that overweight (BMI, 25-29.9) and obesity (BMI \geq 30) had an increased relative risk (RR) of the risk factors of CVD (e.g., hypertension) and CVD itself for middle-aged adults.⁷⁰ All the RRs of cardiovascular risk factors and CVD were higher in obesity status compared with in overweight status. The risk of cardiovascular risk factors and CVD increased with the severity of overweight. Another 10-year cohort study also showed that the incidence of diabetes, hypertension, and heart disease increased with the degree of obesity for middle-aged women and men, while stroke risk increased with obesity for men only.⁷¹ Persons who were overweight and obese significantly had an increased risk of developing one or more cardiovascular morbidities compared with their leaner peers (BMI, 18.5-21.9). Particularly, in both obese women and men, individuals who had high cholesterol or high blood pressure were at a higher risk of developing 1 or more adverse health conditions relative to those who did not. A meta-analysis of 21 cohort studies found that around 45% of the increased risk of coronary heart disease was mediated by high cholesterol and blood pressure.⁷²

Abdominal fat, another measure of fatness, is also of importance for unfavorable cardiovascular risk factors. In children and adolescents, abdominal fat was a predictor of plasma triglycerides, HDL cholesterol, systolic blood pressure, and left ventricular mass which are important cardiovascular risk factors.⁷³ As young people become adults, their central fat may accumulate, leading to unfavorable cardiovascular risk factor status. Haarbo and colleagues⁷⁴⁻⁷⁵

suggested that central fat in postmenopausal women was positively associated with triglycerides and LDL cholesterol while it was negatively associated with HDL cholesterol.

There is little doubt that obesity is a crucial determinant of the increasing prevalence of cardiometabolic risk factors and CVD. The relationship between obesity and CVD may be mediated through the major risk factors (hypercholesterolemia, hypertension, hyperglycemia) and emerging risk factors (atherogenic dyslipidemia, insulin resistance, proinflammatory state, prothrombotic state).⁷⁶ These mediation associations vary by individual differences in genetic and environment factors. However, individuals who are overweight are more likely to simultaneously have multiple major and emerging risk factors causing CVD risk.

1.2.4 High cholesterol

1.2.4.1 Definition

Cholesterol is essential for our body, but it harms when its level is too high in the body. High cholesterol can cause plaque in the arteries, which leads to heart disease and stroke, the top causes of death in the U.S.⁷⁷ Total cholesterol is made up of high-density cholesterol (HDL), low-density cholesterol (LDL) and triglycerides. High cholesterol is defined as the total cholesterol level of 240 mg/dL or higher.⁷⁸ HDL cholesterol is known as “good” cholesterol, which can bring blood cholesterol back to the liver.⁷⁷ Higher levels of HDL cholesterol are better for health. On the other hand, high levels of LDL cholesterol, or “bad” cholesterol, can clog the arteries and increase the risk of heart disease and stroke. Hence, the optimal levels of LDL cholesterol should be maintained less than 100 mg/dL. High levels of triglycerides are associated with coronary heart disease.

1.2.4.2 Prevalence

American Heart Association reported that in 2006, 46.8% of adults aged 20 years and over had total cholesterol levels of 200 mg/dL or above.⁵ Moreover, 16.2 percent of adults showed total cholesterol levels of 240 mg/dL or higher. There are around 35.7 million adults (15.9 million males and 19.7 million females). The 2007 prevalence of high blood cholesterol in adults was 37.6 percent.

The 2006 data indicated that 71.2 million adults had LDL cholesterol 130 mg/dL or above (34.9 million men and 36.3 million women).⁵ On the other hand, 35.1 million adults had HDL cholesterol levels less than 40 mg/dL (26.4 million men and 8.7 million women). The mean level of LDL cholesterol for adults was 115 mg/dL, which is beyond the optimal level. The mean levels of HDL cholesterol and triglyceride were 54.3 mg/dL and 144.2 mg/dL, respectively.

1.2.4.3 Relation of TV viewing to high cholesterol

Sedentary lifestyle can put people at an increased risk of developing high cholesterol. Particularly, TV viewing can cause physical inactivity and unhealthy diet, both of which are risk factors for high cholesterol. The following epidemiological studies show that TV viewing is a predictor of high cholesterol.

1.2.4.4 Cross-sectional studies

Sidney et al.¹⁹ categorized participants into heavy TV viewers (≥ 4 h/day) and light TV viewers (0 to 1 h/day). Total cholesterol (≥ 240 mg/dL), LDL cholesterol (≥ 160 mg/dL), and HDL cholesterol (< 35 mg/dL) were measured.

The authors observed that the duration of TV viewing daily was longer in Blacks than in Whites and was inversely related to education and income. They found that TV viewing was not

significantly associated with the lipid profile in all race/gender groups. These results may be explained by small percentages of subjects who were defined as high cholesterol (less than 9% in all race/gender groups) so that power was limited. On the other hand, the model was adjusted for some covariates including physical activity which is one risk factor of high cholesterol. The association between TV viewing and high cholesterol might be attenuated by physical activity. Also, cholesterol accumulates when people age, but the subjects were only 25-35 year-old young adults.

Limited TV viewing categories including heavy viewers (≥ 4 h/day) and light viewers (0 to 1 h/day) could cause residual confounding. For example, white women represented 8.2% of heavy TV viewers and 52.3% of light TV viewers. The uneven proportion could attenuate the association between TV viewing and high cholesterol. If individuals diagnosed with CVD underreport the duration of TV viewing, this also might result in an attenuated association.

Fung et al.⁷⁹ examined the associations between leisure-time physical activity, TV viewing, and CVD risk factors. This study provided cross-sectional and prospective analyses among 468 healthy male health professionals aged 40-75 years at baseline from 1986 to 1994. Blood samples were only collected in 1993-1994, when physical activity (starting in 1986) and TV viewing (starting in 1988) were measured biennially. The definition of TV viewing was the number of TV viewing hours and video-cassette recordings per week. The lipid biomarkers included total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides. Vigorous leisure-time physical activity was defined as MET.hr/wk ≥ 6 .

Cross-sectional results showed that TV viewing hours were associated positively with LDL cholesterol ($p < 0.05$), whereas they were inversely associated with HDL cholesterol ($p < 0.01$). TV viewing was not significantly associated with the other lipid biomarkers. After

adjusting for covariates plus BMI, the associations between TV hours and the lipid biomarkers decreased. Prospective results suggested that no significant associations between average TV viewing hours in 1988-1994 and the lipid biomarkers. The authors further analyzed the joint effects of TV viewing and vigorous physical activity on HDL cholesterol. They found the associations between TV viewing and HDL cholesterol were independent of physical activity. Vigorous physical activity, however, was a stronger predictor of HDL cholesterol than TV viewing.

This study indicated that TV viewing duration was significantly and positively associated with LDL cholesterol and significantly and inversely associated with HDL cholesterol. This relationship may be partially explained by obesity (BMI). The subjects were all from a unique occupation so results might not be generalized to other populations.

A British study conducted by Jakes et al.⁴⁹ reported that after adjusting for covariates including participation in vigorous activity, total physical activity and BMI, total cholesterol, LDL cholesterol, and triglyceride significantly increased with TV viewing duration in both men and women, whereas HDL cholesterol significantly decreased with TV viewing. TV viewing is associated with the lipid profile independent of physical activity and obesity. That is, the association may be mediated through the other biological processes other than weight gain and reduction in physical activity.

A Danish cross-sectional analysis by Aadahl et al.⁸⁰ sought to examine the relationships between TV viewing and vigorous intensity physical activity and cardiovascular biomarkers including serum lipids. The study included 1693 men and women aged 33-64 years (mean age = 51.3 for men and 50.3 for women). Information on physical activity level was measured by MET score. Diet was categorized into three groups; 1) unhealthy diet: low fruit/vegetable and fish

intake and high fat intake; 2) medium healthy diet: medium fruit/vegetable, fish and fat intake; 3) healthy diet: high fruit/vegetable and fish intake and a low fat intake.

In the linear regression model, total cholesterol, LDL cholesterol, and triglycerides significantly increased with the amount of TV viewing, but the coefficients were relatively small. HDL cholesterol showed no association with TV viewing. This model controlled for several covariates including diet and physical activity.

The authors concluded that amount of time spent on TV viewing is a predictor of CVD biomarkers independent of physical activity. Minimal associations between TV viewing and serum lipids may be partially explained by the adjustment of physical activity and diet. In order to clarify the mechanisms from TV viewing to serum lipids, separate mediator analyses of physical activity and diet are needed. However, more detail on the timeline for recruitment and variables measured was needed, since these factors could reduce the validity of this study.

The Australian Diabetes, Obesity and Lifestyle (AusDiab) study examined the influence of TV viewing on CVD biomarkers longitudinally. This population-based study started recruiting 11,247 Australians aged ≥ 25 years in 1999/2000 with a follow-up measurement in 2004/2005.^{11-12, 81} In 2008, Healy et al.¹¹ analyzed the relationship between TV viewing time and continuous metabolic risk among physically active adults through the cross-sectional data of AusDiab in 1999/2000. There were 2031 males and 2033 females who met the physical activity guideline of at least 2.5 hr/week of moderate- to vigorous-intensity exercise. Participants were asked for the amount of time spent watching TV or videos in the previous week. Triglycerides and HDL cholesterol levels were obtained at the same time.

The authors found that triglycerides increased and HDL cholesterol decreased with the ordinal TV viewing categories for women controlling for covariates including diet quality and

physical activity time. Significant dose-response relationships between TV viewing and triglycerides and HDL cholesterol were observed only in women. The findings showed a gender difference, which may be attributable to physiological sex difference in response to TV viewing.

In 2010, Thorp et al.⁸¹ measured triglycerides and HDL cholesterol among 2,761 women and 2,103 men from the 2004/2005 survey of the AusDiab Study. Again, the findings supported that triglycerides increased and HDL cholesterol decreased with the TV viewing time independent of diet quality and physical activity in women. Moreover, the association was attenuated gradually when four models adjusted for age-only (Model A), demographic covariates plus diet quality and physical activity time (Model B), all covariates plus waist circumference (Model C), and all covariates plus sitting time (Model D). After adjusting for all covariates plus sitting time, TV viewing showed no association with triglycerides. After adjusting for covariates plus waist circumference, HDL cholesterol was not related to TV viewing. The association between TV viewing and cholesterol may be mediated by diet, physical activity, central adiposity, and prolonged sitting time.

Ekelund et al.⁸² assessed if TV viewing and physical activity are independently related to metabolic risk for children through the European Youth Heart Study which provided a cross-sectional survey from 1997 to 2000. They recruited 1,921 children aged 9-10 years and 15- 16 years from three regions in Europe. The participants self-reported daily TV viewing habits and wore an accelerometer over two weekdays and two weekend days to measure daily activity counts per minute (cpm). The clustered metabolic-risk score included skin folds, hypertension, hyperglycemia, insulin resistance, HDL cholesterol and triglycerides.

TV viewing had no significant associations with HDL cholesterol and triglycerides before and after adjusting for physical activity and adiposity in children. There was no

significant association of TV viewing with clustered metabolic risk ($p = 0.053$). The findings suggested that children may be too young to have high cholesterol due to prolonged TV watching.

In a Spanish cross-sectional analysis examined by Martinez-Gomez et al.⁶⁸, the authors tried to estimate the effect of TV viewing on CVD risk factors in 425 adolescents aged 13 to 18.5 years. This study collected self-reported TV viewing information and blood samples between 2000 and 2002. TV viewing was categorized into “low TV viewing” (≤ 3 hr/day) and “high TV viewing” (> 3 hr/day). A combined CVD risk score included triglycerides, HDL cholesterol, LDL cholesterol, and glucose levels.

The analysis showed that high TV viewers had less favorable values of HDL cholesterol and CVD risk score compared with low TV viewers. There were no significant associations of TV viewing with total cholesterol, triglycerides, and LDL cholesterol. In adolescents, an unfavorable effect of excessive TV viewing on CVD risk was observed in this study.

1.2.4.5 Prospective cohort studies

Hancox et al.⁸³ utilized a birth cohort study to assess the association of TV viewing in childhood with adult health in 1000 subjects born in New Zealand, in 1972/1973 at a follow-up until age 26 years. Parents provided the information on TV viewing weekly at ages 5, 7, 9, and 11 years. At ages 13, 15, and 21 years, subjects answered the amount of time spent watching TV on weekdays and at weekends. At age 26 years, adult health was analyzed.

The authors found moderate correlations between mean TV viewing hours at different ages. Average TV viewing at age 5-15 significantly predicted higher cholesterol at age 26 years. Additionally, TV viewing in childhood (age 5-11 years), adolescence (13-15), and young adulthood (21) were also significantly associated with raised cholesterol levels at age 26 years.

Results from population-attributable fractions showed that 15% of increased cholesterol in adulthood could be related to daily watching TV for more than 2 hours during childhood and adolescence (age 5-15 years).

This prospective analysis suggested that TV viewing habits might persist from childhood into young adulthood. Excessive TV viewing in childhood and adolescence may cause long-lasting unfavorable effects on adult health. However, only one type of measurement of adult health cannot provide complete information on health change over time. Also, information on TV viewing reported by parents might underestimate the real viewing time, which may attenuate the association.

Another prospective study conducted by Wijndaele et al.¹² assessed whether greater TV viewing time was associated with increased cardiometabolic risk through 1999/2000 and 2004/2005 measurements of the AusDiab Study. The authors found that increased TV viewing predicted an increase in clustered cardiometabolic risk score and triglycerides in women independent of diet quality and physical activity. A series of analyses in the AusDiab Study only found the TV-lipid association in women so sex variation should be taken into account for further analyses. Furthermore, country and race need to be considered as possible covariates for this association.

1.2.4.6 Summary

Cross-sectional and prospective studies both provide data that TV viewing is associated with serum cholesterol. TV viewing and physical activity may have independent effects and be differently related to cholesterol levels. The association between TV viewing and high cholesterol may be mediated by sedentary lifestyle, unhealthy diet, and adiposity, which were caused by excessive TV viewing. Older age, which is another risk factor of high cholesterol, may

explain that the association between TV viewing and cholesterol level was rarely observed in children and adolescents. Hence, it is reasonable for us to investigate cardiometabolic risk among young adults. However, there is an increasing trend of obesity in children and adolescents so more evidence linking TV viewing to serum cholesterol in those populations is needed. It is still unknown whether the effect of TV viewing on cardiometabolic risk is short-term or long-term. That is another important issue to solve in the future.

1.2.4.7 Relation of high cholesterol to CVD

High blood cholesterol is one major modifiable risk factor for CVD.⁸⁴ Importantly, the age of diagnosis of hypercholesterolemia is decreasing. Around one in five youth aged 12-19 had abnormal cholesterol levels.⁸⁵ Li et al. monitored children for 22 years to examine the longitudinal influence of LDL cholesterol on carotid vascular changes.⁸⁶ They reported that LDL cholesterol level in childhood was a good predictor of adult carotid intima-media thickness which affects poor cardiovascular condition in adolescents and cardiovascular morbidity and mortality in adults.⁸⁷ Frontini et al. indicated that high blood cholesterol in adolescence was associated with atherosclerosis in adulthood.⁸⁸ Amarenco et al. systematically reviewed epidemiological evidence to find that HDL cholesterol was inversely associated with stroke risk.⁸⁹ Additionally, a meta-regression analysis reported by Labreuche et al. showed that plasma triglyceride levels significantly predicted the risk of stroke (adjusted relative risk = 1.05 per 10 mg/dL increase; 95% CI = 1.03-1.07).⁹⁰ When other risk factors (such as hypertension) exist together, CVD risk increases even more.⁹¹ Hence, it is crucial to control cholesterol levels as early as possible.

1.2.5 High blood pressure

1.2.5.1 Definition

High blood pressure is defined as (1) mean systolic blood pressure \geq (SBP) 140 mm Hg or (2) mean diastolic blood pressure (DBP) \geq 90 mm Hg or (3) taking high blood pressure medicine.⁹²

High blood pressure is highly prevalent in the United States in which around 1 of 3 adults has high blood pressure. It raises the risk of heart disease and stroke, the first and third contributing causes of mortality in the United States.

1.2.5.2 Prevalence

In 2006, an estimated 74.5 million adults had high blood pressure (35.7 million men and 38.8 million women).⁵ Data from the CDC showed that the percentage of adults with high blood pressure was 33%.⁹³ American Africans are more likely to develop high blood pressure than Caucasians. According to the American Heart Association statistics, the number of death caused by high blood pressure was 56,561 in 2006.⁵ That is, 17.8% of all-cause death was from high blood pressure. Additionally, from 1996 to 2006, there was a 19.5% increase in high blood pressure related death rate.⁵

1.2.5.3 Relation of TV viewing to high blood pressure

Unhealthy behaviors including unfavorable diet, adiposity, and physical inactivity can put people at a raised risk of developing hypertension.⁹⁴ Evidence shows that TV viewing is associated with these unhealthy behavioral risk factors. Excessive TV viewing may be an initial trigger for high blood pressure risk. The following epidemiological evidence suggests a negative effect of TV on high blood pressure.

1.2.5.4 Cross-sectional studies

Sidney et al. reported that heavy TV viewing had no association with hypertension compared with light TV viewing in young adults aged 23 to 35 years.¹⁹

The UK study examined by Jakes et al. found that TV viewing was positively associated with diastolic blood pressure and systolic blood pressure adjusted for physical activity and BMI in adults aged 45 to 74 years. In addition, the authors suggested that reduction in TV viewing and vigorous activity had a joint-effect on decreased diastolic blood pressure. For participants who had vigorous exercise 1 hr/week and watched TV less than 2 hr/day, diastolic blood pressure was 3.6 mmHg in men and 2.7 mmHg in women lower than for those who did not have vigorous exercise and watched TV more than 4 hr/day.⁴⁹

In a Danish study, Aadahl et al. showed us that adjusting for physical activity and diet, time spent on TV viewing had a linear association with both systolic and diastolic blood pressure in 33-64 year-old adults.⁸⁰

In the 1999-2000 survey of AusDiab study, Healy et al. found a dose-response relationship of TV time with systolic blood pressure independent of physical activity and diet for physically active adults (mean age, 47 years).¹¹

Another AusDiab survey in 2004-2005 suggested that TV viewing time had a linear association with diastolic and systolic blood pressure in women. The associations became non-significant after controlling for diet, physical activity, and waist circumference. It indicated that diet, physical activity, and adiposity are important mediators between TV viewing and high blood pressure.⁸¹

To date, limited studies estimated whether children show the association of TV viewing with high blood pressure. By using data from the European Youth Heart Study, Ekelund et al.

found that TV viewing had no association with blood pressure in children of age 9-10 and 15-16 years.⁸²

1.2.5.5 Prospective cohort studies

The New Zealand cohort study conducted by Hancox et al. showed that TV viewing during childhood (age 5-11 years), adolescence (age 13-15 years), and young adulthood (age 21 years) had no association with blood pressure at age 26 years.⁸³ These null results might be caused by underestimation from parents' TV-viewing reports. Additionally, youth may be too young to develop high blood pressure.

The AusDiab 5-yr follow up study conducted by Wijndaele et al. observed that change in TV viewing time had a linear increase in diastolic blood pressure change among adults independent of diet quality (women: 0.48mmHg, 95% CI = 0.03-0.92). This linear relationship was slightly attenuated by additional adjustment for physical activity (women: 0.47mmHg, 95% CI = 0.02-0.92), and it became non-significant after additional adjustment for waist circumference (women: 0.33mmHg, 95% CI = -0.11-0.77).¹²

1.2.5.6 Summary

Most studies seem to support the harmful influence of TV viewing on blood pressure, especially in adults older than middle-age. This indicates that development of high blood pressure may be a long-term issue. Diet, physical activity, and weight play a role in this association between TV viewing and cardiometabolic risk. Intervention to reduce TV viewing time as early as possible could be effective for reduction of CVD development. However, more evidence for the effect of TV viewing on hypertension is needed.

1.2.5.7 Relation of high blood pressure to CVD

Besides high cholesterol, high blood pressure is another well-known modifiable risk factor for CVD.⁸⁴ Data from American Heart Association suggest that 77% of Americans who have a first stroke have hypertension; 69% for first heart attack; 74% for first heart failure.⁹⁵ A large cohort study in the Asia Pacific region including Australia, mainland China, Hong Kong, Japan, New Zealand, Singapore, South Korea, and Taiwan throughout 3 million person-years of follow-up indicated that reduction in systolic blood pressure was associated with lower stroke risk and lower ischaemic heart disease risk.⁹⁶ Clinical trials also demonstrated that lowering blood pressure levels could decrease risk for heart failure.⁹⁷⁻⁹⁸ To control blood pressure is an important prevention targeted to ease the world's cardiovascular burden.

1.2.6 Hostility

1.2.6.1 Definition

Anger, aggression, and hostility are highly correlated but are seldom well-distinguished. Anger has been seen as an unpleasant feeling that may be expressed as disinhibition (temper tantrums to rages) or overinhibition (silent seething or resentment), which stimulates high levels of sympathetic arousal.⁹⁹ Aggression refers to verbal and physical behaviors to attack, hurt or injure others intentionally. Hostility has been defined as a personality trait that includes a set of cognitive, emotional, and behavioral components encompassing cynicism, anger, mistrust, and aggression.¹⁰⁰ It also represents a combination of negative attitudes, beliefs, and appraisals toward others. Hostile people have a propensity to show malicious intent or feel anger toward others and a tendency to respond to frustrating situations with anger or irritation.^{28, 99} These negative attitudes and angry feelings may motivate aggression.

1.2.6.2 Relation of TV viewing with hostility

Oakley and colleagues¹⁰¹ suggest that television has become the most influential provider of information, replacing family, schools, religion, and community sources. Over the past 40 years, numerous epidemiological studies have supported the notion that TV viewing is associated with hostility and aggression.¹⁰²⁻¹⁰⁶ Among cross-sectional studies, Sidney et al. observed that TV viewing was positively associated with hostility.¹⁹ Among experimental studies, children who watched a violent program were more likely to behave in a hostile way during social interaction.³⁰⁻³² Among longitudinal studies, Johnson and colleagues mentioned a positive association between amount of time spent watching TV during adolescence and young adulthood and the likelihood of subsequent threatening aggression and assaults or fights.¹⁰⁵ This relation remained after controlling for baseline aggression along with other relevant variables.

Moreover, psychological theories proposed explanations for the association between TV viewing and increased risk of both short- and long-term aggressive and hostile behaviors.¹⁰⁷⁻¹¹⁰ Social cognitive theory suggests that children often imitate new behaviors that they witness, and thus they are more likely susceptible to high-risk behaviors if they watch more television.¹¹¹⁻¹¹² Excitation transfer theory states that media violence and high risk activities increase psychological stimulation, which causes subsequent hostile feelings and behaviors.¹¹²⁻¹¹⁴ In addition, TV violence may desensitize an individual to cruel and violent scenes.¹¹⁵⁻¹¹⁶ Rooted in these theories, prolonged TV exposure contributes to hostility. However, the relationship between TV viewing and hostility could be bidirectional because hostile people may tend to watch more TV than their counterparts.

1.2.6.3 Relation of hostility to CVD

Epidemiologic evidence suggests that hostility is a predictor of coronary atherosclerosis^{23, 117-118}, coronary artery disease^{27, 119-121}, coronary artery calcification^{25, 122}, all-cause mortality and cardiovascular mortality^{119-120, 123}. A meta-analysis of 25 studies suggested a positive effect of anger and hostility on coronary heart disease (hazard ratio [HR], 1.19; 95% CI, 1.05 to 1.35). It has also been shown that hostility is predictive of cardiovascular risk factors. Hostility is related to increase risk of physiological factors (BMI, fasting glucose, lipid levels, and blood pressure)^{27, 124-125} and behavioral factors (alcohol consumption and smoking)^{25, 124, 126}. Barefoot et al.¹¹⁹ found that hostility was positively correlated with BMI and physical activity in older Danish adults. Additionally, the authors reported that the cynicism subset of hostility was marginally associated with myocardial infarction and mortality. The hostile attribution and hostile affect subsets were significant predictors for both outcomes, while the aggressive responding subset was not. For children and adolescents, Raikkonen et al.²⁰ found that hostility was associated with BMI. This association was significant in hostile aggressive and aggressive responding subsets but not in the cynical attitudes subset. These findings may be explained by the age differences for these various aspects of hostility, in which suspicion and cynicism values tend to be higher in later life, while aggressiveness values are lower.¹²⁷ Meanwhile, Raikkonen et al. showed that hostility could predict insulin resistance (IRI) and metabolic syndrome defined as the presence of at least two of the following risk factors above the 75th percentile of their counterpart groups: BMI, IRI, ratio of TG to HDL-G, and mean arterial blood pressure. In adults, Sutin et al.¹²⁸ also indicated that angry hostility and impulsiveness were associated with metabolic syndrome defined as having at least three of the following risk factors: abdominal obesity, elevated triglycerides, HDL, blood pressure and blood glucose.

1.2.6.4 Potential mechanisms linking hostility and CVD

Smith provided a series of models to explain the relationship between hostility and health.²⁸

Certainly, the models also can unveil how hostility affects CVD.

A. Psychophysiological effect

Negative emotions affect sympathetic and parasympathetic functions of the autonomic nervous system which mediates cardiovascular and neuroendocrine responses. Hostility contributes to an increase in blood pressure, heart rate, and stress-related hormones. Further, it impairs immune functioning, increasing inflammation and thrombogenesis (i.e., exaggerated platelet reactivity, endothelial dysfunction, and aggregation of blood clot).

B. Psychosocial vulnerability effect

Given cynical and hostile characteristics, it is possible that hostile people have more interpersonal conflicts and less social supports. These interpersonal conflicts may occur at work, in their family of origin and marriage. Hostile people also tend to report severe daily hassles and increases in negative life events. This psychosocial difficulty may cause hostile people to be more vulnerable to CVD.

C. Transactional effect

The transactional model is an advanced interaction of the psychophysiological and psychosocial models. Hostile people do not just simply react to the stressors via physiological responses. Further, cognitive, emotional, and behavioral traits cause hostile people to 'create' more stressors. By tending to attribute negative intent to the actions of others, behaving aggressively to others, and mistrusting others easily, hostile people could exacerbate interpersonal conflicts and undermine the availability of social support, which leads to continuing their hostile thoughts and

behaviors. A reciprocal effect between hostile people and unfavorable social environment increases the risk of the psychosocial vulnerability.

D. Health-behavior effect

Hostile people report a tendency toward poor health habits including smoking, alcohol assumption, unhealthy diet, and less physical activity. These negative health behaviors are also important predictors of CVD. It is not surprising that hostile people have an increased risk of CVD.

**2.0 PROJECT#1: LONGITUDINAL ASSOCIATION BETWEEN TELEVISION
VIEWING AND CARDIOMETABOLIC RISK: CROSS-LAGGED ANALYSES OF TV
VIEWING AND CARDIOMETABOLIC RISK**

Manuscript in Preparation

Chung-Yu Chen¹, Mark A. Pereira², Kevin H. Kim³, Janice C. Zgibor¹, Tammy Chung⁴,
Anthony Fabio¹

¹ Department of Epidemiology, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, Pennsylvania

² Division of Epidemiology and Community Health, School of Public Health, University of Minnesota, Minneapolis, Minnesota

³ Department of Psychology, School of Education, University of Pittsburgh, Pittsburgh, Pennsylvania

⁴ Departments of Psychiatry and Epidemiology, School of Medicine, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, Pennsylvania

Address correspondence to Anthony Fabio, PhD, MPH

University of Pittsburgh, Graduate School of Public Health, Epidemiology Data Coordinating

Center, 130 DeSoto Street, 127 Parran Hall, Pittsburgh, PA 15261

Phone: 412-624-4612

Email: afabio@pitt.edu

2.1 ABSTRACT

Background: Little attention has been paid to the longitudinal association between television (TV) viewing and cardiometabolic risk in adults. Particularly, no cohort studies have been conducted for the U.S. adults.

Objective: We aimed to investigate prospective effect of TV viewing on cardiometabolic risk variables (waist circumference, HOMA insulin resistance, fasting triglycerides, HDL-cholesterol, systolic blood pressure, and clustered cardiometabolic risk score) from young to middle adulthood in U.S. adults.

Methods: We analyzed data on 3,269 participants from the Coronary Artery Risk Development in Young Adults (CARDIA) study – a prospective study of Black and White adults aged 23 to 35 years at exam year 5 in 1990-91. We used cross-lagged panel models at exam years 5, 10, 15, and 20 over 15 years to test whether TV viewing predicts cardiometabolic risk variables while adjusting for physical activity and diet quality and other potential confounders.

Results: The cross-lagged effect of TV viewing on clustered cardiometabolic risk score was significant ($B = .058$ and $.051$) except for TV viewing at year 10 to cardiometabolic risk at year 15. TV viewing was positively associated with waist circumference, HOMA insulin resistance, and systolic blood pressure, whereas negatively associated with HDL-cholesterol and triglycerides.

Conclusion: TV viewing is positively predictive of cardiometabolic risk variables from young to middle adulthood independent of physical activity and diet quality.

2.2 INTRODUCTION

Cardiovascular disease is the leading cause of death not only in the United States⁷ but also worldwide¹²⁹. Heart disease and stroke rank as the first and third cause of death in the U.S.⁷ 81 million American adults live with one or more types of cardiovascular disease, of which, 53% are estimated to be under age 60.⁵ The prevalence of metabolic syndrome is 20-40% in U.S. adults < 60 years of age.¹³⁰ According to the statistics of World Health Organization (WHO), around 80% of coronary heart disease and cerebrovascular disease are associated with these behavioral risk factors.⁶ It is crucial to investigate the behavioral risk factors of cardiovascular disease in young population to prevent the disease early.

Sedentary behaviors, such as television viewing, have been reported as one of the factors underlying the increasing prevalence of cardiometabolic risk.^{12-13 8-11, 14} Time spent engaged in TV viewing has increased over time. In 2010, Americans on average watched 5.3 hours of TV per day, suggesting that this leisure-time activity occupies a significant amount of time.² TV viewing may be the most ubiquitous recreational pastime in the United States.³³

Positive associations between TV exposure and cardiometabolic risk are consistently observed for adults in many populations including American⁸, French⁹, Australian¹⁰⁻¹², British¹³, and Flemish¹⁴. These studies also indicated that TV viewing was associated with many individual cardiometabolic risk variables such as obesity, hypertension, dyslipidaemia, and increased plasma glucose, respectively.

The association between TV viewing and cardiometabolic risk remains unclear, but may include lowering total energy expenditure due to increased sedentary time, or increasing intake of high caloric density and low nutrient foods due to snacking while watching TV or through the influence of food advertisements on TV.¹⁹ However, studies of adults have shown a positive

association between TV viewing and cardiometabolic risk independent of physical activity and diet consumption^{8-14, 19}, suggesting either residual confounding or other mechanistic pathways.

Several well-conducted cross-sectional studies have found TV viewing is an important determinant of the prevalence of metabolic syndrome.^{8-9, 11, 14} To date, only two prospective studies have reported that TV viewing is positively related to cardiometabolic risk in adults.¹²⁻¹³ One study showed that 5-year change in TV exposure predicted increasing clustered cardiometabolic risk in 3,846 Australian adults at mean age 48 years at baseline.¹² The other study reported a positive association between TV viewing frequency at age 23 years and increased cardiometabolic risk at age 44 years in 5,972 British adults, suggesting that there was a long-term longitudinal relationship between TV viewing and cardiometabolic risk.¹³ However, no studies have tested the prospective association between TV viewing and clustered cardiometabolic risk in the U.S. population. This study is also the first longitudinal study assessing repeated measures and cross-lagged effects which cannot be observed in traditional regression models. The Coronary Artery Risk Development in Young Adults (CARDIA) study is a prospective population-based study recruiting 5,115 U.S. adults aged 18 to 30 since 1985/6.¹³¹ Using this cohort, we assessed the longitudinal relationship between TV viewing and cardiometabolic risk among young U.S. adults. The specific aim was to examine the prospective association between TV viewing and clustered cardiometabolic risk. We employed cross-lagged panel models to examine autoregressive effects and cross-lagged effects in depicting a thorough picture for plausible pathways between TV viewing and cardiometabolic risk. Additionally, cross-lagged panel models with multiple repeated measures allowed us to observe whether the association between TV viewing and cardiometabolic risk was consistent from young to middle adulthood.

2.3 MATERIALS AND METHODS

Participants - CARDIA

The Coronary Artery Risk Development in Young Adults (CARDIA) Study is a prospective study designed to investigate the development and risk factors of cardiovascular disease in young adults since 1985-1986. At baseline, 5,115 Black and White young adults who were between the ages of 18 and 30 years were recruited.¹³¹ Recruitment selection at the first assessment made the study population approximately balanced for race (Blacks and Whites), gender, age (18-24 years and 25-30 years), and education (high school or less and more than high school). Participants were recruited from four geographic locations 1) Birmingham, Alabama, 2) Chicago, Illinois, 3) Minneapolis, Minnesota, and 4) Oakland, California. The same participants were followed up during 1987/8 (year 2), 1990/1 (year 5), 1992/3 (year 7), 1995/6 (year 10), 2000/1 (year 15), and 2005/6 (year 20). Participation was high, with 70% retention through year 20.¹³² Because measurements of TV viewing were collected at year 5, year 10, year 15, and year 20, this study used data at these four follow-up examinations. Participants were excluded based on these conditions: (1) pregnant women at any of exam years; (2) medication use for hypertension, hypercholesterolemia, or diabetes at year 5; (3) medication history of hypertension, hypercholesterolemia, or diabetes at year 5; (4) missing data of covariates at year 5. This study included 3,269 adults.

Measures

Television Viewing - Number of daily TV viewing hours, the primary exposure of interest for the currently proposed analyses, was assessed by a self-administered questionnaire asked at years 5, 10, 15, and 20. Participants were asked, “On average, about how many hours per day do you watch television?” We therefore had a continuous measure of self-reported hours

of TV viewing per day for each of four examinations throughout a span of 15 years. Outliers were not excluded since the results did not change significantly before or after removing outliers.

Cardiometabolic Risk - Participants were standing and dressed in light clothing without shoes for anthropometric measures, which was assessed at each CARDIA examination. Body weight was measured to the nearest 0.2 kg with a calibrated balance beam scale. Height was measured with a vertical ruler to the nearest 0.5 cm. BMI was calculated as weight in kilograms divided by height in meters squared. Waist was measured with a tape measure in duplicate to the nearest 0.5 cm around the minimal abdominal girth. Prior to each CARDIA exam participants were asked to fast for ≥ 8 hours and avoid smoking and heavy activity for two hours for blood analyses at each CARDIA examination, but fasting insulin and glucose were only measured at years 0, 7, 10, 15, and 20. Vacuum tubes containing no preservative were used to draw blood. Serum was separated by centrifugation at 4°C and stored in cryovials at -70 °C. Northwest Lipid Research Clinic Laboratory (Seattle, Wash) was used to measure serum cholesterol and triglycerides. For participants who did not fast for ≥ 8 hours prior to clinic exams, data on triglycerides, insulin and glucose were considered missing. Blood pressure was measured on the right arm using a Hawksley random zero sphygmomanometer (WA Baum Company, Copague, NY, USA) with the participant seated and following a 5-minute rest. Three measurements were taken at 1-min intervals. The second and third measurements were averaged. A continuous clustered cardiometabolic risk score was created according to a metabolic syndrome cluster score developed by Drs. Jacobs and Pereira in the CARDIA study. A similar score has been published by other studies.^{12, 133-134} Each participant was assigned a Z-score for each of the following components: waist circumference, HOMA insulin resistance (fasting glucose x fasting insulin / 22.5) (natural log), fasting triglycerides (natural log), HDL-cholesterol,

and systolic blood pressure. The Z-scores ($z = (\text{value} - \text{mean}) / \text{SD}$) were then summed within participant to create the clustered score at years 7, 10, 15, and 20. Means and SD of year 7 were used for standardization at each following exam year. The Z-score for HDL-cholesterol was assigned a negative sign. A higher Z-score meant higher cardiometabolic risk. Due to the lack of fasting glucose and insulin at year 5, the clustered cardiometabolic risk score at year 7 was a surrogate for year 5.

Covariates – All covariates were assessed by interviewer-based questionnaire at each CARDIA examination with the exception of diet. The demographic and behavioral covariates included highest level of education completed (high school or further education), family income (<24,999, 25,000-49,999, or $\geq 50,000$), alcohol use (0, 1-6, or ≥ 7 drinks/week), smoking status (never or former/current). Medication use at follow-ups was assessed by interviewer-based questionnaire for hypertension, hypercholesterolemia, or diabetes (yes/no). The continuous physical activity score was measured by intensity level and the number of months spent in 13 different activities of heavy (≥ 5 metabolic equivalents (METS)) and moderate (3-4 METS) intensity during the past year.¹³⁵ Diet was assessed at years 0, 7, and 20 using the CARDIA Diet History questionnaire.¹³⁶ The continuous dietary pattern score was assessed by types and amounts of food consumption over the past month. According to comparable nutrient characteristics and biological effects, foods were assigned into 46 groups which, in turn, were categorized as beneficial (N = 20), adverse (N = 13), and neutral (N = 13).¹³⁷ Depending on the quintiles of consumption among participants, each participant was assigned scores 0–4 for beneficial foods plus scores in reverse order 4–0 for adverse foods. We coded non-consumers of a certain type of food group as 0, and classified consumers into quartiles with scores from 1 to 4. The dietary pattern score was the sum of scores for the 46 food groups so the maximum dietary

pattern score was 132. A higher dietary pattern score represented better diet quality.¹³⁷ Assessment of diet at year 7 was a surrogate for year 5 of this study. There are several reasons to support that diet at year 7 is a more appropriate surrogate compared with diet at year 0. The dietary pattern at year 5 should be closer to year 7 than year 0. Additionally, longitudinal trends in diet for the CARDIA study participants this current study assessed indicated similar dietary patterns by matching age group at exam years 0, 7, and 20, especially from year 7 to year 20.¹³⁷ Older people remained higher diet quality compared with younger people over time. It was assumed that diet at year 7 would be a better representative of diet quality throughout this study relative to diet at year 0.

Statistical Analyses

SAS version 9.2 was used to conduct descriptive analyses and correlation matrices. The Chi-square tests were used to assess the significance of bivariate associations for categorical outcomes. One-way ANOVA tests were used to assess the significance of bivariate associations for continuous outcomes. Wilcoxon signed rank sum tests were used to examine the significance of the median difference between year 20 and year 5. Spearman correlation coefficients were performed for correlation analysis. A p-value < 0.05 was considered statistically significant. Cross-lagged panel model was specified to examine the prospective relationships between TV viewing and cardiometabolic risk variables (clustered cardiometabolic risk score waist circumference, HOMA insulin resistance, fasting triglycerides, HDL-cholesterol, and systolic blood pressure, respectively) over a total of four five-year intervals (Figure 2-1). Cross-lagged panel model was assessed using structural equation modeling (SEM) in MPlus version 6. Data may be missing due to participants dropping out of the study or due to missing data, such as a missed or insufficient blood sample or missing questionnaires. Our missing data were

demonstrated as Missing Completely at Random (MCAR) so SEM techniques in general (and MPlus specifically) can accommodate/estimate missing data using either maximum likelihood or multiple imputation.

Our model was adjusted for stable variables at baseline and time-varying variables at each exam year. A cross-lagged panel model included autoregressive effects within the same variables, cross-lagged effects for TV viewing and cardiometabolic risk to prospectively predict each other, and adjustment for covariates. This model allowed us to examine each of the cross-lagged paths individually while adjusting for important covariates. Since the scales of our variables were very different, we standardized variables to help build models. Weighted least squares means with variances adjustment (WLSMV) was used to minimize the difference between observed and implied variances and covariances. WLSMV doesn't have any distribution assumption; therefore, we did not test the normality of variables. Under this estimation, a Chi-Square test was evaluated for the null hypothesis that our model fits the data by computing the ratio of the two log-likelihoods from the observed and model-implied covariance matrices. However, the Chi-Square test is sensitive to sample size. Hence, several Goodness of Fit measures were used in the SEM analyses to assess model fit. Comparative Fit Index (CFI) $\geq .95$, Tucker-Lewis Index (TLI) $\geq .95$, and root mean squared error of approximation (RMSEA) $\leq .06$ are usually considered 'good'.¹³⁸ Modification indices were used to identify a model with good model to the data.

2.4 RESULTS

2.4.1 Participant characteristics

Table 2-1 shows the demographic and behavioral distribution of 3269 participants by TV viewing time at baseline (Year 5). Mean age was 29.9 ± 3.6 years. Younger people spent more time watching TV at baseline. People who watched more TV had lower diet scores (mean = 59.7 for the ≥ 4 hours of TV viewing per day group; mean = 72.8 for the 0 hour group) and physical activity scores (mean = 389.4 for the ≥ 4 hours group; mean = 428.9 for the 0 hour group). Males were more likely to spend more time watching TV compared with females (23% of males vs. 20.6% of females in the ≥ 4 hours group). Blacks spent more time on TV viewing than Whites (35.9% of Blacks vs. 8.6% of Whites in the ≥ 4 hours group). Most participants (81.1%) completed some education beyond high school during the CARDIA study. People who had further education than high school spent less time on TV viewing than those who had less education (40.8% of people who had less education vs. 17.4% of those who had further education than high school in the ≥ 4 hours group). People whose annual family income was $\geq 50,000$ watched less TV than those whose $< 50,000$ family income. People who smoked or drank alcohol ≥ 7 drinks/week were more likely to engage in TV viewing compared with those who never smoked or drank alcohol.

2.4.2 Distribution of TV viewing, cardiometabolic risk variables, and physical activity score by year

As Table 2-2 shows, TV viewing time decreased slightly over the 15-year span (means \pm SD = 2.5 ± 2.2 , 2.4 ± 2.0 , 2.3 ± 1.9 , and 2.4 ± 2.3 at years 5, 10, 15, and 20, respectively). Physical activity also decreased over time. Clustered cardiometabolic risk score, waist circumference, fasting triglycerides, and systolic blood pressure levels increased over time. Although fasting glucose and fasting insulin varied over time, the levels of these variables were higher at year 20 than year 5. HDL-cholesterol was assumed to decrease over time, but average HDL-cholesterol was similar at year 5 and year 20.

2.4.3 Correlations among TV viewing and cardiometabolic risk variables

As shown in Table 2-3, there were significantly positive correlations among TV viewing and clustered cardiometabolic risk score, waist circumference, HOMA insulin resistance, triglycerides, and systolic blood pressure. A significantly negative correlation was reported between TV viewing and HDL-cholesterol at year 15 ($r = -.06$, $p = .007$) and year 20 ($r = -.08$, $p \leq .001$), but TV viewing at year 5 was not significantly associated with HDL-cholesterol at year 10 ($r = -.02$, $p = .350$). The correlations among TV viewing and clustered cardiometabolic risk score, waist circumference, triglycerides, HDL-cholesterol, and systolic blood pressure became a little stronger over time. The correlations among TV viewing and triglycerides ($r = .04$, $.06$, and $.07$, respectively) and HDL-cholesterol ($r = -.02$, $-.06$, and $-.08$, respectively) were smaller than 0.1.

2.4.4 Cross-lagged panel model between TV viewing and clustered cardiometabolic risk score

The results for the model summarized in Figure 2-1 supported the prospective effect from TV viewing to cardiometabolic risk assumption and fit the data well, $\chi^2(381) = 3566.31$, CFI = .962; TLI = .950; RMSEA = .051. TV viewing exhibited significant temporal stability ($B = .640, .793$, and $.730$), as did clustered cardiometabolic risk score ($B = .834, .902$, and $.859$). In addition, the five-year lagged effect of TV viewing on clustered cardiometabolic risk score was significant and positive ($B = .058$ and $.051$) except for TV viewing at year 10 to clustered cardiometabolic risk score at year 15. In other words, an increase of 1 standardized unit of TV viewing at year 5 was associated with an increase of .058 standardized unit of clustered cardiometabolic risk score at year 10. An increase of 1 standardized unit of TV viewing at year 15 was associated with an increase of .051 standardized unit of clustered cardiometabolic risk score at year 20. The five-year lagged effect of clustered cardiometabolic risk score on TV viewing was significant, while the direction showed negative, positive, and negative over time ($B = -.208, .315$, and $-.069$); however, the inconsistent effect might imply that clustered cardiometabolic risk score is not an important predictor of TV viewing. The autoregressive effects of TV viewing and clustered cardiometabolic risk score were much stronger than the cross-lagged effects. The values of R^2 indicated that the estimated proportion of clustered cardiometabolic risk score ($R^2 = .237, .874, .819$, and $.773$) was mostly explained by the model better compared with TV viewing ($R^2 = .342, .639, .744$, and $.590$).

2.4.5 Cross-lagged effects of TV viewing on five cardiometabolic risk variables

Table 2-4 represents the differential cross-lagged effects from TV viewing to five cardiometabolic risk variables from young to middle adulthood. TV viewing time at year 5 was positively associated with waist circumference and HOMA insulin resistance at year 10. In addition, TV viewing predicted an increase in systolic blood pressure 5 year later at each examination. A negative association was observed between TV viewing and HDL-cholesterol except for a positive effect of TV viewing at year 5 on HDL-cholesterol at year 10. TV viewing time at year 10 was negatively related to triglycerides at year 15. The associations between TV viewing and HDL-cholesterol and triglycerides were not consistent and robust as our hypotheses.

2.5 DISCUSSION

Over 15 years of follow-up of a cohort of American adults aged from young to middle adulthood, we observed a significant prospective association between TV viewing and cardiometabolic risk. In particular, higher levels of TV viewing predicted an increase in clustered cardiometabolic risk score in adults from the ages of 23-35 to 28-40 and 33-45 to 38-50. However, the effects of clustered cardiometabolic risk score on TV viewing were bi-directional throughout the whole study. TV viewing was positively associated with waist circumference, HOMA insulin resistance, and systolic blood pressure, while negatively associated with HDL-cholesterol and triglycerides. The significant stability within TV viewing implies that people continued this behavior over time, which may accumulate adverse health outcomes such as cardiometabolic risk. Our data suggested that prolonged TV viewing may have been an upstream contributing risk factor toward

cardiometabolic risk independent of several potential confounders including diet and physical activity. These associations of TV viewing and clustered cardiometabolic risk score, waist circumference, and systolic blood pressure were also observed by two other longitudinal studies, although the components of clustered cardiometabolic risk score were slightly different among studies.¹²⁻¹³

Prospective associations of TV viewing on clustered cardiometabolic risk score varied over time. From year 10 to 15 (ages 28-40 to 33-45), TV viewing was not associated with clustered cardiometabolic risk score. There are several potential explanations. First, participants spent less time watching TV, on average, at these two examination years so the association of TV viewing on clustered cardiometabolic risk score might be smaller than the associations at the other examination years. Second, the association might be reduced after adjusting for the covariates. Third, it remains unclear whether the effect of TV viewing on clustered cardiometabolic risk score is short-term (≤ 5 years) or long-term (> 5 years). One British Birth Cohort study reported a positive association between TV viewing at age 23 years and adverse cardiometabolic profiles at age 44 years,¹³ suggesting that the TV viewing effect, if real, might be long-term. Hence, it is possible that our panel model with 5-year intervals may have been underpowered. Future research on the effect of time intervals of the association between TV viewing and cardiometabolic risk variables at different ages is needed.

The prospective association of clustered cardiometabolic risk score on TV viewing is still unknown. To our knowledge, there is only cross-sectional evidence supporting a positive association between TV viewing and clustered cardiometabolic risk score.^{8-9, 11, 14} Our data suggested that clustered cardiometabolic risk score may not be an important risk factor of excessive TV use. However, without adjusting for medication use, the three cross-lagged effects

of clustered cardiometabolic risk score on TV viewing were all positive and significant (data not shown). The results might imply that medication use is a “suppressor” for the effect. People who took medication might live a physically active life so their TV viewing time might reduce.

Cross-sectionally, TV viewing is associated positively with triglycerides and negatively with HDL-cholesterol.¹⁰⁻¹¹ However, it has been reported that either baseline TV watching time or 5-year change in TV viewing had no association with changes in triglycerides and HDL-cholesterol from baseline to 5 years.¹² Another study also found no associations between TV viewing frequency in early adulthood and adverse triglycerides and HDL-cholesterol profiles in middle age.¹³ In addition to our inconsistent findings and small values of correlation, TV viewing might not be an important predictor of triglycerides and HDL-cholesterol.

The positive association of TV viewing on waist circumference, HOMA insulin resistance, and systolic blood pressure may reflect the increased risk of obesity, diabetes, and hypertension induced by prolonged sedentary behaviors. The effect of TV viewing seems stronger on systolic blood pressure than on waist circumference and HOMA insulin resistance. Another CARDIA study reported an improvement in food consumption over 20 years.¹³⁷ It may imply that no adjustment for diet at each year could bias the impact of TV viewing toward the null. This may explain the absence of associations between TV viewing and waist circumference and HOMA insulin resistance after year 10. Our physical activity score did not include light-intensity physical activity which could be beneficially associated with waist circumference and clustered metabolic risk.¹³⁹ People might do light-intensity exercise during leisure time, which could reduce the impact of TV viewing on cardiometabolic risk.

Strengths of our study included the repeated measurements in a population-based cohort. This is the first prospective study to explore the association between TV viewing and

cardiometabolic risk variables in American adults from young to mid-age. Also, this is the first study to examine the autoregressive and cross-lagged effects of TV viewing and cardiometabolic risk variables with regular time intervals. Our study is also appropriate for investigating dose-response relationships. However, our study is not without limitations. The replacement of clustered cardiometabolic risk score at year 5 by year 7 could cause our data to not actually reflect years. It is possible that measurement error happened due to the self-reported behavioral variables including hours of TV viewing. We cannot rule out the possibility of residual confounding. Our population only included Black and White non-Hispanics so the results could not be generalized to other populations. The association between TV viewing and cardiometabolic risk could be biased toward the null by differential loss to follow-up.

In conclusion, our data provided longitudinal evidence that generally suggest that TV viewing leads to higher levels of clustered cardiometabolic risk score, waist circumference, HOMA insulin resistance, and systolic blood pressure independent of diet and physical activity. Our findings support the importance of reducing sedentary behaviors, especially TV viewing, in the prevention of cardiometabolic risk and, in turn, cardiovascular disease and type 2 diabetes. Further longitudinal research to explore the mechanisms between TV viewing and cardiometabolic risk variables is needed.

2.6 TABLES AND FIGURES

Table 2-1. Demographic and behavioral distribution of participants by TV viewing time at baseline (year 5)

Characteristic	Hours Spent Watching TV per day					<i>p</i> value	Total (N = 3269) N (%)
	0 (N = 328) N (%)	1 (N = 826) N (%)	2 (N = 854) N (%)	3 (N = 549) N (%)	≥ 4 (N = 712) N (%)		
Age (y)	30.4 ± 3.5 ^a	30.4 ± 3.4	29.9 ± 3.6	29.6 ± 3.8	29.2 ± 3.8	<.001 ^c	29.9 ± 3.6
Diet score	72.8 ± 12.2	71.2 ± 11.2	66.8 ± 11.7	63.7 ± 11.8	59.7 ± 10.3	<.001 ^c	66.6 ± 12.2
Physical activity score	428.9 ± 278.1	419.6 ± 293.1	422 ± 307.1	360.6 ± 297.3	319.4 ± 284.1	<.001 ^c	389.4 ± 297.2
Sex (%)							
Male	130 (8.2) ^b	419 (26.5)	412 (26.0)	257 (16.3)	364 (23.0)	.006 ^d	1582 (48.4)
Female	198 (11.7)	407 (24.1)	442 (26.2)	292 (17.3)	348 (20.6)		1687 (51.6)
Race (%)							
Black non-Hispanic	68 (4.3)	218 (13.8)	380 (24.1)	344 (21.8)	566 (35.9)	<.001 ^d	1576 (48.2)
White non-Hispanic	260 (15.4)	608 (35.9)	474 (28.0)	205 (12.1)	146 (8.6)		1693 (51.8)
Highest education (%)							
≤ 12 years	32 (5.18)	88 (14.2)	115 (18.6)	131 (21.2)	252 (40.8)	<.001 ^d	618 (18.9)
> 12 years	296 (11.2)	738 (27.8)	739 (27.9)	418 (15.8)	460 (17.4)		2651 (81.1)
Annual family income (%)							
< 24,999	139 (11.0)	213 (16.9)	293 (23.3)	208 (16.5)	406 (32.3)	<.001 ^d	1259 (38.5)
25,000-49,999	102 (8.5)	312 (26.0)	328 (27.4)	241 (20.1)	215 (18.0)		1198 (36.7)
≥ 50,000	87 (10.7)	301 (37.1)	233 (28.7)	100 (12.3)	91 (11.2)		812 (24.8)
Smoking status (%)							
Never	212 (11.4)	517 (27.7)	504 (27.0)	319 (17.1)	314 (16.8)	<.001 ^d	1866 (57.1)
Former/Current	116 (8.3)	309 (22.0)	350 (25.0)	230 (16.4)	398 (28.4)		1403 (42.9)
Alcohol use (%)							
0 (drinks/week)	164 (11.3)	350 (24.2)	391 (27.0)	255 (17.6)	288 (19.9)	<.001 ^d	1448 (44.3)
1-6	118 (10.6)	312 (28.1)	282 (25.4)	181 (16.3)	218 (19.6)		1111 (34.0)
≥ 7	46 (6.5)	164 (23.1)	181 (25.5)	113 (15.9)	206 (29.0)		710 (21.7)

^a Mean ± SD (all such values)

^b N (%) (all such values)

^c Results were tested by one-way ANOVA analysis

^d Results were tested by χ^2 test

Table 2-2. Distribution of TV viewing, cardiometabolic risk variables, and physical activity by year

Variables	Year 5	Year 10	Year 15	Year 20	year 5 vs. year 20
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	<i>p</i> value ^b
Exposure					
Hours of TV viewing daily	2.5 ± 2.2	2.4 ± 2.0	2.3 ± 1.9	2.4 ± 2.3	.434
Outcome					
Clustered cardiometabolic risk score	-.003 ± 3.5 ^a	0.5 ± 3.6	1.3 ± 3.8	2.1 ± 4.0	<.001
Waist circumference (cm)	83.5 ± 13.7	85.3 ± 14.3	89.1 ± 15.3	91.5 ± 15.3	<.001
Fasting glucose (ug/dl)	89.0 ± 13.4 ^a	87.2 ± 14.0	85.5 ± 16.5	97.2 ± 23.9	<.001
Fasting insulin (uU/ml)	13.6 ± 10.2 ^a	13.5 ± 9.5	14.1 ± 10.7	16.0 ± 10.5	<.001
Fasting triglycerides (mg/dl)	82.1 ± 70.3	88.8 ± 70.5	102.0 ± 84.5	107.5 ± 77.8	<.001
HDL-cholesterol (mg/dl)	51.8 ± 13.9	50.2 ± 13.9	50.5 ± 14.4	53.9 ± 16.4	.803
Systolic blood pressure (mmHg)	107.7 ± 11.0	109.1 ± 11.6	112 ± 13.7	115.5 ± 14.4	<.001
Covariate					
Physical activity score	389.4 ± 297.2	344.6 ± 278.8	358.7 ± 288.4	353.3 ± 281.8	<.001

^a Results were assessed at year 7 but not at year 5

^b Results were tested by Wilcoxon signed rank sum test for the median difference between year 20 and year 5

Table 2-3. Correlations between TV viewing and cardiometabolic risk variables

		Clustered10	WST 10	HOMA10 ^b	TRI 10 ^b	HDL 10	SBP 10
TV viewing 5	r ^a	.16***	.16***	.18***	.04*	-.02	.13***
	p	<.001	<.001	<.001	.033	.350	<.001
		Clustered 15	WST 15	HOMA 15 ^b	TRI 15 ^b	HDL 15	SBP 15
TV viewing 10	r	.20***	.19***	.19***	.06**	-.06**	.18***
	p	<.001	<.001	<.001	.004	.007	<.001
		Clustered 20	WST 20	HOMA 20 ^b	TRI 20 ^b	HDL 20	SBP 20
TV viewing 15	r	.21***	.20***	.18***	.07**	-.08***	.20***
	p	<.001	<.001	<.001	.001	<.001	<.001

Clustered = clustered cardiometabolic risk; WST = waist circumference; HOMA = HOMA insulin resistance; TRI = triglycerides; HDL = HDL-cholesterol; SBP = systolic blood pressure

^a Results were tested by Spearman's rank correlation test

^b Variables were log-transformed (natural log)

* $p < .05$; ** $p < .01$; *** $p < .001$

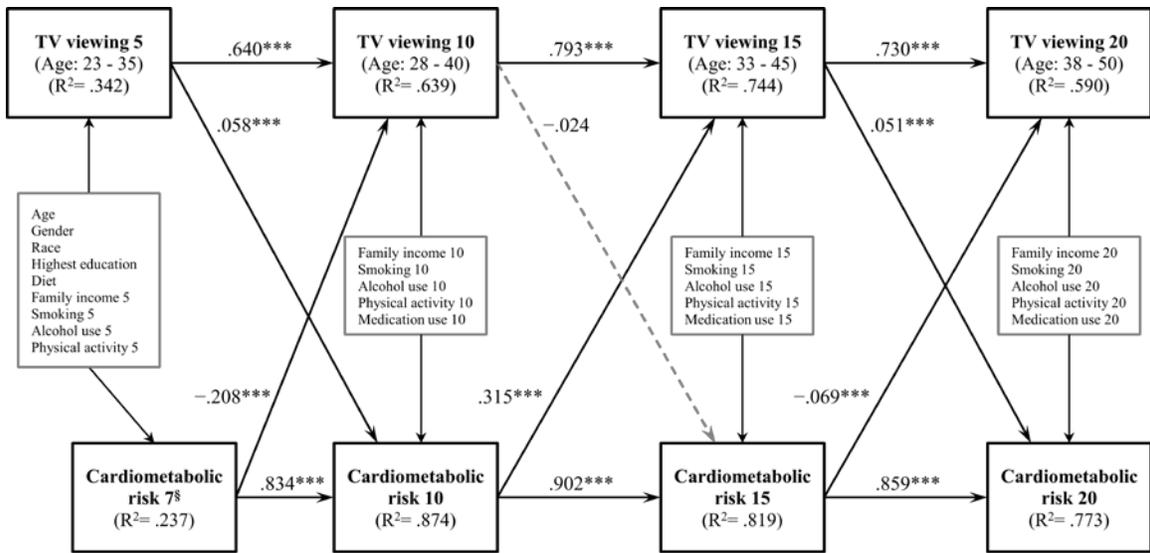


Figure 2-1. A cross-lagged panel model showing that TV viewing predicts increases in clustered cardiometabolic risk score independent of covariates. Regression weights are standardized. R² represents the estimated proportion of the assumed underlying continuous variable explained by the model.

[§] Cardiometabolic risk 7 is a surrogate of cardiometabolic risk 5

* $p < .05$; ** $p < .01$; *** $p < .001$

Table 2-4. Cross-lagged effects from TV viewing to five cardiometabolic risk variables respectively

		WST 10	HOMA 10 ^a	TRI 10 ^a	HDL 10	SBP 10
TV viewing 5	<i>B</i>	.036*	.098***	-.032	.099***	.138***
	<i>p</i>	.019	<.001	.121	<.001	<.001
		WST 15	HOMA 15 ^a	TRI 15 ^a	HDL 15	SBP 15
TV viewing 10	<i>B</i>	-.018	.006	-.079**	-.047**	.046*
	<i>p</i>	.131	.742	.001	.003	.012
		WST 20	HOMA 20 ^a	TRI 20 ^a	HDL 20	SBP 20
TV viewing 15	<i>B</i>	.002	.039	.018	-.047**	.117***
	<i>p</i>	.836	.057	.405	.001	<.001

WST = waist circumference; HOMA = HOMA insulin resistance; TRI = triglycerides; HDL = HDL-cholesterol; SBP = systolic blood pressure. All cross-lagged panel models were adjusted for the same covariates as the clustered cardiometabolic risk model. Regression weights are standardized.

^a Variables were log-transformed (natural log).

* $p < .05$; ** $p < .01$; *** $p < .001$

3.0 PROJECT#2: PROSPECTIVE CROSS-LAGGED ANALYSES OF TELEVISION VIEWING AND OBESITY IN THE CORONARY ARTERY RISK DEVELOPMENT IN YOUNG ADULTS (CARDIA) STUDY

Manuscript in Preparation

Chung-Yu Chen¹, Mark A. Pereira², Kevin H. Kim³, Janice C. Zgibor¹, Tammy Chung⁴,
Anthony Fabio¹

¹ Department of Epidemiology, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, Pennsylvania

² Division of Epidemiology and Community Health, School of Public Health, University of Minnesota, Minneapolis, Minnesota

³ Department of Psychology, School of Education, University of Pittsburgh, Pittsburgh, Pennsylvania

⁴ Departments of Psychiatry and Epidemiology, School of Medicine, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, Pennsylvania

Address correspondence to Anthony Fabio, PhD, MPH

University of Pittsburgh, Graduate School of Public Health, Epidemiology Data Coordinating

Center, 130 DeSoto Street, 127 Parran Hall, Pittsburgh, PA 15261

Phone: 412-624-4612

Email: afabio@pitt.edu

3.1 ABSTRACT

Background: The prospective association between television (TV) viewing and obesity in adults has been paid little attention.

Objective: We aimed to investigate prospective association between TV viewing and obesity using measures of body mass index (BMI) and waist circumference (WST) over 15 years of observation.

Methods: We analyzed data on 3,269 participants from the Coronary Artery Risk Development in Young Adults (CARDIA) study – a prospective study of Black and White adults aged 23 to 35 years at exam year 5 in 1990-91. We used cross-lagged panel models at exam years 5, 10, 15, and 20 over 15 years to assess the association between TV viewing and obesity while adjusting for physical activity and diet quality and other potential confounders.

Results: The cross-lagged effects of TV viewing on obesity were significant from exam year 5 to year 10 ($B = .034$ for BMI; $.036$ for WST).

Conclusion: The findings indicate that higher levels of TV viewing predicted obesity in young adulthood but not in middle adulthood.

3.2 INTRODUCTION

TV viewing is the most prevalent leisure-time sedentary activity in the United States, in which people spend, on average, 5 hours per day watching TV.^{2, 140} Emerging data suggest that prolonged TV watching is an important determinant for the increasing prevalence of obesity among different populations.^{47-49, 51-53} The underlying mechanisms include reduced overall energy expenditure due to lower energy required for TV viewing and increased overall energy intake due to the influence of food advertising and food references in TV programs.⁴⁷

Obesity remains a significant public health threat and is linked to numerous adverse chronic conditions including hypertension, hypercholesterolemia, diabetes, and coronary heart disease.^{70-72, 74-75} The prevalence of obesity in the United States has consistently increased and remained high over decades.¹⁴¹ The prevalence of overweight or obesity in children and adolescents is 16%, and increases to 66% in adults.¹⁴¹ The prevalence of overweight and obesity increase starting at adults 20 years of age.¹⁴¹ The prevalence of cardiovascular disease associated with obesity by age also consistently increases in adults aged 20 and older.⁵ The increasing trends of obesity support that examining changes in potential risk factors for obesity (including TV viewing) in young adults is needed.

Studies have shown a positive association between TV viewing and obesity in adults cross-sectionally^{47-48, 51, 57} and prospectively^{53-55, 142-143}. The magnitude of the increases in obesity risk varied across studies depending on the covariates studied and by the way in which TV viewing and obesity were measured and categorized. A 6-year prospective study conducted by Hu *et al.* reported that the positive association between TV viewing and obesity was independent of physical activity and dietary factors.⁵³ In addition, TV viewing showed a stronger significant effect compared with other sedentary behaviors including sitting at work, standing at work or

home, and brisk walking. This could be explained by a lower relative energy expenditure for TV viewing compared to other sedentary behaviors such as sewing, reading, writing, and driving a car.¹⁴⁴

Although previous studies have shown a prospective association between TV viewing and obesity, most prospective studies in adults were less than 10 years in duration. One of the prospective studies, the 1970 British birth cohort study found no association between TV viewing habits in childhood (at age 5 and 10 years) and obesity in adulthood (at age 30 years).⁵⁴ The lag effect of TV viewing on obesity could decay due to the long time interval used in that study. In addition, the 1958 British birth cohort suggested that frequency of TV viewing influenced BMI gains from adolescence (at age 11 and 16 years) to adulthood (at age 23, 33, and 45 years), but the frequency of TV viewing may not be an appropriate indicator of the effect.⁵⁵

The positive relationship between TV viewing and obesity may be mutually influenced. TV viewing may increase obesity risk, while overweight and obese people may have a propensity for sedentary behaviors like TV viewing and consumption of high caloric food compared to normal-weight people. A Spanish cross-sectional study showed a significant interaction effect of TV viewing and weight status on abdominal obesity.⁶⁸ In the overweight group, there was a significant positive association between TV viewing and waist circumference but there was no association in the normal-weight group. Nevertheless, to date, no research has examined the mutual or reciprocal influence of TV viewing and obesity to investigate the complicated relationship between TV viewing and obesity.

We assessed duration of TV viewing per day and continuous values of BMI and waist circumference (WST) in young adults over a 15-year span using cross-lagged panel models. We aimed to examine the cross-lagged associations between TV viewing and obesity in adults with

four repeated measures separated by 5-year intervals. Additionally, cross-lagged panel models with multiple repeated measures allowed us to observe whether the association between TV viewing and obesity was consistent from young to middle adulthood.

3.3 MATERIALS AND METHODS

Participants - CARDIA

The Coronary Artery Risk Development in Young Adults (CARDIA) study, which began in 1985-1986, is a prospective study designed to investigate the development and risk factors of cardiovascular disease in young adults. At baseline, 5,115 Black and White adults who were between the ages of 18 and 30 years were recruited.¹³¹ Recruitment selection at the first assessment made the study population approximately balanced for race (Blacks and Whites), gender, age (18-24 years and 25-30 years), and education (high school or less and more than high school). Participants were recruited from four geographic locations 1) Birmingham, Alabama, 2) Chicago, Illinois, 3) Minneapolis, Minnesota, and 4) Oakland, California. The same participants were followed up during 1987/8 (year 2), 1990/1 (year 5), 1992/3 (year 7), 1995/6 (year 10), 2000/1 (year 15), and 2005/6 (year 20). Participation was high, with 70% retention through year 20.¹³² Because measurements of TV viewing were collected at year 5, year 10, year 15, and year 20, this study used data at these four follow-up examinations. Participants were excluded based on these conditions: (1) pregnant women at any of exam years; (2) medication use for hypertension, hypercholesterolemia, or diabetes at year 5; (3) medication history of hypertension, hypercholesterolemia, or diabetes at year 5; (4) missing data of covariates at year 5. This study included 3,269 adults.

Measures

Television Viewing - Number of daily TV viewing hours, the primary exposure of interest for the currently proposed analyses, was assessed by a self-administered questionnaire asked at years 5, 10, 15, and 20. Participants were asked, “On average, about how many hours per day do you watch television?” We therefore had a continuous measure of self-reported hours of TV viewing per day for each of four examinations throughout a span of 15 years. Outliers were not excluded since the results did not change significantly before or after removing outliers.

Obesity - Participants were standing and dressed in light clothing without shoes for anthropometric measures, which was assessed at each CARDIA examination. Body weight was measured to the nearest 0.2 kg with a calibrated balance beam scale. Height was measured with a vertical ruler to the nearest 0.5 cm. BMI was calculated as weight in kilograms divided by height in meters squared. Waist was measured with a tape measure in duplicate to the nearest 0.5 cm around the minimal abdominal girth.

Covariates – All covariates were assessed by interviewer-based questionnaire at each CARDIA examination except diet. The demographic and behavioral covariates included highest level of education completed (high school or further education), family income (<24,999, 25,000-49,999, or \geq 50,000), alcohol use (0, 1-6, or \geq 7 drinks/week), smoking status (never or former/current). Medication use at follow-ups was assessed by interviewer-based questionnaire for hypertension, hypercholesterolemia, or diabetes (yes/no). The continuous physical activity score was measured by intensity level and the number of months spent in 13 different activities of heavy (\geq 5 metabolic equivalents (METs)) and moderate (3-4 METs) intensity during the past year.¹³⁵ Diet was assessed at years 0, 7, and 20 using the CARDIA Diet History questionnaire.¹³⁶ The continuous dietary pattern score was assessed by types and amounts of food consumption

over the past month. According to comparable nutrient characteristics and biological effects, foods were assigned into 46 groups which, in turn, were categorized as beneficial (N = 20), adverse (N = 13), and neutral (N = 13).¹³⁷ Depending on the quintiles of consumption among participants, each participant was assigned scores 0–4 for beneficial foods plus scores in reverse order 4–0 for adverse foods. We coded non-consumers of a certain type of food group as 0, and classified consumers into quartiles with scores from 1 to 4. The dietary pattern score was the sum of scores for the 46 food groups so the maximum dietary pattern score was 132. A higher dietary pattern score represented better diet quality.¹³⁷ Assessment of diet at year 7 was a surrogate for year 5 of this study. There are several reasons to support that diet at year 7 is a more appropriate surrogate compared with diet at year 0. The dietary pattern at year 5 should be closer to year 7 than year 0. Additionally, longitudinal trends in diet for the CARDIA study participants this current study assessed indicated similar dietary patterns by matching age group at exam years 0, 7, and 20, especially from year 7 to year 20.¹³⁷ Older people remained higher diet quality compared with younger people over time. It was assumed that diet at year 7 would be a better representative of diet quality throughout this study relative to diet at year 0.

Statistical Analyses

SAS version 9.2 was used to conduct descriptive analyses and correlation matrix. Chi-square test was used to assess the significance of bivariate associations for categorical outcomes. One-way ANOVA tests were used to assess the significance of bivariate associations for continuous outcomes. Wilcoxon signed rank sum tests were used to exam the significance of the median difference between year 20 and year 5. Spearman correlation coefficients were performed for correlation analysis. A p-value < 0.05 was considered statistically significant. Cross-lagged panel model was specified to examine the prospective relationships between TV viewing and obesity

(i.e., continuous values of BMI and waist circumference) over a total of four five-year intervals (Figure 3-1 and 3-2). Cross-lagged panel model was assessed using structural equation modeling (SEM) in MPlus version 6. Data may be missing due to participants dropping out of the study or due to missing data, such as a missed or insufficient BMI measures or missing questionnaires. Our missing data were demonstrated as Missing Completely at Random (MCAR) so SEM techniques in general (and MPlus specifically) can accommodate/estimate missing data using either maximum likelihood or multiple imputation.

Our model was adjusted for stable variables at baseline and time-varying variables at each exam year. A cross-lagged panel model included autoregressive effects within the same variables, cross-lagged effects for TV viewing and obesity to prospectively predict each other, and adjustment for covariates. This model allowed us to examine each of the cross-lagged paths individually while adjusting for important covariates. Weighted least squares means and variances adjusted (WLSMV) was used to minimize the difference between observed and implied variances and covariances. WLSMV doesn't have any distribution assumption; therefore, we did not test the normality of variables. Under this estimation, a Chi-Square test was evaluated for the null hypothesis that our model fits the data by computing the ratio of the two log-likelihoods from the observed and model-implied covariance matrices. However, the Chi-Square test is sensitive to sample size. Hence, several Goodness of Fit measures were used in the SEM analyses to assess model fit. Comparative Fit Index (CFI) $\geq .95$, Tucker-Lewis Index (TLI) $\geq .95$, and root mean squared error of approximation (RMSEA) $\leq .06$ are usually considered 'good'.¹³⁸ Modification indices were used to identify a model with good model to the data.

3.4 RESULTS

3.4.1 Participant characteristics

Table 3-1 shows the demographic and behavioral distribution of 3269 participants by TV viewing time at baseline (Year 5). Mean age was 29.9 ± 3.6 years. Younger people spent more time watching TV at baseline. People who watched more TV had lower diet scores (mean = 59.7 for the ≥ 4 hours group; mean = 72.8 for the 0 hour group) and physical activity scores (mean = 389.4 for the ≥ 4 hours group; mean = 428.9 for the 0 hour group). Males were more likely to spend more time watching TV compared with females (23% of males vs. 20.6% of females in the ≥ 4 hours group). Blacks spent more time on TV viewing than Whites (35.9% of Blacks vs. 8.6% of Whites in the ≥ 4 hours group). Most participants (81.1%) completed some education beyond high school during the CARDIA study. People who had further education than high school spent less time on TV viewing than those who had less education (40.8% of people who had less education vs. 17.4% of those who had further education than high school in the ≥ 4 hours group). People whose annual family income was $\geq 50,000$ watched less TV than those whose $< 50,000$ family income. People who smoked or drank alcohol ≥ 7 drinks/week were more likely to engage in TV viewing compared with those who never smoked or drank alcohol.

3.4.2 Means of TV viewing, BMI, WST, and physical activity score by year

As Table 3-2 shows, TV viewing time decreased slightly over the 15-year span (mean in hours \pm SD = 2.5 ± 2.2 , 2.4 ± 2.0 , 2.3 ± 1.9 , and 2.4 ± 2.3 at years 5, 10, 15, and 20, respectively). Physical activity also decreased over time. In contrast, BMI and WST increased over time.

3.4.3 Correlations among TV viewing and BMI and WST

Table 3-3 shows the correlations of prospective effects of TV viewing on BMI and WST 5 years later. Throughout the whole study, there were significant and positive correlations between TV viewing and obesity ($r = .18, .20, \text{ and } .22$ for BMI; $r = .16, .19, \text{ and } .20$ for WST). The correlations among TV viewing and BMI and WST slightly increased over time.

3.4.4 Cross-lagged panel model between TV viewing and BMI and WST

The results for the BMI and WST models summarized separately in Figure 3-1 and Figure 3-2 supported the prospective association of the TV viewing to obesity hypothesis and fit the data well, CFI= .964; TLI= .954; RMSEA= .050 for BMI; CFI= .963; TLI= .952; RMSEA= .050 for WST. The TV viewing exhibited significant temporal stability ($B = .637, .782, \text{ and } .720$ for BMI; $B = .628, .803, \text{ and } .718$ for WST), as did the obesity risk ($B = 1.012, .940, \text{ and } .871$ for BMI; $B = .952, .914, \text{ and } .908$ for WST). In addition, the five-year lagged effects of TV viewing at year 5 on BMI and WST at year 10 were significant ($B = .034$ and $.036$, individually), but were no longer significant after year 10. In other words, an increase of 1 standardized unit of TV viewing at year 5 was associated with an increase of .034 standardized unit of BMI at year 10. An increase of 1 standardized unit of TV viewing at year 5 was associated with an increase of .036 standardized unit of WST at year 10. This result implied that TV viewing was associated with obesity in young adulthood but not in middle adulthood. The five-year lagged effect of obesity on TV viewing was significant but showed two directions ($B = -.087, .151, \text{ and } -.061$ for BMI; $B = -.147, .210, \text{ and } -.072$ for WST). These inconsistent results imply that obesity, as indicated by BMI and WST, may be not an important determinant of TV viewing when controlling for

other factors. The values of R^2 indicated that the estimated proportion of BMI and WST was mostly explained by the model better compared with TV viewing.

3.5 DISCUSSION

Our study found a positive association between TV viewing and obesity in young adulthood, whereas this association was not observed during middle age. The direction of influence from obesity on TV viewing varied from young to middle adulthood. This is the first study to assess the reciprocal effects between TV viewing and obesity simultaneously. The stable relationship within TV viewing indicated that people continued this sedentary behavior over time. The high levels of stability within BMI and WST implied that previous obesity was strongly contributing to subsequent risk of obesity. Our data suggest that TV viewing is predictive of risk of obesity for young adults independent of several possible confounders including dietary pattern and physical activity. This finding is consistent with previous studies of the association between TV viewing and obesity using measures of BMI^{19, 51, 53, 145} and WST^{12-13, 52, 145}. In addition, the prospective effect of TV viewing on obesity appears to diminish as people age.

Interestingly, TV viewing was positively associated with subsequent obesity for young adults but not for middle-aged. This finding was unexpected and suggests that middle-aged adults may differ from young adults in how they respond to the influence of TV viewing. Although studies in young adults⁵² and middle-aged adults⁵³ indicate that TV watching is positively associated with obesity, it is plausible that young adults may be more susceptible to the effects of TV viewing on energy intake and expenditure than middle-aged adults. In the aspect of energy intake, it is possible that younger people are more likely to snack during TV

viewing and consume unhealthy food due to the seduction of TV advertising. It is also possible that people improve their dietary quality as they age because older people are more likely to care about their health and be capable of affording healthy but pricy food. One CARDIA study examining the longitudinal trends in diet found that diet quality of our CARDIA population increased with age at each exam year.¹³⁷ In addition, dietary recommendations have become more prevalent and practical for the public to follow since 1985. Compared to the 1985 US Dietary Guidelines¹⁴⁶ only encouraging more fruit and vegetables intake, the 2010 US Dietary Guidelines¹⁴⁷ provide more specific dietary pattern such as increasing consumption of fruit, vegetables, whole grains, nuts, and seeds, seafood and low-fat dairy and controlling intake of meats, poultry, and eggs. The CARDIA study also found an improved dietary quality over 20 years of observation from 1985/6 to 2005/6. These hypotheses and studies may explain our unexpected finding, suggesting that aging or secular trends may play a role in the associations between TV viewing and obesity.

With regard to energy expenditure, although one CARDIA study¹⁴⁸ assessing the longitudinal trends in physical activity and our data suggest that people decreased their levels of physical activity over time, we had no measure of light-intensity activity. Healy *et al.* found that sedentary behavior had a strong and negative correlation with light-intensity exercise.¹³⁹ Additionally, light-intensity exercise was a contributor to a reduction in obesity. It is possible that middle-aged people substitute light-intensity activity for TV viewing given that the mean TV viewing time slightly declined throughout the study. Rooted in these possibilities, TV viewing would become too minor a factor in middle-aged adults relative to other risk factors for obesity. Of course, we cannot rule out the possibility that 5-year interval is not appropriate to detect the prospective effect of TV viewing on obesity in middle-aged adults. Additionally, we

cannot rule out the role of attrition over time. Dudley *et al.* indicated that Blacks, those who are younger, less educated and smoke are more likely to drop out of a cohort study.¹⁴⁹ Our findings showed that those people are often susceptible to a less healthy lifestyle such as prolonged TV viewing. Therefore, the association between TV viewing and obesity could be biased toward the null by differential follow-up loss of those spending more time watching TV.

The prospective influence of obesity on TV viewing has not been explored. Overweight and obese people may spend more time engaging TV watching due to the less capability to move freely relative to people of normal weight. Our data suggested that obesity may not be an important risk factor of excessive TV use. However, the cross-lagged effects from obesity to TV viewing were all positive and significant without adjusting for medication use (data not shown). The results might imply that medication use is a “suppressor” for the effect. People who took medication might live a physically active life so their TV viewing time might reduce.

This study provides potentially very important public health implications. The exposure of interest, TV viewing, and the outcome, obesity, are highly prevalent in many populations around the world. Sedentary behaviors, especially TV watching, may be an important factor of the epidemic of obesity in the United States. Reducing sedentary time should be addressed in lifestyle guidelines to the public. The time-varying effect of TV viewing on obesity possibly implies that young adulthood is an important period of intervention for reducing TV viewing time. Also, our study implies that more and more people may have accepted healthy lifestyle concepts over time. Our findings showed that previous obesity was a stronger predictor of subsequent obesity than TV viewing. Hence, healthy lifestyle behaviors should start as young as possible.

Strengths of our study include repeated occasions in a population-based cohort and detailed measures of dietary quality and physical activity. This is the first prospective study to examine the autoregressive and cross-lagged effects of TV viewing and obesity with regular time interval. Our study also reported dose-response relationships. The follow-up time over 1990 to 2005 is appropriate to examine the association between TV viewing and obesity for this current society. However, our study has limitations. The lack of light activity measure may underestimate the effect of TV viewing with obesity. It is possible that measurement error happened due to the self-reported behavioral variables including hours of TV viewing. Our population only included the Black and White non-Hispanic so the results cannot be generalized to other populations. Further studies are needed to examine the association in different populations and age groups. When using categorical covariates, we cannot rule out the possibility of residual confounding (i.e., that an association might be stronger for certain categories than others).

In conclusion, our data provided longitudinal evidence that higher levels of TV viewing leads to greater risk for obesity independent of physical activity and diet. However, this prospective relationship varies with age or period. Our findings support the importance of reducing sedentary behaviors, especially TV viewing, in the prevention of obesity.

3.6 TABLES AND FIGURES

Table 3-1. Demographic and behavioral distribution of participants by TV viewing time at baseline (year 5)

Characteristic	Hours Spent Watching TV per day					<i>p</i> value	Total (N = 3269) N (%)
	0 (N = 328) N (%)	1 (N = 826) N (%)	2 (N = 854) N (%)	3 (N = 549) N (%)	≥ 4 (N = 712) N (%)		
Age (y)	30.4 ± 3.5 ^a	30.4 ± 3.4	29.9 ± 3.6	29.6 ± 3.8	29.2 ± 3.8	<.001 ^c	29.9 ± 3.6
Diet score	72.8 ± 12.2	71.2 ± 11.2	66.8 ± 11.7	63.7 ± 11.8	59.7 ± 10.3	<.001 ^c	66.6 ± 12.2
Physical activity score	428.9 ± 278.1	419.6 ± 293.1	422 ± 307.1	360.6 ± 297.3	319.4 ± 284.1	<.001 ^c	389.4 ± 297.2
Sex (%)							
Male	130 (8.2) ^b	419 (26.5)	412 (26.0)	257 (16.3)	364 (23.0)	.006 ^d	1582 (48.4)
Female	198 (11.7)	407 (24.1)	442 (26.2)	292 (17.3)	348 (20.6)		1687 (51.6)
Race (%)							
Black non-Hispanic	68 (4.3)	218 (13.8)	380 (24.1)	344 (21.8)	566 (35.9)	<.001 ^d	1576 (48.2)
White non-Hispanic	260 (15.4)	608 (35.9)	474 (28.0)	205 (12.1)	146 (8.6)		1693 (51.8)
Highest education (%)							
≤ 12 years	32 (5.18)	88 (14.2)	115 (18.6)	131 (21.2)	252 (40.8)	<.001 ^d	618 (18.9)
> 12 years	296 (11.2)	738 (27.8)	739 (27.9)	418 (15.8)	460 (17.4)		2651 (81.1)
Annual family income (%)							
< 24,999	139 (11.0)	213 (16.9)	293 (23.3)	208 (16.5)	406 (32.3)	<.001 ^d	1259 (38.5)
25,000-49,999	102 (8.5)	312 (26.0)	328 (27.4)	241 (20.1)	215 (18.0)		1198 (36.7)
≥ 50,000	87 (10.7)	301 (37.1)	233 (28.7)	100 (12.3)	91 (11.2)		812 (24.8)
Smoking status (%)							
Never	212 (11.4)	517 (27.7)	504 (27.0)	319 (17.1)	314 (16.8)	<.001 ^d	1866 (57.1)
Former/Current	116 (8.3)	309 (22.0)	350 (25.0)	230 (16.4)	398 (28.4)		1403 (42.9)
Alcohol use (%)							
0 (drinks/week)	164 (11.3)	350 (24.2)	391 (27.0)	255 (17.6)	288 (19.9)	<.001 ^d	1448 (44.3)
1-6	118 (10.6)	312 (28.1)	282 (25.4)	181 (16.3)	218 (19.6)		1111 (34.0)
≥ 7	46 (6.5)	164 (23.1)	181 (25.5)	113 (15.9)	206 (29.0)		710 (21.7)

^a Mean ± SD (all such values)

^b N (%) (all such values)

^c Results were tested by one-way ANOVA analysis

^d Results were tested by χ^2 test

Table 3-2. Means of TV viewing, body mass index, waist circumference, and physical activity score by year

Variables	Year 5	Year 10	Year 15	Year 20	year 5 vs. year 20 <i>p</i> value ^a
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	
Exposure					
Hours of TV viewing daily	2.5 ± 2.2	2.4 ± 2.0	2.3 ± 1.9	2.4 ± 2.3	.434
Outcome					
Body mass index (kg/m**2)	25.9 ± 5.7	27.2 ± 6.3	28.5 ± 6.7	29.1 ± 7.2	<.001
Waist circumference (cm)	81.5 ± 12.4	85.3 ± 14.3	89.1 ± 15.3	91.5 ± 15.3	<.001
Covariate					
Physical activity score	389.4 ± 297.2	344.6 ± 278.8	358.7 ± 288.4	353.3 ± 281.8	<.001

^a Results were tested by Wilcoxon signed rank sum test for the median difference between year 20 and year 5

Table 3-3. Correlations between TV viewing and BMI and waist circumference

		BMI 10	WST 10
TV viewing 5	<i>r</i> ^a	.18***	.16***
	<i>p</i>	<.001	<.001
		BMI 15	WST 15
TV viewing 10	<i>r</i>	.20***	.19***
	<i>p</i>	<.001	<.001
		BMI 20	WST 20
TV viewing 15	<i>r</i>	.22***	.20***
	<i>p</i>	<.001	<.001

BMI = body mass index; WST = waist circumference

^a Results were tested by Spearman's rank correlation test

* *p* < .05; ** *p* < .01; ****p* < .001

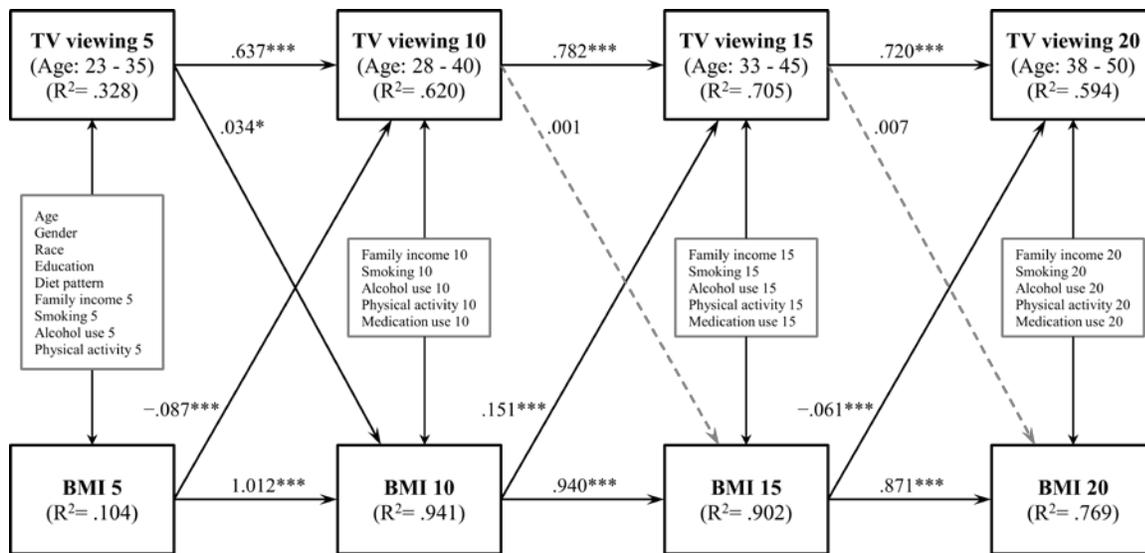


Figure 3-1. A cross-lagged panel model showing the effects of duration of TV viewing on BMI. Regression weights are standardized. R² represents the estimated proportion of the assumed underlying continuous variable explained by the model.

* $p < .05$; ** $p < .01$; *** $p < .001$

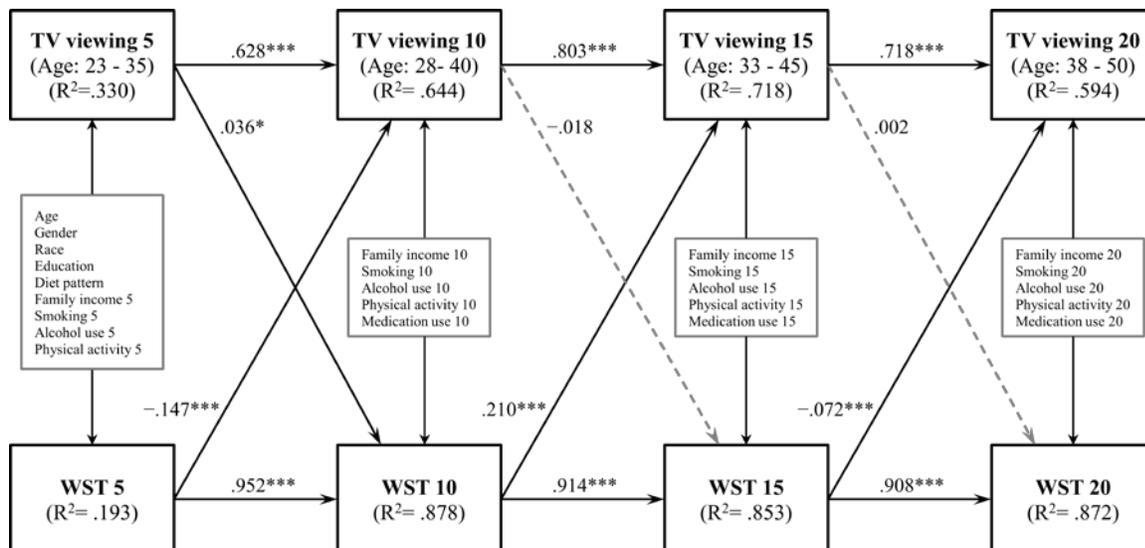


Figure 3-2. A cross-lagged panel model showing the effects of duration of TV viewing on waist circumference.

**4.0 PROJECT#3: THE INFLUENCE OF HOSTILE PERSONALITY TRAIT ON THE
RELATIONSHIP BETWEEN TELEVISION VIEWING AND CARDIOMETABOLIC
RISK**

Manuscript in Preparation

Chung-Yu Chen¹, Mark A. Pereira², Kevin H. Kim³, Janice C. Zgibor¹, Tammy Chung⁴,
Anthony Fabio¹

¹ Department of Epidemiology, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, Pennsylvania

² Division of Epidemiology and Community Health, School of Public Health, University of Minnesota, Minneapolis, Minnesota

³ Department of Psychology, School of Education, University of Pittsburgh, Pittsburgh, Pennsylvania

⁴ Departments of Psychiatry and Epidemiology, School of Medicine, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, Pennsylvania

Address correspondence to Anthony Fabio, PhD, MPH

University of Pittsburgh, Graduate School of Public Health, Epidemiology Data Coordinating

Center, 130 DeSoto Street, 127 Parran Hall, Pittsburgh, PA 15261

Phone: 412-624-4612

Email: afabio@pitt.edu

4.1 ABSTRACT

Background: Hostile people may be more susceptible to TV-induced negative emotions and harmful health habits which increase occurrence of cardiometabolic risk relative to less hostile counterparts. The associations between TV viewing and cardiometabolic risk may be stronger for those with high hostility compared to those with low levels of hostility.

Objective: We aimed to examine whether the hostile personality trait is an important modifier for the association between TV viewing on cardiometabolic risk variables (waist circumference, HOMA insulin resistance, fasting triglycerides, HDL-cholesterol, systolic blood pressure, and clustered cardiometabolic risk score).

Methods: We analyzed data on 3,269 participants from the Coronary Artery Risk Development in Young Adults (CARDIA) study – a prospective study of Black and White adults aged 23 to 35 years at exam year 5 in 1990-91. We used cross-lagged panel models stratified by levels of hostility at exam years 5, 10, 15, and 20 over 15 years to evaluate whether the prospective association between TV viewing and cardiometabolic is modified by the hostile personality traits.

Results: The cross-lagged effect of TV viewing on clustered cardiometabolic risk score was significant for those with high hostility ($B = .068$ for exam years 5 to 10; $B = .057$ for exam years 15 to 20), but not for those with low hostility. A significant difference between these two models was observed using multi-group structural equation modeling ($\chi^2(109) = 450.91, p < .001$).

Conclusion: TV viewing predicts an increase in clustered cardiometabolic risk score for people with high hostility but not for those with low hostility. Our study indicated that the psychological characteristic of hostility can modify the association between TV viewing and cardiometabolic risk.

4.2 INTRODUCTION

Over the past several decades, television (TV) viewing has emerged as a nearly ubiquitous recreational pastime in the United States.^{2, 33} Epidemiological evidence supports excessive TV viewing as a social/environmental exposure that negatively affects cardiovascular disease – the leading cause of morbidity and mortality in the United States and globally.⁵⁻⁶ There are a large number of predictive risk factors for cardiovascular disease including cardiometabolic risk. Two mechanisms may explain how TV viewing increases the risk of cardiovascular disease, including 1) lowering total energy expenditure due to increased sedentary time, and 2) increasing intake of high caloric and low nutrient density foods due to snacking while watching TV or through the influence of food advertisements on TV.¹⁹

Positive associations between excessive TV exposure and cardiometabolic risk are consistently observed in many populations including American⁸, French⁹, Australian¹⁰⁻¹², British¹³, and Flemish¹⁴. These studies also found that there is an adverse effect of TV viewing on cardiometabolic risk such as obesity, hypertension, dyslipidaemia, and increased plasma glucose, respectively.

Hostility has been reported to predict an increased risk of cardiovascular disease through a number of pathways among different populations.²⁰⁻²⁷ Hostility may contribute to cardiovascular disease through increases in blood pressure, heart rate, and stress-related hormones in response to potential stressors.²⁸ Hostility is generally believed to be a personality trait which often exists with anger, cynicism, and aggressive response.¹⁰⁰ Hostile people express anger more frequently and intensely than calm counterparts.²⁸ The contents of TV programs may evoke negative emotional responses and aggressive behaviors, especially among people with a predisposition towards hostility. For instance, data suggest that viewers react to violent TV news

with moral emotions, including anger and contempt.²⁹ Among experimental studies, children who watched a violent program were more likely to behave in a hostile way during social interaction.³⁰⁻³² Data indicate that amount of TV viewing and hostility are highly correlated, which implies that people with high hostility may spend more time watching TV than those with low hostility.¹⁹ Given that people with high hostility are more susceptible to TV-induced negative emotions and more likely to be exposed to TV violence, they may have a higher risk of CVD than agreeable people. Additionally, hostile people show a propensity toward unfavorable health behaviors including smoking, drinking problems, unhealthy diet, and less physical activity, which in turn may be influenced by TV viewing.²⁸ Thus, the associations between TV viewing and cardiometabolic risk may be stronger for those with high hostility compared to those with low levels of hostility.

It was hypothesized that the hostile personality traits may be an important effect modifier for the association between TV viewing and cardiometabolic risk. The hypothesis that the direct association between TV viewing and cardiometabolic risk may be modified by a propensity towards a hostile disposition has yet to be examined. In order to test hypotheses from this theoretical framework, we identified a rich longitudinal dataset with multiple assessments of the exposure (TV), outcomes, and various demographic and lifestyle factors that were examined as confounders. The Coronary Artery Risk Development in Young Adults (CARDIA) Study is a 25-year multi-center prospective study of cardiovascular disease risk evolution in Black and White young adults (18 – 30 years of age at baseline in 1985/6) in four U.S. metropolitan regions. We aimed to evaluate whether the association between TV viewing and clustered cardiometabolic risk is modified by hostile personality trait.

4.3 MATERIALS AND METHODS

Participants - CARDIA

The Coronary Artery Risk Development in Young Adults (CARDIA) study is a prospective study designed to investigate the development and risk factors of cardiovascular disease in young adults since 1985-1986. At baseline, 5,115 Black and White young adults who were between the ages of 18 and 30 years were recruited.¹³¹ Recruitment selection at the first assessment made the study population approximately balanced for race (Blacks and Whites), gender, age (18-24 years and 25-30 years), and education (high school or less and more than high school). Participants were recruited from four geographic locations 1) Birmingham, Alabama, 2) Chicago, Illinois, 3) Minneapolis, Minnesota, and 4) Oakland, California. The same participants were followed up during 1987/8 (year 2), 1990/1 (year 5), 1992/3 (year 7), 1995/6 (year 10), 2000/1 (year 15), and 2005/6 (year 20). Participation was high, with 70% retention through year 20.¹³² Because measurements of TV viewing were collected at year 5, year 10, year 15, and year 20, this study used data at these four follow-up examinations. Participants were excluded based on these conditions: (1) pregnant women at any of exam years; (2) medication use for hypertension, hypercholesterolemia, or diabetes at year 5; (3) medication history of hypertension, hypercholesterolemia, or diabetes at year 5; (4) missing data of covariates at year 5. This study included 3,269 adults.

Measures

Television Viewing - Number of daily TV viewing hours, the primary exposure of interest for the currently proposed analyses, was assessed by a self-administered questionnaire asked at years 5, 10, 15, and 20. Participants were asked, “On average, about how many hours per day do you watch television?” We therefore had a continuous measure of self-reported hours

of TV viewing per day for each of four examinations throughout a span of 15 years. Outliers were not excluded since the results did not change significantly before or after removing outliers.

Cardiometabolic Risk - Participants were standing and dressed in light clothing without shoes for anthropometric measures, which was assessed at each CARDIA examination. Body weight was measured to the nearest 0.2 kg with a calibrated balance beam scale. Height was measured with a vertical ruler to the nearest 0.5 cm. BMI was calculated as weight in kilograms divided by height in meters squared. Waist was measured with a tape measure in duplicate to the nearest 0.5 cm around the minimal abdominal girth. Prior to each CARDIA exam participants were asked to fast for ≥ 8 hours and avoid smoking and heavy activity for two hours for blood analyses at each CARDIA examination, but fasting insulin and glucose were only measured at years 0, 7, 10, 15, and 20. Vacuum tubes containing no preservative were used to draw blood. Serum was separated by centrifugation at 4°C and stored in cryovials at -70 °C. Northwest Lipid Research Clinic Laboratory (Seattle, Wash) was used to measure serum cholesterol and triglycerides. For participants who did not fast for ≥ 8 hours prior to clinic exams, data on triglycerides, insulin and glucose will be considered missing. Blood pressure was measured on the right arm using a Hawksley random zero sphygmomanometer (WA Baum Company, Copaigue, NY, USA) with the participant seated and following a 5-minute rest. Three measurements were taken at 1-min intervals. The second and third measurements were averaged. A continuous clustered cardiometabolic risk score was created according to a metabolic syndrome cluster score developed by Drs. Jacobs and Pereira in CARDIA study. A similar score has been published by other studies.^{12, 133-134} Each participant was assigned a Z-score for each of the following components: waist circumference, HOMA insulin resistance (fasting glucose x fasting insulin / 22.5) (natural log), fasting triglycerides (natural log), HDL-cholesterol, and

systolic blood pressure. The Z-scores ($z = (\text{value} - \text{mean}) / \text{SD}$) were then summed within participant to create the clustered score at years 7, 10, 15, and 20. Means and SD of year 7 were used for standardization at each following exam year. The Z-score for HDL-cholesterol was assigned a negative sign. A higher Z-score meant worse cardiometabolic situation. In order to make a stable cross-lagged model, the clustered cardiometabolic risk score at year 7 was a surrogate for year 5.

Hostility - Participants rated their levels of hostility by using the Cook-Medley hostility questionnaire, a 50-item scale with a true-false format at year 5 exam.¹⁰⁰ This survey can reflect participant's feelings of mistrust, anger, suspicion, and aggression. This questionnaire shows good convergent and discriminate validity.¹⁰⁰ The score ranges from 0 to 50, where higher scores represent higher levels of hostility. A median cut point was used to define high and low hostility, consistent with previously published approaches.²⁵

Covariates – All covariates were assessed by interviewer-based questionnaire at each CARDIA examination except diet. The demographic and behavioral covariates included highest level of education completed (high school or further education), family income (<24,999, 25,000-49,999, or $\geq 50,000$), alcohol use (0, 1-6, or ≥ 7 drinks/week), smoking status (never or former/current). Medication use at follow-ups was assessed by interviewer-based questionnaire for hypertension, hypercholesterolemia, or diabetes (yes/no). The continuous physical activity score was measured by intensity level and the number of months spent in 13 different activities of heavy (≥ 5 metabolic equivalents (METS)) and moderate (3-4 METS) intensity during the past year.¹³⁵ Diet was assessed at years 0, 7, and 20 using the CARDIA Diet History questionnaire.¹³⁶ The continuous dietary pattern score was assessed by types and amounts of food consumption over the past month. According to comparable nutrient characteristics and biological effects,

foods were assigned into 46 groups which, in turn, were categorized as beneficial (N = 20), adverse (N = 13), and neutral (N = 13).¹³⁷ Depending on the quintiles of consumption among participants, each participant was assigned scores 0–4 for beneficial foods plus scores in reverse order 4–0 for adverse foods. We coded non-consumers of a certain type of food group as 0, and classified consumers into quartiles with scores from 1 to 4. The dietary pattern score was the sum of scores for the 46 food groups so the maximum dietary pattern score was 132. A higher dietary pattern score represented better diet quality.¹³⁷ Assessment of diet at year 7 was a surrogate for year 5 of this study. There are several reasons to support that diet at year 7 is a more appropriate surrogate compared with diet at year 0. The dietary pattern at year 5 should be closer to year 7 than year 0. Additionally, longitudinal trends in diet for the CARDIA study participants this current study assessed indicated similar dietary patterns by matching age group at exam years 0, 7, and 20, especially from year 7 to year 20.¹³⁷ Older people remained higher diet quality compared with younger people over time. It was assumed that diet at year 7 would be a better representative of diet quality throughout this study relative to diet at year 0.

Statistical Analyses

SAS version 9.2 was used to conduct descriptive analyses and correlation matrix. The Chi-square test was used to assess the significance of bivariate associations for categorical outcomes. One-way ANOVA tests were used to assess the significance of bivariate associations for continuous outcomes. Wilcoxon signed rank sum tests were used to exam the significance of the median difference between year 20 and year 5. Spearman correlation coefficients were performed for correlation analysis. A p-value < 0.05 was considered statistically significant. Cross-lagged panel model was specified to examine hostility personality traits as a modifier of the association between TV viewing and cardiometabolic risk variables (clustered cardiometabolic risk score,

waist circumference, HOMA insulin resistance, fasting triglycerides, and systolic blood pressure, respectively) over a total of four five-year intervals (Figure 4-1). Cross-lagged panel model was assessed using structural equation modeling (SEM) in MPlus version 6. Data may be missing due to participants dropping out of the study or due to missing data, such as a missed or insufficient blood sample or missing questionnaires. Our missing data were demonstrated as Missing Completely at Random (MCAR) so SEM techniques in general (and MPlus specifically) can accommodate/estimate missing data using either maximum likelihood or multiple imputation.

Models of high and low hostility groups were adjusted for stable variables at baseline and time-varying variables at each exam year. A cross-lagged panel model included autoregressive effects within the same variables, cross-lagged effects for TV viewing and cardiometabolic risk variables to prospectively predict each other, and adjustment for covariates. This model allows us to examine each of the cross-lagged paths individually while adjusting for important covariates. Since the scales of our variables were very different, we standardized variables to help build models. Weighted least squares means and variances adjusted (WLSMV) was used to minimize the difference between observed and implied variances and covariances. WLSMV doesn't have any distribution assumption; therefore, we did not test the normality of variables. Under this estimation, a Chi-Square test was evaluated for the null hypothesis that our model fits the data by computing the ratio of the two log-likelihoods from the observed and model-implied covariance matrices. However, the Chi-Square test is sensitive to sample size. Hence, several Goodness of Fit measures were used in the SEM analyses to assess model fit. Comparative Fit Index (CFI) $\geq .95$, Tucker-Lewis Index (TLI) $\geq .95$, and root mean squared error of approximation (RMSEA) $\leq .06$ are usually considered 'good'.¹³⁸ Modification indices were used to identify a model with good model to the data. Multi-group structural equation modeling

analysis was examined to verify whether the high and low hostility models were significantly different.

4.4 RESULTS

4.4.1 Participant characteristics by hostility

Table 4-1 shows the demographic and behavioral characteristics of participants by levels of hostility at baseline. Mean age was 30.4 ± 3.5 years for people with low hostility and 29.4 ± 3.7 years for those with high hostility. The high hostility group reported lower mean age. People with high hostility had lower diet score (mean = 63.8 for the high hostility group; 69.6 for the low hostility group), but higher physical activity score compared with those with low hostility (mean = 400.4 for the high hostility group; 377.2 for the low hostility group). The high hostility group had more males (58.3% of males vs. 47.4% of females in the high hostility group) and Black non-Hispanic people (67.4% of Black vs. 39.0% of White in the high hostility group). People who had less education than high school were more likely to be hostile (72.8% of people who had less education than high school vs. 48.0% of those who had further education than high school in the high hostility group). People whose annual family income was $< 24,999$ showed higher percentage of high hostility. In the high hostile group, there was a larger proportion that smoked or drank ≥ 7 drinks/week than never smoked or drank.

4.4.2 Distribution of TV viewing, cardiometabolic risk variables, and physical activity score by hostility at each examination year

As Table 4-2 shows, the high hostility group reported higher levels of TV viewing time, clustered cardiometabolic risk score, waist circumference, fasting glucose, insulin, triglycerides, and systolic blood pressure and lower values of HDL-cholesterol at each examination year. The high hostility group showed higher physical activity score at year 5 and 10, but lower physical activity score at year 15 and 20 compared with the low hostility group.

4.4.3 Correlations among TV viewing and cardiometabolic risk variables by hostility

As shown in Table 4-3, there were significantly positive correlations among TV viewing and clustered cardiometabolic risk score, waist circumference, HOMA insulin resistance, and systolic blood pressure for both hostility groups. No significant correlations were observed between TV viewing and triglycerides and HDL-cholesterol for both groups except a negative correlation between TV viewing at year 15 to HDL-cholesterol at year 20 in the low hostility group.

4.4.4 Cross-lagged panel model between TV viewing and clustered cardiometabolic risk score by hostility

The results for the model summarized in Figure 4-1 supported the modifier assumption and fit the data well, CFI= .967; TLI= .957; RMSEA= .046 for the low hostility group; CFI= .970; TLI= .961; RMSEA= .050 for the high hostility group. For both groups, TV viewing exhibited significant temporal stability ($B=$.674, .923, and .670 for the low hostility group; $B=$.599, .716,

and .721 for high hostility group), as did clustered cardiometabolic risk score ($B = .868, .893,$ and $.859$ for the low hostility group; $B = .774, .919,$ and $.851$ for the high hostility group). The five-year lagged effect of TV viewing on clustered cardiometabolic risk score was significant for those with high hostility ($B = .068$ for exam years 5 to 10 and $.057$ for exam years 15 to 20), whereas the effect was non-significant for those with low hostility. In other words, for those with high hostility, an increase of 1 standardized unit of TV viewing at year 5 was associated with an increase of $.068$ standardized unit of clustered cardiometabolic risk score at year 10. Also, an increase of 1 standardized unit of TV viewing at year 15 was associated with an increase of $.057$ standardized unit of clustered cardiometabolic risk score at year 20. Table 4-4 reports chi-square tests for difference testing between the low and high hostility groups, suggesting whether clustered and individual effects are significantly different between these two cross-lagged panel models by hostility. A significant difference between baseline and structural invariance meant that these two overall models were significantly different ($\chi^2(109) = 450.91, p < .001$). The results of difference between structural invariance and partial structural invariance showed that all clustered and individual cross-lagged effects of TV viewing and clustered cardiometabolic risk score were significantly different except the effect of clustered cardiometabolic risk score at year 15 on TV viewing at year 20. This result suggests that hostility personality traits modify the association between TV viewing and cardiometabolic risk. The five-year lagged effect of clustered cardiometabolic risk score on TV viewing was significant but showed reverse directions over time in both hostility groups. The results demonstrate that effect of clustered cardiometabolic risk score on TV viewing is not stable, and provides only partial support for the hypotheses. Results suggest that cardiometabolic risk may not be an important risk factor for TV

viewing. For both groups, the values of R^2 indicated that the estimated proportion of cardiometabolic risk was mostly explained by the model better compared with TV viewing.

4.4.5 Cross-lagged effects of TV viewing on five cardiometabolic risk variables by hostility

Table 4-5 represents the differential cross-lagged effects from TV viewing to five cardiometabolic risk variables by hostility group from young to middle adulthood. There was no association between TV viewing and waist circumference for both groups. TV viewing time was positively associated with HOMA insulin resistance and systolic blood pressure for both groups. The values of HOMA insulin resistance and systolic blood pressure were higher in the high hostility group than the low hostility group. The association between TV viewing and HDL-cholesterol was inconsistent. TV viewing at year 5 for the low hostility group and at year 10 for the high hostility group was negatively related to triglycerides.

4.5 DISCUSSION

In this large prospective cohort of adults from young to middle adulthood, we found that the association between TV viewing and cardiometabolic risk was modified by hostility. Higher levels of TV viewing predicted an increase in clustered cardiometabolic risk score for people with high hostility but not for those with low levels of hostility. The associations between TV viewing and HOMA insulin resistance and systolic blood pressure were stronger for those with high hostility relative to those with low hostility. Our study suggests that the association between

TV viewing and cardiometabolic risk is stronger for those with high hostility relative to those with low levels of hostility. To our knowledge, this is the first study to assess the important hypothesis that the association between TV viewing and cardiometabolic risk may be modified by a propensity towards a hostile disposition.

In the high hostility group, the positive cross-lagged association observed for TV viewing and clustered cardiometabolic risk score is consistent with that observed in our previous study (paper in preparation). The positive association between TV viewing and cardiometabolic risk was also observed in several cross-sectional studies^{8-9, 11, 150-151} and two longitudinal studies¹²⁻¹³ for adults. Potential mechanisms by which excessive TV exposure contributes to cardiometabolic risk may include reduced physical activity and poor dietary quality. However, this present study indicated that the association between TV viewing and cardiometabolic risk was independent of physical activity and dietary quality, although we cannot rule out residual confounding. Another mechanism that is possible for this association is through a relative decrease in energy expenditure from increased TV watching. Metabolic rate is lower during TV watching than during other sedentary behaviors including sewing, playing board games, reading, and driving a car.¹⁴⁴ The Nurses' Health Study offers support for this hypothesis by showing that TV viewing is a stronger predictor of obesity relative to other sedentary behaviors.⁵³ Our findings that psychological attributes could be another potential mechanism through which TV viewing increases the likelihood of cardiometabolic risk add to this existing work. Two findings could support this hypothesis. The prospective association between TV viewing and clustered cardiometabolic risk score was found in hostile people who are more likely to have negative emotions induced by TV viewing. Additionally, the effect of TV viewing on systolic blood pressure was stronger for those with high hostility compared with those with low hostility.

Epidemiological studies have supported the notion that effects from TV viewing may be modified by hostility and aggression.¹⁰²⁻¹⁰⁶ Among experimental studies, children were more likely to behave in a hostile way during social interaction after watching violent programs.³⁰⁻³² Among longitudinal studies, Johnson and colleagues found that watching TV during adolescence and young adulthood increased the likelihood of subsequent threatening aggression and assaults or fights.¹⁰⁵ Psychological theories propose explanations for the association between TV viewing and increased risk of both short- and long-term hostility and aggression.¹⁰⁷⁻¹¹⁰ Social cognitive theory suggests that children often imitate behaviors from the TV programs they watch, and thus they are more likely to become aggressive and violent due to excessive media violence exposure.¹¹¹⁻¹¹² Excitation transfer theory states that media violence and high risk activities increase psychological arousal, which causes subsequent hostile feelings and behaviors.¹¹²⁻¹¹⁴ In addition, TV violence may desensitize an individual to cruel and violent scenes.¹¹⁵⁻¹¹⁶ Rooted in these theories, prolonged TV exposure contributes to a propensity towards a hostile disposition.

Numerous studies have indicated that hostility may exacerbate cardiometabolic risks such as fasting glucose and blood pressure.^{20, 27, 124-125} A meta-analysis of 25 studies suggested a positive effect of anger and hostility on coronary heart disease (hazard ratio [HR], 1.19; 95% CI, 1.05 to 1.35). It has also been shown that hostility is predictive of cardiovascular risk factors.¹⁵² Hostile people have a tendency to feel anger from frustrating situations, raising the possibility that images and messages on TV may promote stronger psychological responses and subsequent cardiometabolic risk for hostile people than for agreeable counterparts. Our findings supported the hypothesis derived from the finding that people with high hostility show a stronger association between TV viewing and clustered cardiometabolic risk score, HOMA insulin resistance, and systolic blood pressure relative to those with low levels of hostility.

There are several limitations in our study. Due to the lack of TV content data, we could not assess what kind of TV program can evoke negative emotional reactions. The replacement of cardiometabolic risk at year 5 by year 7 could underestimate the association because of more follow-up missing data over time. It is possible that measurement error happened due to the self-reported behavioral variables including hours of TV viewing. Our population only included the Black and White non-Hispanic participants so the results cannot be generalized to other populations. The association between TV viewing and cardiometabolic risk could be biased toward null by differential loss to follow-up.

This study is the first study to explore the association between TV viewing and cardiometabolic risk modified by a psychological factor. Our findings are of public health significance given that TV viewing and cardiometabolic risk are highly prevalent in the world. Additionally, future studies are needed to assess whether negative psychological effect is a potential mechanism mediating the relationship of TV viewing on cardiometabolic risk. Future studies with measures of TV programming preferences and cardiometabolic risk could specify warnings of TV content rating systems not only by age but also by personality type. Results of these studies may be very helpful in the prevention of cardiovascular disease.

4.6 TABLES AND FIGURES

Table 4-1. Demographic and behavioral distribution of participants by levels of hostility at baseline (year 5)

Characteristic	Low hostility (N = 1547)	High hostility (N = 1722)	<i>p</i> value	Total (N = 3269) N (%)
Age (y)	30.4 ± 3.5 ^a	29.4 ± 3.7	<.001 ^c	29.9 ± 3.6
Diet score	69.6 ± 11.7	63.8 ± 12	<.001 ^c	66.6 ± 12.2
Physical activity score	377.2 ± 280.8	400.4 ± 310.8	.026 ^c	389.4 ± 297.2
Sex				
Male	660 (41.7) ^b	922 (58.3)	<.001 ^d	1582 (48.4)
Female	887 (52.6)	800 (47.4)		1687 (51.6)
Race				
Black non-Hispanic	514 (32.6)	1062 (67.4)	<.001 ^d	1576 (48.2)
White non-Hispanic	1033 (61.0)	660 (39.0)		1693 (51.8)
Highest education				
≤ 12 years	168 (27.2)	450 (72.8)	<.001 ^d	618 (18.9)
> 12 years	1379 (52.0)	1272 (48.0)		2651 (81.1)
Family income (year)				
< 24,999	471 (37.4)	788 (62.6)	<.001 ^d	1259 (38.5)
25,000-49,999	577 (48.2)	621 (51.8)		1198 (36.7)
≥ 50,000	499 (61.5)	313 (38.6)		812 (24.8)
Alcohol use (drinks/week)				
0	731 (50.5)	717 (49.5)	<.001 ^d	1866 (57.1)
1-6	562 (50.6)	549 (49.4)		1403 (42.9)
≥ 7	254 (35.8)	456 (64.2)		
Smoking status				1448 (44.3)
Never	975 (52.3)	891 (47.8)	<.001 ^d	1111 (34.0)
Former/Current	572 (40.8)	831 (59.2)		710 (21.7)

^a Mean ± SD (all such values)

^b N (%) (all such values)

^c Results were tested by one-way ANOVA analysis

^d Results were tested by χ^2 test

Table 4-2. Distribution of TV viewing, cardiometabolic risk variables, and physical activity by year in the low hostility and high hostility groups

Variables	Year 5	Year 10	Year 15	Year 20	year 5 vs. year 20 <i>p</i> value ^b
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	
<u>Low hostility</u>					
Exposure					
Hours of TV viewing daily	1.9 ± 1.6	1.9 ± 1.5	1.8 ± 1.5	1.9 ± 1.6	.600
Outcome					
Clustered cardiometabolic risk score	-0.4 ± 3.4 ^a	0.04 ± 3.5	0.9 ± 3.8	1.6 ± 4.0	<.001
Waist circumference (cm)	81.9 ± 13.2	83.6 ± 13.6	87.6 ± 15.2	90.1 ± 14.9	<.001
Fasting glucose (ug/dl)	88.2 ± 10.2 ^a	86.1 ± 10.7	84.6 ± 14.2	95.1 ± 17.7	<.001
Fasting insulin (uU/ml)	12.6 ± 7.7 ^a	12.8 ± 9.5	13.5 ± 9.7	15.2 ± 9.5	<.001
Fasting triglycerides (mg/dl)	80.4 ± 76.7	85.1 ± 57.9	101.2 ± 92.3	105.9 ± 79.4	<.001
HDL-cholesterol (mg/dl)	51.9 ± 13.4	50.4 ± 13.2	51 ± 13.1	54.9 ± 16.0	.008
Systolic blood pressure (mmHg)	106.3 ± 10.5	107.8 ± 10.9	110.2 ± 12.7	113.7 ± 13.9	<.001
Covariate					
Physical activity score	377.8 ± 280.8	340.4 ± 260.3	359.2 ± 284.9	365.9 ± 285.8	.030
<u>High hostility</u>					
Exposure					
Hours of TV viewing daily	3 ± 2.4	2.8 ± 2.3	2.7 ± 2.2	2.9 ± 2.7	.145
Outcome					
Clustered cardiometabolic risk score	0.4 ± 3.5 ^a	0.9 ± 3.6	1.7 ± 3.8	2.5 ± 3.9	<.001
Waist circumference (cm)	85 ± 13.9	86.9 ± 14.8	90.6 ± 15.3	93 ± 15.5	<.001
Fasting glucose (ug/dl)	89.9 ± 15.8 ^a	88.2 ± 16.5	86.4 ± 18.4	99.2 ± 28.6	<.001
Fasting insulin (uU/ml)	14.5 ± 12.1 ^a	14.2 ± 9.5	14.7 ± 11.6	16.7 ± 11.4	<.001
Fasting triglycerides (mg/dl)	83.7 ± 63.8	92.4 ± 80.4	102.7 ± 76.2	109.2 ± 76.2	<.001
HDL-cholesterol (mg/dl)	51.6 ± 14.3	50 ± 14.6	49.9 ± 14.6	53 ± 16.7	.020
Systolic blood pressure (mmHg)	108.9 ± 11.2	110.2 ± 12.2	114.1 ± 14.4	117.2 ± 14.8	<.001
Covariate					
Physical activity score	400.4 ± 310.8	348.5 ± 295	358.2 ± 291.7	340.7 ± 277.2	<.001

^a Results were assessed at year 7 but not at year 5

^b Results were tested by Wilcoxon signed rank sum test for the median difference between year 20 and year 5

Table 4-3. Correlations between TV viewing and cardiometabolic risk variables by levels of hostility

<u>Low hostility</u>		Clustered10	WST 10	HOMA 10 ^b	TRI 10 ^b	HDL 10	SBP 10
TV viewing 5	r ^a	.14***	.16***	.19***	.02	-.04	.11***
	p	<.001	<.001	<.001	.394	.172	<.001
		Clustered 15	WST 15	HOMA 15 ^b	TRI 15 ^b	HDL 15	SBP 15
TV viewing 10	r	.22***	.21***	.22***	.08**	-.08**	.16***
	p	<.001	<.001	<.001	.008	.009	<.001
		Clustered 20	WST 20	HOMA 20 ^b	TRI 20 ^b	HDL 20	SBP 20
TV viewing 15	r	.22***	.23***	.21***	.06*	-.11***	.16***
	p	<.001	<.001	<.001	.050	<.001	<.001
<u>High hostility</u>		Clustered10	WST 10	HOMA 10 ^b	TRI 10 ^b	HDL 10	SBP 10
TV viewing 5	r	.12***	.12***	.14***	.03	.02	.11***
	p	<.001	<.001	<.001	.189	.462	<.001
		Clustered 15	WST 15	HOMA 15 ^b	TRI 15 ^b	HDL 15	SBP 15
TV viewing 10	r	.14***	.12***	.14***	.02	-.01	.15***
	p	<.001	<.001	<.001	.568	.666	<.001
		Clustered 20	WST 20	HOMA 20 ^b	TRI 20 ^b	HDL 20	SBP 20
TV viewing 15	r	.16***	.12***	.13***	.05	-.02	.20***
	p	<.001	<.001	<.001	.079	.445	<.001

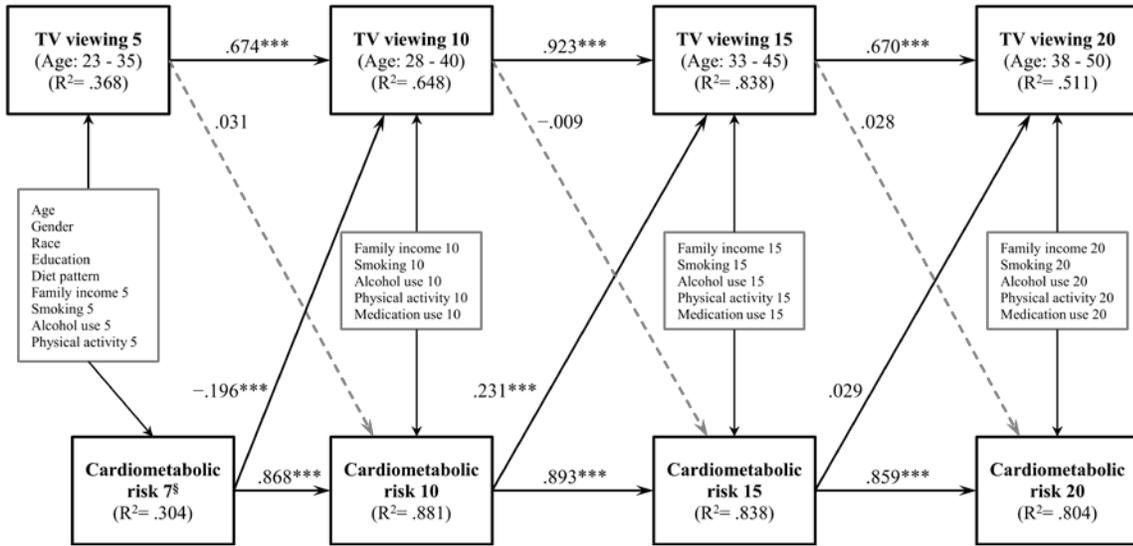
Clustered = clustered cardiometabolic risk; WST = waist circumference; HOMA = HOMA insulin resistance; TRI = triglycerides; HDL = HDL-cholesterol; SBP = systolic blood pressure.

^a Results were tested by Spearman's rank correlation test

^b Variables were log-transformed (natural log)

* $p < .05$; ** $p < .01$; *** $p < .001$

Low hostility group



High hostility group

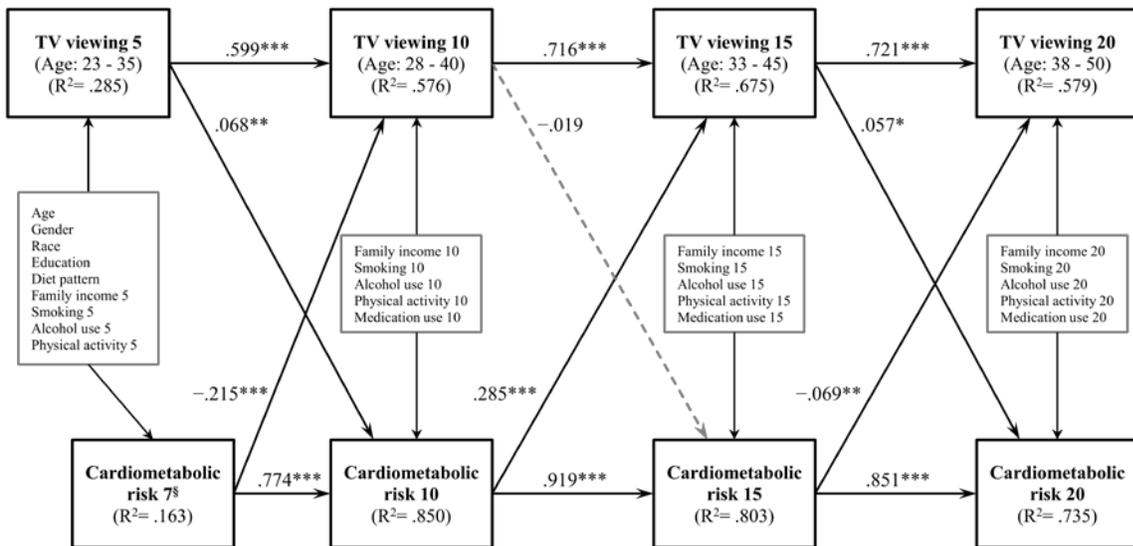


Figure 4-1. Two cross-lagged panel models showing the associations between duration of TV viewing and clustered cardiometabolic risk score are stronger for people with high hostility relative to those with low levels of hostility. Regression weights are standardized. R² represents the estimated proportion of the assumed underlying continuous variable explained by the model.

[§] Cardiometabolic risk 7 is a surrogate of cardiometabolic risk 5.

* $p < .05$; ** $p < .01$; *** $p < .001$

Table 4-4. Chi-square tests for difference testing between the low and high hostility groups

Chi-Square Test for Difference Testing	value	df ^a	<i>p</i>
Baseline vs. Structural Invariance	450.91	109	<.001
Structural Invariance vs. Partial Structural Invariance			
6 cross-lagged effects	27.99	6	<.001
3 effects of TV on Cardiometabolic risk	21.26	3	<.001
TV 5 → Cardiometabolic risk 10	8.72	1	.003
TV 10 → Cardiometabolic risk 15	15.21	1	<.001
TV 15 → Cardiometabolic risk 20	8.99	1	.003
Cardiometabolic risk 7 → TV 10	4.84	1	.028
Cardiometabolic risk 10 → TV 15	6.9	1	.009
Cardiometabolic risk 15 → TV 20	1.37	1	.241

^a df = degree of freedom

Table 4-5. Cross-lagged effects from TV viewing to five cardiometabolic risk variables by levels of hostility

<u>Low hostility</u>		WST 10	HOMA 10 ^a	TRI 10 ^a	HDL 10	SBP 10
TV viewing 5	<i>B</i>	.009	.046*	-.079**	.075**	.119***
	<i>p</i>	.633	.027	.005	.002	<.001
		WST 15	HOMA 15 ^a	TRI 15 ^a	HDL 15	SBP 15
TV viewing 10	<i>B</i>	.025	.055**	-.051	.000	.037
	<i>p</i>	.135	.009	.082	.997	.096
		WST 20	HOMA 20 ^a	TRI 20 ^a	HDL 20	SBP 20
TV viewing 15	<i>B</i>	.005	.024	-.008	-.056**	.053*
	<i>p</i>	.746	.249	.778	.006	.024
<u>High hostility</u>		WST 10	HOMA 10 ^a	TRI 10 ^a	HDL 10	SBP 10
TV viewing 5	<i>B</i>	.035	.064**	-.02	.065**	.177***
	<i>p</i>	.165	.001	.484	.001	<.001
		WST 15	HOMA 15 ^a	TRI 15 ^a	HDL 15	SBP 15
TV viewing 10	<i>B</i>	-.032	.002	-.064*	-.086***	-.006
	<i>p</i>	.085	.950	.034	<.001	.854
		WST 20	HOMA 20 ^a	TRI 20 ^a	HDL 20	SBP 20
TV viewing 15	<i>B</i>	-.006	.04	.02	-.034	.160***
	<i>p</i>	.746	.676	.511	.074	<.001

WST = waist circumference; HOMA = HOMA insulin resistance; TRI = triglycerides; HDL = HDL-cholesterol; SBP = systolic blood pressure. All cross-lagged panel models were adjusted for the same covariates as the clustered cardiometabolic risk model. Regression weights are standardized.

^a Variables were log-transformed (natural log).

* $p < .05$; ** $p < .01$; *** $p < .001$

5.0 DISCUSSION AND CONCLUSION

5.1 SUMMARY

The estimated average time of TV viewing is five hours per day, occupying one fifth of a day in our daily life. This demonstrates the importance of examining the effects of TV exposure. Studies indicate that TV viewing is related to numerous adverse chronic health conditions such as obesity and cardiovascular disease. In developed countries, chronic diseases and obesity have been considered as top public health threats due to unbalanced food consumption and prolonged sedentary behaviors. Obesity, diabetes and cardiovascular disease are the leading causes of morbidity and mortality and they often occur together.

Regular physical activity and good dietary habits are important but not enough to fully prevent the development of cardiovascular disease. Our study found that TV viewing predicted an increase in subsequent cardiometabolic risk and obesity independent of several important confounders including physical activity and dietary quality. That is, reducing sedentary behaviors, particularly TV viewing, is important for improving health.

5.2 PROSPECTIVE ASSOCIATION BETWEEN TV VIEWING AND CARDIOMETABOLIC RISK

In the first project, TV viewing was predictive of clustered cardiometabolic risk score from young to middle adulthood in the U.S. population. This impact of TV viewing on cardiometabolic risk varies with age, given that the positive association was observed only at the ages of 23-35 to 28 -40 and 33-45 to 38-50. At the ages of 40 and older, some other physiological factors may have a stronger effect on cardiometabolic risk than TV viewing, and hence, it would wash out the effect of TV viewing.

TV viewing was also a predictor of waist circumference, insulin resistance, and systolic blood pressure. In particular, higher levels of TV viewing consistently predicted the risk of high blood pressure which can be caused by psychological stimuli. It implies that TV viewing may lead to cardiovascular disease through increasing moral emotions and feelings.

5.3 PROSPECTIVE ASSOCIATION BETWEEN TV VIEWING AND OBESITY

The second project showed a cross-lagged of TV viewing on obesity in young adulthood but not in middle adulthood. The association between TV viewing and obesity varied with age, suggesting that young adults are more susceptible to TV exposure compared with middle-age adults. It is possible that middle-age adults improve dietary quality because of their concern about health conditions more than young people. Additionally, there might be a period effect to decrease the influence of TV viewing on obesity. Today, the US Dietary Guidelines have become more practical and easier to apply in daily life.¹³⁷ Follow-up loss and lack of adjustment

of light-intensity activity might bias our results toward the null. A positive association between TV viewing and obesity suggests that TV viewing is a determinant of obesity.

5.4 THE IMPACT OF HOSTILITY ON THE ASSOCIATION BETWEEN TV VIEWING AND CARDIOMETABOLIC RISK

Our third project indicated that hostility moderated the association between TV viewing and cardiometabolic risk. A prospective effect of TV viewing on clustered cardiometabolic risk score was observed in people with high hostility but not in those with low levels of hostility. Additionally, the effect of TV viewing on systolic blood pressure was stronger for those with high hostility compared with those with low hostility. Rooted in these findings, psychological attributes such as strong reactivity to anger arousing TV content could be another potential mechanism underlying the association between TV viewing and cardiometabolic risk.

5.5 PUBLIC HEALTH SIGNIFICANCE

TV viewing and cardiometabolic risk are highly prevalent in our society. As such, even a modest association between the exposure and outcomes could have high public health significance. Psychological characteristics could be one potential factor to modify and mediate the association between TV viewing and cardiometabolic risk, especially blood pressure. Besides energy intake and energy expenditure, our data suggest that psychological attributes could be another potential mechanism through which TV viewing increases the likelihood of cardiometabolic risk. TV

programming and commercials are rich in messages and scenes which may promote harmful behaviors, including alcohol, cigarettes, poor nutritional choices, and violence. Reduction in TV viewing time is a straightforward strategy to decrease the risk of CVD. Restrictions on harmful behaviors and unhealthy food promotions on TV programming and commercials by public policies could be another strategy. Reducing TV viewing time, especially for people who reported high viewing time and with a propensity towards a hostile disposition, could reduce cardiometabolic risk.

5.6 LIMITATIONS OF THIS STUDY

There are some limitations in our study. Self-reported behavioral variables such as hours of TV viewing could lead to measurement error. We expect the magnitude of exposure misclassification to be consistent across levels of cardiometabolic risk (non-differential misclassification) such that any association that we observe between TV viewing and cardiometabolic risk will likely be underestimated (bias toward the null). Additionally, this longitudinal study could have some bias caused by differential loss to follow-up. We expect that most of the missing participants are people with higher levels of poor life behaviors because they are likely to have difficulty attending interviews such that the follow-up loss could bias our data toward the null. The lack of light activity measure may underestimate the effect of TV viewing with cardiometabolic risk. Future studies assessing all types of physical activity are needed.

5.7 FUTURE STUDIES

This is the first study to examine the prospective relationship between TV viewing and cardiometabolic risk variables in the U.S. population. Our findings suggest that TV viewing is associated with adverse cardiometabolic risk profiles. Moreover, TV viewing increases the risk of obesity in young adulthood. This is also the first study to assess whether the relation between TV viewing and cardiometabolic risk is modified by hostile personality trait. Our results show a stronger association between TV viewing and cardiometabolic risk for those with high hostility relative to those with low levels of hostility. The next step is to better examine whether psychosocial attributes mediate the prospective association between TV viewing and cardiometabolic risk using. Additionally, experimental studies examining whether reducing TV viewing may impact psychosocial characteristics and lifestyle habits would be essential to addressing whether any associations observed in the present observational study may be truly causal.

Violent TV programs are associated with hostile and aggressive behaviors.¹⁰²⁻¹⁰⁶ As such, future studies are needed to address specific TV viewing habits, especially programming preferences. Studies with detailed measures of TV contents may provide information to build specific warnings of TV content rating systems not only by age but also by personality traits. Reduction in TV viewing time and TV content rating systems may reduce people from cardiometabolic risk, especially among those who are high in hostility.

APPENDIX

SUPPLEMENTAL TABLES AND FIGURES

Table A-1. Coefficients and *p* values for individual path in the cross-lagged panel model of TV viewing and cardiometabolic risk for the whole population (N = 3,269)

Path	<i>B</i>	<i>p</i>
TV viewing 5		
← Age	.014	.374
← Gender	-.006	.786
← Race	-.316	<.001
← Education	-.103	<.001
← Diet pattern	-.248	<.001
← Family income 5	-.066	.001
← Smoking 5	.075	<.001
← Alcohol use 5	.183	<.001
← Physical activity 5	-.106	<.001
Cardiometabolic risk 7		
← Age	.087	<.001
← Gender	-.437	<.001
← Race	-.072	.002
← Education	.002	.922
← Diet pattern	-.135	<.001
← Family income 5	-.071	.020
← Smoking 5	.055	.030
← Alcohol use 5	-.108	<.001
← Physical activity 5	-.136	<.001

Table A-1 continued

TV viewing 10

← TV viewing 5	.640	<.001
← Cardiometabolic risk 7	-.208	<.001
← Family income 10	-.192	<.001
← Smoking 10	.051	.024
← Alcohol use 10	.020	.329
← Physical activity 10	.067	.001
← Medication 10	.374	<.001

Cardiometabolic risk 10

← TV viewing 5	.058	<.001
← Cardiometabolic risk 7	.834	<.001
← Family income 10	.080	<.001
← Smoking 10	.007	.708
← Alcohol use 10	-.023	.195
← Physical activity 10	.064	<.001
← Medication 10	.180	<.001

TV viewing 15

← TV viewing 10	.793	<.001
← Cardiometabolic risk 10	.315	<.001
← Family income 15	-.186	<.001
← Smoking 15	-.074	.002
← Alcohol use 15	.033	.153
← Physical activity 15	-.095	<.001
← Medication 15	-.471	<.001

Cardiometabolic risk 15

← TV viewing 10	-.024	.101
← Cardiometabolic risk 10	.902	<.001
← Family income 15	-.036	.046
← Smoking 15	.022	.202
← Alcohol use 15	-.027	.102
← Physical activity 15	-.041	.008
← Medication 15	-.003	.877

Table A-1 continued

TV viewing 20

← TV viewing 15	.730	<.001
← Cardiometabolic risk 15	-.069	<.001
← Family income 20	-.045	.018
← Smoking 20	.068	.001
← Alcohol use 20	-.004	.840
← Physical activity 20	-.026	.094
← Medication 20	.166	<.001

Cardiometabolic risk 20

← TV viewing 15	.051	<.001
← Cardiometabolic risk 15	.859	<.001
← Family income 20	.011	.575
← Smoking 20	-.017	.315
← Alcohol use 20	-.002	.925
← Physical activity 20	.007	.624
← Medication 20	.028	.193

Table A-2. Coefficients and *p* values for individual path in the cross-lagged panel model of TV viewing and BMI for the whole population (N = 3,269)

	Path	<i>B</i>	<i>p</i>
TV viewing 5			
	← Age	.010	.526
	← Gender	-.017	.396
	← Race	-.304	<.001
	← Education	-.101	<.001
	← Diet pattern	-.235	<.001
	← Family income 5	-.080	<.001
	← Smoking 5	.084	<.001
	← Alcohol use 5	.157	<.001
	← Physical activity 5	-.093	<.001
BMI 5			
	← Age	.064	.001
	← Gender	-.056	.008
	← Race	-.208	<.001
	← Education	.020	.343
	← Diet pattern	-.063	.001
	← Family income 5	-.031	.313
	← Smoking 5	.023	.379
	← Alcohol use 5	-.095	.001
	← Physical activity 5	-.115	<.001
TV viewing 10			
	← TV viewing 5	.637	<.001
	← BMI 5	-.087	<.001
	← Family income 10	-.202	<.001
	← Smoking 10	.036	.089
	← Alcohol use 10	.026	.185
	← Physical activity 10	-.012	.495
	← Medication 10	.256	<.001
BMI 10			
	← TV viewing 5	.034	.023
	← BMI 5	1.012	<.001
	← Family income 10	.025	.146
	← Smoking 10	.010	.522
	← Alcohol use 10	-.043	.004
	← Physical activity 10	.035	.011
	← Medication 10	-.108	<.001

Table A-2 continued

TV viewing 15			
	← TV viewing 10	.782	<.001
	← BMI 10	.151	<.001
	← Family income 15	-.158	<.001
	← Smoking 15	-.063	.001
	← Alcohol use 15	.056	.005
	← Physical activity 15	.007	.641
	← Medication 15	-.221	<.001
BMI 15			
	← TV viewing 10	.001	.917
	← BMI 10	.940	<.001
	← Family income 15	-.003	.818
	← Smoking 15	-.003	.809
	← Alcohol use 15	-.033	.007
	← Physical activity 15	-.025	.027
	← Medication 15	.003	.847
TV viewing 20			
	← TV viewing 15	.720	<.001
	← BMI 15	-.061	<.001
	← Family income 20	-.061	.001
	← Smoking 20	.064	.001
	← Alcohol use 20	-.010	.606
	← Physical activity 20	-.060	<.001
	← Medication 20	.137	<.001
BMI 20			
	← TV viewing 15	.007	.471
	← BMI 15	.871	<.001
	← Family income 20	.003	.825
	← Smoking 20	-.010	.508
	← Alcohol use 20	-.050	<.001
	← Physical activity 20	-.059	<.001
	← Medication 20	-.042	.006

Table A-3. Coefficients and *p* values for individual path in the cross-lagged panel model of TV viewing and waist circumference (WST) for the whole population (N = 3,269)

	Path	<i>B</i>	<i>p</i>
TV viewing 5			
	← Age	.011	.493
	← Gender	-.007	.745
	← Race	-.308	<.001
	← Education	-.100	<.001
	← Diet pattern	-.238	<.001
	← Family income 5	-.077	<.001
	← Smoking 5	.077	<.001
	← Alcohol use 5	.174	<.001
	← Physical activity 5	-.093	<.001
WST 5			
	← Age	.096	<.001
	← Gender	-.400	<.001
	← Race	-.104	<.001
	← Education	.029	.172
	← Diet pattern	-.096	<.001
	← Family income 5	-.062	.043
	← Smoking 5	.035	.169
	← Alcohol use 5	-.053	.046
	← Physical activity 5	-.148	<.001
TV viewing 10			
	← TV viewing 5	.628	<.001
	← WST 5	-.147	<.001
	← Family income 10	-.209	<.001
	← Smoking 10	.038	.076
	← Alcohol use 10	.016	.387
	← Physical activity 10	-.015	.390
	← Medication 10	.338	<.001
WST 10			
	← TV viewing 5	.036	.019
	← WST 5	.952	<.001
	← Family income 10	.021	.223
	← Smoking 10	-.009	.581
	← Alcohol use 10	-.026	.074
	← Physical activity 10	.027	.051
	← Medication 10	-.034	.063

Table A-3 continued

TV viewing 15			
← TV viewing 10	.803	<.001	
← WST 10	.210	<.001	
← Family income 15	-.144	<.001	
← Smoking 15	-.056	.004	
← Alcohol use 15	.040	.040	
← Physical activity 15	.004	.801	
← Medication 15	-.323	<.001	
WST 15			
← TV viewing 10	-.018	.131	
← WST 10	.914	<.001	
← Family income 15	-.010	.509	
← Smoking 15	.014	.363	
← Alcohol use 15	-.024	.075	
← Physical activity 15	-.040	.003	
← Medication 15	.019	.290	
TV viewing 20			
← TV viewing 15	.718	<.001	
← WST 15	-.072	<.001	
← Family income 20	-.064	<.001	
← Smoking 20	.061	.002	
← Alcohol use 20	-.007	.708	
← Physical activity 20	-.062	<.001	
← Medication 20	.171	<.001	
WST 20			
← TV viewing 15	.002	.836	
← WST 15	.908	<.001	
← Family income 20	-.001	.931	
← Smoking 20	-.020	.195	
← Alcohol use 20	-.020	.149	
← Physical activity 20	-.019	.115	
← Medication 20	.062	<.001	

Table A-4. Coefficients and *p* values for individual path in the cross-lagged panel model of TV viewing and cardiometabolic risk for the low hostility group (N = 1,547)

	Path	<i>B</i>	<i>p</i>
TV viewing 5			
	← Age	-.011	.640
	← Gender	.010	.738
	← Race	-.407	<.001
	← Education	-.121	<.001
	← Diet pattern	-.288	<.001
	← Family income 5	.031	.382
	← Smoking 5	.146	<.001
	← Alcohol use 5	.096	.004
	← Physical activity 5	-.091	.001
Cardiometabolic risk 7			
	← Age	.046	.082
	← Gender	-.512	<.001
	← Race	-.108	.001
	← Education	-.025	.368
	← Diet pattern	-.118	<.001
	← Family income 5	-.031	.429
	← Smoking 5	.103	.004
	← Alcohol use 5	-.105	.003
	← Physical activity 5	-.145	<.001
TV viewing 10			
	← TV viewing 5	.674	<.001
	← Cardiometabolic risk 7	-.196	<.001
	← Family income 10	-.139	<.001
	← Smoking 10	-.020	.559
	← Alcohol use 10	.105	.001
	← Physical activity 10	.038	.240
	← Medication 10	.438	<.001
Cardiometabolic risk 10			
	← TV viewing 5	.031	.143
	← Cardiometabolic risk 7	.868	<.001
	← Family income 10	.033	.196
	← Smoking 10	-.041	.121
	← Alcohol use 10	.011	.647
	← Physical activity 10	.037	.120
	← Medication 10	.123	.004

Table A-4 continued

TV viewing 15		
← TV viewing 10	.923	<.001
← Cardiometabolic risk 10	.231	<.001
← Family income 15	-.099	.002
← Smoking 15	-.051	.118
← Alcohol use 15	-.049	.097
← Physical activity 15	-.059	.034
← Medication 15	-.375	<.001
Cardiometabolic risk 15		
← TV viewing 10	-.009	.631
← Cardiometabolic risk 10	.893	<.001
← Family income 15	-.051	.045
← Smoking 15	.038	.098
← Alcohol use 15	-.005	.822
← Physical activity 15	-.048	.022
← Medication 15	.024	.500
TV viewing 20		
← TV viewing 15	.670	<.001
← Cardiometabolic risk 15	.029	.226
← Family income 20	-.064	.005
← Smoking 20	.034	.228
← Alcohol use 20	-.051	.049
← Physical activity 20	-.018	.392
← Medication 20	.047	.116
Cardiometabolic risk 20		
← TV viewing 15	.028	.119
← Cardiometabolic risk 15	.859	<.001
← Family income 20	.021	.401
← Smoking 20	-.041	.075
← Alcohol use 20	.021	.305
← Physical activity 20	.002	.933
← Medication 20	.071	.029

Table A-5. Coefficients and *p* values for individual path in the cross-lagged panel model of TV viewing and cardiometabolic risk for the high hostility group (N = 1,722)

	Path	<i>B</i>	<i>p</i>
TV viewing 5			
	← Age	.043	.076
	← Gender	-.029	.370
	← Race	-.235	<.001
	← Education	-.061	.005
	← Diet pattern	-.223	<.001
	← Family income 5	-.143	<.001
	← Smoking 5	.042	.098
	← Alcohol use 5	.204	<.001
	← Physical activity 5	-.108	<.001
Cardiometabolic risk 7			
	← Age	.128	<.001
	← Gender	-.353	<.001
	← Race	-.033	.281
	← Education	.012	.701
	← Diet pattern	-.141	<.001
	← Family income 5	-.084	.046
	← Smoking 5	.016	.644
	← Alcohol use 5	-.106	.013
	← Physical activity 5	-.117	.002
TV viewing 10			
	← TV viewing 5	.599	<.001
	← Cardiometabolic risk 7	-.215	<.001
	← Family income 10	-.212	<.001
	← Smoking 10	.085	.005
	← Alcohol use 10	-.017	.578
	← Physical activity 10	.060	.033
	← Medication 10	.322	<.001
Cardiometabolic risk 10			
	← TV viewing 5	.068	.002
	← Cardiometabolic risk 7	.774	<.001
	← Family income 10	.097	<.001
	← Smoking 10	.045	.100
	← Alcohol use 10	-.043	.093
	← Physical activity 10	.079	.001
	← Medication 10	.250	<.001

Table A-5 continued

TV viewing 15			
	← TV viewing 10	.716	<.001
	← Cardiometabolic risk 10	.285	<.001
	← Family income 15	-.227	<.001
	← Smoking 15	-.080	.009
	← Alcohol use 15	.096	.003
	← Physical activity 15	-.069	.011
	← Medication 15	-.396	<.001
Cardiometabolic risk 15			
	← TV viewing 10	-.019	.408
	← Cardiometabolic risk 10	.919	<.001
	← Family income 15	-.020	.429
	← Smoking 15	.004	.877
	← Alcohol use 15	-.043	.082
	← Physical activity 15	-.032	.185
	← Medication 15	-.049	.145
TV viewing 20			
	← TV viewing 15	.721	<.001
	← Cardiometabolic risk 15	-.069	.002
	← Family income 20	-.037	.193
	← Smoking 20	.070	.007
	← Alcohol use 20	.006	.814
	← Physical activity 20	-.047	.038
	← Medication 20	.152	<.001
Cardiometabolic risk 20			
	← TV viewing 15	.057	.013
	← Cardiometabolic risk 15	.851	<.001
	← Family income 20	.010	.721
	← Smoking 20	.002	.940
	← Alcohol use 20	-.030	.248
	← Physical activity 20	.009	.683
	← Medication 20	-.007	.808

BIBLIOGRAPHY

1. U.S. Census Bureau "Facts for Features, Conversion from Analog to Digital-TV". 2008; http://www.census.gov/newsroom/releases/archives/facts_for_features_special_editions/cb08-ffse03.html.
2. The Nielson Company, "Television, Internet and Mobile Usage in the U.S.," Three Screen Report, Volume 8, 1st Quarter. 2010.
3. Television Bureau of Advertising, Inc., "TV Basics". 2011; <http://www.tvb.org/trends/95487>.
4. Nielsen A. *Nielson report on television* Northbrook, IL1987.
5. American Heart Association. *Heart Disease & Stroke Statistics 2010 Update AT-A-Glance*.
6. World Health Organization. Cardiovascular diseases (CVDs). 2009; <http://www.who.int/mediacentre/factsheets/fs317/en/index.html>.
7. *Centers for Disease Control and Prevention. National Vital Statistics Reports 2010*.
8. Ford ES, Kohl HW, 3rd, Mokdad AH, Ajani UA. Sedentary behavior, physical activity, and the metabolic syndrome among U.S. adults. *Obes Res*. Mar 2005;13(3):608-614.
9. Bertrais S, Beyeme-Ondoua JP, Czernichow S, Galan P, Hercberg S, Oppert JM. Sedentary behaviors, physical activity, and metabolic syndrome in middle-aged French subjects. *Obes Res*. May 2005;13(5):936-944.
10. Dunstan DW, Salmon J, Owen N, et al. Associations of TV viewing and physical activity with the metabolic syndrome in Australian adults. *Diabetologia*. Nov 2005;48(11):2254-2261.
11. Healy GN, Dunstan DW, Salmon J, Shaw JE, Zimmet PZ, Owen N. Television time and continuous metabolic risk in physically active adults. *Med Sci Sports Exerc*. Apr 2008;40(4):639-645.
12. Wijndaele K, Healy GN, Dunstan DW, et al. Increased cardiometabolic risk is associated with increased TV viewing time. *Med Sci Sports Exerc*. Aug 2010;42(8):1511-1518.
13. Stamatakis E, Hamer M, Mishra GD. Early adulthood television viewing and cardiometabolic risk profiles in early middle age: results from a population, prospective cohort study. *Diabetologia*. Feb 2012;55(2):311-320.
14. Wijndaele K, Duvigneaud N, Matton L, et al. Sedentary behaviour, physical activity and a continuous metabolic syndrome risk score in adults. *Eur J Clin Nutr*. Mar 2009;63(3):421-429.
15. Powell LM, Szczycka G, Chaloupka FJ. Adolescent exposure to food advertising on television. *Am J Prev Med*. Oct 2007;33(4 Suppl):S251-256.
16. Madden PA, Grube JW. The frequency and nature of alcohol and tobacco advertising in televised sports, 1990 through 1992. *Am J Public Health*. Feb 1994;84(2):297-299.

17. Grube JW, Wallack L. Television beer advertising and drinking knowledge, beliefs, and intentions among schoolchildren. *Am J Public Health*. Feb 1994;84(2):254-259.
18. Strasburger VC. Children, adolescents, and television--1989: II. The role of pediatricians. *Pediatrics*. Mar 1989;83(3):446-448.
19. Sidney S, Sternfeld B, Haskell WL, Jacobs DR, Jr., Chesney MA, Hulley SB. Television viewing and cardiovascular risk factors in young adults: the CARDIA study. *Ann Epidemiol*. Mar 1996;6(2):154-159.
20. Raikonen K, Matthews KA, Salomon K. Hostility predicts metabolic syndrome risk factors in children and adolescents. *Health Psychol*. May 2003;22(3):279-286.
21. Raikonen K, Matthews KA, Kuller LH, Reiber C, Bunker CH. Anger, hostility, and visceral adipose tissue in healthy postmenopausal women. *Metabolism*. Sep 1999;48(9):1146-1151.
22. Raikonen K, Matthews KA, Flory JD, Owens JF. Effects of hostility on ambulatory blood pressure and mood during daily living in healthy adults. *Health Psychol*. Jan 1999;18(1):44-53.
23. Matthews KA, Owens JF, Kuller LH, Sutton-Tyrrell K, Jansen-McWilliams L. Are hostility and anxiety associated with carotid atherosclerosis in healthy postmenopausal women? *Psychosom Med*. Sep-Oct 1998;60(5):633-638.
24. Markovitz JH, Matthews KA, Kiss J, Smitherman TC. Effects of hostility on platelet reactivity to psychological stress in coronary heart disease patients and in healthy controls. *Psychosom Med*. Mar-Apr 1996;58(2):143-149.
25. Iribarren C, Sidney S, Bild DE, et al. Association of hostility with coronary artery calcification in young adults: the CARDIA study. *Coronary Artery Risk Development in Young Adults*. *Jama*. May 17 2000;283(19):2546-2551.
26. Eaker ED, Sullivan LM, Kelly-Hayes M, D'Agostino RB, Sr., Benjamin EJ. Anger and hostility predict the development of atrial fibrillation in men in the Framingham Offspring Study. *Circulation*. Mar 16 2004;109(10):1267-1271.
27. Chaput LA, Adams SH, Simon JA, et al. Hostility predicts recurrent events among postmenopausal women with coronary heart disease. *Am J Epidemiol*. Dec 15 2002;156(12):1092-1099.
28. Smith TW. Hostility and health: current status of a psychosomatic hypothesis. *Health Psychol*. 1992;11(3):139-150.
29. Dagmar Unz FS, Peter Winterhoff-Spurk. TV News - The Daily Horror? *Journal of Media Psychology: Theories, Methods, and Applications*. 2008;20(4):141-155.
30. Geen RG. Some effects of observing violence upon the behavior of the observer. *Prog Exp Pers Res*. 1978;8:49-92.
31. Geen RG. Behavioral and physiological reactions to observed violence: effects of prior exposure to aggressive stimuli. *J Pers Soc Psychol*. May 1981;40(5):868-875.
32. Geen RG, Berkowitz L. Some conditions facilitating the occurrence of aggression after the observation of violence. *J Pers*. Dec 1967;35(4):566-576.
33. *American Time Use Survey-2009 Results* United States Department of Labor;2009.
34. Powell LM, Szczyepka G, Chaloupka FJ. Trends in exposure to television food advertisements among children and adolescents in the United States. *Arch Pediatr Adolesc Med*. Sep 2010;164(9):794-802.
35. Tirodkar MA, Jain A. Food messages on African American television shows. *Am J Public Health*. Mar 2003;93(3):439-441.

36. Henderson VR, Kelly B. Food advertising in the age of obesity: content analysis of food advertising on general market and african american television. *J Nutr Educ Behav*. Jul-Aug 2005;37(4):191-196.
37. DuRant RH, Rome ES, Rich M, Allred E, Emans SJ, Woods ER. Tobacco and alcohol use behaviors portrayed in music videos: a content analysis. *Am J Public Health*. Jul 1997;87(7):1131-1135.
38. DuRant RH, Rich M, Emans SJ, Rome ES, Allred E, Woods ER. Violence and weapon carrying in music videos. A content analysis. *Arch Pediatr Adolesc Med*. May 1997;151(5):443-448.
39. Anderson CR. Television commercial violence during nonviolent programming: the 1998 major league baseball playoffs. *Pediatrics*. Oct 2000;106(4):E46.
40. Tamburro RF, Gordon PL, D'Apolito JP, Howard SC. Unsafe and violent behavior in commercials aired during televised major sporting events. *Pediatrics*. Dec 2004;114(6):e694-698.
41. Media education. American Academy of Pediatrics. Committee on Public Education. *Pediatrics*. Aug 1999;104(2 Pt 1):341-343.
42. Cardiovascular Disease Types. 2008; <http://heart-disease.emedtv.com/cardiovascular-disease/cardiovascular-disease-types.html>.
43. Brunzell JD, Davidson M, Furberg CD, et al. Lipoprotein management in patients with cardiometabolic risk: consensus statement from the American Diabetes Association and the American College of Cardiology Foundation. *Diabetes Care*. Apr 2008;31(4):811-822.
44. Centers for Disease Control and Prevention. About BMI for Adults. http://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/index.html.
45. World Health Organization. *Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee, 1995. WHO Technical Report Series 854.*
46. Ford ES, Li C, Zhao G, Tsai J. Trends in obesity and abdominal obesity among adults in the United States from 1999-2008. *Int J Obes (Lond)*. Sep 7 2010.
47. Dietz WH, Jr., Gortmaker SL. Do we fatten our children at the television set? Obesity and television viewing in children and adolescents. *Pediatrics*. May 1985;75(5):807-812.
48. Vioque J, Torres A, Quiles J. Time spent watching television, sleep duration and obesity in adults living in Valencia, Spain. *Int J Obes Relat Metab Disord*. Dec 2000;24(12):1683-1688.
49. Jakes RW, Day NE, Khaw KT, et al. Television viewing and low participation in vigorous recreation are independently associated with obesity and markers of cardiovascular disease risk: EPIC-Norfolk population-based study. *Eur J Clin Nutr*. Sep 2003;57(9):1089-1096.
50. Parsons TJ, Power C, Manor O. Physical activity, television viewing and body mass index: a cross-sectional analysis from childhood to adulthood in the 1958 British cohort. *Int J Obes (Lond)*. Oct 2005;29(10):1212-1221.
51. Bowman SA. Television-viewing characteristics of adults: correlations to eating practices and overweight and health status. *Prev Chronic Dis*. Apr 2006;3(2):A38.
52. Cleland VJ, Schmidt MD, Dwyer T, Venn AJ. Television viewing and abdominal obesity in young adults: is the association mediated by food and beverage consumption during viewing time or reduced leisure-time physical activity? *Am J Clin Nutr*. May 2008;87(5):1148-1155.

53. Hu FB, Li TY, Colditz GA, Willett WC, Manson JE. Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. *JAMA*. Apr 9 2003;289(14):1785-1791.
54. Viner RM, Cole TJ. Television viewing in early childhood predicts adult body mass index. *J Pediatr*. Oct 2005;147(4):429-435.
55. Parsons TJ, Manor O, Power C. Television viewing and obesity: a prospective study in the 1958 British birth cohort. *Eur J Clin Nutr*. Dec 2008;62(12):1355-1363.
56. Marshall SJ, Biddle SJ, Gorely T, Cameron N, Murdey I. Relationships between media use, body fatness and physical activity in children and youth: a meta-analysis. *Int J Obes Relat Metab Disord*. Oct 2004;28(10):1238-1246.
57. Fitzgerald SJ, Kriska AM, Pereira MA, de Courten MP. Associations among physical activity, television watching, and obesity in adult Pima Indians. *Med Sci Sports Exerc*. Jul 1997;29(7):910-915.
58. Tucker LA. Television viewing and physical fitness in adults. *Res Q Exerc Sport*. Dec 1990;61(4):315-320.
59. Tucker LA. The relationship of television viewing to physical fitness and obesity. *Adolescence*. Winter 1986;21(84):797-806.
60. Eisenmann JC, Bartee RT, Wang MQ. Physical activity, TV viewing, and weight in U.S. youth: 1999 Youth Risk Behavior Survey. *Obes Res*. May 2002;10(5):379-385.
61. Schneider M, Dunton GF, Cooper DM. Media use and obesity in adolescent females. *Obesity (Silver Spring)*. Sep 2007;15(9):2328-2335.
62. Samdal O, Tynjala J, Roberts C, Sallis JF, Villberg J, Wold B. Trends in vigorous physical activity and TV watching of adolescents from 1986 to 2002 in seven European Countries. *Eur J Public Health*. Jun 2007;17(3):242-248.
63. Burke V, Beilin LJ, Durkin K, Stritzke WG, Houghton S, Cameron CA. Television, computer use, physical activity, diet and fatness in Australian adolescents. *Int J Pediatr Obes*. 2006;1(4):248-255.
64. Klesges RC, Shelton ML, Klesges LM. Effects of television on metabolic rate: potential implications for childhood obesity. *Pediatrics*. Feb 1993;91(2):281-286.
65. Utter J, Neumark-Sztainer D, Jeffery R, Story M. Couch potatoes or french fries: are sedentary behaviors associated with body mass index, physical activity, and dietary behaviors among adolescents? *J Am Diet Assoc*. Oct 2003;103(10):1298-1305.
66. Salmon J, Campbell KJ, Crawford DA. Television viewing habits associated with obesity risk factors: a survey of Melbourne schoolchildren. *Med J Aust*. Jan 16 2006;184(2):64-67.
67. Coon KA, Goldberg J, Rogers BL, Tucker KL. Relationships between use of television during meals and children's food consumption patterns. *Pediatrics*. Jan 2001;107(1):E7.
68. Martinez-Gomez D, Rey-Lopez JP, Chillon P, et al. Excessive TV viewing and cardiovascular disease risk factors in adolescents. The AVENA cross-sectional study. *BMC Public Health*. 2010;10:274.
69. Villafranco JE, Lustigman AB. Regulation of dietary supplement advertising: current claims of interest to the Federal Trade Commission, Food and Drug Administration and National Advertising Division. *Food Drug Law J*. 2007;62(4):709-725.
70. Wilson PW, D'Agostino RB, Sullivan L, Parise H, Kannel WB. Overweight and obesity as determinants of cardiovascular risk: the Framingham experience. *Arch Intern Med*. Sep 9 2002;162(16):1867-1872.

71. Field AE, Coakley EH, Must A, et al. Impact of overweight on the risk of developing common chronic diseases during a 10-year period. *Arch Intern Med.* Jul 9 2001;161(13):1581-1586.
72. Bogers RP, Bemelmans WJ, Hoogenveen RT, et al. Association of overweight with increased risk of coronary heart disease partly independent of blood pressure and cholesterol levels: a meta-analysis of 21 cohort studies including more than 300 000 persons. *Arch Intern Med.* Sep 10 2007;167(16):1720-1728.
73. Daniels SR, Morrison JA, Sprecher DL, Khoury P, Kimball TR. Association of body fat distribution and cardiovascular risk factors in children and adolescents. *Circulation.* Feb 2 1999;99(4):541-545.
74. Haarbo J, Hassager C, Schlemmer A, Christiansen C. Influence of smoking, body fat distribution, and alcohol consumption on serum lipids, lipoproteins, and apolipoproteins in early postmenopausal women. *Atherosclerosis.* Oct 1990;84(2-3):239-244.
75. Haarbo J, Hassager C, Riis BJ, Christiansen C. Relation of body fat distribution to serum lipids and lipoproteins in elderly women. *Atherosclerosis.* Nov 1989;80(1):57-62.
76. Grundy SM. Obesity, metabolic syndrome, and cardiovascular disease. *J Clin Endocrinol Metab.* Jun 2004;89(6):2595-2600.
77. Centers for Disease Control and Prevention. Cholesterol <http://www.cdc.gov/cholesterol/index.htm>.
78. American Heart Association. What Your Cholesterol Levels Mean. http://www.heart.org/HEARTORG/Conditions/Cholesterol/AboutCholesterol/What-Your-Cholesterol-Levels-Mean_UCM_305562_Article.jsp.
79. Fung TT, Hu FB, Yu J, et al. Leisure-time physical activity, television watching, and plasma biomarkers of obesity and cardiovascular disease risk. *Am J Epidemiol.* Dec 15 2000;152(12):1171-1178.
80. Aadahl M, Kjaer M, Jorgensen T. Influence of time spent on TV viewing and vigorous intensity physical activity on cardiovascular biomarkers. The Inter 99 study. *Eur J Cardiovasc Prev Rehabil.* Oct 2007;14(5):660-665.
81. Thorp AA, Healy GN, Owen N, et al. Deleterious associations of sitting time and television viewing time with cardiometabolic risk biomarkers: Australian Diabetes, Obesity and Lifestyle (AusDiab) study 2004-2005. *Diabetes Care.* Feb 2010;33(2):327-334.
82. Ekelund U, Brage S, Froberg K, et al. TV viewing and physical activity are independently associated with metabolic risk in children: the European Youth Heart Study. *PLoS Med.* Dec 2006;3(12):e488.
83. Hancox RJ, Milne BJ, Poulton R. Association between child and adolescent television viewing and adult health: a longitudinal birth cohort study. *Lancet.* Jul 17-23 2004;364(9430):257-262.
84. Ford ES, Mokdad AH, Ajani UA. Trends in risk factors for cardiovascular disease among children and adolescents in the United States. *Pediatrics.* Dec 2004;114(6):1534-1544.
85. Prevalence of abnormal lipid levels among youths --- United States, 1999-2006. *MMWR Morb Mortal Wkly Rep.* Jan 22 2010;59(2):29-33.
86. Li S, Chen W, Srinivasan SR, et al. Childhood cardiovascular risk factors and carotid vascular changes in adulthood: the Bogalusa Heart Study. *JAMA.* Nov 5 2003;290(17):2271-2276.

87. Oren A, Vos LE, Uiterwaal CS, Gorissen WH, Grobbee DE, Bots ML. Change in body mass index from adolescence to young adulthood and increased carotid intima-media thickness at 28 years of age: the Atherosclerosis Risk in Young Adults study. *Int J Obes Relat Metab Disord*. Nov 2003;27(11):1383-1390.
88. Frontini MG, Srinivasan SR, Xu J, Tang R, Bond MG, Berenson GS. Usefulness of childhood non-high density lipoprotein cholesterol levels versus other lipoprotein measures in predicting adult subclinical atherosclerosis: the Bogalusa Heart Study. *Pediatrics*. May 2008;121(5):924-929.
89. Amarenco P, Labreuche J, Touboul PJ. High-density lipoprotein-cholesterol and risk of stroke and carotid atherosclerosis: a systematic review. *Atherosclerosis*. Feb 2008;196(2):489-496.
90. Labreuche J, Deplanque D, Touboul PJ, Bruckert E, Amarenco P. Association between change in plasma triglyceride levels and risk of stroke and carotid atherosclerosis: systematic review and meta-regression analysis. *Atherosclerosis*. Sep 2010;212(1):9-15.
91. American Heart Association. Why Cholesterol Matters. 2011; http://www.heart.org/HEARTORG/Conditions/Cholesterol/WhyCholesterolMatters/Why-Cholesterol-Matters_UCM_001212_Article.jsp.
92. National Health and Nutrition Examination Survey, CDC, NCHS. Healthy People 2010 Operational Definition.
93. Centers for Disease Control and Prevention. Hypertension. <http://www.cdc.gov/nchs/fastats/hyprtens.htm>.
94. Centers for Disease Control and Prevention. High Blood Pressure. <http://www.cdc.gov/bloodpressure/about.htm>.
95. American Heart Association. Why Blood Pressure Matters. 2011; http://www.heart.org/HEARTORG/Conditions/HighBloodPressure/WhyBloodPressureMatters/Why-Blood-Pressure-Matters_UCM_002051_Article.jsp.
96. Lawes CM, Rodgers A, Bennett DA, et al. Blood pressure and cardiovascular disease in the Asia Pacific region. *J Hypertens*. Apr 2003;21(4):707-716.
97. Prevention of stroke by antihypertensive drug treatment in older persons with isolated systolic hypertension. Final results of the Systolic Hypertension in the Elderly Program (SHEP). SHEP Cooperative Research Group. *JAMA*. Jun 26 1991;265(24):3255-3264.
98. Dahlof B, Lindholm LH, Hansson L, Schersten B, Ekbom T, Wester PO. Morbidity and mortality in the Swedish Trial in Old Patients with Hypertension (STOP-Hypertension). *Lancet*. Nov 23 1991;338(8778):1281-1285.
99. Dornelas. EA. *Psychotherapy with cardiac patients : behavioral cardiology in practice* Washington, DC : American Psychological Association, c2008; 2008.
100. Barefoot JC, Dodge KA, Peterson BL, Dahlstrom WG, Williams RB, Jr. The Cook-Medley hostility scale: item content and ability to predict survival. *Psychosom Med*. Jan-Feb 1989;51(1):46-57.
101. Oakley A, Bendelow G, Barnes J, Buchanan M, Husain OA. Health and cancer prevention: knowledge and beliefs of children and young people. *BMJ*. Apr 22 1995;310(6986):1029-1033.
102. Centerwall BS. Television and violence. The scale of the problem and where to go from here. *JAMA*. Jun 10 1992;267(22):3059-3063.
103. Christakis DA, Zimmerman FJ. Violent television viewing during preschool is associated with antisocial behavior during school age. *Pediatrics*. Nov 2007;120(5):993-999.

104. Huesmann LR, Moise-Titus J, Podolski CL, Eron LD. Longitudinal relations between children's exposure to TV violence and their aggressive and violent behavior in young adulthood: 1977-1992. *Dev Psychol.* Mar 2003;39(2):201-221.
105. Johnson JG, Cohen P, Smailes EM, Kasen S, Brook JS. Television viewing and aggressive behavior during adolescence and adulthood. *Science.* Mar 29 2002;295(5564):2468-2471.
106. Singer MI, Miller DB, Guo S, Flannery DJ, Frierson T, Slovak K. Contributors to violent behavior among elementary and middle school children. *Pediatrics.* Oct 1999;104(4 Pt 1):878-884.
107. Bandura A. *Social learning theory.* NJ: Prentice Hall: Englewood cliffs; 1977.
108. Berkowitz. *Aggression: Its causes, consequences, and control.* New York: McGraw-Hill 1993.
109. Huesmann LR. Aggressive Behavior. *An information processing model for the development of aggression.* Vol 14 1988:13-24.
110. Zillmann D. *Hostility and aggression.* NJ: Erlbaum: Hillsdale; 1979.
111. Bandura A, Ross D, Ross SA. Vicarious Reinforcement and Imitative Learning. *J Abnorm Psychol.* Dec 1963;67:601-607.
112. Cantor J. Media violence. *J Adolesc Health.* Aug 2000;27(2 Suppl):30-34.
113. Zillman DW, James B III Effects of prolonged exposure to gratuitous media violence on provoked and unprovoked hostile behavior. *Journal of Applied Social Psychology.* Jan 1999 1999;29(1):145-165.
114. Zillman DW, James B III. Psychoticism in the effect of prolonged exposure to gratuitous media violence on the acceptance of violence as a preferred means of conflict resolution. *Personality and Individual Differences.* May 1997 1997;22(5):613-627.
115. Cline VB, Croft RG, Courier S. Desensitization of children to television violence. *J Pers Soc Psychol.* Sep 1973;27(3):360-365.
116. Mullin CR, Linz D. Desensitization and resensitization to violence against women: effects of exposure to sexually violent films on judgments of domestic violence victims. *J Pers Soc Psychol.* Sep 1995;69(3):449-459.
117. Barefoot JC, Patterson JC, Haney TL, Cayton TG, Hickman JR, Jr., Williams RB. Hostility in asymptomatic men with angiographically confirmed coronary artery disease. *Am J Cardiol.* Sep 1 1994;74(5):439-442.
118. Knox SS, Adelman A, Ellison RC, et al. Hostility, social support, and carotid artery atherosclerosis in the National Heart, Lung, and Blood Institute Family Heart Study. *Am J Cardiol.* Nov 15 2000;86(10):1086-1089.
119. Barefoot JC, Larsen S, von der Lieth L, Schroll M. Hostility, incidence of acute myocardial infarction, and mortality in a sample of older Danish men and women. *Am J Epidemiol.* Sep 1 1995;142(5):477-484.
120. Everson SA, Kauhanen J, Kaplan GA, et al. Hostility and increased risk of mortality and acute myocardial infarction: the mediating role of behavioral risk factors. *Am J Epidemiol.* Jul 15 1997;146(2):142-152.
121. Low KG, Fleisher C, Colman R, Dionne A, Casey G, Legendre S. Psychosocial variables, age, and angiographically-determined coronary artery disease in women. *Ann Behav Med.* Summer 1998;20(3):221-226.

122. Baron KG, Smith TW, Butner J, Nealey-Moore J, Hawkins MW, Uchino BN. Hostility, anger, and marital adjustment: concurrent and prospective associations with psychosocial vulnerability. *J Behav Med.* Feb 2007;30(1):1-10.
123. Barefoot JC, Dahlstrom WG, Williams RB, Jr. Hostility, CHD incidence, and total mortality: a 25-year follow-up study of 255 physicians. *Psychosom Med.* Mar 1983;45(1):59-63.
124. Siegler IC, Peterson BL, Barefoot JC, Williams RB. Hostility during late adolescence predicts coronary risk factors at mid-life. *Am J Epidemiol.* Jul 15 1992;136(2):146-154.
125. Georgiades A, Lane JD, Boyle SH, et al. Hostility and fasting glucose in African American women. *Psychosom Med.* Jul 2009;71(6):642-645.
126. Scherwitz LW, Perkins LL, Chesney MA, Hughes GH, Sidney S, Manolio TA. Hostility and health behaviors in young adults: the CARDIA Study. Coronary Artery Risk Development in Young Adults Study. *Am J Epidemiol.* Jul 15 1992;136(2):136-145.
127. Barefoot JC, Beckham JC, Haney TL, Siegler IC, Lipkus IM. Age differences in hostility among middle-aged and older adults. *Psychol Aging.* Mar 1993;8(1):3-9.
128. Sutin AR, Costa PT, Jr., Uda M, Ferrucci L, Schlessinger D, Terracciano A. Personality and metabolic syndrome. *Age (Dordr).* Dec 2010;32(4):513-519.
129. World Health Organization. Cardiovascular diseases (CVDs)2011.
130. Ervin RB. Prevalence of metabolic syndrome among adults 20 years of age and over, by sex, age, race and ethnicity, and body mass index: United States, 2003-2006. *Natl Health Stat Report.* May 5 2009(13):1-7.
131. Friedman GD, Cutter GR, Donahue RP, et al. CARDIA: study design, recruitment, and some characteristics of the examined subjects. *J Clin Epidemiol.* 1988;41(11):1105-1116.
132. Coronary Artery Risk Development in Young Adults 2011; <http://www.cardia.dopm.uab.edu/index.htm>.
133. Batey LS, Goff DC, Jr., Tortolero SR, et al. Summary measures of the insulin resistance syndrome are adverse among Mexican-American versus non-Hispanic white children: the Corpus Christi Child Heart Study. *Circulation.* Dec 16 1997;96(12):4319-4325.
134. Brage S, Wedderkopp N, Ekelund U, et al. Features of the metabolic syndrome are associated with objectively measured physical activity and fitness in Danish children: the European Youth Heart Study (EYHS). *Diabetes Care.* Sep 2004;27(9):2141-2148.
135. Sidney S, Jacobs DR, Jr., Haskell WL, et al. Comparison of two methods of assessing physical activity in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Am J Epidemiol.* Jun 15 1991;133(12):1231-1245.
136. McDonald A, Van Horn L, Slattery M, et al. The CARDIA dietary history: development, implementation, and evaluation. *J Am Diet Assoc.* Sep 1991;91(9):1104-1112.
137. Sijtsma FP, Meyer KA, Steffen LM, et al. Longitudinal trends in diet and effects of sex, race, and education on dietary quality score change: the Coronary Artery Risk Development in Young Adults study. *Am J Clin Nutr.* Feb 1 2012.
138. Hu L, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal.* 1999;6(1):1-55.
139. Healy GN, Wijndaele K, Dunstan DW, et al. Objectively measured sedentary time, physical activity, and metabolic risk: the Australian Diabetes, Obesity and Lifestyle Study (AusDiab). *Diabetes Care.* Feb 2008;31(2):369-371.

140. *American Time Use Survey-2004 Results Announced by BLS*: United States Department of Labor;2004.
141. Wang Y, Beydoun MA. The obesity epidemic in the United States--gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. *Epidemiol Rev.* 2007;29:6-28.
142. Tucker LA, Tucker JM. Television viewing and obesity in 300 women: evaluation of the pathways of energy intake and physical activity. *Obesity (Silver Spring)*. Oct 2011;19(10):1950-1956.
143. Ching PL, Willett WC, Rimm EB, Colditz GA, Gortmaker SL, Stampfer MJ. Activity level and risk of overweight in male health professionals. *Am J Public Health*. Jan 1996;86(1):25-30.
144. Ainsworth BE, Haskell WL, Leon AS, et al. Compendium of physical activities: classification of energy costs of human physical activities. *Med Sci Sports Exerc*. Jan 1993;25(1):71-80.
145. Kronenberg F, Pereira MA, Schmitz MK, et al. Influence of leisure time physical activity and television watching on atherosclerosis risk factors in the NHLBI Family Heart Study. *Atherosclerosis*. Dec 2000;153(2):433-443.
146. *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, to the Secretary of Agriculture and the Secretary of Health and Human Services*1985.
147. *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 2010*2010.
148. Anderssen N, Jacobs DR, Jr., Sidney S, et al. Change and secular trends in physical activity patterns in young adults: a seven-year longitudinal follow-up in the Coronary Artery Risk Development in Young Adults Study (CARDIA). *Am J Epidemiol*. Feb 15 1996;143(4):351-362.
149. Dudley J, Jin S, Hoover D, Metz S, Thackeray R, Chmiel J. The Multicenter AIDS Cohort Study: retention after 9 1/2 years. *Am J Epidemiol*. Aug 1 1995;142(3):323-330.
150. Chang PC, Li TC, Wu MT, et al. Association between television viewing and the risk of metabolic syndrome in a community-based population. *BMC Public Health*. 2008;8:193.
151. Gardiner PA, Healy GN, Eakin EG, et al. Associations between television viewing time and overall sitting time with the metabolic syndrome in older men and women: the Australian Diabetes, Obesity and Lifestyle study. *J Am Geriatr Soc*. May 2011;59(5):788-796.
152. Chida Y, Steptoe A. The association of anger and hostility with future coronary heart disease: a meta-analytic review of prospective evidence. *J Am Coll Cardiol*. Mar 17 2009;53(11):936-946.