

**Associations Between Social Risk Factors and the American Heart Association's Life's
Simple 7 in US Adults: NHANES 2015-2016**

by

Brandon Matthew Herbert

BS, University of Pittsburgh, 2017

Submitted to the Graduate Faculty of the
Department of Epidemiology
Graduate School of Public Health in partial fulfillment
of the requirements for the degree of
Master of Public Health

University of Pittsburgh

2019

UNIVERSITY OF PITTSBURGH
GRADUATE SCHOOL OF PUBLIC HEALTH

This essay is submitted

by

Brandon Matthew Herbert

on

December 19, 2019

and approved by

Essay Advisor:

Maria Mori Brooks, PhD
Professor of Epidemiology and Biostatistics
Department of Epidemiology
Graduate School of Public Health
University of Pittsburgh

Essay Readers:

Jared W Magnani, MD, MSc
Associate Professor of Medicine
Department of Medicine
School of Medicine
University of Pittsburgh

Christina Mair, PhD
Assistant Professor
Behavioral and Community Health Sciences
Graduate School of Public Health
University of Pittsburgh

Copyright © by Brandon Matthew Herbert

2019

Associations Between Social Risk Factors and the American Heart Association's Life's

Simple 7 in US Adults: NHANES 2015-2016

Brandon Matthew Herbert, MPH

University of Pittsburgh, 2019

Abstract

Background: Risk of cardiovascular disease (CVD) is attenuated by healthy lifestyle choices. Identifying patients at increased risk for developing CVD based on modifiable risk factors is crucial for reducing the burden of disease. The American Heart Association's Life's Simple 7 (LS7) is commonly used to assess modifiable cardiovascular risk factors in individuals and communities.

Hypothesis: Individuals with (a) limited English proficiency or (b) lower socioeconomic status (SES) will have lower rates of ideal cardiovascular health when compared to participants proficient in English or with higher SES.

Methods: Data on adults ages ≥ 25 years from the 2015-2016 National Health and Nutrition Examination Survey (NHANES) cycle were analyzed. The AHA's LS7 was used to measure cardiovascular health. The LS7 score ranges from 0 to 14 points where 7 categories are assigned as either poor, intermediate, or ideal health status. SES was divided into three categories: low, middle, and high. Each category of SES was defined as a combination of poverty income ratio and education level. The LS7 score was analyzed as a continuous variable using regression analyses and each individual dichotomized LS7 component was examined by race, English proficiency, and SES categories.

Results: Among 5,053 participants, we observed significant differences in LS7 scores by race/ethnicity, SES and limited English proficiency. With multivariable adjusted linear regression, low and middle SES was associated with significantly lower average LS7 scores than those in the high SES category ($\beta=-1.41$ [$p<0.001$], $\beta=-0.89$ [$p<0.001$]). Limited English proficiency was not significantly related with the LS7 score. The LS7 components that differed between low and high SES participants were glycohemoglobin, smoking status, physical activity, and diet. Non-Hispanic blacks had lower rates of ideal cardiovascular health in 6 of the 7 components that comprise the LS7 score ($p<0.01$ for all 6 components).

Conclusions: This study provides insight into the relations between cardiovascular health, SES, limited English proficiency, and race/ethnicity. Large disparities in ideal cardiovascular health metrics across SES and racial/ethnic groups are significant to the field of public health. Population-level interventions need to be refined in order to address these disparities. The contribution of limited English proficiency to CVD risk requires further study.

Key words: Cardiovascular disease; American Heart Association's Life's Simple 7; limited English proficiency; socioeconomic status.

Table of Contents

Preface.....	ix
1.0 Introduction.....	1
1.1 Overview of Cardiovascular Disease in the United States.....	1
1.2 Common Types of Cardiovascular Disease.....	1
1.3 Economic Impact in the United States.....	2
1.4 Known Cardiovascular Disease Risks	3
1.4.1 Social and Environmental Risk Factors for CVD.....	6
1.5 Cardiovascular Disease Measurement Tools	9
1.5.1 The American Heart Association’s Life’s Simple 7	11
1.6 The National Health and Nutrition Examination Survey (NHANES)	14
1.7 Gaps in Knowledge.....	16
1.8 Public Health Significance	16
1.9 Objective.....	17
2.0 Methods.....	18
3.0 Results	25
4.0 Discussion.....	43
Bibliography	50

List of Tables

Table 1. Definitions of Poor, Intermediate, and Ideal Cardiovascular Health for Each Metric of the American Heart Association’s Life Simple 7.....	12
Table 2. Predictor Variable Classifications.....	22
Table 3. Demographic Profiles of Total, Non-missing LS7, and Missing LS7 Samples.	26
Table 4. Demographic Characteristics Stratified by Limited English Proficiency.	28
Table 5. Mean LS7 by Race/Ethnicity, SES, and Limited English Proficiency.	30
Table 6. Compartmental LS7 Score Distribution Across All Participants.	31
Table 7. Regression Parameters, Estimates, and 95% Confidence Intervals for Univariate Models.	34
Table 8. Regression Parameters, Estimates, and 95% Confidence Intervals for Multivariable Models.	35

List of Figures

Figure 1. Flow Diagram for the Final Study Sample.....	24
Figure 2. Distribution of LS7 Score Across the Final Population with Weighted Frequencies and Standard Deviations.....	29
Figure 3. Compartmental LS7 Scores by SES Category.....	36
Figure 4. Compartmental LS7 Scores by SES Category (Laboratory Measurements).	37
Figure 5. LS7: Smoking Status by Race/Ethnicity.....	38
Figure 6. LS7: BMI by Race/Ethnicity.	39
Figure 7. LS7: Physical Activity by Race/Ethnicity.....	39
Figure 8. LS7: Diet Components by Race/Ethnicity.....	40
Figure 9. LS7: Total Cholesterol by Race/Ethnicity.....	40
Figure 10. LS7: Blood Pressure by Race/Ethnicity.....	41
Figure 11. LS7: Glycohemoglobin by Race/Ethnicity.	41

Preface

I would like to express my gratitude to the members of my committee which include Dr. Maria Brooks, Dr. Jared Magnani, and Dr. Christina Mair for their guidance and mentorship throughout this process.

1.0 Introduction

1.1 Overview of Cardiovascular Disease in the United States

Across the United States, roughly 630,000 people die of cardiovascular disease (CVD) every year and this accounts for 1 in every 4 deaths. For both men and women, cardiovascular disease is the leading cause of death in the US.¹ Since CVD is a chronic disease that develops over a long period of time without causing immediate death, the prevalence of the disease is extremely high. Nearly 102.7 million American adults were living with one CVD condition in 2015.² By 2035, it is projected that the number of Americans with CVD will rise to 131.2 million. The incidence of CVD conditions, or the number of newly diagnosed cases, varies widely among the different forms of CVD.³ Importantly, many forms of the disease are preventable and treatable with healthy lifestyle choices while few are significantly related to genetics.³

1.2 Common Types of Cardiovascular Disease

Generally, the term cardiovascular disease refers to medical conditions that either involve narrow or blocked blood vessels or conditions that affect the heart's muscle, valves, or rhythms.⁴ The term "cardiovascular disease" is often synonymous with the term "heart disease." The most common types of CVD include coronary heart disease (CHD), myocardial infarction (i.e., heart attack), arrhythmia, heart failure, congenital heart defects, cardiomyopathy, and peripheral artery disease (PAD).⁵ CHD is the most common form and occurs when the build-up of plaque narrows or hardens arteries supplying blood to the heart.⁶ Plaque build-up is also known as atherosclerosis and the site of the build-up determines the type of heart disease. Coronary artery disease (CAD) is

classified as the build-up of plaque in arteries supplying blood to the heart while PAD is classified as the build-up of plaque in arteries supplying blood to the arms and legs.⁶ Oftentimes CAD and CHD are terms used interchangeably by health professionals, but technically CHD is a result of CAD. This distinction is important for understanding the disease etiology of CHD.

1.3 Economic Impact in the United States

Due to the fact that CVD is often treated over the course of decades after diagnosis, the economic impact of the disease is immense. Based on the American Heart Association's (AHA) 2019 Heart Disease and Stroke Statistics Update, between 2014 and 2015, \$351.2 billion was spent on total cardiovascular disease and stroke across the US in terms of direct and indirect costs.³ Direct costs totaled \$213.8 billion while an estimated \$137.4 billion was lost indirectly through lost productivity and mortality.³ A separate analysis conducted by RTI International estimated current medical costs for all CVD in the US to be \$318 billion and indirect costs to be \$237 billion.^{2,7} By 2035, these costs are expected to rise 135% and 55%, respectively. If accurate, this projection will put the total cost of CVD above \$1 trillion dollars come 2035.

On an individual level, Nichols et al. investigated the medical care costs among patients with established CVD.⁸ The team identified 12,278 patients from the Kaiser Permanente Northwest CVD registry and found a mean annual direct medical cost for a patient to be \$18,953 with a standard deviation of \$39,036. The causes for the greatest differences in cost were determined to be a secondary CVD hospitalization, presence of diabetes, an estimated glomerular filtration rate of less than 60 mL/min/1.73 m², and depression. Nichols et al. published these data in 2010 and the cost for a patient to live with CVD has likely increased significantly since 9 years ago.

1.4 Known Cardiovascular Disease Risks

The primary risk factors for CVD can be broken down into three broad categories: medical conditions, behaviors, and family history. Medical conditions that contribute to increased risk for CVD include high blood pressure, high cholesterol, diabetes and obesity.¹ Symptoms of high blood pressure are commonly not noticed by patients and requires regular blood pressure monitoring to detect early. High cholesterol levels can occur when more cholesterol is taken in through a person's diet than the body can use. The extra cholesterol in the blood contributes to the build-up of plaque in arteries that can result in atherosclerosis. Importantly, not all cholesterol contributes negatively to the development of CVD. High-density lipoprotein cholesterol (HDL) is considered "good" as it provides some protection against heart disease while low-density lipoprotein cholesterol (LDL) is considered "bad" because it increases risk for the build-up of plaque.¹

Additional medical conditions that contribute to the risk for developing CVD are diabetes and obesity. In the US, the prevalence of obesity among adults in 2014 was estimated to be 37.7%.² Obesity is linked with higher LDL cholesterol levels, higher triglyceride levels in the blood, and to lower HDL lipoprotein cholesterol concentrations. High blood pressure is another potential adverse health outcome for those who are obese.¹ Using data from 2013-2016, it is estimated that 26 million (9.8%) American adults had diagnosed diabetes and over 80,000 deaths were attributed to diabetes alone in 2016.² Diabetes causes the build-up of sugar in the blood which damages blood vessels and the nerves that control the heart and blood vessels.¹

Almost all of the medical conditions associated with CVD risk may be the consequence of lifestyle or behavior choices. An unhealthy diet, physical inactivity, excessive alcohol intake and tobacco use can increase risk for CVD.¹ Salas-Salvadó et al. conducted a meta-analysis of high-quality prospective cohorts and randomized clinical trials that investigated the relation between

the Mediterranean Diet and CVD risk.⁹ The Mediterranean Diet is a diet characterized by olive oil as a main source of fat, high intake of fruit and vegetables, low to moderate amounts of animal products, and wine in moderation with meals. A review of recent studies shows an inverse relation between adherence to the diet and diabetes and metabolic syndrome incidence.⁹ Additionally, beneficial effects on blood pressure, triglycerides, LDL cholesterol, and body weight were detected. The meta-analysis concluded that the Mediterranean Diet has a beneficial role in CVD prevention, but the effect size cannot be certain due to high heterogeneity between studies.⁹ A diet with high levels of saturated fats, trans fat, cholesterol, and salt can greatly increase the risk for CVD.¹

A second major behavioral risk factor for CVD is physical inactivity. Wahid et al. conducted a systematic review and meta-analysis on the association between physical activity (PA) and CVD / diabetes.¹⁰ Across 36 studies, 3,439,874 participants, and 179,393 events, the team was able to conclude that achieving recommended PA levels (150 minutes of moderate-intensity aerobic activity per week) was associated with a 23% lower risk of CVD mortality, 17% decrease in risk for incident CVD, and a 26% decrease in risk for being diagnosed with type 2 diabetes when compared to being inactive, adjusting for body weight. Additionally, physical inactivity is associated with other medical conditions previously discussed such as obesity, high blood pressure and high cholesterol.¹

Two other well-known risk factors for CVD are high alcohol consumption and tobacco use. Drinking too much alcohol can increase blood pressure and triglyceride levels which can harden arteries.¹ Smoking cigarettes can damage the heart and blood vessels because nicotine raises blood pressure and carbon monoxide reduces the capacity of red blood cells to carry oxygen. In 2016, Lubin et al. analyzed synergistic and non-synergistic associations for cigarette smoking

and non-tobacco risk factors for CVD incidence.¹¹ The researchers found that there may be potential for smoking to interact with other risk factors for CVD in additive or multiplicative forms; however, research in this regard to smoking still needs to be performed before conclusions can be drawn.

Over 50 years ago, scientists began making some of the first observations that CVD had a genetic component that predisposed certain individuals to increased risk.¹² It was understood that if parents had CVD their children have increased risk for development of CVD, but the exact gene(s) that were responsible was not clear. Genome-wide association studies (GWAS) have rapidly accelerated our understanding of the genetic component of CVD over the past 10 years. Due to each form of CVD being unique, specific gene(s) have been associated with certain forms of CVD such as CAD and CHD.

Of the millions of single nucleotide polymorphisms (SNPs) analyzed, chromosome 9p21 locus is perhaps the strongest single marker of CAD today.¹² However, this locus and many others identified with it have had a marginal impact as effect sizes were quite small (odds ratio less than 1.2) and only about 10% of the estimated heritability is explained.¹² A new joint association analysis was performed by Nikpay et al. in a meta-analysis that discovered 202 variants in 129 loci associated with CHD. Together, these loci explained a much larger proportion of the estimated heritability of CHD than previous studies at almost 28%.¹³ Yet, 72% of the heritability is still uncertain. Genetic studies for various forms of CVD, including both CAD and CHD, are still needed. Future research is exploring the interaction of environmental factors with genetic predisposition to try to estimate an even larger portion of the heritability for CHD.¹²

1.4.1 Social and Environmental Risk Factors for CVD

The environment can play a critical role in the contribution of risk for developing CVD, and environmental factors can be broken down into two large categories: natural and social. The natural environment includes circadian rhythms, seasons, sunlight, altitude, and greenspaces. Greenspaces and the physical environment have been a point of recent discussion.¹⁴ It is understood that the built environment, or the human-made environment, is associated with CVD, but which dimensions of the built environment contribute the most to CVD risk is not yet known. Jia et al. assessed the effects of different measures of an individual's neighborhood walkability score along with a greenness score in relation to CVD risk. Those that lived in neighborhoods with high walkability scores or greenness scores had significant reductions in risk for hypertension, CHD, and stroke.¹⁵ Particulate matter 2.5 (PM_{2.5}), which is air pollution with a diameter less than 2.5 micrometers, has been declared to have a causal relation with cardiovascular morbidity and mortality by the American Heart Association.¹⁶ Short-term exposure to PM_{2.5} can trigger CVD-related mortality and nonfatal events while long-term exposure can shorten an individual's lifetime by months or even years.¹⁶ Researchers have also proposed credible pathological mechanisms which give strength to the observational epidemiological studies.^{16,17}

Similarly, the social environment can have a large impact on an individual's risk for developing CVD. The social environment includes certain characteristics of the built environment (e.g. presence of food deserts, lack of exercise facilities), environmental noise, social networks, and socioeconomic status.¹⁴ Low socioeconomic status (SES) is a major risk factor for cardiovascular disease. The evidence is fairly consistent in supporting the association between SES and CVD risk across studies and systematic reviews in high-income countries.¹⁸ Sommer et al. conducted a review of high quality systematic reviews and found studies agreed on higher

incidence of stroke and acute MI among low SES and educational attainment groups. Interestingly, research has not been conclusive about the relation between childhood socioeconomic inequalities and adult risk for CVD.¹⁸ SES is associated with incidence of CVD, but the best interventions for addressing this disparity in cardiovascular health is still under investigation.¹⁹

Schultz et al. conducted a review of the current challenges and interventions for addressing the disparity of cardiovascular outcomes between SES strata.¹⁹ Patients with low SES have been traditionally targeted with interventions aiming to change modifiable traditional risk factors for CVD. Particularly, structured physical activity interventions have had success in low SES populations.¹⁹ The authors advocate for SES to be used more frequently in risk prediction models and for more research to investigate cultural and regional differences in SES. Underlying mechanisms of CVD risk in low SES populations still need to be better understood to create tailored interventions to vulnerable subpopulations in the US.¹⁹

In the AHA's Heart Disease and Stroke Statistics – 2019 Update, differences in cardiovascular health and disease are documented well by race and ethnicity.³ From 1999 to 2016, non-Hispanic (NH) black persons were more than twice as likely to die of heart disease compared to NH Asians. NH black communities experience a greater burden of MI, HF, stroke, and several other CVD outcomes. This is likely due to the higher prevalence of unrecognized risk factors that go untreated.²⁰ In an analysis of Atherosclerosis Risk in Communities (ARIC) Study participants, CVD events in blacks were more likely to be explained by elevated or borderline risk factors when compared to whites (90% of events compared to 65% of events, respectively).³ African American males have the highest overall death rate from CVD, and African American females have higher CVD-related death rates compared to white females.²⁰ Importantly, African Americans die at younger ages from CVD compared to NH whites.

Different minority groups have unique risk factors that increase their risk for developing CVD. Across the world, African Americans and Mexican Americans living in the US have among the highest rate of hypertension and elevated blood pressure.²⁰ South Asians tend to have more nontraditional CVD risk factors and differences in inflammatory markers and insulin resistance have been documented.²⁰ Smoking rates are significantly higher in Korean, Vietnamese, and Filipino Americans when compared to NH whites. Metabolic syndrome is significantly more prevalent in Mexican Americans than other racial/ethnic groups. In summary, the levels of various risk factors for CVD vary across populations and this is critical for health care professionals to recognize.

To address the disparities in risk factors for CVD between populations, medical and public health professionals need to understand the communities they serve.²⁰ This idea has been commonly referred to as “cultural competency” which briefly means to provide tailored care to each individual based on their linguistic, social, and cultural needs.²⁰ Although disparities in CVD incidence and prevalence are understood across racial and ethnic groups, the most effective interventions for various modifiable risk factors across these groups are still being investigated.³

Another component of the social environment is spoken language and limited English proficiency. While socioeconomic and ethnic differences are better understood inequalities in cardiovascular health, the role of not speaking a majority language is less certain. Mackay et al. conducted a retrospective, cross-sectional analysis of the relation between non-English language preferences and cardiovascular health in London communities.²¹ Although the sizes of the non-English speaking populations were small, the study was able to identify that the non-English preference community had a greater likelihood of having CHD (OR=1.18, 95% CI: 1.03, 1.34); however, no differences were detected in the prevalence of hypertension or stroke. From our

review of the literature, limited research has been conducted in the non-English speaking populations in regard to their risk for developing CVD. Language preference has the potential to serve as an indicator variable that defines a number of other shared cultural and behavioral risk factors for developing CVD.²¹

1.5 Cardiovascular Disease Measurement Tools

In order for practitioners to provide better care to patients and for policy makers to decide on better population level interventions, the identification of individuals at high risk for CVD is critical. Studies have demonstrated that accurate CVD risk assessments result in better clinical outcomes for patients.²² However, the number of CVD risk calculators that can be found online, in literature, and from professional organizations is staggering. In 2017, Bonner et al. conducted a systematic review on the clinical validity and understandability of online CVD risk calculators.²³ The group identified 73 unique calculators. In 2011, the Vanderbilt Evidence-based Practice Center and Institute for Medicine and Public Health prepared a report on CVD clinical risk prediction models for the Agency for Healthcare Research and Quality that is a part of the US Department of Health and Human Services.²⁴ The group identified 102 different risk models cited in literature over a 10 year period from 1999 to 2009. Within all of the existing CVD risk assessments that can be found, few have been validated externally.²⁴

In Bonner et al.'s systematic review, the most common clinical model was the Framingham risk score. The Framingham risk prediction model was updated by D'Agostino et al. in 2008 and is the current 10-year CVD risk calculator the Framingham Heart Study hosts on their website.²⁵ The risk calculator was developed by performing Cox proportional-hazards regression on 8,491 Framingham study participants for developing a first CVD event. The participants were followed

for an extended period of time, 12 years, to allow the outcome ample time to occur. The predictors of the model include age, diabetes, smoking, treated and untreated systolic blood pressure (SBP), total cholesterol, and HDL cholesterol. Notably, BMI can replace lipid measurements if lipid measurements are not attainable.²⁵ In the original regression model for individual CVD outcomes, high cholesterol had a stronger association with CHD and intermittent claudication, a potential indicator of PAD, high SBP had a stronger association with stroke and CHF, and smoking had a stronger association with intermittent claudication.^{25, 26} The primary limitation of this risk score is its lack of generalizability to populations not similar to the Framingham cohort which is mostly Caucasian.

Bonner et al. identified the second most common model used in online risk calculators was another that is used in clinical practice guidelines: the American College of Cardiology / American Heart Association's Atherosclerotic Cardiovascular Disease (ASCVD) risk estimator. This estimator allows healthcare providers to estimate 10-year risk for ASCVD. Currently, the definition of ASCVD has two distinct components that can classify an outcome: coronary death or nonfatal myocardial infarction, or fatal or nonfatal stroke. The first component is based on the Pooled Cohort Equations while the second is based on work performed by Lloyd-Jones et al.²⁷ Unlike the Framingham Risk Score calculator, five community and population-based studies' regression estimates were pooled together to form the Pooled Cohort Equations for estimating ASCVD risk.²⁸ The studies included the Framingham Heart Study, Framingham Offspring Study, Atherosclerosis Risk In Communities (ARIC) Study, Cardiovascular Health Study (CHS), and the Coronary Artery Risk Development in Young Adults (CARDIA) Study. The estimator considers the following predictors when determining the 10-year ASCVD risk: sex, age, race, total cholesterol, HDL cholesterol, SBP, treatment for high blood pressure (BP), diabetes, and smoking

status.²⁷ The data incorporated over 25,000 individuals aged 40 to 79 years old and came from a wider range of ethnic and racial backgrounds to create estimates more accurate for the US population as a whole.²⁸

1.5.1 The American Heart Association's Life's Simple 7

In 2010, the American Heart Association constructed a new definition of ideal cardiovascular health based on seven risk factors that can be changed by patients through lifestyle choices and are readily accessible in healthcare system databases.²⁹ Since the creation of the metric, recognized as AHA's Life's Simple 7 (LS7), several adverse health outcomes have been associated with a poor overall score. These outcomes include incident heart failure,³⁰⁻³² atrial fibrillation,^{33, 34} silent myocardial infarction (SMI), and worse prognosis after SMI and MI.³⁵⁻³⁷ Lloyd-Jones et al. conducted an analysis on the Framingham Heart Study participants and found that the absence of poor LS7 metrics at age 50 was associated with longer survival and a very low lifetime risk for developing CVD.³⁸

The four modifiable and three biometric risk factors that compose the LS7 metric are smoking, body mass index (BMI), diet, physical activity, total cholesterol, blood pressure, and fasting plasma glucose.²⁹ Each of the seven components are classified into three levels: poor, intermediate, and ideal. The following table provides the definition for each health metric:²⁹

Table 1. Definitions of Poor, Intermediate, and Ideal Cardiovascular Health for Each Metric of the American Heart Association's Life Simple 7.

	Poor	Intermediate	Ideal
Smoking status	Current	Former ≤12 months	Never or quit >12 months Never tried; never smoked whole cigarette
BMI (kg/m ²)	≥30	25≥BMI<30	18.5≥BMI<25
Physical activity	None	1-149 min/week moderate or 1-74 min/wk vigorous 1-149 min/wk moderate + 2x vigorous >0 min <60 min of moderate or vigorous every day	≥150 min/wk moderate or ≥75 min/wk vigorous ≥150 min/wk moderate + 2x vigorous ≥60 of moderate or vigorous every day
Healthy diet pattern, number of components**	0-1	2-3	4-5
Total cholesterol (mg/dL)	≥240	200≥total cholesterol<240	<200
Blood pressure (mmHg)	SBP≥140 or DBP≥90	SBP 120-139 or DBP 80-89 or treated to goal	<120 SBP / <80 DBP
Fasting plasma glucose (mg/dL)	≥126	100-125	<100

*Taken directly from the American Heart Association.²⁹

**The five healthy eating components are as follows: consume ≥ 4.5 cups/d of fruits and vegetables, ≥ 2 servings/wk of fish, ≥ 3 servings/d of whole grains, no more than 36 oz/wk of sugar-sweetened beverages, and no more than 1500 mg/d of sodium.

Although the AHA's LS7 has several strengths, several weaknesses exist that become problematic when comparing studies. The primary weakness associated with AHA's LS7 is the lack of a defined and clinically meaningful overall score outcome which results in the inconsistency of scoring and scoring scales between studies. Studies define the LS7 scores as

dichotomous based on the presence of poor health metrics³⁸ or the presence of a pre-specified number of ideal health metrics.³¹ Studies have analyzed the score as a continuous variable with a range of 0 to 14 where 0 points are given for poor health metrics, 1 point for intermediate health metrics, and 2 for ideal health metrics.^{30, 36} Additionally, the overall LS7 score has been split into a categorical variable where scores of 0-4 are classified as inadequate, 5-9 average, and 10-14 optimal.^{33, 35} Lin, M. et al. created a LS7 score that gave participants one point for obtaining an ideal health metric and 0 points for both poor and intermediate health metrics (resulting in a score that ranged from 0 to 7).³⁹ Lin, A. et al. followed the same scoring method as Lin, M. et al., but then categorized this score into three categories: low was 0-1, medium was 2-3, and greater than 4 was high.^{39, 40}

The LS7 scoring inconsistencies create a difficult situation for researchers attempting to establish consistency and dose-response relations across studies. In 2016, Younus et al. aggregated the findings from numerous studies that incorporated an outcome of ideal cardiovascular health using AHA's LS7.⁴¹ Importantly, the group only included studies that defined the AHA LS7 score as 0-7 where 1 point was given for an ideal metric status and 0 for poor and intermediate. They found the prevalence of ideal cardiovascular health, defined as a score of 6 or 7, ranged from 0.3% to 15% across US and international cohorts. Six mortality studies analyzed agreed upon an inverse relation between the number of ideal cardiovascular health measures and all-cause / CVD-related mortality risk. Ideal cardiovascular health metrics were also negatively associated with incident cardiovascular events.⁴¹

In summary, there are numerous cardiovascular risk measurement tools that exist throughout the literature and online. Among them, few have been validated in a clinical setting and each needs to be used with its limitations in mind. The accuracy of the Framingham risk model

has been replicated across multiple studies, but it is important to keep in mind that this model is based on a primarily white population. The ASCVD risk model overcomes the race and ethnic barriers by aggregating data across diverse cohorts, but only contains a portion of the predictors that may be crucial for estimating CVD risk. The AHA's LS7 combines modifiable / behavioral risk factors with biometric risk factors, but the overall scoring and interpretation of the score appear to have high heterogeneity in the literature. Additionally, each risk factor is evaluated with equal importance to cardiovascular health as regression coefficients are not used to assess risk like the Framingham risk score and ASCVD risk score.

1.6 The National Health and Nutrition Examination Survey (NHANES)

The National Health and Nutrition Examination Survey (NHANES) program consists of a series of cross-sectional and multi-stage studies. The studies are designed to assess the health and nutritional status of a subpopulation of the US population that is weighted and stratified to be generalizable to the entire non-institutionalized population of the country. Each cycle, the study combines interviews and physical examinations. The National Center for Health Statistics (NCHS), which falls under the Centers for Disease Control and Prevention, manages and funds NHANES as the NCHS is responsible for producing vital and health statistics for the US.⁴² Roughly 5,000 people across the US participate in the NHANES program each year and are interviewed about their demographics, socioeconomic status, dietary intake, and health. The medical examination component consists of medical, dental, physiological measurements, and laboratory tests. The data from all NHANES studies are made publicly available on the NHANES website in order to enable epidemiological studies and health sciences research.

In the health examination surveys, the primary focus of NHANES is to collect information on chronic diseases and conditions and their prevalence.⁴³ A large number of risk factors are included that give details on a participant's lifestyle, heredity, and environment. In order to create a reliable sub-sample, NHANES over samples certain segments of the population. Primarily, these segments include those that are 60 years of age and older, African Americans, and Hispanics.⁴³ Participants in these segments of the population are then weighted appropriately to represent their demographic profiles across the US. Importantly, all participants visit the physician, have body measurements taken, and take dietary interviews. Individual characteristics were associated with a willingness to participate in various study components; for example, older individuals tend to have more extensive examinations performed compared to younger people.⁴³

Participants are asked to provide data at different locations and at different times. A health interview is conducted in the respondent's home.⁴⁴ NHANES operates a number of mobile centers which are specially-designed and equipped to travel across the country and conduct the health measurements. The staff that conduct the interviews and health examinations consists of a team of physicians, medical and health technicians, and professional dietary and health interviewers. A large proportion of these staff are bilingual in order to survey respondents whose first language may not be English.⁴⁴

For each location NHANES plans to visit, a recruitment effort is launched prior to the arrival of NHANES staff. First, local health and government officials are notified that the survey is coming to their respective area. Households in a pre-specified area receive a letter from the NHANES program that introduces the survey and asks for their participation.⁴⁴ Additionally, NHANES reaches out to the local media in order to increase coverage in the area through stories broadcasted on television. Notably, steps are taken in order to prevent barriers to participation. For

example, transportation is provided for participants who have no means to get to the mobile center. Participants are compensated for participation and receive a detailed medical report at the end of the survey and examination.⁴⁴

1.7 Gaps in Knowledge

The AHA's Life's Simple 7 measurement tool for ideal cardiovascular health is based on modifiable risk factors and is often accessible through existing patient records. LS7 is relatively new compared to other commonly used CVD risk measurement tools. Research has classified the outcome of the LS7 score in a variety of forms and the definitions proposed by the AHA have not been strictly followed. More analyses that are generalizable to the entire US population that strictly follow the AHA guidelines for LS7 scoring and analyzes social factors that contribute to CVD risk is needed. Of these social factors, limited English proficiency has been under-researched and warrants more study in diverse populations that live in the US.

An in-depth analysis that incorporates, rather than omits, missing data for the LS7 score is also needed to help determine if those participants with missing LS7 components in NHANES are inherently different than those who with complete LS7 data. Numerous studies that use NHANES data to analyze LS7 exclude participants who do not have a complete score and this may be leading to biased conclusions.

1.8 Public Health Significance

The following study has the potential to contribute significant findings to the field of cardiovascular epidemiology for diverse populations. The relations among behavioral risk factors,

race/ethnicity and social factors, such as SES and limited English proficiency, with CVD are complex. With the following analysis, we hope to better understand the relations between LS7 score and limited English proficiency, SES, and race/ethnicity. By using NHANES data, the following analysis provides nationally representative estimates that are generalizable to the United States population. The usefulness of the LS7 tool may benefit the medical community as it is easier to implement and measure when compared to other CVD risk measurement tools. When patients are empowered to modify their risk for developing CVD, the LS7 tool offers simple, interpretable, and valid scales and standards for change. Eventually, more public health efforts and interventions may be curated specifically for minority and vulnerable populations that enter clinics across the US with a high number of easily identifiable risk factors.

1.9 Objective

The objective of this study is to assess the relations between limited English proficiency, socioeconomic status, and race/ethnicity with cardiovascular disease risk, using AHA's LS7, using cross-sectional data. We hypothesize that NHANES participants who have limited English proficiency or are in the low SES category will have lower overall LS7 scores when compared to those who are proficient in English or are in the high SES category. Additionally, we will describe the compartmental LS7 scores by race/ethnicity, SES and limited English Proficiency.

2.0 Methods

For this analysis, we used data from participants for one NHANES cycle that spanned from 2015-2016. In order to create the AHA's LS7 outcome variable, 12 different data sets were downloaded from the NHANES website for the 2015-2016 cycle. These data sets include Demographic Variables and Sample Weights, Blood Pressure, Body Measures, Cholesterol – Total, Plasma Fasting Glucose, Glycohemoglobin, Dietary Interview – Individual Foods, First Day, Dietary Interview – Individual Foods, Second Day, Dietary Interview – Total Nutrient Intakes, First Day, Dietary Interview – Total Nutrient Intakes, Second Day, Physical Activity, and Smoking – Cigarette Use.⁴⁵

Creating the LS7 score began with analyzing the diets of each participant for daily and weekly intake of foods. USDA codes were used to identify the specific foods of interest for the LS7 score. USDA codes were obtained from the NHANES data set, but food descriptions and portion sizes were obtained from the USDA Food and Nutrient Database for Dietary Studies (FNDDS).⁴⁶ Fruits and vegetables were identified by the USDA code ranges 61100500 – 67600100 and 71000100 – 78101120, respectively. Portions were calculated as the reported number of grams the participant provided divided by that food code's portion size in grams provided by the USDA FNDDS. The number of servings were calculated for both day 1 and day 2 of the dietary interviews and the mean was calculated.

Whole grains were identified by performing a proximity match for 'whole grain' on the 'Main Food Description' variable obtained from the USDA FNDDS 2015-2016 data set. USDA food codes were not able to be used for the classification of whole grains due to the lack of defined coding. Using the proximity match, 99 unique whole grain descriptions were identified. One

serving of whole grains was interpreted as a 16 g serving. The number of servings of whole grains was calculated over day 1 and day 2 of the interview and the mean was calculated.

For fish intake, participants were interviewed on their monthly intake of a number of different fish types. Each fish type was counted for each participant over the course of the reported month and each report of a fish type eaten was interpreted as being equal to one serving. The monthly fish count was then divided by four to obtain an estimate for the weekly intake of fish for each participant.

For sugar-sweetened beverages, intake was defined by USDA codes. The following list of USDA codes were used to identify sugar-sweetened beverages: 92101820, 92101900-92102110, 92102450-92104000, 92121000-92193025, 92305040-92305110, 92308000-92308010, 92400000-92900300, 94100200-94100300, and 95310200-95342000. In the NHANES Dietary data set, beverages are only reported in grams. Grams were converted to fluid ounces by assuming 30 grams was equal to 1 fluid ounce across all beverage types. In order to calculate weekly intake, the mean of the two dietary interview days was calculated and then multiplied by 3.5.

Daily intake of sodium was calculated through the use of the Dietary Interview – Total Nutrient Intakes, First / Second Day data sets. Mean sodium intake was calculated over the two-day period and was already reported in milligrams by NHANES.

The components of each dietary component were then assessed according to the definitions provided by the American Heart Association.²⁹ To reiterate, these components are as follows: consume ≥ 4.5 cups/d of fruits and vegetables, ≥ 2 servings/wk of fish, ≥ 3 servings/d of whole grains, no more than 36 oz/wk of sugar-sweetened beverages, and no more than 1500 mg/d of sodium. Participants then received a diet LS7 score that ranged from 0 to 2 based on the number

of components they had: 0 to 1 component were allotted 0 points for poor, 2-3 components allotted 1 point for intermediate, and 4-5 components allotted 2 points for ideal.

Next, the LS7 component of physical activity (PA) was classified according to the AHA's definitions. The amount of vigorous and moderate PA in minutes per week was calculated by multiplying the reported number of days for each activity type by the respective average number of minutes for each day. Vigorous and moderate activity were asked in the questionnaire based on it being performed at work, on the way to work, or recreationally. All of these categories were weighed equally. Participants received a PA LS7 component score of 2 for reporting more than 75 minutes per week of vigorous activity or 150 minutes of moderate activity. Participants received a score of 1 for reporting less than 75 minutes of vigorous activity or 150 minutes of moderate activity. Participants received a score of 0 if they reported not participating in any vigorous or moderate activity over the last week.

Importantly, fasting plasma glucose was not assessed in this analysis due to the high proportion of missing data for that variable. Similar to other studies, we used blood glycohemoglobin in place of fasting plasma glucose and split it into three scoring categories: poor was classified as $\geq 6.5\%$ glycohemoglobin (HbA1c), intermediate was classified as $\geq 5.7\%$ but $< 6.5\%$ glycohemoglobin, and ideal was classified as $< 5.7\%$ glycohemoglobin.⁴⁷ The 2015 American Diabetes Association defines diabetes as $Hb1Ac \geq 6.5\%$.⁴⁸

For the other components of LS7, body mass index (BMI) was calculated by NHANES as the participant's weight (kg) divided by their height (m) squared. Blood pressure was measured using mercury sphygmomanometers and all measurements provided for participant by NHANES were averaged and then classified by the AHA definitions. The smoking component was classified as ideal if a participant responded 'No' to smoking at least 100 cigarettes over the course of their

lifetime as a large proportion of data is missing for the follow-up question ‘Ever smoked a cigarette even 1 time?’. Participants were assigned an intermediate score for quitting smoking within the last 12 months while those currently smoking received a poor score. Total cholesterol was classified according to the AHA definition.²⁹ The components of LS7 were then summed for each participant into a variable that ranges from 0 to 14.

After creation of the LS7 score, the predictor variables of interest were included into the aggregated data set. These variables included ratio of family income to poverty, education, socioeconomic status (SES), limited English proficiency, marital status, gender, and age. The classification of these variables is defined in the following table:

Table 2. Predictor Variable Classifications.

Variable	Classification	Definition	Notes
Ratio of Family Income to Poverty (PIR)	Low	Less than 1.3	1.3 is equal to 130% of the federal poverty line
	Medium	Equal to or greater than 1.3 but less than 3.5	
	High	Equal to or greater than 3.5	
Education	1	Less than 12 years of education	
	2	HS graduate, GED, or equivalent	
	3	Some college	
	4	Completed college or greater	
Education_Dichot	1	Equal to or less than HS / GED	
	2	Greater than HS	
SES	Low	PIR = Low and Education = 1	
	Medium	All combinations of PIR and Education that are not the two lowest or highest values	
	High	PIR = High and Education = 4	
Limited English Proficiency	No	Participant did not take the survey in a language other than English and did not use an interpreter	
	Yes	Participant took the survey in a language other than English or used an interpreter	

The data set was then finalized for the analysis phase of the project. Participants under the age of 25 or did not complete both the interview and medical examination component of the survey were excluded (427 participants only completed the survey). Participants with missing data for either components of the LS7 score or the exposures of interest were kept in the dataset.

All analyses utilized the complex survey procedures in SAS 9.4 (SAS Institute) where sample weights were incorporated to produce nationally representative estimates. Additionally, the complexity of the NHANES survey design was taken into account through the inclusion of masked variance pseudo-stratum and masked variance pseudo-PSU variables in all analyses. Continuous variables are expressed as means with the associated standard error or 95% confidence interval (CI) estimated by Taylor Series Linearization. For the regression analyses, missing data for the LS7 score were incorporated into each analysis and considered not missing completely at random by including the 'NOMCAR' option in the 'PROC SURVEYREG' and 'PROC SURVEYLOGISTIC' statements in SAS. This option computes variance estimates by analyzing both missing and non-missing outcomes and treating the non-missing values as a domain or a subpopulation thereby increasing the standard errors of the point estimates.

The distribution of the outcome variable, a continuous LS7 score, was evaluated through the construction of a weighted histogram and tested for normality. The overall LS7 score was compared by race/ethnicity, SES and English proficiency groups using t-tests or ANOVA tests. Weighted histograms were created for each component of the LS7 score and were examined by race/ethnicity, SES and English Proficiency. A dichotomous ideal cardiovascular health variable was constructed for each LS7 component score where ideal was coded as 1 and poor and intermediate was coded as 0. The associations between each of the dichotomous ideal cardiovascular health variables and by race/ethnicity, SES and English Proficiency were assessed using chi-square tests.

Missing data for the outcome variable, the continuous LS7 score, was assessed by comparing characteristics for the sample population with a missing LS7 variable and those with a non-missing LS7 using chi-squared tests and t-tests. Multicollinearity across the predictor

variables of interest was assessed using Spearman rank correlation coefficient for ordinal variables and chi-square for nominal variables. Continuous independent variables, such as age, were grand mean centered.

Using PROC SURVEYREG, weighted unadjusted and multivariable adjusted linear regression was performed to model the continuous LS7 outcome variables based on a series of pre-specified predictor variables. For all analyses, a two-tailed *P* value of <0.05 was considered significant.

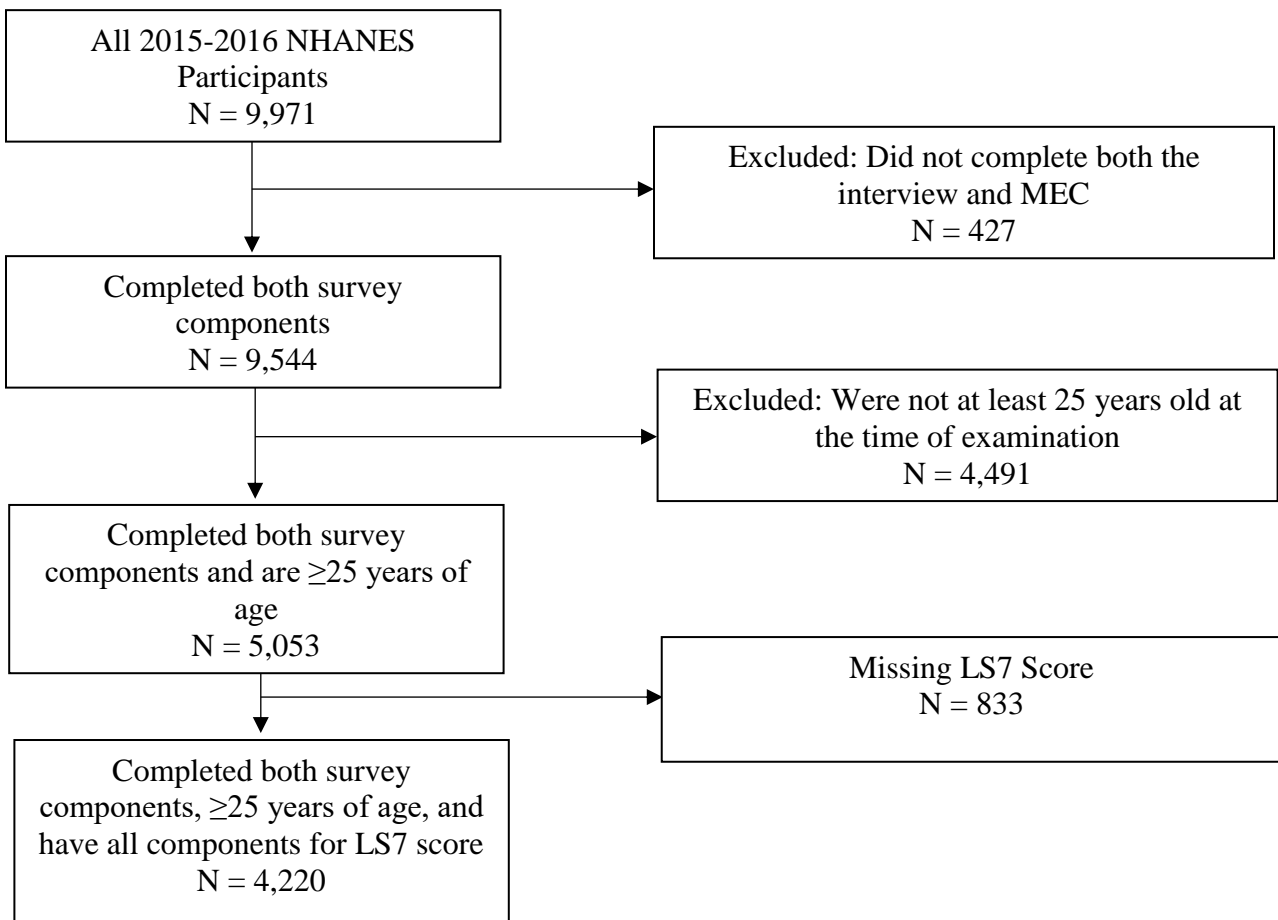


Figure 1. Flow Diagram for the Final Study Sample.

3.0 Results

Table 3 outlines the weighted demographic profiles for the total study sample (n=5,053), the subsample with all components of the AHA's LS7 score (n=4,220), and the subsample of participants who are missing at least one component of the LS7 score (n=833). Table 3 reports crude frequency counts in the sample, and the proportion (%) reflects the proportion of the weighted sample corresponding to the target population.

The demographic profile of the total sample is as follows: mean age is 50.3 years (SE=0.6), 47.8% male, and majority non-Hispanic white ethnicity (64.8%). Most participants had an educational background that went beyond the high school level (65.1%) and were in the medium SES category (68.9%).

A number of significant associations between missingness of the LS7 score and predictor variables were detected: race/ethnicity ($p<0.01$), education ($p=0.01$), PIR ($p<0.01$), SES ($p<0.01$), marital status ($p=0.01$), use of an interpreter ($p<0.01$), and limited English proficiency ($p<0.01$). The most notable differences between the non-missing and missing samples are the lower proportion of non-Hispanic (NH) Whites (66.5% vs 53.1%) and the higher proportions of the NH Asians (4.9% vs 12.3%), less than 12 years education (13.8% vs 21.2%), low PIR and low SES (18.9% vs 28.6% and 6.2% vs 12.2%, respectively), and use of an interpreter (2.1% vs 6.6%) in those with missing LS7 measures.

Table 3. Demographic Profiles of Total, Non-missing LS7, and Missing LS7 Samples.

	Total Population	Non-missing LS7 Score	Missing LS7 Score	
	Mean (SE) or Freq (%)	Mean (SE) or Freq (%)	Mean (SE) or Freq (%)	P-value**, weighted
Age (years)	50.3 (0.6)	50.4 (0.6)	50.2 (1.3)	0.90
Male	2420 (47.8)	2018 (47.7)	402 (49.0)	0.67
Race/ethnicity (n=5,053)				0.01
Mexican American	878 (8.6)	761 (8.6)	117 (8.7)	
Other Hispanic	670 (6.2)	569 (6.0)	101 (7.1)	
NH White	1667 (64.8)	1452 (66.5)	215 (53.1)	
NH Black	1063 (11.0)	864 (10.4)	199 (15.4)	
NH Asian	601 (5.9)	427 (4.9)	174 (12.3)	
Other	174 (3.5)	147 (3.6)	27 (3.4)	
Education (n=5,050)				0.01
Less than 12	1239 (14.8)	984 (13.8)	255 (21.2)	
GED or equiv	1071 (20.1)	931 (20.7)	140 (16.3)	
Some college	1441 (31.4)	1210 (31.7)	231 (29.6)	
College grad+	1299 (33.7)	1095 (33.9)	204 (32.8)	
Education_Dichot (n=5,050)				0.15
≤HS	2310 (34.9)	1915 (34.5)	395 (37.6)	
>HS	2740 (65.1)	2305 (65.5)	435 (62.4)	
PIR (n=4,523)				0.01
Low	1438 (20.1)	1161 (18.9)	277 (28.6)	
Medium	1792 (36.1)	1545 (36.4)	247 (34.3)	
High	1293 (43.8)	1121 (44.8)	172 (37.2)	
SES (n=4,521)				0.01
Low	618 (7.0)	481 (6.2)	137 (12.2)	
Medium	3220 (68.9)	2756 (69.3)	464 (65.9)	
High	683 (24.1)	590 (24.5)	93 (21.9)	
Marital Status (n=5,051)				0.01
Married	2727 (58.1)	2291 (59.0)	436 (52.3)	
Widowed	396 (6.5)	305 (5.9)	91 (10.4)	
Divorced	577 (10.5)	490 (10.4)	87 (10.9)	
Separated	180 (2.7)	160 (2.7)	20 (2.3)	
Never Married	715 (13.3)	587 (12.9)	128 (15.7)	
Living w/ partner	456 (9.0)	386 (9.1)	70 (8.4)	

Table 3 Continued

Language of Interview (n=5,053)				0.88
English	4271 (92.9)	3545 (93.0)	726 (92.8)	
Spanish	782 (7.1)	675 (7.0)	107 (7.2)	
Interpreter (n=5,053)				0.01
Yes	289 (2.7)	197 (2.1)	92 (6.6)	
No	4764 (97.3)	4023 (97.9)	741 (93.4)	
Limited English Proficiency (n=5,053)				0.01
Yes	4050 (90.7)	3413 (91.4)	637 (86.3)	
No	1003 (9.3)	807 (8.6)	196 (13.7)	

*Frequencies reported are unweighted in that they are the count in the NHANES population, but proportions determined from those frequencies are weighted.

**p-values based on weighted t-tests or chi-square tests.

Table 4 shows sample characteristics stratified by one of the main predictor variables of interest, limited English proficiency. As expected, large differences between demographic profiles exist based on this variable. Those with limited English proficiency are more likely to be male (51.5% vs 47.3%, $p=0.01$), of Mexican American or other Hispanic background (49.5% vs 4.8% and 33.4% vs 3.4%, $p<0.01$), of a lower educational level background (78.4% vs 30.3%, $p<0.01$), and to be in the lower categories for both PIR and SES (58.1% vs 15.6%, $p<0.01$ and 38.7% vs 3.5%, $p<0.01$, respectively).

Table 4. Demographic Characteristics Stratified by Limited English Proficiency.

	Total (N=4220)	Non-Limited English Proficiency (n=3413)	Limited English Proficiency (n=807)	P-value, weighted
Age (years)	50.4 (0.6)	50.6 (0.6)	48.3 (1.7)	0.22
Male	2018 (47.7)	1631 (47.3)	387 (51.5)	0.01
Race/ethnicity (n=5,053)				<0.01
Mexican American	761 (8.6)	366 (4.8)	395 (49.5)	
Other Hispanic	569 (6.0)	283 (3.4)	286 (33.4)	
NH White	1452 (66.5)	1447 (72.6)	5 (2.1)	
NH Black	864 (10.4)	851 (11.2)	13 (1.5)	
NH Asian	427 (4.9)	323 (4.2)	104 (12.8)	
Other	147 (3.6)	143 (3.8)	4 (0.65)	
Education (n=5,050)				<0.01
Less than 12	984 (13.8)	475 (9.4)	509 (60.9)	
GED or equiv	931 (20.7)	801 (21.0)	130 (17.5)	
Some college	1210 (31.7)	1102 (33.2)	108 (14.7)	
College grad+	1095 (33.9)	1035 (36.4)	60 (6.9)	
Education_Dichot (n=5,050)				<0.01
≤HS	1915 (34.5)	1276 (30.3)	639 (78.4)	
>HS	2305 (65.5)	2137 (69.7)	168 (21.6)	
PIR (n=4,523)				<0.01
Low	1161 (18.9)	747 (15.6)	414 (58.1)	
Medium	1545 (36.4)	1312 (36.4)	233 (36.3)	
High	1121 (44.8)	1081 (48.1)	40 (5.6)	
SES (n=4,521)				<0.01
Low	481 (6.2)	199 (3.5)	282 (38.7)	
Medium	2756 (69.3)	2356 (70.0)	400 (60.7)	
High	590 (24.5)	585 (26.5)	5 (0.6)	
Marital Status (n=5,051)				0.01
Married	2291 (59.0)	1790 (58.7)	501 (61.3)	
Widowed	305 (5.9)	256 (6.1)	49 (4.2)	
Divorced	490 (10.4)	428 (10.8)	62 (6.2)	
Separated	160 (2.7)	111 (2.4)	49 (5.8)	
Never Married	587 (12.9)	534 (13.4)	53 (7.9)	
Living w/ partner	386 (9.1)	293 (8.6)	93 (14.6)	

*Note: This table is representative of those who have a LS7 score.

The mean LS7 score in the study population is 8.43 (95% CL: 8.24, 8.62) and Figure 2 portrays the distribution of the score as approximately normal. Although normality was rejected for the distribution of the continuous LS7 score (Kolmogorov-Smirnov Goodness-of-Fit test, $p < 0.01$), the large sample size combined with the unimodal distribution with minor skew validates further analysis of the variable as approximately normally distributed.

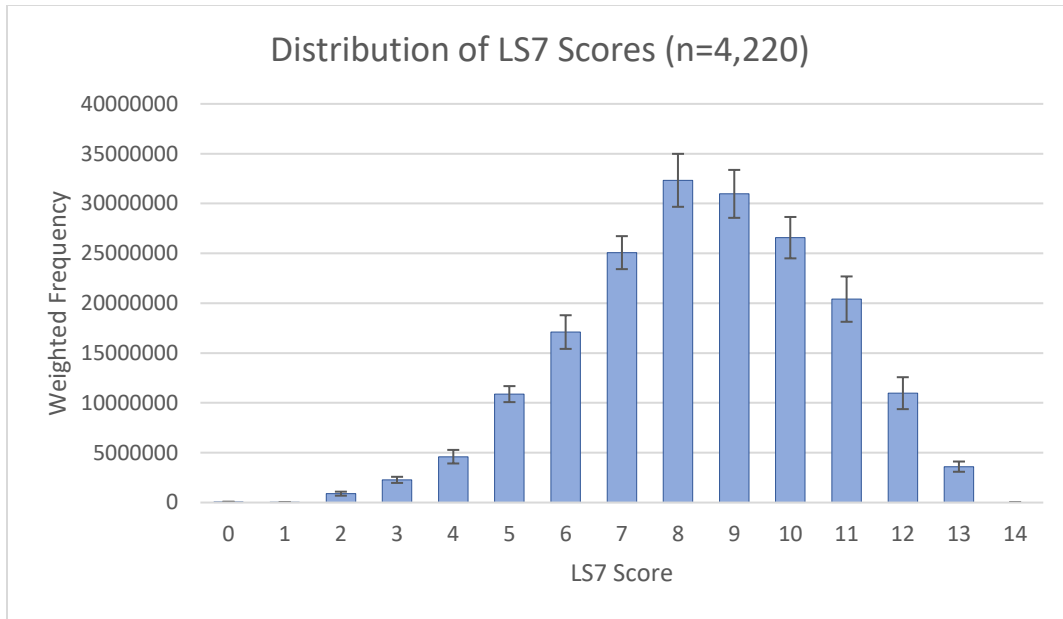


Figure 2. Distribution of LS7 Score Across the Final Population with Weighted Frequencies and Standard Deviations.

Table 5 shows the LS7 means by race/ethnicity, SES, and limited English proficiency. Means for LS7 score varied significantly by race/ethnicity ($p < 0.01$), SES ($p < 0.01$), and limited English proficiency ($p = 0.03$). Mexican Americans and NH Blacks had the lowest mean LS7 scores (7.89 and 7.88, respectively) across all racial/ethnic groups. Participants in the low SES strata had the lowest mean LS7 score across all groups in Table 5. Participants who have limited English proficiency, on average, had 0.42 lower LS7 scores when compared to participants proficient in English.

Table 5. Mean LS7 by Race/Ethnicity, SES, and Limited English Proficiency.

	LS7 Score Mean (SD)	P-value, weighted
Race/ethnicity		<0.01
Mexican American	7.89 (0.24)	
Other Hispanic	8.27 (0.27)	
NH Black	7.88 (0.17)	
NH Asian	9.34 (0.22)	
Other	8.13 (0.41)	
NH White	8.55 (0.18)	
SES		<0.01
Low	7.43 (0.25)	
Middle	8.25 (0.18)	
High	9.32 (0.24)	
Limited English Proficiency		0.03
Yes	8.04 (0.37)	
No	8.46 (0.17)	

*P-values calculated by ANOVA and t-tests, as appropriate.

Table 6 presents the LS7 score distribution for the compartmentalized scoring categories. The proportion of the sample and population that are characterized as ideal, intermediate and poor varies widely across the seven characteristics. For example, the majority of the study population had an ideal status for the smoking score (79%), but almost no participants had an ideal status for the dietary score (0.3%). A relatively small proportion of the population had an ideal status for BMI (26%). A majority of participants had an ideal status for the categories of physical activity (63%), total cholesterol (58%), and glycohemoglobin (HbA_{1c}) (63%). Blood pressure ideal health status was calculated for 42% of the population.

Table 6. Compartmental LS7 Score Distribution Across All Participants.

	Frequency (UNW)	Weighted Freq	Std Err of Weighted Freq	Percent	Std Err of Percent
Smoking					
Current	953	39221069	1698516	18.48	0.97
Former (≤12 mo.)	116	5253214	607594	2.48	0.27
Never or quit >12 months	3919	167712468	11104973	79.04	1.09
BMI					
≥30	2061	86410321	5803604	40.70	1.62
25-29.9	1621	68470793	4163253	32.25	0.55
18.5-24.9	1255	55087255	4547158	25.95	1.52
PA					
None	1414	47800662	2998587	22.33	1.43
Intermediate	758	32112591	2594777	15.00	0.63
Ideal	2869	134157986	8932282	62.67	1.32
Diet					
0-1 components	3187	140001682	8675349	69.69	1.32
2-3 components	1436	60254150	4353444	29.99	1.28
4-5 components	9	627228	247500	0.31	0.12
Total Cholesterol					
≥240 mg/dL	578	27221139	2896408	13.28	0.94
200-239	1320	58839803	5175440	28.71	1.01
<200	2875	118880137	5317965	58.01	1.52
Blood Pressure					
SBP≥140 or DBP≥90	1049	37821622	2870936	18.05	0.90
SBP 120-139 or DBP 80-89	1972	82942254	4542258	39.58	1.23
SBP<120 or DBP<80	1892	88770237	6706440	42.37	1.55
HbA _{1c}					
≥6.5	693	20860779	1277229	10.11	0.90
5.7-6.5	1494	55265619	3245419	26.78	0.94
<5.7	2630	130257388	9901056	63.11	1.59

Evaluation of multicollinearity between predictor variables of interest revealed that education, PIR, and SES were highly associated with one another (Spearman correlation coefficient p -values < 0.01 for all relations). Table 4 shows the associations between limited English proficiency and gender, race, education, PIR, SES, and marital status.

The results from the linear regression models are shown in Tables 7 and 8. Models A, E, and H include the predictor variable of interest, limited English proficiency. The unadjusted model (Model A) (Table 7) demonstrates that limited English proficiency is associated with a 0.42 lower estimated LS7 score ($p=0.03$). Adjusting for age, race/ethnicity and additional demographic factors (Model E and Model H) (Table 8), the association between the LS7 score and limited English proficiency is attenuated and not statistically significant. Due to the very small number of participants that had high SES and limited English proficiency, SES was coded as a dichotomous variable (high/medium versus low) in the model that included both factors. This model (Model H) had an R^2 of 0.131.

Socioeconomic status was strongly associated with LS7 score. When compared to participants in the high SES category, participants in the middle SES category had estimated LS7 scores that were 0.89 lower (95% CI: -1.24, -0.54, $p < 0.01$) and those in the low SES category 1.41 lower (95% CI: -1.79, -1.02, $p < 0.01$) adjusting for age, race/ethnicity and other demographic factors. The multivariable model including the three-level SES variable, model E, had an R^2 value of 0.158. The multivariable model including PIR and education, rather than SES, had a relatively similar R^2 value of 0.160.

The relation between LS7 score and SES and was further explored by examining the individual components of the LS7 score by SES category. Figures 3 and 4 show the results from

this analysis. The greatest differences across SES categories appeared to be in the smoking, physical activity, diet, and glycohemoglobin components.

Table 7. Regression Parameters, Estimates, and 95% Confidence Intervals for Univariate Models.

Parameter	Model A Coefficient (95% CI)	Model B Coefficient (95% CI)	Model C Coefficient (95% CI)
Intercept	8.46 (8.28, 8.65)	9.03 (8.86, 9.20)	9.32 (9.05, 9.58)
Limited English Proficiency	-0.42 (-0.79, -0.06)*		
Poverty-Income Ratio			
Low		-0.66 (-1.04, -0.29)*	
Medium		-0.42 (-0.70, -0.13)*	
Education			
≤HS		-0.87 (-1.12, -0.61)**	
SES			
Low			-1.89 (-2.27, -1.52)**
Medium			-1.06 (-1.43, -0.70)**
R-Squared	0.00285	0.063	0.056

* p<0.05, ** p<0.001, ¹ Variables centered to the mean, References include: proficient in English, female, NH White, PIR and SES (High), Education (>HS).

Table 8. Regression Parameters, Estimates, and 95% Confidence Intervals for Multivariable Models.

Parameter	Model D Coefficient (95% CI)	Model E Coefficient (95% CI)	Model F Coefficient (95% CI)	Model G Coefficient (95% CI)	Model H Coefficient (95% CI)
Intercept	8.84 (8.63, 9.05)	8.84 (8.63, 9.05)	9.43 (9.14, 9.71)	9.15 (8.93, 9.37)	8.85 (8.63, 9.07)
Limited English Proficiency		-0.10 (-0.32, 0.13)			0.13 (-0.15, 0.40)
Age ¹	-0.04 (-0.05, -0.04)**	-0.04 (-0.05, -0.04)**	-0.04 (-0.05, -0.04)**	-0.04 (-0.05, -0.03)**	-0.04 (-0.050, -0.04)**
Gender (ref=F)	-0.13 (-0.29, 0.04)	-0.12 (-0.29, 0.04)	-0.10 (-0.27, 0.08)	-0.08 (-0.25, -0.09)	-0.11 (-0.27, 0.05)
Race/ethnicity					
Mexican American	-0.96 (-1.19, -0.73)**	-0.91 (-1.14, -0.69)**	-0.67 (-0.95, -0.39)**	-0.55 (-0.84, -0.27)**	-0.91 (-1.19, -0.63)**
Other Hispanic	-0.48 (-0.76, -0.19)*	-0.43 (-0.67, -0.19)*	-0.28 (-0.56, -0.01)*	-0.23 (-0.50, 0.05)	-0.48 (-0.75, -0.20)*
NH Black	-0.78 (-0.94, -0.62)**	-0.78 (-0.94, -0.62)**	-0.69 (-0.86, -0.53)**	-0.66 (-0.81, -0.51)**	-0.83 (-0.99, -0.66)**
NH Asian	0.46 (0.22, 0.70)**	0.49 (0.26, 0.72)**	0.46 (0.23, 0.69)**	0.55 (0.32, 0.78)**	0.53 (0.27, 0.78)**
Other race	-0.50 (-1.00, -0.002)*	-0.50 (-1.00, -0.001)*	-0.43 (-0.96, 0.09)	-0.44 (-0.91, 0.036)	-0.53 (-1.03, -0.03)*
PIR					
Low				-0.56 (-0.94, -0.17)*	
Medium				-0.38 (-0.66, -0.41)*	
Education					
≤HS				-0.66 (-0.90, -0.41)**	
SES					
Low			-1.41 (-1.79, -1.02)**		
Medium			-0.89 (-1.24, -0.54)**		
SES (ref=Low)					-0.69 (-0.94, -0.43)**
Marital Status					
Widowed	-0.30 (-0.61, 0.01)	-0.30 (-0.61, 0.01)	-0.05 (-0.39, 0.29)	-0.01 (-0.37, 0.35)	-0.23 (-0.55, 0.10)
Divorced	-0.60 (-0.87, -0.32)**	-0.60 (-0.87, -0.32)**	-0.41 (-0.69, -0.12)*	-0.43 (-0.72, -0.14)*	-0.55 (-0.84, -0.25)**
Separated	-1.06 (-1.55, -0.56)**	-1.06 (-1.55, -0.56)**	-0.87 (-0.14, -0.35)*	-0.77 (-1.31, -0.23)*	-0.99 (-1.52, -0.45)**
Never Married	-0.19 (-0.55, 0.17)	-0.19 (-0.55, 0.17)	-0.08 (-0.45, 0.30)	-0.05 (-0.44, 0.34)	-0.14 (-0.52, 0.25)
Living with Partner	-0.35 (-0.73, 0.03)	-0.35 (-0.73, 0.03)	-0.21 (-0.55, 0.12)	-0.20 (-0.55, 0.16)	-0.29 (-0.66, 0.08)
R-Squared	0.126	0.126	0.158	0.160	0.131

* p<0.05, ** p<0.001, ¹ Variables centered to the mean, References include: proficient in English, female, NH White, PIR and SES (High), Education (>HS).

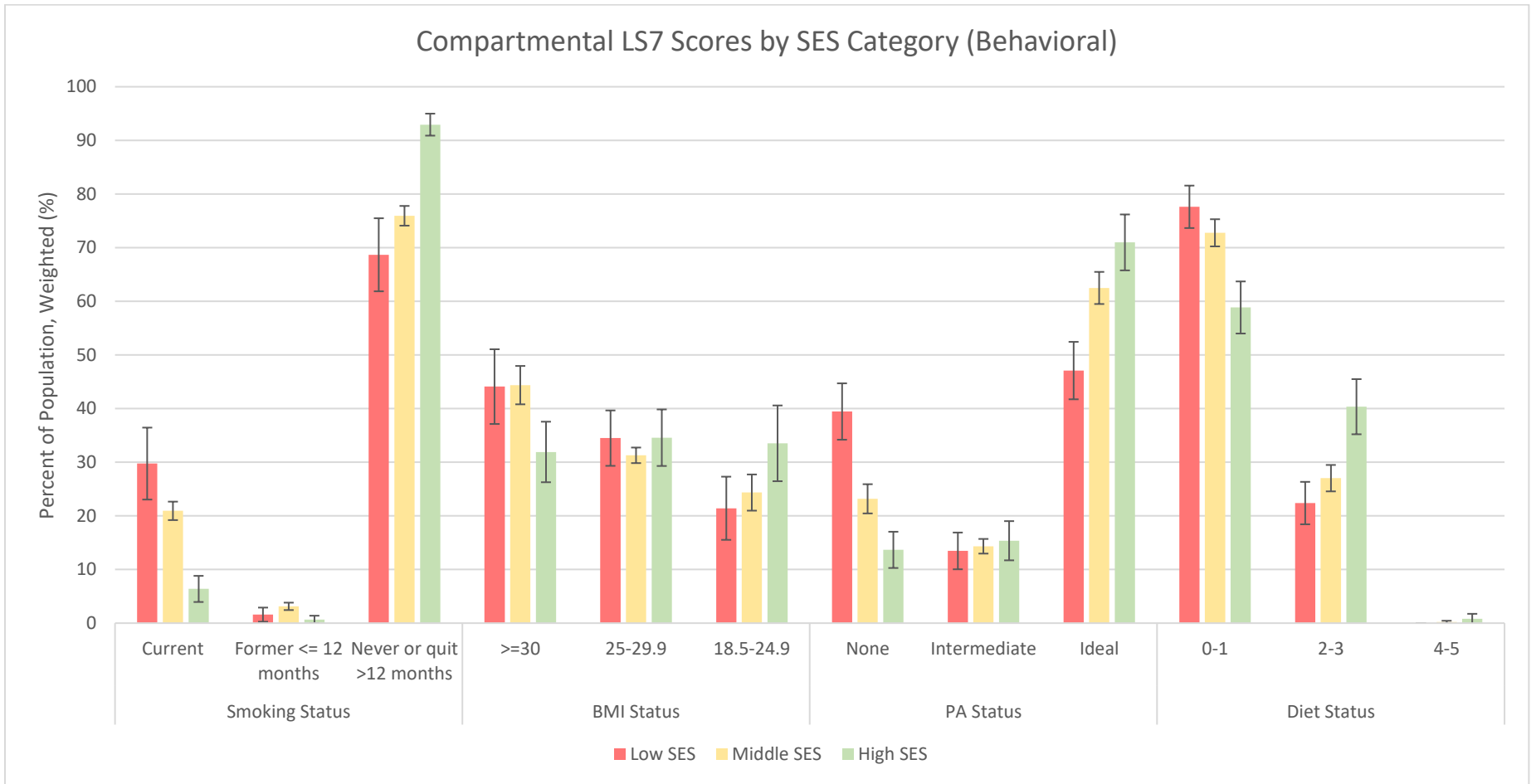


Figure 3. Compartmental LS7 Scores by SES Category.

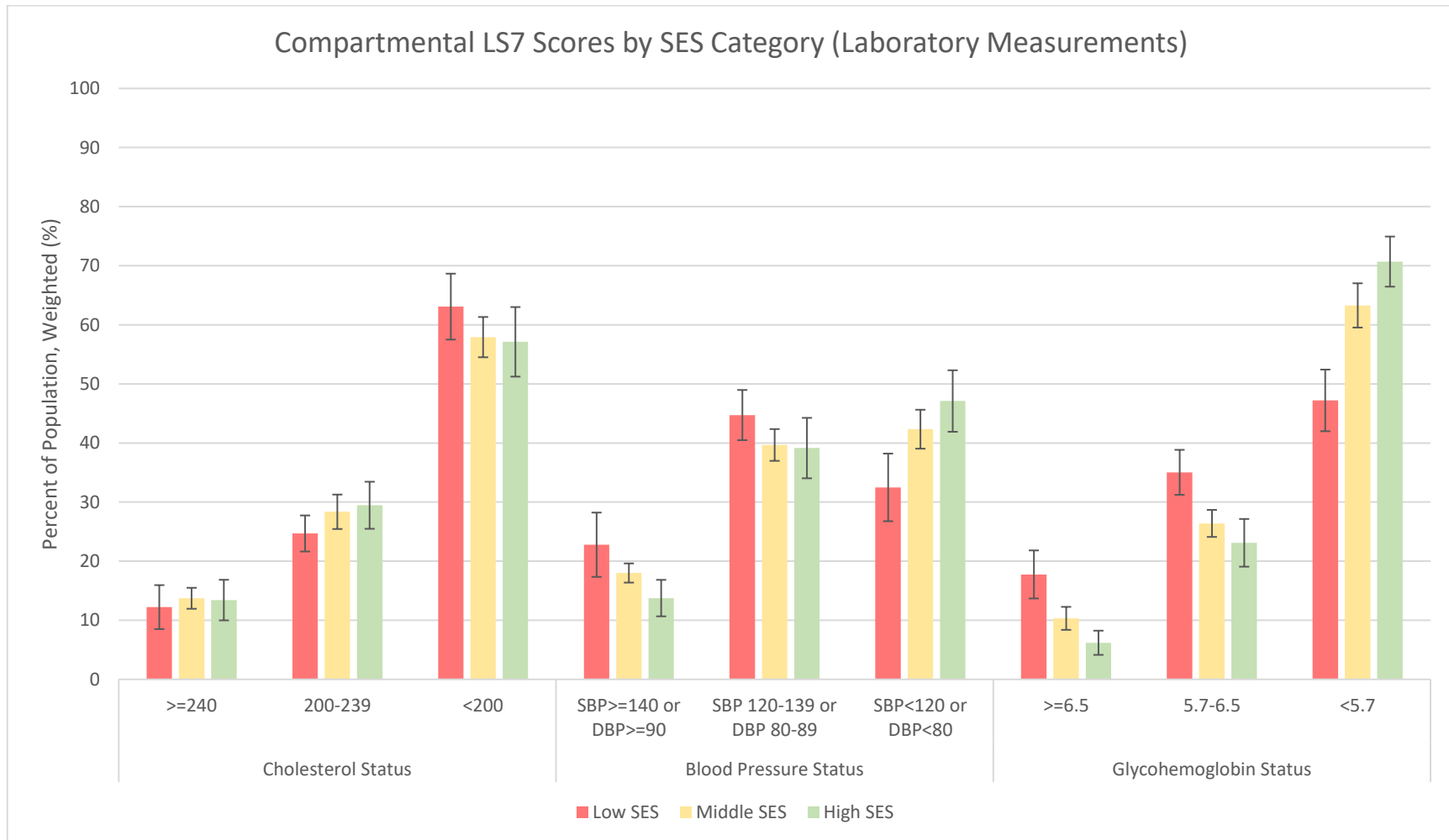


Figure 4. Compartmental LS7 Scores by SES Category (Laboratory Measurements).

Figures 5-11 show smoking status, BMI, PA, diet, total cholesterol, blood pressure, glycohemoglobin by race/ethnicity.

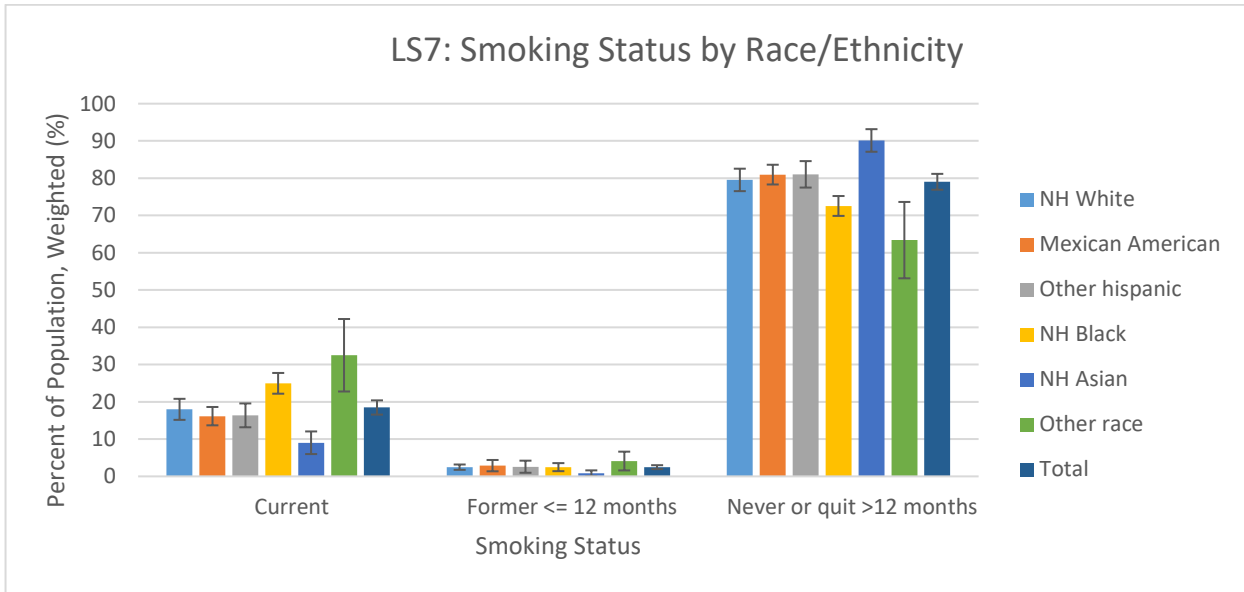


Figure 5. LS7: Smoking Status by Race/Ethnicity.

Distribution of smoking status stratified by ethnicity with standard deviations (95% confidence intervals calculated as $\pm 1.96 \times$ standard error), weighted. Standard deviation calculations include uncertainty from the population missing a LS7 score.

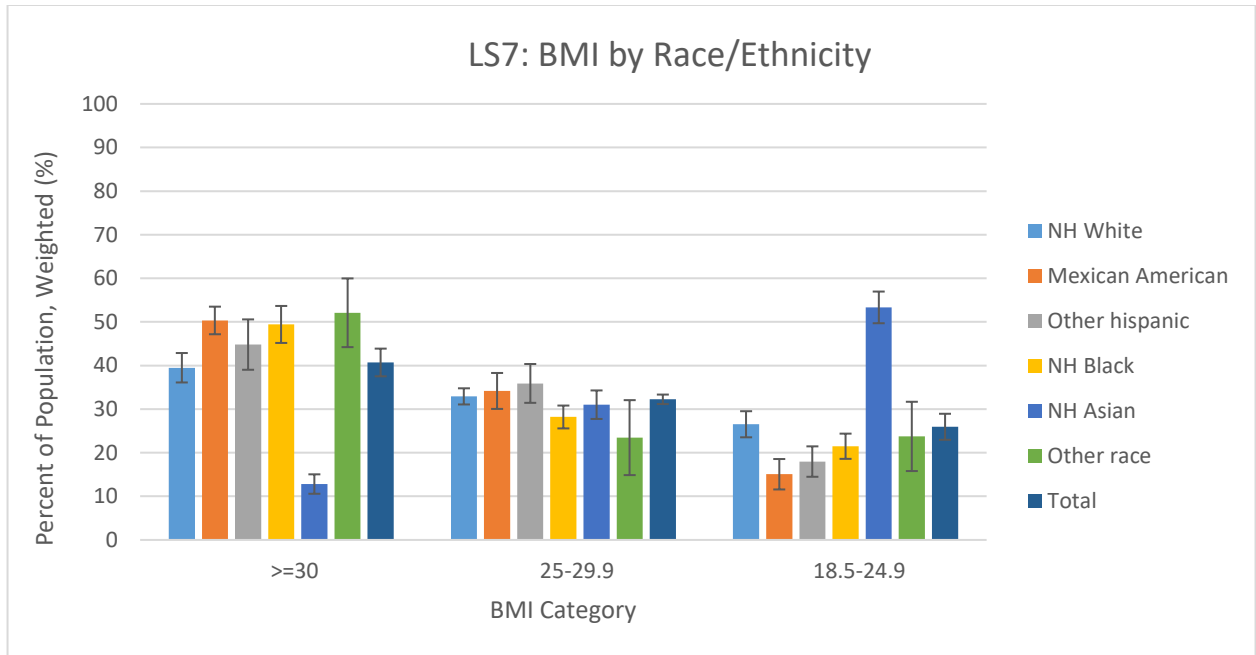


Figure 6. LS7: BMI by Race/Ethnicity.

Distribution of BMI status stratified by ethnicity with standard deviations (95% confidence intervals calculated as $\pm 1.96 \times$ standard error), weighted. Standard deviation calculations include uncertainty from the population missing a LS7 score.

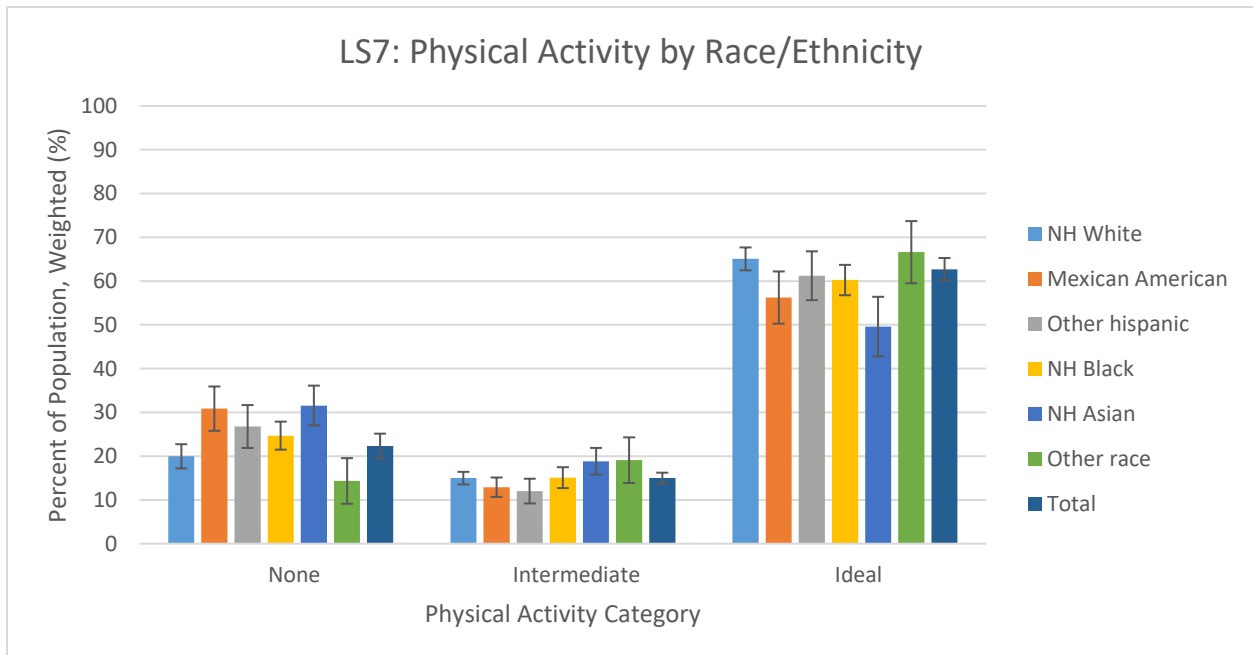


Figure 7. LS7: Physical Activity by Race/Ethnicity.

Distribution of PA status stratified by ethnicity with standard deviations (95% confidence intervals calculated as $\pm 1.96 \times$ standard error), weighted. Standard deviation calculations include uncertainty from the population missing a LS7 score.

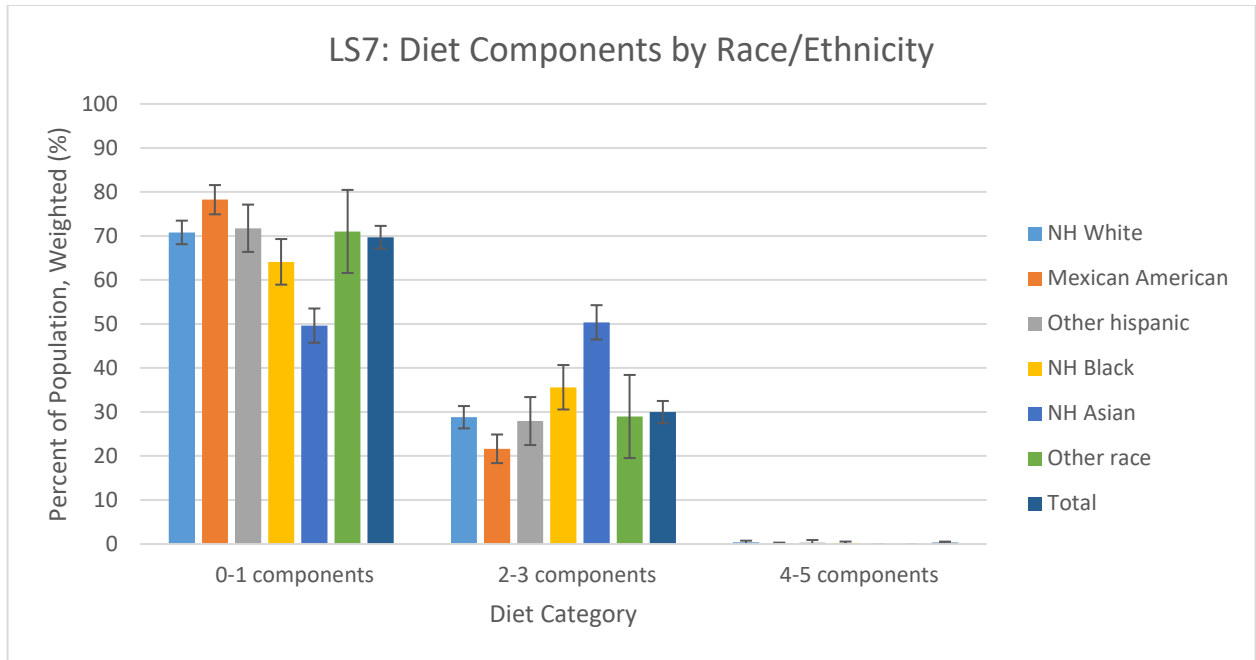


Figure 8. LS7: Diet Components by Race/Ethnicity.

Distribution of diet status stratified by ethnicity with standard deviations (95% confidence intervals calculated as $\pm 1.96 \times \text{standard error}$), weighted. Standard deviation calculations include uncertainty from the population missing a LS7 score.

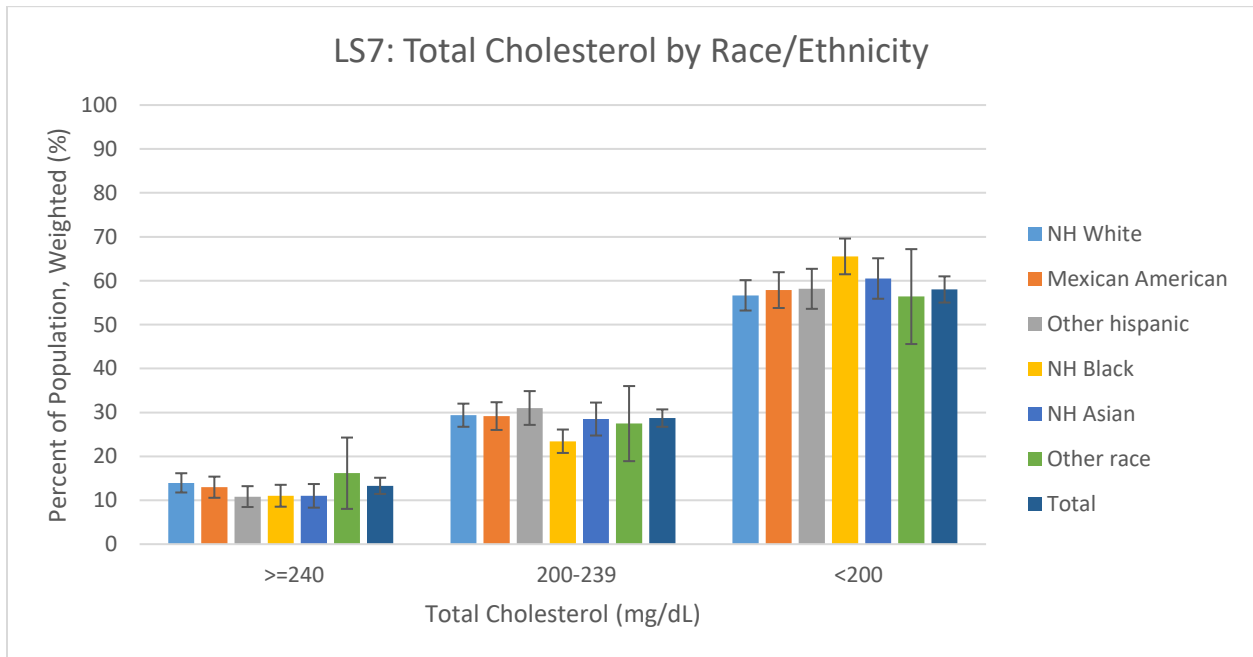


Figure 9. LS7: Total Cholesterol by Race/Ethnicity.

Distribution of total cholesterol status stratified by ethnicity with standard deviations (95% confidence intervals calculated as $\pm 1.96 \times \text{standard error}$), weighted. Standard deviation calculations include uncertainty from the population missing a LS7 score.

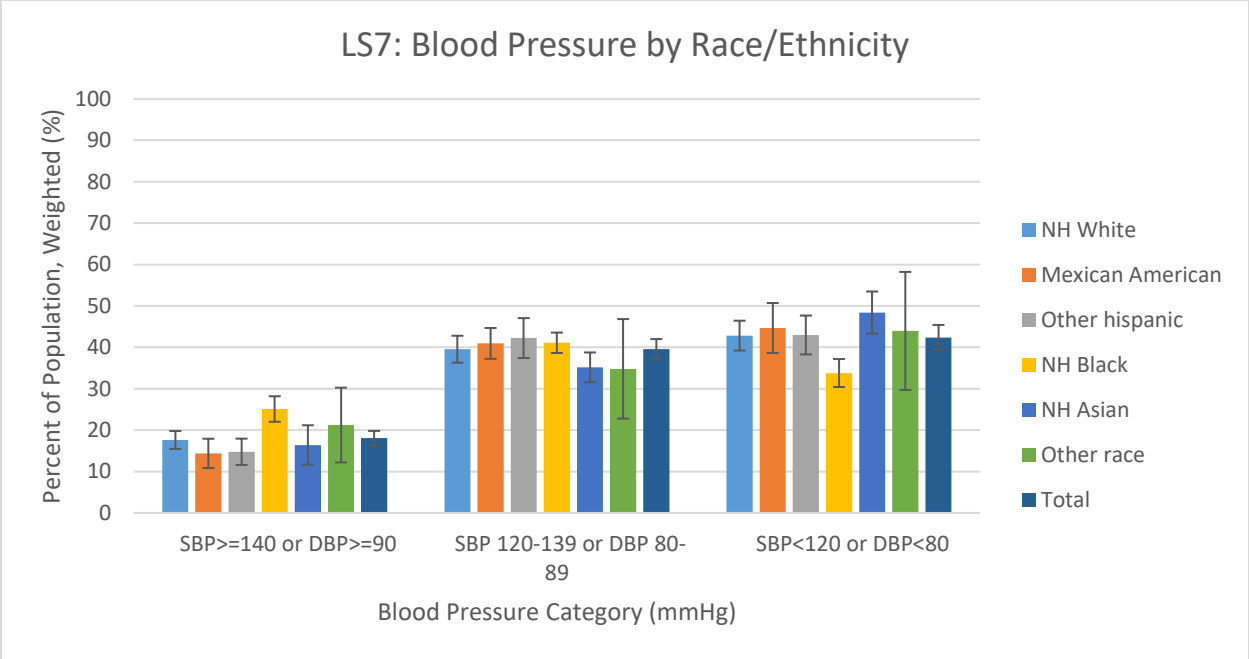


Figure 10. LS7: Blood Pressure by Race/Ethnicity.

Distribution of blood pressure status stratified by ethnicity with standard deviations (95% confidence intervals calculated as $\pm 1.96 \times \text{standard error}$), weighted. Standard deviation calculations include uncertainty from the population missing a LS7 score.

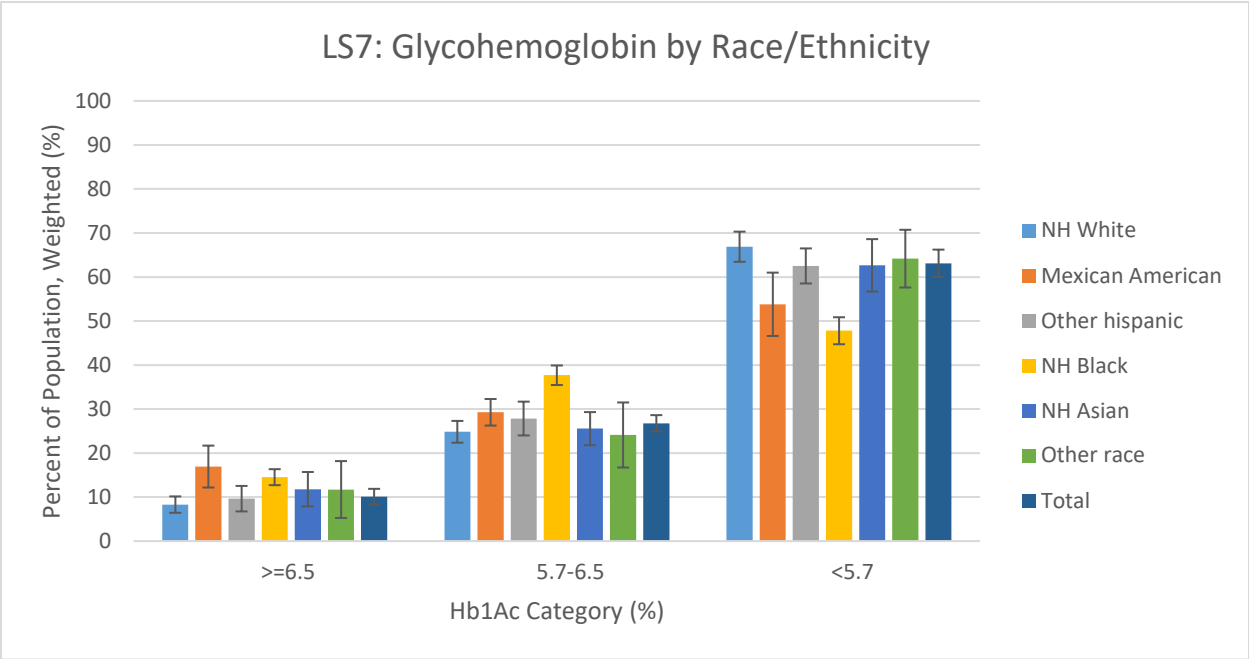


Figure 11. LS7: Glycohemoglobin by Race/Ethnicity.

Distribution of glycohemoglobin status stratified by ethnicity with standard deviations (95% confidence intervals calculated as $\pm 1.96 \times \text{standard error}$), weighted. Standard deviation calculations include uncertainty from the population missing a LS7 score.

Notably, the NH Black subpopulation had lower rates of ideal cardiovascular health in the smoking, BMI, blood pressure, and glycohemoglobin LS7 components when compared to NH Whites ($p < 0.01$ for all). The NH Asian subpopulation had significantly higher rates of ideal cardiovascular health in the BMI and smoking components ($p < 0.01$ for both) and lower rates of ideal cardiovascular health in the physical activity component ($p < 0.01$). Overall, the other Hispanic and multiracial ethnicities were the most similar to the NH White reference group across all components.

4.0 Discussion

The objective of this study was to assess the relations between limited English proficiency, socioeconomic status, and race/ethnicity with cardiovascular disease risk calculated by AHA's LS7. We hypothesized that NHANES participants who have limited English proficiency or are in the low SES category will have lower LS7 scores when compared to those who are proficient in English or are in the high SES category. Our study was able to address these hypotheses and contribute meaningfully to the field of public health through valid and generalizable findings on the relation between various social risk factors and LS7 score.

This study first included an analysis of the missing outcome data for our selected NHANES population. In the total population (n=5,053), a modest proportion of participants were missing one or more components of the LS7 score (n=833, 16.5%), but it is important to note that the proportion with missing data was much higher in certain subpopulations of interest. Many variables of interest were significantly associated with missingness of the outcome variable such as low PIR, low SES, and having limited English proficiency. These findings suggest that the population that is missing a LS7 score is likely inherently different than those who have a score, and the relation between select factors and the LS7 score may vary across different subpopulations.

A substantial portion of the sample who had a non-missing LS7 score (n=4,220) were classified as having limited English proficiency (n=807, 19.1%). This can be attributed to the NHANES oversampling of certain understudied segments of the population to obtain more generalizable estimates. Importantly, limited English proficiency was strongly associated with many other predictor variables of interest with the exception of age. Given the cross-sectional design of this analysis, it is difficult to disentangle the temporal ordering and causal associations

among many of these variables. Race/ethnicity and education are likely associated with limited English proficiency and LS7 score while PIR may be a consequence of limited English proficiency and thus in the causal pathway between limited English proficiency and LS7 score. It is therefore challenging to determine what factors should be included in a multivariable adjusted linear regression model designed to analyze the effect of limited English proficiency on the LS7 score.

The multivariable models adjusting for SES, age, gender, race/ethnicity, and marital status explained 15.8% of the variance in the LS7 score outcome. SES was found to have a stronger relation with LS7 score when compared to limited English proficiency. Participants in the low SES category had the lowest LS7 scores adjusting for other key demographic risk factors. Additionally, PIR and education were strongly associated with a decrease in LS7 score. Limited English proficiency was statistically significant in the unadjusted model, but this relation did not hold when confounders were controlled for in the full models.

An examination of the component LS7 scores confirmed this finding that the proficient in English and limited English proficiency groups were more similar than hypothesized (data not shown). Kim et al. identified differences in elevated blood pressure for limited English proficiency speakers in 2003-2012 NHANES data where the primary outcome of elevated blood pressure was defined as $SBP \geq 140$ mmHg and/or $DBP \geq 90$ mmHg. Here we identify no differences in blood pressure status in the poor, intermediate, or ideal categories among those not proficient in English compared to those who are proficient.⁴⁹ It is possible that selection bias is masking relations between LS7 score and limited English proficiency. Those that participate in the NHANES surveys and do not speak fluent English may be significantly healthier than those who are not proficient in English and do not participate in the survey.

The regression analyses result for race/ethnicity were similar to what was hypothesized and in agreement with the trends identified in the latter compartmentalized LS7 score analysis. In multivariable models, NH Asians had an average 0.46 higher LS7 score than NH Whites while NH Blacks and Mexican Americans had lower average scores when compared to NH Whites (-0.69 and -0.67, respectively). These findings are in agreement with other studies that have analyzed the LS7 score as categorical based on ideal health status, but quantifies the relation on a continuous scale.³¹ In regard to public health significance, the results from our regression analyses further highlight disparities in CVD risk between racial/ethnic groups and SES strata, but not for individuals with limited English proficiency.

The component LS7 score analysis for SES indicates that the components of glycohemoglobin, smoking status, physical activity, and diet are the primary contributors to the difference in scores between low and high SES categories. Specifically, we identify significant disparities in physical activity levels between high and low SES strata. This analysis reiterates the need for public health efforts to focus on reducing smoking rates and the prevalence of diabetes in lower SES populations while promoting the importance of physical activity and a healthy diet. Our findings support the need for interventions advocated by Schultz et al. to engage low SES communities with physical activity programs to reduce CVD risk.¹⁹ Higher levels of physical activity have the potential to influence several other components recognized by LS7 and could be very beneficial in increasing average LS7 scores of low SES participants.

The compartmental LS7 scores by race/ethnicity provide valuable insights that are lost when data is analyzed at the aggregated level. The NH Black and Mexican American subpopulations were less likely to have ideal health status for numerous components while NH

Asians were more likely to have more ideal health status components. The Other Hispanic and Other race groups were similar to the NH White reference population.

The NH Black subpopulation was significantly less likely to have an ideal health status for 6 of the 7 components (the only exception being diet). The rate of current smoking in NH Blacks was significantly higher than that reported in the AHA's Heart Disease and Stroke Statistics 2019 Update (25% vs 16.5%).³ The difference in rates may be attributable to selection bias in our relatively small 2015-2016 NHANES subpopulation or our exclusion of adults under the age of 25. Ideal physical activity status was significantly lower for NH Blacks when compared to NH Whites, but the effect size was not very large (59% vs. 61% for NH Whites). The decreased rates of ideal cardiovascular health observed in the components of glycohemoglobin, blood pressure, and total cholesterol are well documented by other studies and replicated here.

Interestingly, the NH Asian subpopulation had some notable differences in ideal cardiovascular health when compared to NH Whites. The proportion of NH Asians that have ideal BMI as compared with NH Whites was 53.3% vs. 26.5%, but NH Whites reported higher rates of physical activity (61% vs 49%). It is known that BMI is influenced by a large number of factors that are not captured in this study, but rates in the NH Asian population may be easier to compare across time periods. The rates of obesity, defined as having a BMI \geq 30, was higher in this analysis of NH Asians when compared to NHANES data from 2011-2014 (12.8% vs. 11.7%); however, the age groups being compared are slightly different (over 25 years old vs. over 20 years old by the AHA's 2019 Update).³ Overall, the NH Asian population met the most ideal cardiovascular health metrics when compared to all races/ethnicities included in this study for the 2015-2016 NHANES cycle.

Overall, the public health relevance of both the regression analyses and compartmental analyses by SES and race/ethnicity is significant. We identify that individuals that have diverse racial/ethnic backgrounds and/or are from lower SES strata are still experiencing disparities in the modifiable risk factors for CVD specified in LS7. Public health efforts need to continue to engage, empower, and motivate diverse populations to make changes to behaviors that may be placing communities at increased risk for CVD. Because regional and cultural differences exist for both SES and racial/ethnic groups, our analysis cannot fully address the challenges faced by generalized implementation of SES or race into CVD risk prediction models.¹⁹ However, we do highlight general disparities that warrant further investigation into how to reduce CVD risk in diverse groups.

This study has numerous strengths and weaknesses. A major limitation of the study was the cross-sectional design. Cross-sectional studies do not have the ability to establish temporality between exposure and outcome. We cannot know whether certain exposures, such as SES and PIR, came before a low or high CVD risk score. On a similar note, NHANES captures survey data at one point in time and certain exposures are not assessed retrospectively. For example, NHANES reports current PIR but the relation between an earlier exposure to having a low or high PIR on the current outcome status is left unknown.

A degree of reporting bias may be present in the NHANES data. In regard to behaviors, it is not unlikely that some participants withheld information (such as in the diet component) or recalled information incorrectly. Fish intake was questioned over the course of a month and the accuracy of this questionnaire is likely not to be high. Physical activity was another measure subject to report bias. In regard to the effect of report bias on the results, it is likely a safe assumption that participant's tended to over-report healthy behaviors and under-report unhealthy

behaviors. This would cause an artificial inflation in the LS7 scores observed. It is difficult to estimate the scope of the bias present in this study.

In order to address the missingness of the outcome variable, we assumed that missing data were not missing completely at random, and data from participants with missing and non-missing outcomes were used to compute variance estimates. Ultimately, this means that an appropriate level of uncertainty is included in the standard errors, but point estimates are unchanged. Other methods for dealing with missing data such as multiple imputation and inverse probability weighting could be used to adjust the estimates to account for potential bias. In future analyses, one of these methods will be used to obtain more accurate point estimates.

Another strength of this analysis is that the NHANES data allow an efficient way of obtaining nationally representative results for the exposures and outcome of interest. NHANES publishes all of its data to be available to the public and they enable a great deal of research by doing so. NHANES uses professionally trained technicians and clinicians in order to obtain information from participants that is not dependent upon who is collecting the sample or asking the question. In regard to the diet questionnaire, multiple coders were used in order to verify what participants have reported and increase accuracy.

Further analyses could be performed to better understand the relations that may be present between social factors and cardiovascular health. As other studies have done, the outcome could be recoded to count the number of ideal health metrics or number of poor health metrics. Until a validated scale is provided by the AHA, it will be important for researchers to assess the score similarly in order to enable replication and understand relations. A further investigation into the data missing for limited English proficiency NHANES participants may demonstrate that they are missing data that is collected in more lengthy and complex interviews. Additionally, it will be

important to assess the social factors of interest, SES and limited English proficiency, with future designs that incorporate temporal relations to be established.

Cardiovascular disease is the cause of 1 out of 4 deaths in the United States. It is important for researchers to establish risk factors and identify where certain interventions may need to be tailored to certain subpopulations. The public health significance of this project was to identify relations between social risk factors and ideal cardiovascular health to inform the development of future interventions. Although those with limited English proficiency were determined to be at higher cardiovascular disease risk in this study, limited English proficiency was not determined to be a significant independent risk factor for cardiovascular disease. The difference observed between the limited English proficiency and English proficient participants may be attributed to other factors such as age and SES. However, disparities between English speakers and non-English speakers still needs investigation. To reduce the tremendous burden of CVD in the US, it will be crucial to understand that the best prevention strategy across each segment of the population may not be the same.

Bibliography

1. Centers for Disease Control and Prevention. Heart Disease. U.S. Department of Health & Human Services; 2017 [updated November 28, 2017; cited 2019 October 23, 2019]; Available from: <https://www.cdc.gov/heartdisease/facts.htm>.
2. American Heart Association and American Stroke Association. Cardiovascular Disease: A Costly Burden for America - Projections Through 2035. The American Heart Association Office of Federal Advocacy 2017.
3. Benjamin EJ, Muntner P, Alonso A, Bittencourt MS, Callaway CW, Carson AP, et al. Heart disease and stroke statistics - 2019 update: a report from the American Heart Association [published online ahead of print January 31, 2019], and AHACoEaPSC, Stroke Statistics Subcommittee; 2019.
4. Mayo Clinic Staff. Heart disease. Mayo Clinic; 2018 [updated March 22, 2018; cited 2019 October 23, 2019]; Available from: <https://www.mayoclinic.org/diseases-conditions/heart-disease/symptoms-causes/syc-20353118>.
5. ThedaCare. Types of Heart & Vascular Diseases. ThedaCare; 2019 [cited 2019 October 27, 2019]; Available from: <https://www.thedacare.org/Health-Library-and-Conditions/cardiovascular/common-conditions-and-risks/Types-of-Heart-and-Vascular-Diseases.aspx>.
6. New York State Department of Health. Types of Cardiovascular Disease. 2012 [updated December 2012; cited 2019 October 27, 2019]; Available from: https://www.health.ny.gov/diseases/cardiovascular/heart_disease/types_of_cv.htm.
7. Khavjou O, Phelps D, Leib A. Projections of Cardiovascular Disease Prevalence and Costs: 2015-2035: RTI International 2016 November 2016.
8. Nichols GA, Bell TJ, Pedula KL, O'Keefe-Rosetti M. Medical care costs among patients with established cardiovascular disease. *Am J Manag Care*. 2010; 16:e86-e93.
9. Salas-Salvado J, Becerra-Tomas N, Garcia-Gavilan JF, Bullo M, Barrubés L. Mediterranean Diet and Cardiovascular Disease Prevention: What Do We Know? *Prog Cardiovasc Dis*. 2018; 61:62-7.
10. Wahid A, Manek N, Nichols M, Kelly P, Foster C, Webster P, et al. Quantifying the Association Between Physical Activity and Cardiovascular Disease and Diabetes: A Systematic Review and Meta-Analysis. *J Am Heart Assoc*. 2016; 5.
11. Lubin JH, Couper D, Lutsey PL, Yatsuya H. Synergistic and Non-synergistic Associations for Cigarette Smoking and Non-tobacco Risk Factors for Cardiovascular Disease Incidence in the Atherosclerosis Risk In Communities (ARIC) Study. *Nicotine Tob Res*. 2017; 19:826-35.
12. Niiranen TJ, Vasan RS. Epidemiology of cardiovascular disease: recent novel outlooks on risk factors and clinical approaches. *Expert Rev Cardiovasc Ther*. 2016; 14:855-69.
13. Nikpay M, Goel A, Won HH, Hall LM, Willenborg C, Kanoni S, et al. A comprehensive 1,000 Genomes-based genome-wide association meta-analysis of coronary artery disease. *Nat Genet*. 2015; 47:1121-30.
14. Bhatnagar A. Environmental Determinants of Cardiovascular Disease. *Circ Res*. 2017; 121:162-80.

15. Jia X, Yu Y, Xia W, Masri S, Sami M, Hu Z, et al. Cardiovascular diseases in middle aged and older adults in China: the joint effects and mediation of different types of physical exercise and neighborhood greenness and walkability. *Environ Res.* 2018; 167:175-83.
16. Brook RD, Rajagopalan S, Pope CA, 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, et al. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. *Circulation.* 2010; 121:2331-78.
17. Bhatnagar A. Environmental cardiology: studying mechanistic links between pollution and heart disease. *Circ Res.* 2006; 99:692-705.
18. Sommer I, Griebler U, Mahlknecht P, Thaler K, Bouskill K, Gartlehner G, et al. Socioeconomic inequalities in non-communicable diseases and their risk factors: an overview of systematic reviews. *BMC Public Health.* 2015; 15:914.
19. Schultz WM, Kelli HM, Lisko JC, Varghese T, Shen J, Sandesara P, et al. Socioeconomic Status and Cardiovascular Outcomes: Challenges and Interventions. *Circulation.* 2018; 137:2166-78.
20. Graham G. Disparities in Cardiovascular Disease Risk in the United States. *Current Cardiology Reviews.* 2015; 11:238-45.
21. Mackay A, Ashworth M. The role of spoken language in cardiovascular health inequalities: a cross-sectional study of people with non-English language preference. *BJGP Open.* 2017.
22. Motamed N, Rabiee B, Perumal D, Poustchi H, Miresmail SJ, Farahani B, et al. Comparison of cardiovascular risk assessment tools and their guidelines in evaluation of 10-year CVD risk and preventive recommendations: A population based study. *Int J Cardiol.* 2017; 228:52-7.
23. Bonner C, Fajardo MA, Hui S, Stubbs R, Trevena L. Clinical Validity, Understandability, and Actionability of Online Cardiovascular Disease Risk Calculators: Systematic Review. *J Med Internet Res.* 2018; 20:e29.
24. Matheny M MM, Glasser A, Mercaldo N, Weaver RB, Jerome RN, Walden R, McKoy JN, Pritchett J, Tsai C. Systematic Review of Cardiovascular Disease Risk Assessment Tools. Evidence Synthesis No. 85. AHRQ Publication No. 11-05155-EF-12011.
25. D'Agostino RB, Sr., Vasan RS, Pencina MJ, Wolf PA, Cobain M, Massaro JM, et al. General cardiovascular risk profile for use in primary care: the Framingham Heart Study. *Circulation.* 2008; 117:743-53.
26. Mayo Clinic Staff. Claudication. *MayoClinic.org: Mayo Clinic*; 2018; Available from: <https://www.mayoclinic.org/diseases-conditions/claudication/symptoms-causes/syc-20370952>.
27. The American College of Cardiology and American Heart Association. 2013 Prevention Guideline Tools: CV Risk Calculator. *Professional.heart.org: American Heart Association: Professional Heart Daily*; 2013; Available from: https://professional.heart.org/professional/GuidelinesStatements/ASCVDRiskCalculator/UCM_457698.
28. Lloyd-Jones DM, Huffman MD, Karmali KN, Sanghavi DM, Wright JS, Pelser C, et al. Estimating Longitudinal Risks and Benefits From Cardiovascular Preventive Therapies Among Medicare Patients: The Million Hearts Longitudinal ASCVD Risk Assessment Tool: A Special Report From the American Heart Association and American College of Cardiology. *Circulation.* 2017; 135:e793-e813.

29. The American Heart Association. Life's Simple 7. Heart.org: The American Heart Association; 2019; Available from: <https://www.heart.org/en/professional/workplace-health/lifes-simple-7>.
30. Folsom AR, Shah AM, Lutsey PL, Roetker NS, Alonso A, Avery CL, et al. American Heart Association's Life's Simple 7: Avoiding Heart Failure and Preserving Cardiac Structure and Function. *Am J Med.* 2015; 128:970-6 e2.
31. Ogunmoroti O, Oni E, Michos ED, Spatz ES, Allen NB, Rana JS, et al. Life's Simple 7 and Incident Heart Failure: The Multi-Ethnic Study of Atherosclerosis. *J Am Heart Assoc.* 2017; 6.
32. Uijl A, Koudstaal S, Vaartjes I, Boer JMA, Verschuren WMM, van der Schouw YT, et al. Risk for Heart Failure: The Opportunity for Prevention With the American Heart Association's Life's Simple 7. *JACC Heart Fail.* 2019; 7:637-47.
33. Garg PK, O'Neal WT, Chen LY, Loehr LR, Sotoodehnia N, Soliman EZ, et al. American Heart Association's Life Simple 7 and Risk of Atrial Fibrillation in a Population Without Known Cardiovascular Disease: The ARIC (Atherosclerosis Risk in Communities) Study. *J Am Heart Assoc.* 2018; 7.
34. Isakadze N BP, B S, Patel R, Baer J, Isiadinso I, Alonso A, Lloyd M, Sperling L. Life's Simple 7 Approach to Atrial Fibrillation Prevention. *Journal of Atrial Fibrillation.* 2018; 11.
35. Ahmad MI, Chevli PA, Barot H, Soliman EZ. Interrelationships Between American Heart Association's Life's Simple 7, ECG Silent Myocardial Infarction, and Cardiovascular Mortality. *J Am Heart Assoc.* 2019; 8:e011648.
36. Mok Y, Sang Y, Ballew SH, Rebholz CM, Rosamond WD, Heiss G, et al. American Heart Association's Life's Simple 7 at Middle Age and Prognosis After Myocardial Infarction in Later Life. *J Am Heart Assoc.* 2018; 7.
37. Patel N, Kalra R, Bhargava A, Arora G, Arora P. Ideal Cardiovascular Health Among American Adults After the Economic Recession of 2008-2009: Insights from NHANES. *Am J Med.* 2019; 132:1182-90 e5.
38. Lloyd-Jones DM, Leip EP, Larson MG, D'Agostino RB, Beiser A, Wilson PW, et al. Prediction of lifetime risk for cardiovascular disease by risk factor burden at 50 years of age. *Circulation.* 2006; 113:791-8.
39. Lin MP, Ovbiagele B, Markovic D, Towfighi A. "Life's Simple 7" and Long-Term Mortality After Stroke. *J Am Heart Assoc.* 2015; 4.
40. Lin AM, Lin MP, Markovic D, Ovbiagele B, Sanossian N, Towfighi A. Less Than Ideal. Stroke. 2018:STROKEAHA118022644.
41. Younus A, Aneni EC, Spatz ES, Osondu CU, Roberson L, Ogunmoroti O, et al. A Systematic Review of the Prevalence and Outcomes of Ideal Cardiovascular Health in US and Non-US Populations. *Mayo Clin Proc.* 2016; 91:649-70.
42. The National Center for Health Statistics. Introduction to the National Health and Nutrition Examination Survey. Centers for Disease Control and Prevention.
43. The National Health and Nutrition Examination Survey. The 1999-2016 Survey Content Brochure. In: The National Center for Health Statistics, editor. CDC.gov: Centers for Disease Control and Prevention; 2016.
44. Zipf G CM, Porter KS, et al., The National Health and Nutrition Examination Survey. National Health and Nutrition Examination Survey: Plan and Operations, 1999-2010:

- Centers for Disease Control and Prevention, Division of Health and Nutrition Examination Surveys;2013. Report No.: 56.
45. Centers for Disease Control and Prevention (CDC). National Health and Nutrition Examination Survey Data. In: National Center for Health Statistics (NCHS), editor. Hyattsville, MD. 2015-2016.
 46. Donna G. Rhodes SM, Carrie L. Martin, Meghan E. Adler, Melanie A. Hymes, Anne O. Garceau, Arminda Kovalchik, Lara H. Sattgast, Lois C. Steinfeldt, John C. Clemens, Randy P. LaComb, and Alanna J. Moshfegh. USDA Food and Nutrient Database for Dietary Studies 2015-2016. In: U.S. Department of Agriculture Agricultural Research Service, editor. Food Surveys Research Group Home Page2018.
 47. Sun X, Du T. Trends in cardiovascular risk factors among U.S. men and women with and without diabetes, 1988-2014. *BMC Public Health*. 2017; 17:893.
 48. American Diabetes A. (2) Classification and diagnosis of diabetes. *Diabetes Care*. 2015; 38 Suppl:S8-S16.
 49. Kim EJ, Kim T, Paasche-Orlow MK, Rose AJ, Hanchate AD. Disparities in Hypertension Associated with Limited English Proficiency. *J Gen Intern Med*. 2017; 32:632-9.