Using an Innovative Approach to Examining Cardiovascular Health

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Dedication

This dissertation is dedicated to my parents.

For their endless love, support, and encouragement.

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Abstract

Cardiovascular disease (CVD) is a serious problem affecting a wide range of populations in the U.S. This dissertation focused on cardiovascular risks in two vulnerable yet understudied populations: women with a history of intimate partner violence (IPV) and Chinese Americans. First, an integrative review of 19 articles was conducted to examine the relationship between IPV and CVD. The overall findings suggested that IPV as a stressor could put women at high risk for CVD development. To reduce cardiovascular complications in this group, it is essential for healthcare providers to routinely screen IPV status in clinical practice and to initiate targeted interventions. Next, a cross-sectional study was carried out to assess four non-traditional CVD risk factors (sleep, physical activity, psychological stress, and fatigue) and subclinical cardiovascular markers (arterial stiffness and blood pressure variability) in a middle-aged cohort consisting of 41 Chinese Americans and 46 non-Hispanic whites. The study results showed that: 1) Chinese Americans exhibited poorer objective sleep outcomes, lower physical activity, higher psychological stress, and higher fatigue in comparison to whites; 2) both Asian race and poor perceived sleep quality independently predicted high psychological stress and fatigue; 3) low sleep quality not only had a direct relationship with high arterial stiffness and increased blood pressure variability but also moderated the relationship between these two subclinical cardiovascular markers. This cross-sectional study suggests that healthcare providers can improve cardiovascular outcomes in minority groups by implementing culturally sensitive interventions to promote their physical activity and sleep outcomes.

Keywords: Intimate partner violence; minority health; cardiovascular health; sleep; arterial stiffness; blood pressure variability.

Table of Contents

Chapter 1: Introduction

Background

Cardiovascular Disease

Cardiovascular disease (CVD), the leading cause of mortality and disability in the U.S., encompasses a wide range of health problems and complications (Cardiovascular Diseases, 2020; Olvera-Lopez et al., 2020). Although the death rates of CVD have steadily declined overall, individuals belonging to disadvantaged and vulnerable groups have been persistently affected by this disease (Mensah et al., 2017). This dissertation sought to better understand cardiovascular health in two understudied populations: females with experiences of intimate partner violence (IPV) and Chinese Americans. An integrative review was conducted to summarize existing evidence on cardiovascular risk and outcomes in female survivors of intimate partner violence (see Manuscript 1). In addition, a cross-sectional study was carried out to understand cardiovascular health in Chinese Americans using an innovative approach. The study was conducted with two purposes: to examine behavioral and psychological-physiological factors between Chinese Americans and non-Hispanic whites (see Manuscript 2) and to explore the relationship between sleep and subclinical cardiovascular markers (see Manuscript 3).

Female Survivors of Intimate Partner Violence

Intimate partner violence (IPV) is a serious public health issue that affects approximately one in three women in the United States (Black et al., 2011). The negative sequelae of IPV can cause short- and long-term consequences on women's physical and psychological well-being (World Health Organization, 2012). The relationship between IPV (physical violence, sexual violence, stalking, and/or psychological aggression) and

CVD has been inconsistent in research to date (Black, 2011; Suglia et al., 2015). Extant research is limited to self-reported measures or use of databases, precluding a comprehensive understanding of the association between IPV and CVD risk (Scott-Storey et al., 2009; Stene et al., 2013). Given that CVD is a major threat to women's health and its symptoms often go unnoticed until a major cardiovascular event occurs, early detection is particularly vital among this large proportion of women who may be at increased risk for CVD (Centers for Disease Control and Prevention, 2017).

Cardiovascular Risks in Chinese Americans

Cardiovascular disease remains the leading cause of death among Asian Americans (Go et al., 2014). Recent studies examining cardiovascular outcomes across racial/ethnic groups have predominantly combined persons of Asian ancestry into a single group. This classification often masks the heterogeneity of cardiovascular outcomes within the diverse group since various factors, such as different cultural practices, can contribute to their development of CVD (Jose et al., 2014; Palaniappan et al., 2010). Thus, it is crucial to examine Asian-American subgroups individually when assessing CVD and its risk factors.

According to the 2015 Census, the number of Chinese American immigrants has dramatically increased more than six-fold since 1980, with over 2.3 million people living in this nation (U.S. Census Bureau, n.d.). Although there have been a handful of studies investigating CVD among Chinese residing in mainland China, much less attention has been paid to addressing cardiovascular health in Chinese Americans, specifically those who migrate to the U.S. Jose et al.(2014) reported that Chinese Americans exhibited greater mortality from hypertension (men: proportionate mortality ratio [PMR] = 1.27;

women: PMR = 1.69) and hemorrhagic stroke (men: PMR = 2.19; women: PMR = 2.28) than non-Hispanic whites. Another study showed that Chinese Americans have experienced a higher prevalence of coronary heart disease, diabetes, hypertension, hypercholesterolemia, obesity, poor diet, and smoking compared to their counterparts who live in mainland China (Gong & Zhao, 2016). Given these facts, it is necessary to explore specific attributable factors associated with CVD among Chinese Americans.

Behavioral Factors: Sleep and Physical Activity. Sleep as a non-traditional risk factor of CVD has received growing attention in the scientific field. Sleep is considered a complex and dynamic process that plays an essential role in cardiovascular homeostasis (Wolk et al., 2005). Currently, limited research studies have thoroughly assessed sleep in Chinese Americans. The most recent study demonstrated that Chinese Americans were more likely than non-Hispanic Caucasians to have sleep-disordered breathing (odds ratio [OR] = 1.37, 95% confidence interval [CI]: 0.81-2.31) and short sleep duration (OR = 2.31, 95% CI: 1.48-3.61) (X. Chen et al., 2015). Because impaired sleep profoundly impacts the cardiovascular system, this problem highlights the necessity of taking sleep into account when assessing Chinese Americans' cardiovascular health.

Regular physical activity is an important protective factor of CVD development. The high rate of cardiovascular events has been found to associate with low physical activity (Myers, 2003). The current literature has consistently shown low physical activity in Chinese Americans. Taylor et al.(2007) found that only 31% of their sample of Chinese Americans (N = 395) reported regular exercise. A different study observed that only 24.3% of Chinese Americans living in New York City engaged in moderate or vigorous exercise in quantities that met the standard physical activity guidelines, showing a

strikingly lower prevalence of aerobic exercise in comparison to other Asian ethnicity cohorts (Yi et al., 2015). Given the available evidence on low exercise levels in Chinese Americans, it is important to take physical activity into consideration when assessing their cardiovascular health.

Psycho-physiological Factors: Psychological Stress and Fatigue. According to data in the 2010 American Community Survey, 76% of Chinese Americans were first-generation immigrants, making up the third-largest foreign-born group in the U.S. (Chinese in the U.S. Fact Sheet, 2017; Keister et al., 2016). Chinese Americans may face various acculturation conflicts when attempting to balance their cultural norms and values with American traditions. The process of acculturation may result in mental and physiological burdens in this minority group. Evidence has suggested that stress and fatigue can play critical roles in the etiology, development, and clinical manifestations of CVD (Casillas et al., 2006; Dimsdale, 2008). Currently, little research has investigated psychological stress and fatigue among Chinese Americans. Therefore, assessing these psycho-physiological risk factors of CVD can help researchers identify high-risk individuals in this minority population and allow healthcare providers to initiate tailored interventions.

Subclinical Cardiovascular Markers: Arterial Stiffness and Blood Pressure Variability. Arterial stiffness is closely associated with the pathogenesis of CVD (Mitchell, 2009). At the beginning of left ventricular contraction, the increase of the aortic pressure creates an incident wave that propagates along the arterial tree. As the wave travels distally, it encounters impedance mismatch (local arterial branching points or lumen narrowing sites) and produces a partial wave reflection that transmits back

towards the aorta during diastole. Optimally, the reflected wave augments the diastolic blood pressure (BP), thereby improving coronary perfusion. However, when central arteries stiffen, the impairment of cushioning function in the arterial wall leads to a rapid travel of the incident wave, quickly returning to the aorta. The early arrival of the reflected wave during systole superimposes on the incident wave, resulting in a marked increase in systolic BP and a reduction in diastolic BP (Shirwany & Zou, 2010). Accordingly, a series of pathophysiological changes will occur, such as isolated systolic hypertension, left ventricular hypertrophy, and end-organ damage (Laurent et al., 2006; London & Pannier, 2010).

Blood pressure is a dynamic-fluctuating parameter that occurs as a result of a complex interplay between extrinsic environmental and behavioral factors, as well as intrinsic cardiovascular regulatory mechanisms (Parati et al., 2013; 2018). Although variation in BP is physiological, there has been increasing evidence showing excessive blood pressure variability (BPV) to be a novel predictor of CVD and death, independently of mean BP (Höcht, 2013; Mehlum et al., 2018; Stevens et al., 2016). This subclinical cardiovascular marker is defined by the size and patterns of BP variations. Clinical studies that focused on hypertensive patients have indicated the prognostic importance of assessing and quantifying BPV (Höcht, 2013).

Relationships between Sleep and Subclinical Cardiovascular Markers. Although sleep, arterial stiffness, and BPV have individual consequences on cardiovascular outcomes, their interactions remain unclear. Investigating the relationships among sleep, arterial stiffness, and BPV could be particularly beneficial to better understanding the

underlying mechanisms of CVD risks, which will help develop nursing interventions for improving cardiovascular health in high-risk populations.

Study Purpose and Specific Aims

The cross-sectional study was intended to examine four factors important to cardiovascular health in Chinese Americans: sleep, physical activity, psychological stress, and fatigue (see Manuscript 2). Further, the study sought to explore the inter-relationships between sleep characteristics, arterial stiffness, and BPV (see manuscript 3). Healthy Chinese Americans and non-Hispanic whites aged between 18 and 64 years were recruited through a convenience sampling in central Virginia. The behavioral and psycho-physiological factors were evaluated by questionnaires. Objective sleep characteristics were assessed by an actigraph to estimate sleep quantity and quality. Arterial stiffness was measured by carotid-femoral pulse wave velocity (cf-PWV) over two nights. BPV was assessed by a 24-hour ambulatory BP monitor. The covariates included in the data analyses were age, sex, race, education, income, smoking history, intake of sleep medication, body mass index (BMI), mean arterial pressure (MAP), and heart rate (HR). The specific aims of this study are listed as follows:

Specific Aim 1: To compare behavioral factors, including sleep and physical activity, between Chinese Americans and whites.

Specific Aim 2: To compare psycho-physiological factors, including psychological stress and fatigue, between Chinese Americans and whites.

Specific Aim 3: To assess whether race and behavioral factors are independent predictors of psychological stress and fatigue.

Specific Aim 4: To examine the relationships between objective sleep characteristics, arterial stiffness, and BPV.

Specific Aim 5: To explore whether objective sleep characteristics moderate the relationship between arterial stiffness and BPV.

Innovation

This dissertation study has several novel and innovative features. First, it focuses on a large yet understudied population that is at great risk of developing CVD. Second, this study has the potential to elucidate the underlying factors that contribute to CVD and provide foundational knowledge for the development of targeted interventions for both Chinese Americans and other high-risk minority populations. Third, this study will encourage nurses to take the steps to improve the awareness of CVD disparities among different ethnic groups and provide timely, evidence-based preventative care. Fourth, this is the first attempt to examine the relationship between objective sleep characteristics and subclinical CVD markers using non-invasive, cost-efficient, and highly reliable measures. The findings will inform the feasibility of employing sleep measures and subclinical cardiovascular markers in clinical assessments.

Conceptual Framework

The figure shown below displays a conceptual framework for the relationships among the variables of interest. The three major parameters of this study—behavioral factors, psycho-physiological factors, and subclinical cardiovascular markers—are present in the proposed model. One of the study foci is to examine cardiovascular risks in Chinese Americans and whites (Specific Aims 1-3). The other focus was to assess the relationships between objective sleep characteristics, arterial stiffness, and BPV (Specific

Aims 5 and 6). The findings will serve as a basis for the future development of targeted interventions that promote cardiovascular health in Chinese Americans. The key factors known to influence the outcome variables were included as covariates: age, sex, race, education, income, smoking history, intake of sleep medication, BMI, MAP, and HR.

Figure

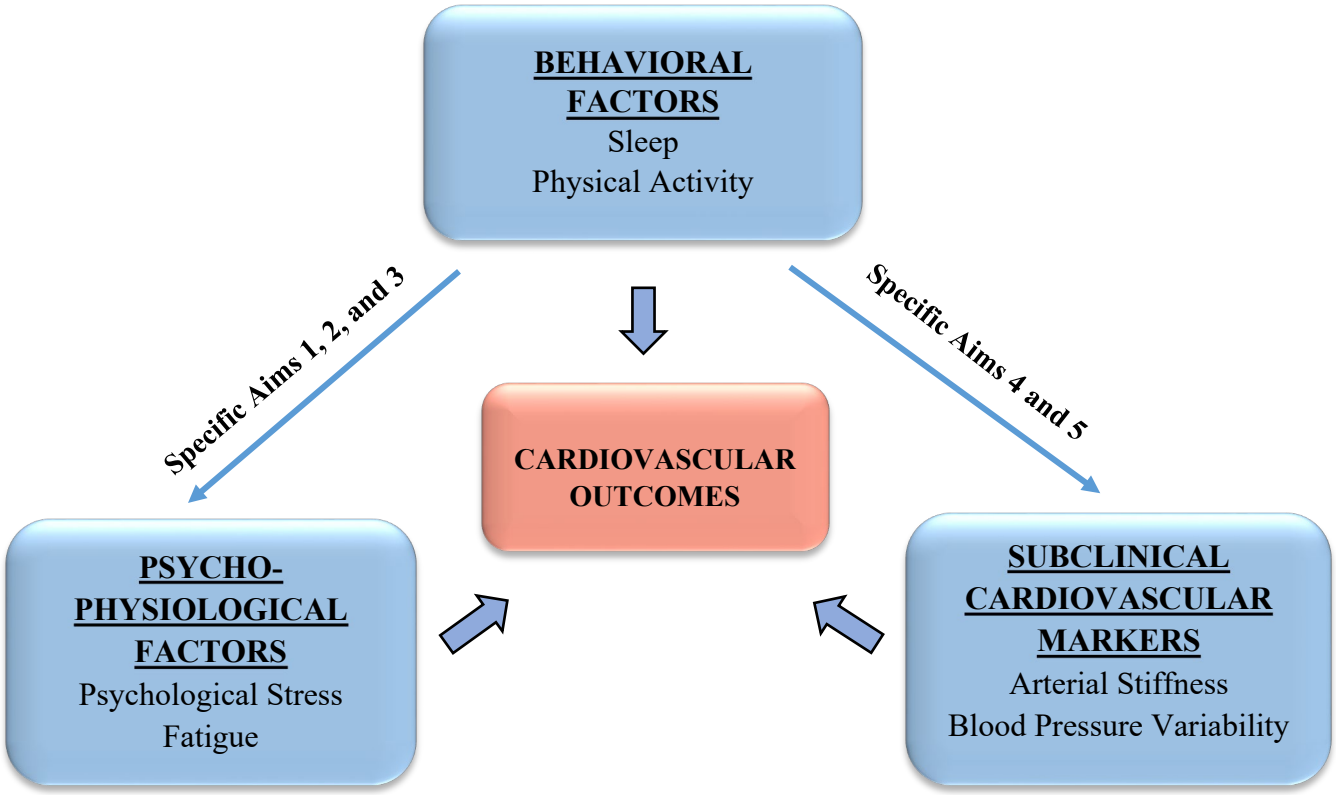

Figure 1. Conceptual Framework of the Dissertation Study

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Chapter 2: Methodology

Research Design

This cross-sectional study aimed to compare behavioral and psycho-physiological factors between Chinese Americans and non-Hispanic whites and to examine the inter-relationships between sleep characteristics and subclinical cardiovascular markers in both racial groups.

Subjects and Setting

A convenience sample of Virginians was selected through opportunistic, purposive, and snowball strategies. The recruitment outreach included: 1) distributing flyers on a university campus and local areas (e.g., churches, restaurants, grocery stores), and 2) distributing study information via local community organizations. Chinese Americans in this study were defined as individuals who identified Chinese as their sole ancestry and were first-generation immigrants who arrived in the U.S. after the age of 12. The study inclusion criteria were 35-64 years old, cognitively competent to understand and follow the study procedures, and no history of CVD. Individuals were excluded if they took medication that could affect heart rate, arterial stiffness, or sleep (e.g., antihypertensive and steroid hormones), had an irregular cardiac rhythm that prohibited the use of a measuring device for arterial stiffness (e.g., frequent premature arterial contractions or premature ventricular contractions), worked night shifts because this may cause a different profile of BPV, or were pregnant because pregnancy is associated with hemodynamic changes in the cardiovascular system (Janić et al., 2014).

Measures

Table 1 displays the information about the study variables. Considering that some Chinese Americans may have limited English proficiency, all questionnaires were

administered in Mandarin Chinese to avoid response bias. The translation of the instruments from English to Mandarin Chinese followed a three-step process (Brislin, 1970). First, the principal investigator (PI) and a nursing doctoral student who are both native Chinese and fluent in English translated questionnaire items from English into Chinese. The translation was thoroughly compared and discussed for resolving inconsistencies. Second, back translation from Chinese to English was conducted by a professor who is bilingual and unfamiliar with the original version of the questionnaires. Third, a professor who was a native English speaker compared the original items and back-translated items to evaluate semantic equivalence and accuracy.

Behavioral Factors

Subjective sleep was evaluated by the Pittsburgh Sleep Quality Index (PSQI), a 19-item questionnaire evaluating sleep quality and disturbances during the previous month (Buysse et al., 1989). The items cover seven domains, including subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. Each component score is equally weighted on a 0 - 3 scale, yielding a sum of total score with a cutoff value of 5 distinguishing “poor sleepers” and “good sleepers.” PSQI has been used in a wide range of clinical populations and it has been translated into various languages. It has demonstrated satisfactory psychometric properties: Internal consistency $\alpha = 0.83$, test-retest reliability ranging from 0.65 to 0.84, and good validity showing strong correlations with sleep log data (Backhaus et al., 2002; Buysse et al., 1989).

Objective sleep characteristics were assessed by an actigraph (GT9X Link, ActiGraph, FL). This is a watch-sized device equipped with an ambient light sensor to

capture and record movements continuously over an extended period. The recordings are then translated to digital counts accumulated across 30-second epoch and stored in the internal memory. Sleep data were analyzed using the ActiLife software (version 6.13.3) to estimate sleep/wake parameters. The Cole-Kripke algorithm was applied to distinguish sleep from wakefulness and calculate rest information by estimating decreased or increased activity count. Sleep quantity was assessed by sleep duration (the duration of sleep start and end time). Sleep quality was assessed by sleep efficiency (the ratio of sleep duration to time spent in bed), sleep onset (the time interval between lights off and sleep start), and wake after sleep onset (WASO; the amount of time spent awake between the initial sleep onset and falling asleep again). To verify the accuracy of actigraphy data, participants were also asked to complete a short sleep log including six questions asking about bed time, sleep time, awake time, and sleep quality. In particular, the information of bed time and awake time was important to determine an individual's sleep duration because the software could potentially count low motion period during the day as sleep time (Lauderdale et al., 2008). Please see Figures 1 and 2 for GT9X Link and sleep measures used in this study.

Physical activity was assessed by the Global Physical Activity Questionnaire (GPAQ), an instrument developed by the World Health Organization (WHO) (2005). GPAQ collects information regarding frequency and duration of physical activity in three domains (activity at work, travel to and from places, and recreational activities) as well as sedentary behaviors (Armstrong & Bull, 2006). This self-administered instrument contains 16 questions: 15 questions on moderate and vigorous activity for each domain, which are asked based on a skip pattern for the preceding question; 1 question on sitting

time, which is assessed by asking, “How much time do you spend sitting or reclining on a typical day?” The questions are supplemented with picture cards to help participants better understand the activity asked in each question. The data were cleaned and analyzed using the GPAQ analysis guidelines provided by the WHO. Mean hours/week for each of the three domains were calculated and summed for total physical activity, total moderate physical activity, and total vigorous physical activity. Sitting time was assessed in hours/day (Yi et al., 2016). The level of total physical activity was quantified using the metabolic equivalent task (MET), which was the ratio of a person's working metabolic rate relative to the resting metabolic rate. One MET was defined as the energy cost of sitting quietly and was equivalent to a caloric consumption of 1 kcal/kg/hour. According to the GPAQ guidelines, 4 METs were assigned to the time spent in moderate activities while 8 METs to the time spent in vigorous activities. To obtain an individual’s overall energy expenditure, the MET scores for each activity were multiplied by the hours spent in that activity each time then added. Participants were classified into three categories based on their levels of physical activity: low (< 10 MET-hours/week), moderate (10-50 MET-hours/week), or high (> 50 MET-hours/week). GPAQ has been tested in large-scale population-based surveys with the general adult populations (*Global Physical Activity Questionnaire Analysis Guide*, n.d.). This instrument has established excellent psychometric properties: short-term test–retest reliability ranging from 0.83 to 0.96 and satisfactory correlation with accelerometer-measured physical activity (Herrmann et al., 2013; Hoos et al., 2012).

Psycho-physiological Factors

Psychological stress was measured by the ten-item Perceived Stress Scale (PSS). The

PSS instrument examines the level at which situations are appraised as stressful in an individual's life (S. Cohen et al., 1983). The PSS estimates how unpredictable, uncontrollable, and overloaded individuals perceive their lives to be (S. Cohen et al., 1983). The participants rated their feelings or thoughts in the past month on a scale of 0 (never) to 4 (very often). In this questionnaire, 4 out of 10 items are negatively worded, and they are scored in the reverse direction. the total score was calculated by summing all ratings across the items, with a higher score indicative of greater psychological stress. This scale has established satisfactory internal consistency of 0.74 to 0.91, test-retest reliability of 0.72 to 0.88, and good construct validity (Lee, 2012).

Fatigue was measured by the fatigue symptom inventory (FSI) (Hann et al., 1998). This 14-item questionnaire evaluates four dimensions of fatigue—severity, frequency, perceived interference on daily functioning, and diurnal variation. The four-item severity subscale evaluates participants' most, least, and average level of fatigue experienced during the past week as well as their current level of fatigue. Each item was rated on an 11-point scale (0 = "not at all fatigued" to 10 = "as fatigued as I could be"). The seven-item functioning subscale measures the degree to which fatigue interferes with general activity level, ability to bathe and dress, work activity, ability to concentrate, relations with others, enjoyment of life, and mood. Those items were assessed on a separate 11-point scale (0 = "no interference" and 10 = "extreme interference"). The two-item frequency subscale evaluates the number of days the participants felt fatigued (from 0 to 7 days) and the average portion of each day they felt fatigued (from 0 = "none of the day" to 10 = "the entire day") during the past week. The last item evaluates the participants' daily pattern of fatigue. Since this item is designed to only provide descriptive

information about their diurnal variation of fatigue, it is not used as a quantitative subscale (Donovan & Jacobsen, 2011). The first 13 items were summed to derive a total score with a higher score indicating more fatigue.

Subclinical Cardiovascular Markers

Arterial stiffness was assessed by carotid-femoral pulse wave velocity (cfPWV) using the SphygmoCor device. The PI placed a pressure-sensitive tonometer over the carotid and femoral pulse points to generate sequential recordings (a minimum of 12 seconds of pulse wave signals) referenced to ECG (Doupis et al., 2016). cfPWV was computed using the formula cfPWV = ΔDistance/ΔTime (Najjar et al., 2008). The distance (ΔDistance) was measured over the body surface by calculating the difference of the distance from the suprasternal notch to the carotid site (S-Carotid) and the distance from the suprasternal notch to the femoral artery site (S-Femoral). ΔTime (pulse transit time) represents the delay of arrival time at the two measurement sites and was quantified by the foot-to-foot time interval (T_1-T_2) between the carotid and femoral pulse waves (Millasseau et al., 2005). Arterial stiffness can be quantified via the velocity at which a pulse wave propagates along the arterial tree. Compared to other non-invasive methods, cfPWV is broadly recognized as the gold-standard assessment of arterial stiffness because it measures along the aorto-iliac pathway, and the thoracic and abdominal aorta makes the largest contribution to the arterial buffering function (Laurent et al., 1994, 2006). Please see Figure 3 for detailed information regarding cfPWV.

Blood pressure variability (BPV) was estimated using an ambulatory BP monitor (ABPM) developed by Spacelabs Healthcare (OnTrak 90227, Spacelabs Healthcare, WA). This device was programmed to measure BP every 30 minutes from 6:00 am to

10:00 pm and every 60 minutes from 10:00 pm and 6:00 am. An appropriate BP cuff size was selected for each participant. Participants initiated the assessment after waking up in the morning, and they wore the device for 24 hours. They were informed to perform their usual daily activities but kept their monitored arm relaxed and still during each BP measurement (Zhou et al., 2018). All recordings were downloaded to compute the mean readings of average systolic BP (SBP) as well as diastolic BP (DBP) over 24 hours. Systolic BPV and diastolic BPV were estimated by average real variability (ARV), which is the average of absolute differences between consecutive BP measurements. This index provides a better estimation of 24-hour BPV than other measures of dispersion, such as standard deviation (SD), because ARV accounts for the order in which BP measurements are obtained and is less sensitive to the low sampling frequency of ambulatory BP monitoring (Höcht, 2013; Mena et al., 2017). According to Mena et al.'s (2017) meta-analysis, ARV is considered an independent indicator of the presence and progression of subclinical organ damage after adjusting for BP and other clinical factors. Please see Figure 4 for OnTrak 90227.

Covariates

Age, sex, race, education (< 12 years of formal education vs. ≥ 12 years of formal education), income (< $75,000 vs. ≥ $75,000), intake of sleep medication (yes vs. no), and smoking history (never smoked vs. lifetime smoking history) were assessed using the Sociodemographic Questionnaire developed by the PI. BP and HR values were obtained by a standard digital sphygmomanometer. BP was recorded twice at a 2-minute rest interval, and the values of SBP, DBP, and MAP were averaged. BMI was calculated as weight (kg)/height squared (m^2). Bodyweight was measured to the nearest 0.1 kg. The

weight scale was recalibrated every four months according to the manufacturer's instructions. Height was measured to the nearest 0.1 cm.

Procedures

The entire study procedure is outlined in Figure 5. After obtaining approval from the UVA Institutional Review Board, participants were informed of the study using the sampling strategies described above. Then, whether in person or over the phone, the PI explained to any interested individuals the study procedures and subsequently screened for eligibility. Those who met the study criteria were asked to sign a consent form on the day of their study visit and were scheduled for participation based on their convenience.

Data collection included two sections: an on-site study visit and off-site data collection. The on-site study visit took place in a laboratory at the UVA School of Nursing. Considering the variation caused by circadian rhythms of cardiovascular functions, all study assessments were conducted between 7:00 am and 11:00 am (Logan et al., 2012). All potential participants were asked to abstain from alcohol use for at least 10 hours and refrain from the consumption of foods or caffeinated drinks for at least 3 hours prior to the on-site visit in order to reduce the impact of these on cardiovascular hemodynamics (Laurent et al., 2006). The PI went through the study procedures orally and then obtained written consent from participants before the study assessments. Participants were asked to fill out five questionnaires, including the Sociodemographic Questionnaire, PSQI, GPAQ, PSS, and FSI. Subsequently, they were asked to change into a hospital gown to measure their height and weight for BMI calculation. Missing responses were checked and then addressed with participants. Next, BP and HR were measured twice at a 2-minute interval using a standard digital sphygmomanometer fitted

with an appropriate cuff size. Immediately thereafter, cfPWV assessment was conducted by positioning a tonometer at the carotid and femoral artery sites to generate pulse waveforms in the system. Upon finishing the on-site study visit, the PI placed an actigraph at the participants' wrist on their non-dominant arm for objective sleep assessment over two consecutive nights and also asked them to complete the sleep log. The participants received instructions on how to complete the off-site data collection. On day two, they were asked to initiate an ABPM in the morning and kept wearing it for 24 hours. On day three, the participants returned the study devices along with the sleep log. Each individual received $60 at the completion of the study.

Data Management and Analysis

Descriptive statistics for all measurements were estimated and identified for outliers. Continuous or interval variables were examined for normality. Log transformation was used if the variables were not normally distributed. Bivariate relationships among all variables were examined by Pearson correlation. The significance level in this study was set at $\alpha = 0.05$ (Cohen, 1992).

Specific Aim 1: To compare behavioral factors, including sleep and physical activity, between Chinese Americans and whites.

Hypothesis: Chinese Americans report poorer perceived sleep quality, have worse objective sleep characteristics (lower sleep duration, lower sleep efficiency, higher sleep onset, and higher WASO), and perform lower levels of physical activity than whites.

Analysis 1: Multiple linear regression analyses were conducted to compare perceived sleep quality, sleep duration, sleep efficiency, sleep onset, WASO, and

physical activity after controlling for age, sex, race, income, education, and smoking history.

Specific Aim 2: To compare psycho-physiological factors, including psychological stress and fatigue, between Chinese Americans and whites.

Hypothesis: Chinese Americans report higher psychological stress and fatigue than whites.

Analysis 2: Multiple linear regression analyses were conducted to compare psychological stress and fatigue after controlling for age, sex, race, income, education, and smoking history.

Specific Aim 3: To assess whether race and behavioral factors were independent predictors of psychological stress and fatigue.

Hypothesis a: Race independently predicts high psychological stress and fatigue.

Hypothesis b: Each of the following sleep variables—poorer perceived sleep quality, longer sleep duration, lower sleep efficiency, higher sleep onset, and higher WASO— independently predicts high psychological stress and fatigue.

Hypothesis c: Low physical activity independently predicts high psychological stress and fatigue.

Analysis 3: In this aim, race and each of the behavioral factors (sleep variables and physical activity) were independent variables. Psychological stress and fatigue were dependent variables. Multivariate analyses of covariance were conducted to test how psychological stress and fatigue levels were influenced by race and each of the behavioral factors. The analyses were adjusted by age, sex, income, education, and smoking history.

Specific Aim 4: To examine the relationships between objective sleep characteristics, arterial stiffness, and BPV.

Hypothesis a: Each of the sleep variables (longer sleep duration, lower sleep efficiency, higher sleep onset, and higher WASO) is related to increased cfPWV.

Hypothesis b: Each of the sleep variables (longer sleep duration, lower sleep efficiency, higher sleep onset, and higher WASO) is related to increased BPV.

Hypothesis c: Higher cfPWV is related to increased BPV.

Analysis 4: First, multiple linear regression analyses were carried out to examine how each of the sleep variables (sleep duration, sleep efficiency, sleep onset, and WASO) was related to cfPWV and BPV, respectively, after adjusting for age, sex, race, intake of sleep medication, smoking history, BMI, MAP, and HR. Second, multiple linear regression analysis was performed to examine how cfPWV was related to BPV after adjusting for age, sex, race, intake of sleep medication, smoking history, BMI, MAP, and HR.

Specific Aim 5: To explore whether objective sleep characteristics moderated the relationship between arterial stiffness and BPV.

Hypothesis: each of the poor objective sleep characteristics (longer sleep duration, lower sleep efficiency, higher sleep onset, and higher WASO) serves as a moderator that influences the relationship between cfPWV and BPV.

Analysis 5: The moderation analysis was examined sequentially with the following three steps: in step 1, the association between cfPWV (independent variable) and systolic BPV (dependent variable) was assessed; in step 2, one sleep variable (moderator) was added; and finally, in step 3, the interaction term cfPWV

× moderator was added. This process was repeated for each proposed moderator including sleep duration, sleep efficiency, sleep onset, and WASO. The analyses were adjusted by age, sex, race, intake of sleep medication, smoking history, BMI, MAP, and HR.

Table

Table 1

Key study variables, measures, and operational definitions

Variable	Measure	Device	Calculation
Sleep			
Sleep quantity	Sleep duration	GT9X Link (ActiGraph, FL) ActiLife software version 6.13.3 (ActiGraph, FL)	sleep start time - sleep end time
Sleep quality	Sleep efficiency		$\frac{\text{Sleep duration}}{\text{Time in bed}}$
	Sleep onset		Lights off time - sleep start time
	wake after sleep onset (WASO)		$\frac{\text{Total wake time after sleep onset}}{\text{Total number of main sleep periods}}$
Subjective sleep	Pittsburgh Sleep Quality Index (PSQI)		
Physical Activity			
Physical activity	Global Physical Activity Questionnaire (GPAQ)		
Psychological Stress			
Psychological stress	Perceived Stress Scale (PSS)		
Fatigue			
Fatigue	Fatigue Symptom Inventory (FSI)		
Subclinical cardiovascular markers			
Arterial stiffness	carotid-femoral pulse wave velocity (cfPWV)	SphygmoCor (AtCor Medical, Australia)	$\frac{\Delta\text{Distance}}{\Delta\text{Time}}$
Blood pressure variability (BPV)	24-hour BPV	OnTrak 90227 (Spacelabs Healthcare, WA)	Average real variability
Covariates			
Age	Sociodemographic Questionnaire	Self-report	N/A
Sex			
Race			
Education			
Income			
Smoking history			
Intake of sleep medication			
Body mass index (BMI)	Weight	Electronic weight scale	$\frac{\text{Weight in kg}}{(\text{Height in m})^2}$

	Height	Wall mounted stadiometer	
Mean arterial pressure (MAP)	Standard digital sphygmomanometer	Omron 3 Series	$\frac{SBP + 2 * (DBP)}{3}$
Heart rate (HR)	Standard digital sphygmomanometer	Omron 3 Series	N/A

Figures

Figure 1. ActiGraph GT9X Link.

Note. The image was derived from https://www.actigraphcorp.com/actigraph-link/

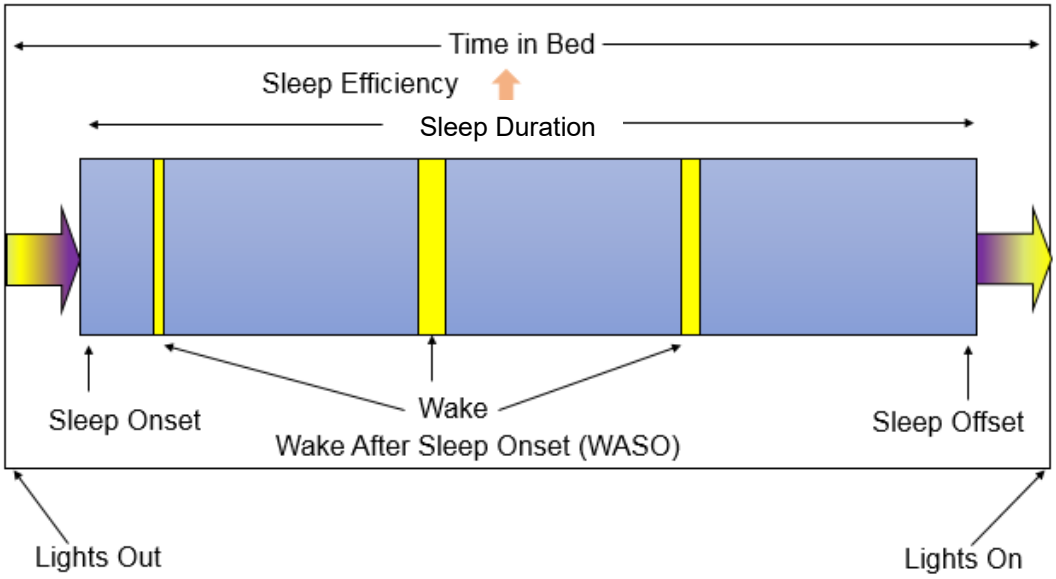

Figure 2. Measures of sleep characteristics.

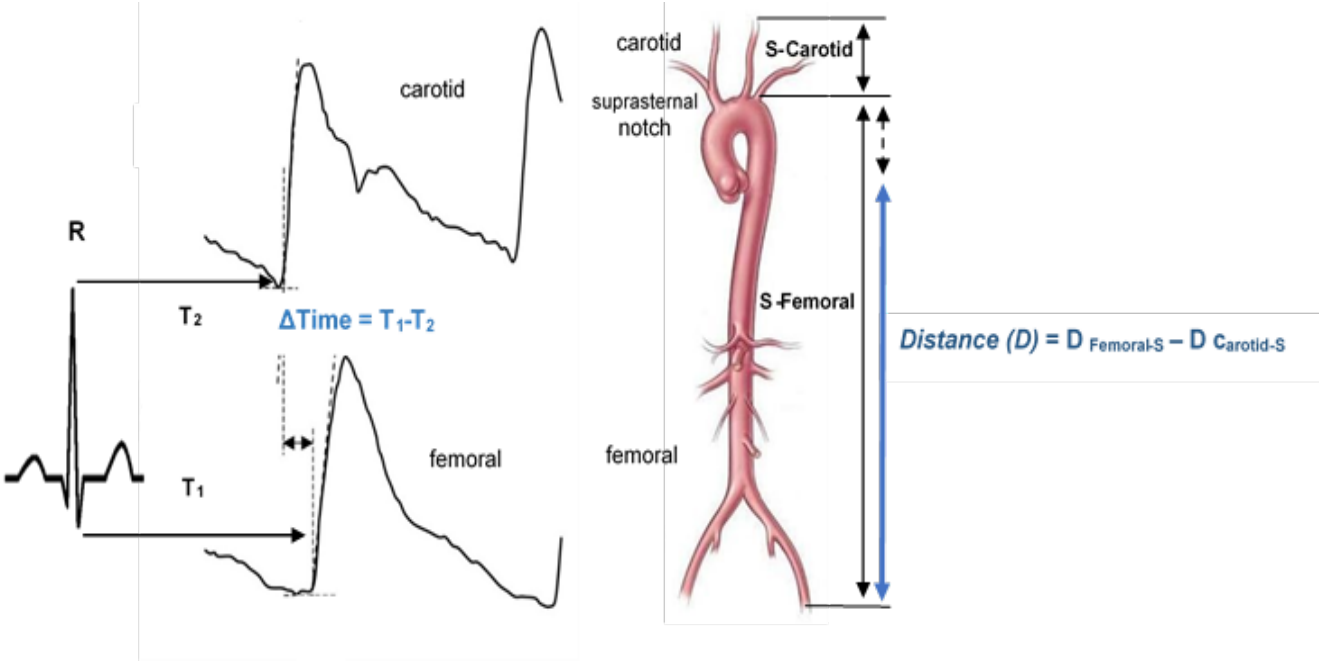

Figure 3. Measurement of arterial stiffness.

Note. The figure was adapted from "Assessment of Pulse Wave Velocity," by P. Boutouyrie, M. Briet, C. Collin, S. Vermeersch, and B. Pannier, 2009, *Artery Research*, *3*, p.5. and "Aortic Stiffness and Microalbuminuria in Patients with Chronic Obstructive Pulmonary Disease." by A. Refaat, M. Abdou, A. Ismael, and I. Alhelali, 2015, *Egyptian Journal of Chest Diseases and Tuberculosis*, *21*, p. 543.

Figure 4. Spacelabs OnTrak 90227.

Note. The image was derived from https://www.spacelabshealthcare.com/products/diagnostic-cardiology/abp-monitoring/ontrak/

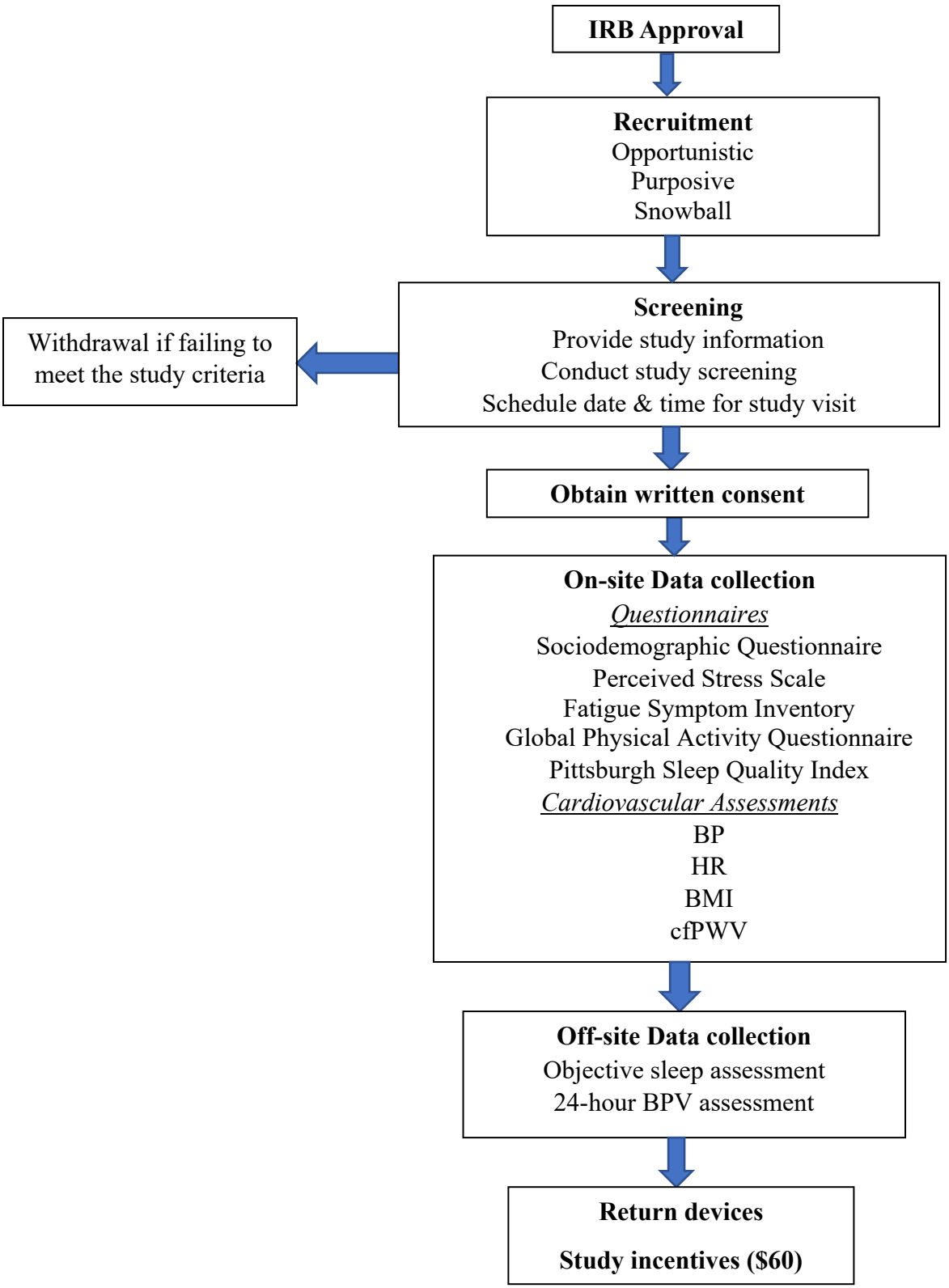

Figure 5. Study Procedures

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Chapter 3: Cardiovascular Risk and Outcomes in Women who Have Experienced Intimate Partner Violence: An Integrative Review

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Abstract

Background: Cardiovascular disease (CVD) and intimate partner violence (IPV) are two major chronic problems that prevalently affect women's health and quality of life in the United States. However, whether IPV female survivors are at risk for developing adverse cardiovascular outcomes has not been clearly understood.

Objective: This integrative review aimed to bridge the literature gap by examining cardiovascular health in adult females with a history of IPV experience.

Methods: Three electronic databases including PubMed, CINAHL, and Web of Science were used to search for studies published between 1998 and 2019. The search process followed the Preferred Reporting Items for Systematic Review and Meta-Analyses (PRISMA) guidelines.

Results: Of the 229 records retrieved from the literature, 19 met the criteria for review. All included studies were quantitative research. Although the overall findings showed a mixed relationship between IPV and CVD, women who experienced abuse were more likely to engage in unhealthy behaviors, have higher levels of CVD biomarkers, experience cardiovascular symptoms, and may exhibit long-term cardiovascular complications when compared to non-abused women.

Conclusions: Intimate partner violence is a stressor which directly and indirectly influences women's cardiovascular health. Therefore, it is essential for healthcare providers to routinely screen IPV status in clinical practice. Targeted interventions, such as assessing women's coping strategies and evaluating their cardiovascular health using a total risk factor approach, are recommended to prevent or reduce the deleterious effects of violence on this large, vulnerable group of women.

Keywords: cardiovascular diseases; intimate partner violence; women's health; risk factors; review literature

Introduction

Cardiovascular disease (CVD) remains the leading cause of morbidity and mortality in the United States, accounting for approximately one female death every 80 seconds.[1] This chronic disease has been widely recognized as the "female silent killer," as the acute onset of cardiovascular events often results in fatal consequences without any prior clinical signs or symptoms.[2] While many factors can contribute to the development of CVD, recent studies have suggested that exposure to violence is a potential risk factor for CVD.[3,4] Increasingly, researchers have begun exploring the role of intimate partner violence (IPV) on cardiovascular risk. Intimate partner violence includes physical violence, sexual violence, stalking, and/or psychological aggression in an effort to gain power and control over another intimate partner.[5,6] An estimated one in three women have experienced some type of violence during their lifetime.[7] Compared to males, females are disproportionately affected by this public health issue regardless of age, socioeconomic status or cultural backgrounds.[8] Given the prevalence of IPV and CVD among women, it is important to examine their relationship in an effort to better understand how the combination of these two factors may impact the overall health among female survivors of IPV.

Previous evidence has suggested two potential pathways linking IPV to adverse cardiovascular outcomes.[9–12] One plausible pathway is alterations at the physiological, biochemical, and endocrine levels caused by the sustained stress from IPV.[10,13] The perception of stress immediately stimulates the autonomic nervous system and the hypothalamic-pituitary-adrenal (HPA) axis, targeting end organs such as the heart and producing glucocorticoid hormones. Examples of related systematic changes include

increased heart rate, elevated blood pressure (BP), reduced insulin sensitivity, and impaired endothelial function in arteries resulted from circulating stress hormones.[14] The stress from IPV continuously triggers immune-inflammatory responses, making female survivors more susceptible to the deleterious effects of biochemical-marker production (e.g., pro-inflammatory cytokines) and inducing the subsequent breakdown of the cardiovascular system.[15,16] Another proposed pathway by which experiencing IPV may increase cardiovascular risk is through the survivors' adoption of unhealthy lifestyles.[10] Notably, these maladaptive stress-coping behaviors—for instance, smoking, alcohol use, and physical inactivity—are not only considered the most prominent behavioral risk factors of CVD, but are also found to influence other cardiovascular risk indicators or conditions such as BP, lipid levels, oxidative stress, or inflammation.[17]

While extensive research studies have documented a consistent relationship between childhood violence exposure and CVD, less research has focused on the cardiovascular health in adults with a history of IPV victimization.[4,10,11] This is an important area of inquiry given adulthood exposure to violence may operate under different biological mechanisms that differ from childhood exposure.[4] Understanding vulnerability in CVD among IPV female survivors is essential for healthcare providers to initiate targeted interventions and prevent further development of CVD. As of 2015, Suglia and colleagues[4] published a systematic review of the association of interpersonal violence with cardiovascular outcomes in adults. Their focus was centered on violence perpetrated by both strangers from wider communities and non-strangers (e.g., family members). Additionally, their search criteria were limited to studies that assessed CVD endpoints, excluding studies that evaluated cardiovascular risk factors such as biomarkers of CVD.

Indeed, cardiovascular risk factors have critical implications for disease prevention. Evidence has supported that some leading modifiable risk factors (e.g., hypercholesterolemia, diabetes, hypertension, obesity, smoking) are responsible for over half of cardiovascular mortality in Americans adults aged from 45 to 79 years.[18] CVD biomarkers, particularly, have been increasingly recognized as valuable tools for early prevention and treatment of CVD.[19] Since the cardiovascular risk and outcomes specifically for IPV survivors have not been well addressed, synthesizing current research on this topic may fill a gap in the literature and further our understanding about the relationship between IPV and CVD. Therefore, the purpose of our integrative review is to examine if IPV is associated with cardiovascular risk and outcomes among female adults.

Methods

The analysis was guided by Whittemore and Knafl's[20] updated integrative review framework, using a systematic approach to synthesize rigorous evidence from empirical resources. Activities involved in the current review included a literature search, data evaluation and extraction, data synthesis and comparison, and interpretation. With the assistance of a university librarian, the researchers employed three electronic databases—PubMed, CINAHL, and Web of Science. The databases were thoroughly searched for the topics of IPV and CVD using the Medical Subject Headings (MeSH) and keywords, namely: "Intimate Partner Violence," "Domestic Violence," "Family Violence," "Partner Abuse," "Spouse Abuse," "Exposure to Violence," "Battered Women," "Cardiovascular Diseases," "Cardiovascular Outcomes," "Cardiovascular Health," "Cardiovascular Risk," "Coronary Diseases," "Blood Pressure," and "CVD." The researchers combined the terms for each topic using "OR" and between the topics using "AND." The search was

initially limited to studies that met the following criteria: peer-reviewed studies, written in English language, published from 1998 to 2019, and included adults aged 18 years or above. This yielded 223 articles retrieved from the data sources described above. Hand searching and reference tracing were also carried out, which yielded 6 additional articles. After removing 30 duplicates, the researchers examined the titles and abstracts of the remaining articles and excluded 162 articles for other reasons (see Figure 1). This process left 37 articles for a full review. After the full review, 18 articles were removed as they didn't meet our search criteria. Consequently, 19 articles were included for analysis. Data were extracted from each study based on the following categories: author and year, study aim, sample characteristics, study methods, main CVD measures, IPV measures, and key findings (see Table 1).

Results

Study Design and Sample Characteristics

Given the nature of the research topic, all 19 articles included in this review utilized a quantitative method to examine cardiovascular health in IPV female survivors. The studies varied in research designs: 13 studies[10,12,13,21–30] used secondary data analyses, 5 studies[11,15,16,31,32] included prospective cross-sectional designs, and 1 study[33] used a prospective longitudinal design. Across the included studies, 14 included women who lived in the United States[12,13,15,16,21–24,28–33] while the remaining focused on women from Canada (n = 1),[10] Oslo (n = 1),[11] Mexico (n = 1),[25] Spain (n = 1),[27] and South Africa (n = 1).[26] The targeted study population in 16 studies was females while the other three studies[13,22,24] included males and females. The majority of studies included female

samples who were in mid-adulthood, with the mean age ranging from 22[22,24] to 55[16] years.

Study Measures

Approximately 79% (n = 15) of the studies compared cardiovascular health between individuals exposed to IPV and their non-abused counterparts with variations in the type and timing of abuse examined.[11–13,15,21,22,24–31,33] Both physical and sexual abuse was the most commonly evaluated forms of IPV among female survivors. Lifetime history of abuse was frequently used as a measure to assess the timing of IPV. Because CVD is a complex, multifaceted condition, the operationalization of this concept varied significantly across the studies. Thus, we reported the findings based on two categories: cardiovascular risk and cardiovascular outcomes.

Cardiovascular Risk

Cardiovascular Disease Risk Factors

High blood pressure (BP) is one of the strongest risk factors for nearly all types of CVD. Of the 9 studies[10,12,13,15,24,26–28,31] reviewed that explored the relationship between BP outcomes and IPV, seven[12,13,15,26–28,31] relied on self-reported measures while two[10,24] used objective measures. Among the studies reviewed, the prevalence of hypertension ranged from 12.21%[24] to 50.7%,[15] and only one study[28] found a statistically significant finding. Breiding and colleagues[13] analyzed the 2005 *Behavioral Risk Factor Surveillance System (*BRFSS), showing that 22.6% of the female IPV sample was diagnosed with hypertension and that lifetime IPV was significantly associated with hypertension after controlling for sociodemographic variables (adjusted odds ratio [aOR] = 1.11, confidence interval [CI]: 1.01-1.23); however, the relationship was attenuated

after adjusting for other health variables such as high cholesterol, stroke, and tobacco and alcohol use (aOR = 0.97, CI: 0.86-1.10). Although the studies did not demonstrate a significant association between IPV and high BP, some evidence suggested that the effect of psychological abuse in contributing to hypertension might be greater than any other forms of abuse. Analyzing a longitudinal dataset that followed female nurses for 6 years (N = 51,434), Mason and colleagues[28] demonstrated that women who were exposed to severe psychological IPV had a 24% increased risk of developing hypertension in comparison to their counterparts who were not exposed to emotional abuse after adjusting for history of childhood abuse, race, parental education, somatogram score at age 5, and BMI at age 18 (adjusted hazard ratio [aHR] = 1.24, CI: 1.02-1.53). Thus, the research that has examined the relationship between IPV and BP is mixed.

Diabetes, disorders of lipid metabolism, and obesity are also well-established risk factors for the development of CVD. A total of six studies examined how these risk factors were linked to IPV.[11–13,21,26,31] Three studies[12,13,26] that assessed IPV and diabetes did find a relationship, and three studies[11,13,31] documented mixed findings regarding the association of IPV with cholesterol and lipid metabolism. In the study carried out by Breiding et al.,[13] female survivors of IPV were 13% more likely to have high cholesterol than their non-abused counterparts (aOR = 1.13, CI: 1.01–1.27). In a population-based study that compared cardiovascular risks and medication use in 5,593 Oslo women with or without the experience of IPV, Stene and colleagues[11] noted that those who reported lifetime IPV exhibited lower high-density lipoproteins (HDL) and higher triglyceride levels (p = .031 and p = .003, respectively); yet, the level of total cholesterol did not significantly differ between the two groups. Bonomi et al.[31] did not find an association

between IPV and lipid metabolism disorder in 272 out of 1,928 women who reported a past-year history of abuse after adjusting for age (aRR [adjusted relative risk] = 0.86, CI: 0.46-1.62). Further, four studies did not note a significant relationship between IPV and obesity, as measured by BMI.[11–13,21] However, when waist-to-hip ratio was used to measure obesity, Stene and colleagues[11] found that women victimized by physical and/or sexual violence (n = 415) exhibited a higher likelihood of abdominal obesity than the control group (p = .001). Taken together, the research examining the associations of diabetes, lipid metabolism, and obesity with experiences of IPV has shown mixed results.

Behavioral Risk Factors

Unhealthy behaviors are adopted by some women as coping mechanisms to deal with IPV-induced stress. These behaviors, such as smoking, alcohol use, and physical inactivity, may confer adverse consequences on cardiovascular health. Of the six studies that included measures of smoking and alcohol use, five revealed significant results.[10,11,13,21,26] The overall prevalence of smoking and alcohol use among the IPV samples was 33.8%[13]-54.8%[12] and 14.5%[13] -89.6%,[11] respectively. In three studies that included nationally-representative samples, abused women were 1.68 to 2.80 times more likely to smoke and 1.39 to 2.37 times more likely to use alcohol as compared to non-abused women.[13,21,26] Limited research has examined specific characteristics of IPV and the association between tobacco and alcohol use. Among a sample of 309 Canadian female survivors of IPV, Scott-Storey et al.[10] hypothesized that current smoking status had a positive relationship with both severity of past IPV and ongoing abuse. The investigators noted a significant correlation between smoking and severity of past abuse ($t = 3.586$, $p \leq .001$); yet, their results showed that women who experienced ongoing

abuse were more likely to be non-smokers, which was contrary to their original hypothesis. Stene and colleagues[11] found that women who were exposed to physical or sexual IPV had higher adoption rates of smoking ($p < .001$) and drinking ($p = .014$) than their counterparts who only reported psychological IPV or no abuse history. However, Vijayaraghavan and colleagues[12] did not find significant differences in the rate of smoking (54.8% vs. 46.4%, $p = .30$) or alcohol use (61.8% vs. 54.2%, $p = .20$) among 329 homeless women with and without history of IPV. This finding could be explained by the unique characteristics of the homeless population, a group which has a strikingly high rate of smoking according to the past literature.[34] Taken together, smoking and alcohol use are negative health behaviors that are more prevalent among women with previous experiences of IPV.

In addition to the two maladaptive coping strategies discussed above, physical inactivity can also play a critical role in the development of CVD.[35] In this review, three studies assessed the relationship between IPV and physical activity using the BRFSS dataset, and did not yield significant findings.[11,13,21] For example, researchers reported that nearly half of their female sample who had a lifetime experience of IPV performed weekly moderate or vigorous exercise, and the level of physical activity did not differ when comparing abused versus non-abused women (aOR = 1.00, CI: 0.92-1.09).[13] Another study by Dichter et al.[21] analyzed a sample of 21,162 women, and the investigators were unable to find an association between lifetime IPV and physical inactivity after adjusting for demographic characteristics and veteran status (aOR = 0.90, CI: 0.80-1.10). In summary, IPV status does not have a direct association with physical inactivity.

Cardiovascular Disease Biomarkers

Intimate partner violence may over-activate the immune system and cause elevated levels of biomarkers such as inflammatory cytokines (e.g., Interleukin-6 [IL-6)]) and acute-phase proteins (e.g., C-reactive protein [CRP]), which have been shown to accelerate the pathogenesis of CVD.[9] Five studies examined the association between IPV victimization and CVD biomarkers.[15,16,23,25,33] Among the studies reviewed, four[15,16,23,33] used serum and/or salivary samples, and one[25] used an ultrasound technique to compute the subclinical marker carotid intima-media thickness (IMT).

Despite their nascency in IPV research, IL-6 and CRP were two of the most frequently examined biomarkers among the studies; overall, the evidence showed their close association with IPV victimization, particularly when females survivors were severely assaulted or exposed to significant psychological trauma. Newton and colleagues[16] examined CVD biomarkers in a sample of 68 women who underwent divorce or separation resulting from stressful relationships— finding that severe physical abuse remained a significantly negative correlation with phytohemagglutinin (PHA)-stimulated IL-6 production after adjusting for BMI, current posttraumatic stress, and depressive symptoms ($\beta = -0.08$; $p = .005$). Another pilot cross-sectional study by Halpern et al.[15] found that IPV was correlated with salivary CVD biomarkers, such as CRP/IL-6, among a sample of women presenting to an oral surgery clinic (51% vs 49%, $p < .01$, N = 37). Yet, the results need to be interpreted cautiously given the small sample size. In addition to the biomarkers IL-6 and CRP, one study[25] used carotid IMT (intima media thickness) to predict adverse cardiovascular function in female victims. Carotid IMT is a novel biomarker of CVD which predicts subclinical carotid atherosclerosiss.[36]

Flores-Torres and colleagues[25] in their study found that the adjusted mean percentage difference in carotid IMT between women who were exposed to either sexual or physical violence and their non-abused counterparts to violence was 2.4% (CI: 0.50-4.30). The investigators also found that women who reported physical violence by their family members had 1.3% of higher mean carotid IMT, and this population was 1.48 times more likely to have subclinical carotid atherosclerosis when compared to those without a history of violence (aOR = 1.48, CI: 0.78-2.83). Taken together, there is strong evidence showing that IPV victimization is related to higher levels of CVD biomarkers.

Cardiovascular Outcomes

Cardiovascular symptoms and diagnosis are important indicators of adverse cardiovascular outcomes. The most commonly reported symptoms in this review were chest pain, palpitations, and shortness of breath.[10,15,31] Scott-Storey et al.[10] analyzed a sample of 309 IPV female survivors living in Canadian communities, hypothesizing that chronic stress (operationalized by severity of past experience of IPV and ongoing abuse) were positively associated with cardiovascular symptoms (chest pain, shortness of breath, palpitations, and symptoms of hypertension). Notably, they found that neither severity of IPV or current abuse was a significant predictor of those cardiovascular symptoms [χ^2 (2, n = 271) = 0.85, p = .655). It was also noted that half of the abused sample reported cardiovascular symptoms which were important indicators of ischemic heart disease, yet only 17.2% of the women received a diagnosis of CVD. This implied that healthcare providers might overlook the presentation of cardiovascular symptoms among IPV female survivors and fail to diagnose and treat CVD in this population.

Four studies documented several CVD diagnoses which were frequently reported by abused women: coronary heart disease, heart attack, cardiac arrhythmia, chronic ischemic heart disease, and cardiac valve disorders.[10,13,26,27] The likelihood of developing coronary heart disease (CHD) in this population was particularly striking. Vives-Cases et al.[27] conducted a secondary analysis of a sample which included 13,094 women from Spain. They found that 0.98% of their sample experienced IPV within past 12 months and that this population was 5.28 times more likely to develop CHD in comparison to those who were never abused (aOR = 5.28, CI: 1.45-19.25) after controlling for sociodemographic characteristics, social support, smoking and BMI. Similarly, another study conducted by Breiding et al.[26] showed that IPV female survivors (26.4%) were nearly 1.5 times (aOR = 1.43, CI: 1.06-1.94) more likely to develop CHD as compared to their non-abused counterparts.

Additionally, five studies reviewed utilized the Framingham Risk Score to assess long-term cardiovascular health over a 10-year or 30-year time frame.[11,22,29,30,32] This comprehensive measure has been widely considered as a validated tool of estimating future CVD events in asymptomatic individuals as it takes into account multiple important risk factors of CVD, including age, sex, systolic BP, total cholesterol, HDL, use of antihypertensive medications, and smoking status.[37,38] Stene and colleagues[11] reported that women who disclosed physical or sexual IPV had a slightly higher risk of CVD in 10 years than those without IPV histories (p = .033). For example, the percentages of women who had over 5.54% risk of 10-year estimated risk of CVD were 27.5% and 24.7% in those with and without IPV histories, respectively. The remaining four studies[22,29,30,32] employed the 30-year risk prediction model and the results

consistently showed higher CVD scores among IPV female survivors. In a study of 7,392 young women that included female survivors (n = 1,161), Wight et al.[29] found that those who experienced past-year IPV had a small yet significant increase in the mean score of 30-year CVD risk when compared with the non-abused group (9.6% vs. 8.7%, $p < .01$). However, the relationship was no longer significant after adjusting for other important predictors in the model including — race and ethnicity, education, pregnancy status, health insurance, and financial stress. These findings highlight the necessity of considering other contextual factors that may affect cardiovascular outcomes in IPV survivors. The same investigators subsequently used the same sample to conduct another analysis, assessing whether depressive symptoms, alcohol dependence, or perceived stress were potential mediators of the relationship between IPV and 30-year CVD risk. They found that stress (total indirect effect = 0.047, standard errors [SE] = 0.010, $p < .01$) and depression (total indirect effect = 0.054, SE = 0.012, $p < .01$) played contributing roles in the context of IPV and cardiovascular health. In summary, the evidence shown above has indicated a potential correlation between IPV and long-term negative cardiovascular outcomes in female survivors.

Discussion

This literature review summarized evidence from 19 articles to investigate cardiovascular health in women with experiences of IPV. While research in this area is limited, our findings suggest that IPV may be an important risk factor for the development of CVD. Compared to their non-abused counterparts, abused women are more likely to engage in unhealthy behaviors, experience cardiovascular symptoms, and have long-term cardiovascular complications. However, the overall mixed findings limit

our ability to definitively establish the relationship between IPV and CVD. There are several factors that may help explain the inconsistent findings. First, the sample size widely ranged from 34 to 7,0156, which could affect power and introduce great variability in the study results. Second, the differences among sample sociodemographics may influence women's responses towards IPV, coping strategies, and their knowledge about cardiovascular health and management. Lastly, the researchers employed different measures of both IPV and cardiovascular outcomes, which may contribute to mixed findings.

One of the major findings identified from this review is the high adoption rate of smoking and alcohol use among abused women, both of which are risk factors for CVD. Even though it is unclear whether IPV experience is a direct cause of those unhealthy behaviors, this type of exposure to violence may precipitate maladaptive behaviors and their relationship has been theoretically grounded in previous investigations on stress and coping.[39,40] According to Lazarus and Folkman[41], an individual's coping strategies are often developed through an interplay between one's internal resources and external demands that are created by the stressful transaction. Those researchers found that people who appraised their problems as changeable tended to use more problem-focused strategies while they were more likely to employ emotion-focused strategies when the situation was appraised as not or less changeable.[42] Hence, it is likely that many abused women select conflict-specific, maladaptive coping strategies with respect to the form, severity, or chronicity of IPV they experience.[43] Therefore, fostering behavioral change should be considered as a top priority for CVD prevention among those women. Healthcare providers should incorporate the evaluation of coping skills and mental

distress into their routine health assessment. Focus can be placed on identifying high-risk situations that most likely trigger tobacco or alcohol use, and empower this vulnerable population with effective coping strategies and emotion regulation skills. In addition, further research is warranted to better understand how IPV characteristics might affect women's choice of unhealthy behaviors and how the engagement of those behaviors might change overtime throughout IPV trajectories.[10]

Our review highlights the importance of employing a total risk approach rather than using single risk factors to estimate the overall cardiovascular health among IPV female survivors. As the study results indicated, the assessment of BP, diabetes, cholesterol, or BMI have shown either an insignificant or mixed correlation with IPV. This could be partially explained by the cardio-protective effect of estrogen in females because a majority of the study participants were pre-menopausal women. Growing research suggests the use of total risk approach to predict cardiovascular outcomes. Many researchers have recognized that the single risk-factor approach may cause individuals with low cardiovascular risk to receive unnecessary treatments while leaving the truly high-risk populations—those who present with high total cardiovascular risk resulting from multiple slightly elevated risk factors—neglected for timely medical interventions. Because many CVD factors tend to coexist and act multiplicatively,[44] the total risk approach employs scoring prediction models which are centered on the profile of all essential cardiovascular risk factors, appearing to be a logical and effective means to predict negative cardiovascular consequences in the abused population. Indeed, our review showed that IPV female survivors generally had higher Framingham risk scores than non-abused women. This evidence not only suggests the long-term influence of IPV

on one's cardiovascular health, but also underscores that the total cardiovascular risk assessment approach could become a routine clinical tool to identify high-risk individuals and reduce premature morbidity and mortality among abused women.

In addition, this review explored the association between IPV and cardiovascular biomarkers, a relatively new focus of linking IPV to health outcomes. The findings of elevated levels of IL-6 and CRP in abused women are consistent with the emerging literature on stress, inflammatory responses, and CVD.[45] The changes of those biomarkers signal an early indication of systemic inflammation and endothelial dysfunction and may subsequently lead to atherosclerosis and promote the development of CVD.[46] In our review, two studies[15,23] tested the feasibility of using salivary specimen to measure cardiovascular biomarkers in the IPV population. Both suggested that this less invasive approach could potentially provide prognostic indication for cardiovascular risk in the IPV population. However, because research on IPV and cardiovascular biomarkers is still quite limited, it is important to explore the validity and clinical utility of using these novel biomarkers in large study samples before introducing them into the routine clinical assessment.

There are several limitations in this review that should be addressed. First, the included studies predominantly employed cross-sectional or secondary data analysis approaches to examine CVD. Although the use of a cross-sectional method is an efficient way to conduct a study, the data collected from each participant cannot be used to infer a causal relationship between IPV and CVD.[47] Similarly, carrying out secondary data analysis enables existing data to be fully and appropriately explored, but this method limits the ability to measure all variables salient to CVD.[10] Future research should include

population-based and longitudinal studies, in an effort to observe cardiovascular change over and beyond the trajectory of violence. Second, a vast majority of the studies reviewed utilized self-report to measure cardiovascular outcomes. Clinical information obtained by self-report may introduce bias to the results.[11] Therefore, objective measures of CVD risk and outcomes are suggested for future studies. Lastly, IPV is a complicated issue that takes various forms of abuse, timing, and severity. Most studies had limited measures of IPV which may not adequately capture the magnitude of the issue. It would be beneficial for future researchers to conduct a more comprehensive review of this problem and explore how the different types, severity, and chronicity of IPV have an impact on women's cardiovascular health.

Nurses, who are ideally positioned to identify women with experiences of IPV, can play a pivotal role to help this vulnerable population to prevent CVD occurrence and achieve optimal health outcomes. The 2019 American College of Cardiology/American Heart Association guideline[48] has provided several recommendations for healthcare providers to improve primary prevention of CVD in clinical practice. Some essential ones are changing lifestyle behaviors (e.g., diet and physical activity) and using traditional risk factors to estimate 10-year cardiovascular risk in every 4-6 years among individuals who are 20-79 years old and free from CVD. Other recommendations include promoting patient-centered approaches (e.g., team-based care, shared decision making, assessment of social determinants of health such as environmental and psychosocial factors) and initiating cost-effectiveness of prevention. Such strategies can be instrumental for nurses in guiding targeted interventions to meet individual needs and promote the overall population health outcomes.

Conclusions

Overall, this review demonstrates that the experience of IPV could increase women's risk for developing CVD. Intimate partner violence serves as a stressor not only affecting cardiovascular health through modifying women's lifestyle behaviors but also triggers a cascade of biological changes that pose long-lasting detrimental influences on one's health. To address cardiovascular risks in abused women, it is extremely vital to prevent IPV from occurring in the first place or avoid this problem from continuing. Healthcare providers are encouraged to adopt routine IPV screening and counseling in both primary care and community settings and refer female survivors for specialty services as early as possible. Once IPV is identified, healthcare providers should consider initiating early recognition of CVD in this vulnerable population through a comprehensive assessment of lifetime experiences of victimization, coping strategies, and total risk factors. Targeted interventions need to be timely implemented at the individual and community levels simultaneously in order to mitigate the effects of violence in this large vulnerable group of women.

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Table

Table 1

Summary of included studies

Ref No.	First Author (Year)	Aim	Sample	Method	Main CVD Measures	IPV Measures	Key Findings
1	Bonomi (2009) [31]	To examine the relative risk of 18 major health outcomes in women with a past-year history of IPV in comparison to those who never experienced IPV	Target population Women who were randomly sampled from a health plan in Washington state and northern Idaho Sample size N=1,928 • IPV+ (12.6%) Age 18-64 y/o Mean- not reported	Prospective, cross-sectional study	Information of cardiovascular signs and symptoms, chest pain, lipid metabolism disorders & HTN were obtained from medical records	Past-year IPV status was screened by using the Women's Experience with Battering Scale. Five questions on BRFSS were used to determine types of IPV (physical, psychological, & sexual abuse).	There was a significant association between past-year IPV and chest pain (aRR=1.53, 1.07-2.20) after adjusting for age. The relative risks of having cardiovascular signs and symptoms (aRR=1.10, 0.71-1.69), HTN (aRR=1.33, 0.96-1.83) and disorders of lipid metabolism (aRR=0.86, 0.46-1.62) did not significantly differ between women who reported past-year abuse and those who never experienced IPV.
2	Breiding (2008) [13]	To examine the impact of lifetime IPV experience on chronic diseases and health behaviors	Target population Adults with and without lifetime IPV exposure in 18 states/territories in the U.S. Sample size N=70,156 • female IPV+ (26.4%)	Secondary analysis of the 2005 Behavioral Risk Factor Surveillance Survey (BRFSS)	Self-report of high cholesterol, high BP, diabetes, heart attack, heart disease, CHD, stroke, high BMI (>25), current heavy/binge alcohol use, current smoking & physical activity	IPV was assessed by asking participants whether they experienced threatened, attempted, or completed physical and/or sexual IPV.	Lifetime IPV victimization among women was significantly associated with high cholesterol (aOR=1.13, 1.01-1.27), CHD (aOR=1.43, 1.06-1.94), stroke (aOR=1.49, 1.08-2.07), smoking (aOR=1.96, 1.76-2.21), and alcohol use (aOR=1.39, 1.18-1.64) after controlling for sociodemographic characteristics and health variables. Heart attack, high BP, diabetes, BMI,

			• male IPV+ (15.9%) <u>Age</u> 18+ y/o Mean- not reported				and physical activity did not differ in women with or without exposure to IPV
3	Clark (2014) [24]	To assess how sex differs in the relationship between IPV (victimization and perpetration) experienced in late adolescence and young adulthood and BP outcomes in young adulthood	<u>Target population</u> Adolescents & young adults in the U.S. <u>Sample size</u> Females (N=5,388) • IPV+ (46.9%) Males (N=4,311) • IPV+ (33.2%) <u>Age</u> Mean age of female participants at Wave 3 =21.72 y/o	Secondary analysis of waves 3 (2001-02) and 4 (2008-09) of the National Longitudinal Study of Adolescent Health	Objective measure of BP at wave 4	Victimization of physical and sexual IPV was examined by four questions based on the Revised Conflict Tactics Scales.	12.21% women who did not report hypertension in Wave 3 had hypertension at Wave 4. Mean values of SBP (moderate victimization [OR=0.36, -0.78-1.56]; severe victimization [OR=0.59, -1.51-2.68] and DBP (moderate victimization [OR=-0.13, -0.99-0.73]; severe victimization OR=1.47, -0.28-3.21]) did not significantly differ by severity of IPV exposure among women. No relationship was found between IPV and incident hypertension in women.
4	Clark (2016) [22]	To examine the relationship between IPV (victimization and perpetration) experienced in late adolescence and young adulthood with CVD	<u>Target population</u> Adolescents & young adults in the U.S. <u>Sample size</u> Females (N=5,504) • IPV+ (8.45%) Males (N=4,472) • IPV+ (12.54%)	Secondary analysis of waves 3 (2001-02) and 4 (2008-09) of the National Longitudinal Study of Adolescent Health	30-year CVD Framingham Risk Score (sex, age, SBP, smoking, diabetes, BMI, and antihypertensive medications)	Physical and sexual IPV was examined by using four items based on the Revised Conflict Tactics Scales	The average mean score of 30-year CVD risk was 9.03 (8.64-9.43) in women at Wave 4. Regression analysis showed that a one-standard deviation increase in IPV victimization score was associated with 0.28% (0.03-0.54, *p*=.03) increase in 30-year CVD risk score among study participants. Other significant variables that predicted higher long-term CVD risk were male

		risk in adulthood and how this relationship differs by sex	<u>Age</u> Mean age of female participants at Wave 3 =21.72 y/o				sex, black race, older age, financial stress, higher neighborhood poverty, and physical neglect.
5	Dichter (2011) [21]	To examine the relationship between lifetime IPV victimization and CVD risk factors and whether the relationship differed by veteran status	<u>Target population</u> Female veterans and non-veterans in the U.S. <u>Sample size</u> N=21,162 • IPV+ (24.3%) <u>Age</u> 18+ y/o Mean- not reported	Secondary analysis of the 2006 BRFSS	Self-report of depression symptoms, current smoking, binge/heavy drinking, physical inactivity & overweight/obese	Lifetime victimization of physical and sexual IPV was assessed by self-report	Veteran women who experienced IPV were more likely to be overweight/obese than those without IPV history (66.4% vs 48.5%, $p<.05$). IPV victimization significantly associated with depression (aOR=3.80, 3.20-4.50), smoking (aOR=2.80, 2.40-3.2), and binge/heavy drinking (aOR=1.8, 1.50-2.10). No association between IPV victimization and physical inactivity or overweight/obese.
6	Flores-Torres (2017) [25]	To assess the association between different types of violence and carotid intima-media thickness (IMT)	<u>Target population</u> Female Mexican teachers <u>Sample size</u> N=634 • IPV+ (not clearly reported) <u>Age</u> 25+ y/o Mean=48.9 y/o	Secondary analysis of the Mexican Teachers' Cohort Study	Carotid IMT	Physical and/or sexual violence by family members or strangers was assessed by using the violence-related items on the Life Stressor Checklist	The multivariable-adjusted mean percentage difference in carotid IMT between women who experienced violence exposure and those who did not was 2.4% (0.50-4.30). Women who reported exposure to violence had 60% higher odds of subclinical carotid atherosclerosis than those with no history of violence (OR=1.60, 1.10-2.32).
7	Gass (2010) [26]	To assess the relationship between physical	<u>Target population</u> Married and cohabiting	Secondary analysis of the South African	Self-report of heart attack, stroke, high BP, diabetes, current and	Physical IPV in current or most recent	Physical IPV was significantly associated with smoking (current: aOR=1.90, 1.09-3.30; past: aOR=1.68, 1.06-

		IPV and chronic physical illnesses, health-risk behaviors, and health-seeking behaviors	women in South Africa Sample size N=1,229 • IPV+ (31%) Age 18+ y/o Mean- not reported	Stress and Health (SASH) study	lifetime use of tobacco and alcohol	intimate relationships was assessed by self-report	2.68) and drinking (current: aOR=2.37, 1.28-4.41; past: aOR=1.89, 1.30-2.75). No relationship was found between physical IPV and stroke within the past 12 months (aOR=1.32, 0.62-2.79), heart attack within past 12 months (aOR=1.82, 1.00-3.32), history of heart disease (aOR=1.23, 0.69-2.18), high BP (aOR=1.45, 0.96-2.20), and diabetes (aOR=1.36, 0.67-2.75).
8	Halpern (2017) [15]	To identify stress-related CVD outcomes in IPV survivors and to compare CVD inflammatory biomarkers between women with or without exposure to IPV	Target population African American (AA) and non-AA females (N=37) from an oral surgery clinic in the U.S. Sample size N=37 • IPV+ (51%) Age 19-63 y/o Mean- not reported	Prospective cross-sectional study	10 salivary CVD biomarkers including CD40L, IL-1β, IL-6, TNF-α, Adiponectin, PAI-1, MMP9, Myoglobin, ICAM-1 & CRP Self-report of in- vivo CVD events including chest pain, heart palpitations & HTN	IPV was screened by using three items on the Partner Violence Screen. Long-term injuries resulted from abuse and physical symptoms were examined by using the Partner Abuse Symptom Scale	Abused women showed significant correlations with respect to IL-1β/sCD40L; TNF-α/sCD40L; Myoglobin/IL-1β; CRP/sCD40L; CRP/IL-6; CRP/TNF-α; TNF-α/sICAM; CRP/MMP9 & TNF-α/Adiponectin in comparison to their non-abused counterparts (p<.05). Significant association was found between IPV victimization and chest pain (p=.01) and heart palpitations (p=.02).
9	Mason (2012) [28]	To examine the relationship between IPV in adulthood and HTN	Target population Female nurses in the U.S. who did not have HTN or use of anti-hypertensive medication at baseline	Secondary analysis of the Nurses' Health Study II (NHSII), 2001-2007	Self-report of HTN	Lifetime victimization of physical and sexual IPV was assessed by two questions. Lifetime emotional abuse was	No significant association was found between HTN and physical (aHR=1.06, 1.00-1.12) or sexual IPV (aHR=0.99, 0.91-1.07) after adjusting for history of childhood abuse, race, parental education, somatogram score at age 5, and BMI at age 18. Only women who suffered severe emotional

			Sample size N=51,434 • IPV+ (62% physical IPV; 10% sexual IPV; 1.5% severe emotional IPV) Age 37-54 y/o at baseline Mean= 46 y/o at baseline			assessed by using the Women's Experiences with Battering Scale.	abuse were 24% more likely to be diagnosed with HTN compared to those experienced less degree of psychological abuse (aHR=1.24, 1.02-1.53).
10	Newton (2011) [16]	To assess the relationship of remitted IPV with CRP, IL-6, and *in vitro* IL-6 production	Target population Postmenopausal females with histories of divorce/separation in the U.S. Sample size N=68 • IPV+ (100%) Age 45-60 y/o Mean=55 y/o	Prospective cross-sectional study	plasma CRP & IL-6 and ambulatory BP	Lifetime victimization of physical, sexual, psychological IPV was assessed by using the Revised Conflict Tactics Scale. Lifetime occurrence of stalking was assessed by using the items on the National Violence Against Women Survey.	The average levels of ambulatory SBP and DBP were 119.35 mmHg and 74.55mmHg, respectively. Among the different types of IPV, only stalking was significantly associated with higher CRP levels (r=0.37, $p \leq .04$). Severe physical IPV had a significantly negative association with PHA-stimulated IL-6 production (β=-.08, p=.005) after adjusting for BMI, current posttraumatic stress, and depressive symptoms.
11	Out (2012) [23]	To examine the	Target population	Secondary analysis of a large	Plasma and salivary CRP	Measures of current victimizati	Saliva CRP significantly discriminated CVD risk based on the clinically

		associations of CRP with systemic inflammation (BMI and smoking) and CVD risk	Female IPV survivors who resided in either a shelter or sought help from community agencies in a Midwestern state <u>Sample size</u> N=157 • IPV+ (100%) <u>Age</u> 18+ y/o Mean=34 y/o	2-year longitudinal study which included seven waves of data collection		on of IPV was not described in the study	standard serum levels of CRP (cutoff = 3mg/L).
12	Renner (2017) [32]	To examine CVD risk in women who experienced poly-victimization of violence (three or more prior violent experiences in addition to IPV).	<u>Target population</u> Female IPV survivors who sought help from group psychotherapy at two community-based organizations in a Midwestern state <u>Sample size</u> N=34 • IPV+ (100%) <u>Age</u> 18+ y/o Mean=38.6 y/o	Prospective cross-sectional study	30-year Framingham Risk (age, sex, smoking status, BMI, SBP, diabetes & use of antihypertensive medications)	Lifetime victimization of child maltreatment and sexual abuse was assessed by four self-report questions.	50% of the sample experienced poly-victimization violence. The mean Framingham CVD risk score was higher in IPV survivors who experienced severe poly-victimization (at least three forms of violence) than those who experienced fewer violence victimizations in addition to IPV in adulthood (31.47 vs 13.18, β=9.25, *p*=.069). Age showed a significantly positive association with the 30-year CVD risk score (β=1.33, *p*<.001)
13	Scott-Storey (2009) [3]	To examine the relationship	<u>Target population</u> Female IPV victims who	Secondary analysis of the Women's	cardiovascular symptoms were measured by	IPV status was screened by using a	44.1% reported current smoking; 53.2% were overweight or obese; 12.3% had hypertension;

		of IPV-associated stress (IPV severity and current abuse), smoking, and cardiovascular risk	left abusive partners between 3 months and 3 years in a Canadian community <u>Sample size</u> N=309 • IPV+ (100%) <u>Age</u> 19-63 y/o Mean=40 y/o	Health Effects Study, 2004-2005	the 4-item cardio-respiratory subscale of the Partner Abuse Symptoms Scale (symptoms of hypertension, chest pain, shortness of breath, heart palpitations), self-reported smoking, CVD diagnoses and CVD medications, and objective measure of BMI and BP	modified version of the Abuse Assessment Screen. Severity of abuse was examined by the Index of Spouse Abuse	50.8% experienced cardiovascular symptoms. Presence of cardiovascular symptoms was significantly associated with age and income, but it was not significantly associated with current abuse (OR=1.15, 0.68-1.94), severity of past IPV (OR=1.00, 0.99-1.02), or smoking status (OR=1.06, 0.63-1.79). Smoking was positively related to severity of past abuse (t=3.586, p≤0.001) and negatively related to current abuse (χ^2=5.19, *p*=.023).
14	Stene (2013) [11]	To examine the association of IPV with cardiovascular risk and drug treatment	<u>Target population</u> Females in Oslo <u>Sample size</u> N=5,593 • IPV+ (13.4%) <u>Age</u> 30-60 y/o Mean - not reported	Cross-sectional study	CVD medications, Framingham 10-year risk score (age, sex, diabetes, smoking, SBP, total cholesterol, HDL), alcohol use, physical activity, BMI, abdominal obesity, non-fasting serum blood lipids, and cardiovascular medication use	Recent or lifetime victimization of psychological, physical, and sexual IPV was examined by using five self-reported questions	Women who experienced physical and/or sexual IPV were more likely to have abdominal obesity, lower HDL level, higher triglyceride level, and higher 10-year CVD risk scores in comparison to non-abused women. Physical and/or sexual IPV were associated with antihypertensive use after adjusting for age, education, and BP (IRR=1.36, 1.09-1.7).
15	Symes (2010) [33]	To assess the alterations	<u>Target population</u>	Prospective	11 serum CVD biomarkers	Physical and sexual IPV were	Only the level of serum vascular cell adhesion molecule-1 (sVCAM-1)

		in serum levels of CVD biomarkers in IPV survivors hospitalized for acute coronary syndrome (ACS) at baseline, 3 months and 6 months after hospitalization.	Women hospitalized for acute coronary syndrome (ACS) in the U.S. Sample size N=45 • IPV+ (55.6%) Age 18+ y/o Mean=57 y/o	longitudinal study	including neuroendocrine markers (Prolactin & Cortisol/DHEA ratio), proinflammatory cytokines (TNF-α, IL-6 & CRP), and cell adhesion molecules and a chemotactic cytokine (VCAM-1, ICAM-1, selectin & MCP-1) obtained from blood	assessed by the Lifetime Trauma and Victimization.	showed a moderate group effect size (multivariate EF=0.19) among the biomarkers examined.
16	Vives-Cases (2011) [27]	To assess the prevalence of different forms of violence against women in Spain and to compare the effect of abuse on women's health outcomes between those who experienced IPV and those who experienced other forms of violence against women	Target population Female adults in Spain Sample size N=13,094 • IPV+ (0.98%) Age 16+ y/o Mean: not reported	Secondary analysis of the Spanish National Health Survey, 2006	Self-reported hypertension and CHD	IPV within the previous 12 months was assessed by two self-reported questions	There was no significant relationship between IPV and hypertension (aOR=0.81, 0.45-1.47). Women who experienced IPV were 5.28 more likely to have CHD compared with those who never experienced IPV (aOR=5.28, 1.45–19.25).
17	Vijayaraghavan (2012) [12]	To assess health characteristics, access	Target population Homeless women living	Secondary analysis of the HIV Risk	self-report of diabetes, HTN and obesity,	Lifetime or current victimization of	Homeless women with and without histories of IPV did not differ significantly in

		to health care, and health care use among homeless women who lived in shelters and compare these outcomes between women with and without histories of IPV	in 28 shelters in New York City <u>Sample size</u> N=329 • IPV+ (44.7%) <u>Age</u> 18+ y/o Mean=37.9 y/o	among Homeless Women Study, 2007-2008	smoking & alcohol use	physical IPV was assessed by self-report	cardiovascular risk factors of HTN, diabetes, and obesity (36.3% vs. 27.4%, *p*=.09), smoking (54.8% vs. 46.4%, *p*=.3), or alcohol use (61.8% vs. 54.2%, *p*=.2).
18	Wright (2018) [29]	To assess the association between past-year IPV exposure and 30-year CVD risk	<u>Target population</u> Women with and without exposure to past-year IPV in the U.S. <u>Sample size</u> N= 7,392 • IPV+ (15.7%) <u>Age</u> 24-32 y/o Mean: 29 y/o	Secondary analysis of Wave IV of the National Longitudinal Study of Adolescent to Adult Health	Framingham 30-year risk (age, sex, SBP, use of antihypertensive medications, diabetes, BMI, smoking status)	Physical and sexual IPV in the past year was assessed by the Revised Conflict Tactic Scales	The mean scores of 30-year CVD risk between abused and non-abused women were 9.6% and 8.7%, respectively (*p*<.01). Women who experienced past-year IPV had a statistically significant .009-unit increase in 30-year CVD risk score when compared to non-abused women (*p*<0.01). However, the relationship became insignificant after adjusting for demographic variables or other important predictors (*p*=.33).
19	Wright (2019) [30]	To examine potential mediators, including depressive symptoms, perceived stress, and alcohol dependence, on the relationship between	<u>Target population</u> Women with and without exposure to past-year IPV in the U.S. <u>Sample size</u> N= 7,392 • IPV+ (15.7%) <u>Age</u>	Secondary analysis of Wave IV of the National Longitudinal Study of Adolescent to Adult Health	Framingham 30-year risk (age, sex, SBP, use of antihypertensive medications, diabetes, BMI, smoking status)	Physical and sexual IPV in the past year was assessed by the Revised Conflict Tactic Scales	Perceived stress (B=0.047, SE=0.010, *p*<.01) and depressive symptoms (B=0.054, SE=0.012, p< .01) partially mediated the relationship between IPV and 30-year CVD risk score. Alcohol dependence did not mediate the relationship between IPV and 30-year CVD risk score. Only depressive symptoms

		IPV and CVD risk	24-32 y/o Mean: 29 y/o				(B=0.04, SE=0.01, *p*=.003) remained as a partial mediator on the relationship between past year IPV and 30-year CVD risk score when including perceived stress, depressive symptoms, and alcohol dependence in the multiple mediation analyses.

Abbreviations and Acronyms: aOR – adjusted odds ratio; aHR – adjusted hazard ratio; aRR – adjusted relative risk; BP - blood pressure; SBP – systolic blood pressure; DBP - diastolic blood pressure; BMI – body mass index; CVD – cardiovascular heart disease; CHD – coronary heart disease; CRP – C-reactive protein; CI – confidence interval; HDL – high-density lipoproteins; HTN - hypertension; IPV – intimate partner violence (IPV+ denotes positive experience of IPV); IRR – incidence rate ratio; IL-6 – interleukin 6; SE – standard errors

Figure

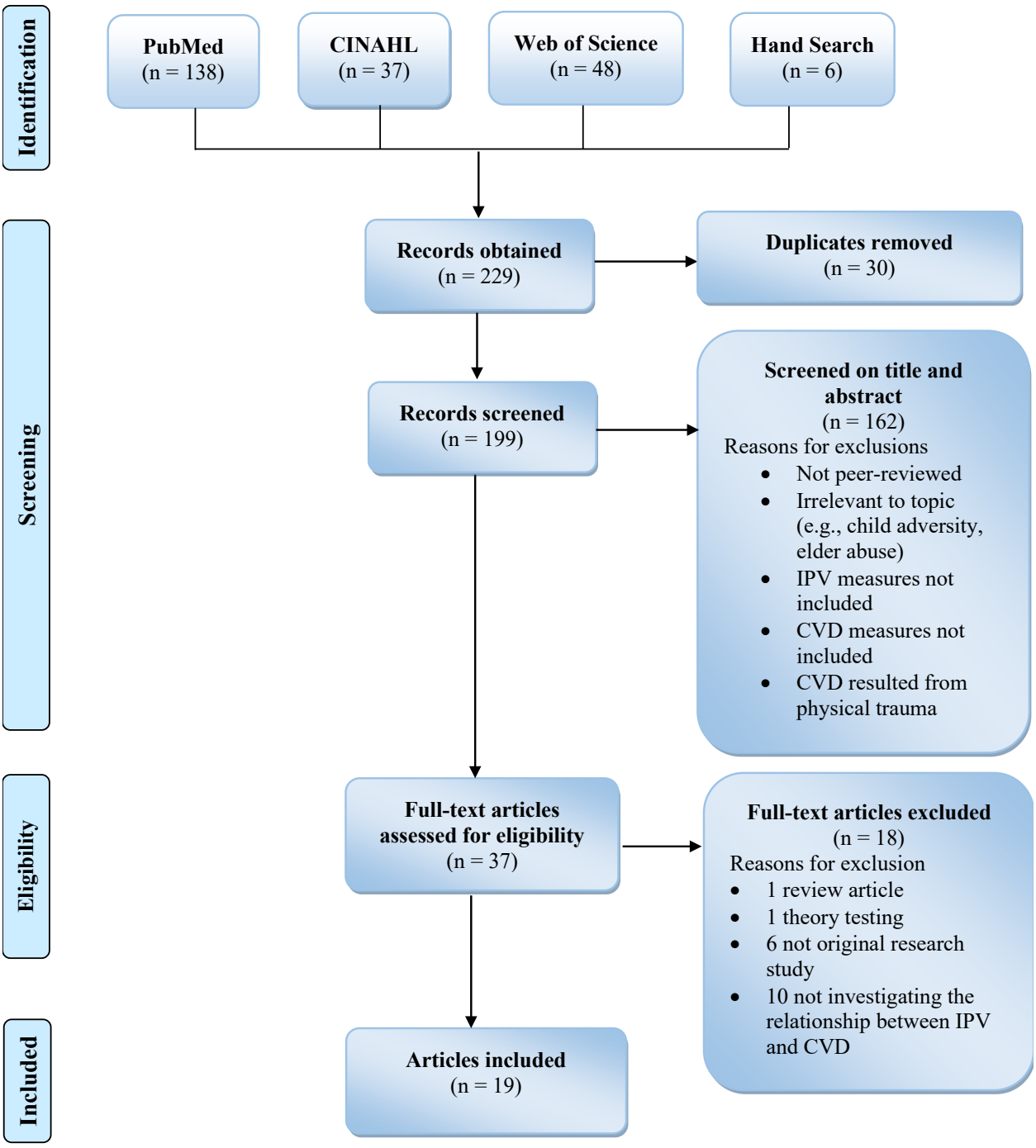

Figure 1. PRISMA flowchart of search process

Chapter 4: Examining Sleep, Physical Activity, Psychological Stress, and Fatigue Between Chinese Americans and Non-Hispanic Whites

Target journal: Journal of Community Health

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Abstract

Chinese Americans make up the largest Asian subgroup in the U.S. Little research has specifically focused on the well-being of this population. This study aimed to 1) examine racial differences in health behaviors (sleep and physical activity) and psycho-physiological health (psychological stress and fatigue) between Chinese Americans and non-Hispanic whites; and 2) investigate whether race and health behaviors were independent predictors of psycho-physiological health. Using a convenience sampling strategy, we recruited 87 middle-aged healthy adults (41 Chinese Americans, 46 non-Hispanic whites). We evaluated variables of interest with questionnaires and measured participants' sleep characteristics using a wrist actigraph. Sociodemographic covariates (age, sex, education, and income) were self-reported. When compared with the white cohort, Chinese Americans demonstrated significantly poorer objective sleep outcomes (shorter sleep duration, lower sleep efficiency, and longer sleep onset), engaged in lower physical activity levels, and reported higher psychological stress and fatigue after controlling for the covariates. The results of multivariate analyses suggested that race and poor perceived sleep quality were independently associated with high psychological stress and fatigue. This study revealed significant disparities in behavioral and psycho-physiological health between Chinese Americans and whites. These findings warrant further explorations of social and cultural determinants of health in this minority group. Such research will facilitate healthcare providers' understanding of the underlying factors and barriers that contribute to health disparities in Chinese Americans, and development of targeted, culturally sensitive interventions.

Keywords: Chinese Americans; sleep; physical activity; psychological stress; fatigue.

Background

Chinese Americans represent the largest Asian subgroup in the U.S. and account for 24% of the national Asian population (López et al., 2017). Seventy-six percent of this group are first-generation Chinese immigrants, comprising the third-largest foreign-born group in the U.S (*Chinese in the U.S. Fact Sheet*, 2017; Keister et al., 2016). Available data demonstrate that Chinese Americans have been disproportionally affected by numerous diseases compared with other racial/ethnic groups in the U.S. For example, Chinese Americans have higher rates of hypertension, left ventricular hypertrophy, and high blood lipids as well as glucose levels than whites (Fang et al., 2004). Chinese Americans have also been found to have worse health outcomes than whites, experiencing longer hospitalizations for mental health issues and higher risks of 30-day potentially preventable readmissions after stroke-related hospitalizations (Nakagawa Kazuma et al., 2016; Tong & Sentell, 2017). Despite the considerable population growth and unique health disparities experienced by Chinese Americans, little research has focused on the well-being of this population. Previous studies that examined health outcomes across racial/ethnic groups have predominantly combined persons of Asian ancestry into a single group. This classification masks the heterogeneity of health issues among the subgroups because factors such as different cultural backgrounds can play unique roles in individual well-being (Jose et al., 2014; Palaniappan et al., 2010). Therefore, our study aimed to identify key risk factors that likely influence health outcomes in Chinese Americans, an large yet overlooked group.

Sleep and physical activity are paramount health behavioral factors that have an impact on well-being. With increased length of residence in the U.S., Chinese Americans

exhibit acculturation-associated changes in lifestyle behaviors (Lv & Cason, 2004). Among factors closely linked to individuals' well-being, sleep plays an indispensable role in maintaining bodily functions and homeostasis. A recent study reported that elderly Chinese Americans were more likely to have sleep-disordered breathing disorders and shorter sleep duration than whites (X. Chen et al., 2015). Despite this alarming finding, it is not clear how other aspects of sleep characteristics (e.g., sleep quantity and quality) present in Chinese Americans. Further, the U.S. Asian populations are frequently reported to be physically inactive, and this major health issue persists in Chinese Americans (Kandula & Lauderdale, 2005; Kao et al., 2016; Yi et al., 2015). One study found that only 24.3% of Chinese Americans living in New York City engaged in moderate or vigorous exercise in quantities that met standard physical activity guidelines. The same study also demonstrated a strikingly lower prevalence of aerobic exercise among Chinese participants in comparison to other Asian ethnicity cohorts (Yi et al., 2015). Recent data reveal that adults aged 30- 64 years old comprise 47% of the Chinese American population (*Chinese in the U.S. Fact Sheet*, 2017). This age group might be expected to be more physically active than seniors (whose exercise habits are more often studied), nonetheless Yi et al. (2016) found that physical activity among Chinese Americans tended to increase with age, with those over 65 years old performing the highest level of physical activity, more than any other age group over 21. Current behavioral studies which included Chinese Americans rarely focus on individuals in middle-adulthood. Given these knowledge gaps, we compared sleep characteristics and physical activity between Chinese Americans and whites in middle-adulthood.

This study also focused on psychological stress and fatigue among Chinese Americans, as both are important factors that indicate psychological and physiological well-being (Schneiderman et al., 2005; Williamson et al., 2005). Chinese Americans are likely to encounter specific challenges after migration, such as language barriers, separation from family, social isolation, cultural value conflicts, and limited access to health insurance and health services. These can result in mental and physiological burden in this population (Y. Chen et al., 2019). In one study of elderly Chinese Americans, almost three-quarters reported experiencing some form of psychological stress in their daily lives (Zhang et al., 2014). Another study on Chinese Americans with Type II diabetes noted that almost two-thirds reported fatigue (Kuo et al., 2020). In fact, the prevalence of psychological stress and fatigue experienced by Chinese Americans is potentially higher than reported, as individuals from Asian cultures are known to underreport these conditions (Lim et al., 2003; Uppaluri et al., 2001). There is scarce research reporting how psychological stress and fatigue levels among Chinese Americans differs from other racial groups; for that reason, we examined these two psycho-physiological factors in this study.

Racial differences in behavioral activities may impact both psychological and physiological health. Therefore, we conducted a cross-sectional study to evaluate the health behaviors and psycho-physiological health among Chinese Americans and whites. We assessed two sets of factors: behavioral factors (sleep and physical activity) and psycho-physiological factors (psychological stress and fatigue). The aims of our study are two-fold. First, we compared how sleep characteristics, physical activity, psychological stress, and fatigue differed between Chinese Americans and whites. Second, we

investigated whether race and health behavioral factors were independent predictors of psychological stress and fatigue.

Methods

Participants

A convenience sample of Chinese Americans and whites was recruited from central Virginia by posting flyers in all communities and disseminating study information through word-of-mouth. Our study inclusion criteria were healthy adults aged 35- 64 years old. To reduce variations within the sample, we recruited participants by age categories (35-44, 45-54, and 55-64 years old). The number of participants in each age category was approximately the same between the two racial groups. Our final sample included 41 Chinese Americans and 46 non-Hispanic whites. We defined Chinese Americans as individuals who self-identified as Chinese and arrived in the U.S. after the age of 12 and whites as individuals who self-identified as being non-Hispanic. We explained detailed study procedures to individuals who expressed interest and obtained informed consent in-person. Subsequently, each participant was asked to fill out questionnaires and take part in a two-night sleep assessment. This study was approved by the university's Institutional Review Board.

Instruments

Sleep Characteristics

Sleep characteristics were measured subjectively and objectively. To measure participants' perception about sleep quality, we used the Pittsburgh Sleep Quality Index (PSQI), a 19-item questionnaire evaluating sleep quality and disturbances during the previous month (Buysse et al., 1989). The items cover seven components, including

subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. Each component score is equally weighted on a 0-3 scale, with 3 indicating the worst function. The sum of all component scores derives a total score ranging from 0 (greatest) to 21 (worst), and a cutoff value of 5 distinguishes "good sleepers" from "poor sleepers." In addition, participants wore an actigraph (GT9X Link, ActiGraph, FL) on their non-dominant wrist over two consecutive nights for objective sleep assessments. The data were processed with the ActiLife 6.13.4 software (ActiGraph, FL) and scored in 1-minute epochs using the Cole-Kripke sleep algorithm to determine sleep/wake state (Cole et al., 1992). Consequently, we obtained the following variables: sleep onset—the first epoch scored as sleep; sleep duration—the amount of time between sleep onset and sleep end; sleep efficiency—the ratio of sleep duration to time spent in bed; and wake after sleep onset (WASO)—the amount of time spent awake between the initial sleep onset and falling asleep again. During the two days of sleep assessments, we also asked participants to complete a sleep diary in which they recorded their lights-off and out-of-bed times. The diary information was used to validate the accuracy of the actigraphy data.

Physical Activity

Physical activity was assessed by the Global Physical Activity Questionnaire (GPAQ). This questionnaire collects information about the intensity, frequency (days), and duration (hours/minutes) of physical activity performed in a typical week. GPAQ contains 15 items assessing moderate and vigorous physical activity on three domains—activity at work, travel to and from places, and recreational activities, as well as one item evaluating a participant's sedentary behavior by asking their average time per day spent

in sitting or reclining (Armstrong & Bull, 2006). The questionnaire is supplemented with picture cards to help participants better understand the activity inquired about in each question. We used the analysis guidelines provided by the World Health Organization to estimate each individual's energy expenditure using the GPAQ data. The intensity of physical activity was quantified using metabolic equivalent of task (MET), which is the ratio of a person's working metabolic rate relative to the resting metabolic rate. Four METs were assigned to the time spent in moderate activities, and 8 METs to the time spent in vigorous activities. We multiplied the assigned MET value by the average minutes per week performing physical activity to derive an energy expenditure score for each of the three domains. The scores were then summed to create an overall physical activity MET value. We originally classified participants' physical activity levels into three groups: low (< 10 MET-hours/week), moderate (10 - 50 MET-hours/week), or high (> 50 MET-hours/week). Since only one Chinese American met the high category of physical activity level, we combined the number of individuals who had moderate or high levels of physical activity into one group.

Psychological Stress

Psychological stress was measured by the 10-item Perceived Stress Scale (PSS) (S. Cohen et al., 1983). The PSS instrument examines the level at which situations are appraised as stressful in an individual's life. The PSS estimates how unpredictable, uncontrollable, and overloaded individuals perceive their lives to be (S. Cohen et al., 1983). Participants rated their feelings or thoughts in the past month on a scale of 0 (never) to 4 (very often). In this questionnaire, 4 out of 10 items were negatively worded,

and they were scored in the reverse direction. We calculated the total score by summing all ratings across the items, with a higher score indicative of greater psychological stress.

Fatigue

Fatigue was measured by the fatigue symptom inventory (FSI) (Hann et al., 1998). This 14-item questionnaire evaluates four dimensions of fatigue—severity, frequency, perceived interference on daily functioning, and diurnal variation. The 4-item severity subscale evaluates participants' most, least, and average level of fatigue experienced during the past week as well as their current level of fatigue. Each item was rated on an 11-point scale (0 = "not at all fatigued" to 10 = "as fatigued as I could be"). The 7-item functioning subscale measures the degree to which fatigue interferes with general activity level, ability to bathe and dress, work activity, ability to concentrate, relations with others, enjoyment of life, and mood. Those items were assessed on a separate 11-point scale (0 = "no interference" and 10 = "extreme interference"). The 2-item frequency subscale evaluates the number of days the participants felt fatigued (from 0 to 7 days) and the average portion of each day they felt fatigued (from 0 = "none of the day" to 10 = "the entire day") during the past week. The last item evaluates participants' daily pattern of fatigue. This item is designed to only provide descriptive information about their diurnal variation of fatigue (Donovan & Jacobsen, 2011). We summed the first 13 items to derive a total score with a higher score indicating more fatigue.

Sociodemographic Covariates

Sociodemographic covariates included self-report of age, sex, education, income, and smoking history. The following variables were analyzed as binary variables: education (<

12 years of formal education vs. ≥ 12 years of formal education), income (< $75,000 vs. ≥ $75,000), and smoking history (never smoked vs. lifetime smoking history).

Statistical Analysis

Descriptive statistics were reported as mean ± standard deviation (SD) for continuous variables and frequency (%) for binary variables. We used two-sample t-tests or Chi-square tests to assess differences between the two racial groups on sociodemographic characteristics, sleep characteristics, physical activity, psychological stress, and fatigue. Additionally, we performed multiple linear regression analyses to compare those factors between Chinese Americans and non-Hispanic whites (as the reference group) with adjustment of the sociodemographic covariates (age, sex, education, income, and smoking history). To examine how stress and fatigue levels were influenced by race and behavioral factors, we used multivariate analyses of covariance in which stress and fatigue were dependent variables, and race and one of the health behavior variables (sleep metrics and physical activity) were entered as independent variables while controlling for the covariates. All data were analyzed with SPSS version 26. The 2-tailed significance level was set at $p < 0.05$.

Results

The sociodemographic and clinical characteristics of the two racial groups are outlined in Table 1. The two groups did not significantly differ on the key sociodemographic variables (age, sex, and income). In this sample, the Chinese participants were more likely to have higher education and a lower rate of smoking history.

Table 2 presents the adjusted comparisons of the main study variables—sleep, physical activity, psychological stress, and fatigue—between Chinese Americans and whites. Although there was no significant difference in perceived sleep quality between the two groups, the actigraphy-measured sleep data demonstrated that Chinese Americans had an average of 0.92 hour (55 minutes) of shorter sleep duration ($p = 0.001$) and 4.5% of lower sleep efficiency ($p = 0.01$) than white participants. This minority group scored 57.1 MET-hours/week fewer physical activity levels than whites ($p = 0.001$). In addition, the levels of reported psychological stress and fatigue were significantly higher in Chinese Americans than their counterparts after adjusting for the covariates ($p < 0.05$).

Table 3 presents the results of multivariate analysis of variables. In the baseline model, race was an independent predictor of psychological stress and fatigue. More specifically, when compared with whites, Chinese Americans had significantly higher levels of psychological stress (mean difference = 3.99, $p = 0.013$) and fatigue (mean difference = 13.87, $p = 0.016$) after adjusting for the covariates. When each of the sleep variables was individually added to the baseline model, race was consistently associated with psychological stress and fatigue (Models 1 to 6). Only perceived sleep quality was deemed as a predictor in Model 1, indicating that subjective poor sleep quality appeared to increase psychological stress ($\beta = 0.67$, $p < 0.001$) and fatigue ($\beta = 2.96$, $p < 0.001$) independent of race. When compared with those who had low physical activity, individuals with moderate or high physical activity showed no difference in the levels of psychological stress and fatigue, after accounting for the racial differences in psychological stress and fatigue.

Discussion

We studied a cohort of healthy, middle-aged Chinese Americans and whites and examined their health behaviors and psycho-physiological health. We found that the Chinese participants experienced more impaired sleep, engaged in less physical activity, and reported higher psychological stress and fatigue in comparison to their white counterparts. We also found that race and perceived sleep quality each served as an independent predictor of psychological stress and fatigue. This is the first study comprehensively assessing behavioral and psycho-physiological health among Chinese Americans. The findings underscore the need to promote sleep outcomes and reduce the burdens of psychological stress and fatigue in this population.

Impaired sleep can affect all bodily systems and increase the risks of cardiovascular disease, metabolic dysfunction, psychiatric disorders, and early mortality (Kline, 2014). To date, there is a limited number of studies addressing sleep health in racial/ethnic minority groups; Asians, in particular, are considerably less likely to be included in sleep disparity research compared with black or Hispanic populations (Johnson et al., 2019). Despite this scarce evidence, a few studies have documented poor sleep outcomes in the larger category of Asian Americans. One study showed that Asian Americans had an average of 37.8 minutes of shorter sleep duration ($p < 0.01$) and more frequent daytime sleepiness in comparison to white participants ($p < 0.01$) (Carnethon et al., 2016). Chen and colleagues (2015) reported that middle-aged Chinese Americans had higher odds of short actigraphy-measured sleep duration (< 6 hours), higher prevalence of habitual snoring, and lower sleep quality than their white counterparts. Our findings of sleep disparities in Chinese Americans echoed those researchers' work and supported the evidence that suboptimal sleep could link between racial/ethnic minorities and health

disadvantage (Johnson et al., 2019). Thus, timely recognition of impaired sleep among minority populations followed by appropriate, culturally sensitive interventions could improve general health outcomes. We further identified that Chinese Americans had a better subjective rating of sleep quality, while they manifested poorer objective sleep outcomes than whites. Underreporting of impaired sleep was not only observed in our Chinese American cohort, but was also noted in Caribbean Americans according to the literature (Jean-Louis et al., 2001; Johnson et al., 2019). For this reason, healthcare providers should be aware of the potential response bias when conducting sleep assessments for minority populations. The findings also implied that applying objective sleep evaluation tools (e.g., actigraphs) in clinical practice could increase the validity of self-report and enhance identification of high-risk minority populations.

Physical activity was another health behavioral factor examined in this study. According to the World Health Organization (2005), achieving an equivalent combination of moderate- and vigorous-intensity activities at least 10 MET-hours/week meets physical activity recommendations. The average MET scores for both racial groups studied exceeded the recommended level of weekly energy expenditure. However, the Chinese cohort had 57.13 fewer MET-hours/week than the white participants after adjusting for the covariates, demonstrating a distinctively lower level of physical activity in this minority group. The literature has suggested a few barriers to performing physical activity in Chinese Americans, such as lack of time, safety concerns and weather conditions, limited resources, high costs of fitness equipment, insufficient education for physical activity, health concerns about exercising, and inadequate English proficiency (Fu et al., 2018; Le et al., 2019; Victoria M. Taylor et al., 2008). In addition, the

traditional Chinese culture might be a contributing factor, as many Chinese Americans prefer to engage in constant, flowing movements with an emphasis on mind and body balance (e.g., Tai Chi) versus performing intense or strenuous activities (Katigbak et al., 2020). Further, the issue of low physical activity in Chinese Americans could be related to acculturation. One study noted that Chinese Americans who were less acculturated tended to participate in a lower amount of physical activity (Yi et al., 2016). Given the fact that filial piety and family harmony are two central values in Chinese culture, Chinese Americans who have a strong identification with their native culture may place family duties as their higher priority versus performing physical activity (Le et al., 2019). It is essential to investigate socio-cultural determinants of health behaviors in this minority group; the results of such investigation should be used to develop targeted interventions that address or potentially eliminate the persistent disparity problem.

Besides health behavioral factors, we examined two important indicators of psycho-physiological health which can greatly impact the general well-being, namely psychological stress and fatigue. Our results strongly suggested that Chinese Americans were more vulnerable to psychological stress and fatigue than whites. This finding should raise questions about a common assumption that Asian Americans are typically viewed as a "model minority" due to the perception that they are better adjusted and thriving more in the U.S. than other racial/ethnic groups (Miller et al., 2011). This assumption may be false as several investigations have illustrated a high prevalence of mental and physical problems among Asian Americans, specifically in Chinese Americans. It has been reported that Chinese Americans commonly experienced depression, anxiety disorders, and neurasthenia (Kung & Lu, 2008). We posit that these mental illnesses and poor

lifestyle behaviors (e.g., impaired sleep and physical inactivity) contribute to high psychological stress and fatigue in Chinese Americans, which may consequently affect one's performance, cognition and emotion, as well as dysregulation of autonomic function and the immune systems (Kocalevent et al., 2011; Kop & Kupper, 2016). Because better stress management skills are associated with lower fatigue levels, healthcare providers can help Chinese Americans acquire effective stress reduction techniques to enhance psycho-physiological health in this population (Lattie et al., 2012).

Another finding of importance is that race and low subjective sleep quality significantly predicted high psychological stress and fatigue. On one hand, this demonstrated crucial of racial difference in contributing health disparities, warranting future health disparity research targeting racial/ethnic minority populations to bridge the gap of health inequity. On the other hand, the effect of perceived sleep quality on psycho-physiological health implied that implementing a behavioral sleep intervention (e.g., relaxation training or stimulus control) could be one approach considered to reduce psychological stress and fatigue in Chinese Americans.

Strengths and Limitations

This study showed significant health disparities between whites and Chinese Americans, an understudied population. Our enrollment of middle-aged adults was important because this age group has often been overlooked, and their current health status may directly reflect future health outcomes. Our study assessed objective as well as subjective sleep, confirming self-reporting bias in this ethnic minority group. Several limitations must also be considered when interpreting the results. This study was limited by the small sample and the highly educated nature of the Chinese American cohort.

These results should be confirmed with a larger, fully powered sample, and Chinese participants with various socioeconomic and educational backgrounds should be recruited to represent the characteristics of the general Chinese American population. Since objective sleep characteristics were only measured over two nights, the data may not accurately capture participants' actual sleep patterns. Future studies are encouraged to measure sleep outcomes for at least seven consecutive nights to derive a better estimation of sleep.

Conclusions

In summary, our study identified several health disadvantages faced by Chinese Americans. Future studies are suggested to investigate social and cultural determinants of health through a culturally sensitive, community-based approach among Chinese Americans. Such research may help healthcare providers better understand the underlying cause of the observed health disparities. Further, longitudinal and interventional research is needed to address poor behavioral and psycho-physiological health. This minority group may benefit from targeted sleep interventions, physical activity programs, and stress management approaches to reduce health disparities.

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Tables

Table 1

Sociodemographic and Clinical Characteristics of Study Participants

	Chinese Americans (n = 41)	Whites (n = 46)	*p*
Age, year	46.4 ± 8.1	48.2 ± 9.7	0.346
Sex, n (%)			
Male	15 (36.6%)	23 (50.0%)	0.208
Female	26 (63.4%)	23 (50.0%)	
Education, n (%)			
College or below	18 (43.9%)	30 (65.2%)	0.046
Above college	23 (56.1%)	16 (34.8%)	
Income, n (%)			
$75,000 or below	17 (42.5%)	24 (47.8%)	0.370
Above $75,000	23 (57.5%)	22 (52.2%)	
Smoking history, n (%)			
Never smoked	36 (87.8%)	24 (52.2%)	< 0.001
Lifetime smoking history	5 (12.2%)	22 (47.8%)	
Perceived sleep quality	6.3 ± 3.7	6.7 ± 4.1	0.597
Poor sleeper, n (%)	19 (46.3%)	28 (60.9%)	0.175
Objective sleep characteristics			
Sleep duration, hour	6.1 ± 1.2	7.0 ± 1.2	0.001
Sleep efficiency, %	81.7 ± 8.2	85.9 ± 5.8	0.008
Sleep onset, minute	4.6 ± 7.2	2.4 ± 3.2	0.082
WASO, minute	73.7 ± 33.7	65.7 ± 29.3	0.247
Physical activity, MET-hours/week	16.0 ± 13.5	71.6 ± 88.0	< 0.001
Low, n (%)	14 (35.0%)	9 (19.6%)	0.107
Moderate and high, n (%)	26 (65.0%)	37 (80.4%)	
Sedentary time, hour	6.8 ± 3.5	5.6 ± 3.9	0.176
Psychological stress	14.8 ± 5.4	12.4 ± 6.7	0.079
Fatigue	45.9 ± 22.2	33.3 ± 23.3	0.017

Note. A higher score of perceived sleep quality indicates poorer sleep quality.

Abbreviations: MET = metabolic equivalent of task; WASO = wake after sleep onset

Table 2

Comparison of Sleep, Physical Activity, Psychological Stress, and Fatigue Between Chinese Americans and Whites

	Estimated mean difference between Chinese Americans and whites	p
Perceived sleep quality	-0.09 ± 0.92	0.926
Sleep duration, hour	-0.92 ± 0.28*	0.001
Sleep efficiency, %	-4.47 ± 1.71*	0.010
Sleep onset, minute	2.93 ± 1.32*	0.029
WASO, minute	8.13 ± 7.74	0.297
Physical activity, MET-hours/week	-57.13 ± 15.78*	0.001
Sedentary time, hour	1.37 ± 0.94	0.147
Psychological stress	3.16 ± 1.44*	0.031
Fatigue	13.87 ± 5.61*	0.016

Note. All models were adjusted for age, sex, education (college or below vs. above college), income (below $75,000 vs. above $75,000), and smoking history (never smoked vs. lifetime smoking history). A higher score of perceived sleep quality indicates poorer sleep quality. Whites were the reference group.

Abbreviations: MET = metabolic equivalent of task; WASO = wake after sleep onset

Table 3

Multivariate Analysis of Psychological Stress and Fatigue

	Psychological Stress		Fatigue	
	Regression coefficient (SE)	*p*	Regression coefficient (SE)	*p*
Model 0				
Chinese Americans vs. whites	3.99 (1.57)*	0.013	13.87 (5.61)*	0.016
Model 1				
Chinese Americans vs. whites	3.89 (1.44)*	0.009	13.42 (4.81)*	0.007
Perceived sleep quality	0.67 (0.17)*	< 0.001	2.96 (0.57)*	< 0.001
Model 2				
Chinese Americans vs. whites	4.25 (1.72)*	0.016	15.15 (6.18)*	0.017
Sleep duration, hour	0.52 (0.63)	0.409	1.47 (2.25)	0.515
Model 3				
Chinese Americans vs. whites	3.79 (1.70)*	0.029	14.36 (6.09)*	0.021
Sleep efficiency, %	-0.002 (0.11)	0.986	0.12 (0.38)	0.758
Model 4				
Chinese Americans vs. whites	4.44 (1.67)*	0.010	14.52 (6.07)*	0.020
Sleep onset, minute	-0.26 (0.17)	0.135	-0.26 (0.62)	0.674
Model 5				
Chinese Americans vs. whites	3.73 (1.65)*	0.027	13.81 (5.92)*	0.023
WASO, minute	0.01 (0.02)	0.750	0.01 (0.09)	0.936
Model 6				
Chinese Americans vs. whites	4.05 (1.70)*	0.020	13.05 (6.00)*	0.033
Moderate and high physical activity vs. low activity, MET-hours/week	0.43 (1.84)	0.816	-6.08 (6.50)	0.353

Note. All models were adjusted for age, sex, education (college or below vs. above college), income (below $75,000 vs. above $75,000), and smoking history (never smoked vs. lifetime smoking history). A higher score of perceived sleep quality indicates poorer sleep quality.

Abbreviations: MET = metabolic equivalent of task; WASO = wake after sleep onset

Chapter 5: Sleep Moderates the Association Between Arterial Stiffness and 24-hour Blood Pressure Variability

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Abstract

Background: Arterial stiffness and increased blood pressure variability (BPV) are important subclinical cardiovascular diseases (CVDs). Evidence is accumulating that poor sleep is associated with subclinical CVDs. The purpose of our study was to investigate how sleep was related to arterial stiffness and BPV. We also explored whether sleep moderated the association between arterial stiffness and BPV.

Methods: We conducted a cross-sectional study including 78 healthy adults aged between 35 and 64 years. Variables of interest were: 1) objective seep characteristics, assessed with a wrist actigraphy for two consecutive nights; 2) arterial stiffness, measured by carotid-femoral pulse wave velocity (cfPWV); and 3) BPV, measured using an ambulatory blood pressure monitor over 24 hours and estimated by average real variability.

Results: Lower sleep efficiency was an independent predictor of higher cfPWV and higher systolic BPV, while longer wake after sleep onset (WASO) was an independent predictor of higher cfPWV only. In addition, cfPWV showed a positive relationship with systolic BPV, and this relationship was moderated by sleep efficiency and WASO, respectively. The relationship between cfPWV and systolic BPV became stronger among individuals who had a level of sleep efficiency lower than 84% and who had WASO higher than 67 minutes, respectively.

Conclusion: Our study showed that poor sleep not only directly linked with arterial stiffness and BPV but also moderated the relationship between these two subclinical CVDs. These findings suggest that improving sleep quality could be a target intervention to promote cardiovascular health in clinical practice.

Keywords: sleep health; sleep quality; actigraph; arterial stiffness; blood pressure variability; cardiovascular disease.

1. Introduction

Sleep is a lifestyle behavior essential for overall health.[1] Accumulating studies have suggested that impaired sleep can contribute to a host of medical conditions, including cardiovascular disease (CVD).[2,3] Notably, there is a growing interest in the relationship between sleep and subclinical CVD. Arterial stiffness and excessive blood pressure variability (BPV) are important subclinical CVDs that indicate an early stage of cardiovascular burdens.[4,5] Arterial stiffness features a degenerative process involving structural and functional alterations in the artery wall.[6] Increased hardening of central arteries can impose an extra load to the left ventricle and influence the efficiency of cardiac ejection, thereby inducing augmentation of end-systolic pressures.[7] Much data have indicated arterial stiffness as an independent predictor of cardiovascular complications (e.g., isolated systolic hypertension, myocardial infarction, and stroke) and total mortality.[8] BPV refers to the spontaneous oscillation of BP values occurring over the short- and long-term.[5] This multifaceted phenomenon results from an interplay of intrinsic cardiovascular mechanisms as well as extrinsic factors, such as environment and emotion changes.[5,9] The fluctuations of BP within 24 hours (short-term) can be mainly driven by central and autonomic modulation, as well as the elastic properties of arteries.[5] Several longitudinal and observational studies have shown the prognostic significance of 24-hour BPV to estimate cardiovascular risks, regardless of mean BP levels.[5,10,11]

Prior studies on the association of objective sleep characteristics with arterial stiffness and BPV have some methodological as well as demographic gaps. Several large-scale studies showed that individuals who reported abnormal sleep duration or low sleep efficiency had higher arterial stiffness than those without sleep issues.[12–16] However,

while these studies predominantly relied on subjective measures of sleep, the accuracy of self-reported data may likely be hampered by recall or social desirability bias.[17] Evaluating objective sleep characteristics with actigraphy has gained increasing popularity in science as this non-invasive measurement approach provides valid and accurate estimations of sleep outcomes.[18,19] Thus, incorporating the actigraphy technology into the study of sleep and subclinical CVDs may provide us with new insights on their relationships. Additionally, existing sleep studies have focused on the effect of obstructive sleep apnea (OSA) on BPV.[20,21] Substantial data have indicated that increased BPV is caused by apnea, which induces chemoreflex-mediated sympathetic activation and changes in the ventricular filling because of intrathoracic pressure during obstructive events.[22] Yet, how impaired sleep—beyond sleep-disordered breathing—is linked to BPV remains unclear. Although research has connected arterial stiffness to BPV,[23,24] much evidence was established on individuals with known CVD conditions or risks (e.g., hypertension and diabetes), leaving this relationship obscure for healthy populations. Moreover, it may be particularly meaningful to discover the relationship between arterial stiffness and BPV in middle-aged adults, as CVD has been shown to prevalently affect people between 35 and 64 years old;[25] this age group may potentially benefit from the stratification of cardiovascular risks through arterial stiffness and BPV.[26] Ultimately, because of the link between arterial stiffness and BPV and because both can be influenced by sleep, we speculated that impaired sleep might serve as a moderator, changing the relationship between these two subclinical CVDs. In this study, we sought to examine the interrelationships among objective sleep characteristics, arterial stiffness, and 24-hour BPV among healthy, middle-aged adults. We hypothesized that 1) impaired

sleep had a positive relationship with arterial stiffness and BPV; and 2) arterial stiffness was related to BPV and this relationship could be moderated by sleep (See Figure 1).

2. Methods

2.1. Sample and Study Procedures

Our cross-sectional study consisted of healthy adults between 35 and 64 years old. This sample was derived from our original study, which investigated racial differences in general health between Asian Americans and Caucasians. We recruited residents within central Virginia through purposeful convenience sampling. The two racial groups were matched for age and sex to reduce the demographic heterogeneity within the sample. Individuals who showed interest in the study were screened to determine eligibility. We excluded individuals who: 1) took antihypertensive medications; 2) had an irregular heart rhythm; 3) worked night shifts; 4) were pregnant; and 5) had a previous cardiovascular event (e.g., coronary artery disease, myocardial infarction, and cerebrovascular disease). We invited participants to our lab in the morning to collect their demographic and biometric information, after which we measured arterial stiffness. Upon completing the on-site assessment, we placed an actigraphy on participants' non-dominant wrist for a two-night sleep evaluation and requested them to complete a sleep diary. After completing the first night of sleep, each individual was instructed to wear and initiate the ambulatory BP monitor in the morning to start the assessment for the next 24 hours. Participants returned the devices and the sleep diary on the third day. After examining the data, we subsequently excluded nine records that contained missing values of arterial stiffness, ambulatory BP parameters, or sleep measures. This exclusion yielded a final

sample size of 78. Our research study was approved by the Institutional Review Board at the University of Virginia.

2.2. Arterial Stiffness

Arterial stiffness was quantified by carotid-femoral pulse wave velocity (cfPWV) using the SphygmoCor device (AtCor Medical, Australia). cfPWV is broadly recognized as the gold-standard measurement of arterial stiffness.[28,29] Our data collection procedure followed the expert consensus on the measurement of arterial stiffness using cfPWV.[30] In order to control for the effects of food and physical activity on cfPWV, we instructed participants to refrain from food, caffeinated beverages, tobacco, medications, or vigorous physical activity within three hours before the assessment. All measurements took place in a quiet, temperature-controlled laboratory between 8:30 am and 11:30 am to minimize the diurnal variation.[30] Participants were first asked to rest in a supine position for 10 minutes. Next, we obtained two sets of readings for office BP and HR at a 2-minute interval using a standard electronic BP monitor. Following, we used a measuring tape to measure the body surface distance from the suprasternal notch to the right common carotid artery site (s-carotid distance) and the body surface distance from the suprasternal notch to the right common femoral artery site (s-femoral distance). A pressure-sensitive tonometer was then lightly pressed over the carotid and femoral pulse points, sequentially, to acquire a minimum of 12-second pulse wave signals referenced to electrocardiogram (ECG) at each arterial site.[31] The SphygmoCor software automatically computed cfPWV by dividing the difference in pulse travel distance between the carotid and femoral arteries (ΔDistance) by the difference in pulse transit time (ΔTime), as denoted in this formula: $\Delta Distance/\Delta Time$.[32] ΔDistance is derived by subtracting the s-

carotid distance from the s-femoral distance. ΔTime represents the time delay between the arrival of the pulse wave at the common carotid artery and at the femoral artery,[30] and this value is calculated by the SphgmoCor software on the basis of ECG using the foot-to-foot method. To maintain consistency, all assessments were carried out by one trained researcher. Each participant had two measurements, and a mean value of the measurements was used. If the first two readings differed more than 0.5 m/s, a third one was taken and used.

2.3. Sleep Characteristics

Participants wore a wrist actigraphy (GT9X Link, ActiGraph, USA) over two consecutive nights. An actigraphy is a watch-sized triaxial accelerometer equipped with an ambient light sensor to capture and record movements. Compared to lab-based polysomnograpy (PSG), which is the "gold standard" for measuring sleep, the actigraphy is less cumbersome and provides reliable information. We initialized the actigraphy to collect acceleration signals at 30Hz and placed the device on participants' non-dominant wrist before they left the lab. During each day of the assessment, participants also completed a sleep diary to track their time in bed, time out of bed, estimated time falling asleep, number of times waking up during the night, and sleep quality (rated on a scale of 1 to 5). We later used the information on the sleep diary to validate the actigraphy data for analysis. All raw data were processed using ActiLife 6.13.3 software, which converted and filtered the raw data into digital activity counts and subsequently summed the counts over 60-second epochs. The Cole-Kripke algorithm was applied to distinguish sleep from wakefulness.[33] We obtained the following sleep measures from the software: sleep onset, sleep duration, sleep efficiency, number of awakenings, and wake after sleep

onset (WASO). Sleep onset represented the time interval between lights off and sleep start. Sleep duration was defined as the total amount of hours asleep in bed. This measure included the time between sleep onset and offset, not accounting for the minutes awake in bed. Sleep efficiency was the ratio of sleep duration to time spent in bed (percentage). Number of awakenings was recorded from the beginning of sleep onset to sleep offset. WASO characterized the total amount of time spent awake between the initial sleep onset and falling asleep again.

2.4. Twenty-four-hour Ambulatory Blood Pressure Monitoring

We assessed BPV with the Spacelabs OnTrak Monitor 90227 (Spacelabs Healthcare, USA). This device was programmed to measure BP every 30 minutes from 6am to 10pm and every 60 minutes from 10pm and 6am. We selected an appropriate cuff size for all individuals by measuring the mid-section circumference of their non-dominant upper arm with a tape measure. Participants initiated the assessment after waking up in the morning, and they wore the device for 24 hours. We asked participants to perform their usual daily activities but keep their monitored arm relaxed and still during each measurement.[24] All recordings were downloaded to compute the mean readings of average systolic BP (SBP) as well as diastolic BP (DBP) over 24 hours. We estimated systolic BPV and diastolic BPV by average real variability (ARV), which is the average of absolute differences between consecutive BP measurements. This index has been shown to provide a better estimation of 24-hour BPV than other measures of dispersion, such as standard deviation, because ARV accounts for the order in which BP measurements are obtained and is less sensitive to the low sampling frequency of ambulatory BP monitoring.[5,35] According to Mena et al.'s meta-analysis,[35] ARV is

considered an independent indicator of the presence and progression of subclinical organ damage after adjusting for BP and other clinical factors.

2.5. Covariates

Covariates include age, sex, race, intake of sleep medication, smoking history, body mass index (BMI), office mean arterial pressure (MAP), and office heart rate (HR). Information about age, sex, race, intake of sleep medication, and smoking history was collected by self-report. Intake of sleep medication was coded as a dichotomous variable (yes or no). Height and weight were measured to compute BMI. Participants were asked to lie in a supine position for 10 minutes before assessing their office BP and HR using a standard electronic BP monitor. The measurements were repeated with a 2-minute interval to obtain mean values of office MAP and office HR.

2.6. Statistical Analysis

All statistical analyses were carried out using SPSS version 26. We summarized participants' demographic and clinical characteristics using mean (standard deviation) and n (%). Continuous variables were examined for normal distribution. All analyses were adjusted for covariates including age, sex, race, sleep medication, BMI, mean office MAP, and mean office HR. We built multiple linear regression models to examine whether each of the sleep variables had associations with cfPWV and systolic BPV, respectively. We then examined the association between cfPWV and systolic BPV in which cfPWV was treated as an independent variable and systolic BPV as a dependent variable. We tested whether any of the sleep variables moderated the relationship between cfPWV and systolic BPV using Model 1 of Hayes' PROCESS macro with the Johnson-Neyman technique in SPSS.[36,37] The moderation analysis was examined

sequentially with the following three steps: in step 1, we examined the association between cfPWV (independent variable) and systolic BPV (dependent variable); in step 2, one sleep variable (moderator) was added; and finally, in step 3, the interaction term cfPWV × moderator was added. This process was repeated for each proposed moderator including sleep onset, sleep duration, sleep efficiency, number of awakenings, and WASO. We set the significance threshold of p-value at 0.050.

3. Results

Eighty-seven participants were originally enrolled in the study. We subsequently excluded nine subjects from the analysis due to missing valid actigraphy data (n = 2), missing valid cfPWV (n = 3), or an unsatisfactory number of ambulatory BP readings (n = 4), which resulted in our final sample size of 78. Table 1 summarizes participants' demographic characteristics and key study variables. Our sex- and race-matched sample (male = 47.4%; Asian Americans = 48.7%) had a mean age of 47 years old. Participants' average sleep duration was 6.55 hours per night, with over one third sleeping fewer than 6 hours. Their mean sleep efficiency was 83.57%. On average, our participants spent 3.60 minutes to fall asleep and their mean WASO (71 minutes) made up approximately 18% of the entire sleep duration. The mean number of awakenings was 23. The sample had a mean cfPWV of 6.97 m/s, which was within the range of reference values for adults aged between 35 and 64 years, according to the literature.[38] Participants' 24-hour ambulatory BP was 117/74 mmHg.

Table 2 presents multiple regression analyses of the relationships between each of the sleep measures and subclinical CVDs, after adjusting for age, sex, race, BMI, smoking history, sleep medication, mean office MAP, and mean office HR. The results

showed that sleep onset, sleep duration, and number of awakenings were not associated with cfPWV or with systolic BPV. Additionally, a one percent decrease in sleep efficiency was associated with an average of 0.03 m/s ($p = 0.029$) increase in cfPWV and 0.08 unit ($p = 0.018$) increase in systolic BPV. Furthermore, a minute increase in WASO was associated with 0.01 m/s ($p = 0.014$) increase in cfPWV. No relationship was found between WASO and systolic BPV.

Of the sleep variables assessed, both sleep efficiency and WASO demonstrated a moderating role that affected the relationship between cfPWV and systolic BPV. As Tables 3 and 4 illustrated, in the baseline model, higher cfPWV was associated with increased systolic BPV ($\beta = 0.93$; $p = 0.007$) after controlling for the covariates. This relationship remained significant after adding sleep efficiency or WASO in Model 2. The inclusion of the interaction term in Model 3 resulted in the interaction of cfPWV and sleep efficiency being marginally significant ($\beta = -0.04$; $p = 0.050$) while the interaction term of cfPWV x WASO exhibited statistical significance ($\beta = 0.01$; $p = 0.026$), indicating that both of these sleep measures could influence the relationship between cfPWV and systolic BPV. To further understand the details of the interaction, we applied the Johnson-Neyman technique using Haye's PROCESS tool to identify the cutoff values for sleep efficiency and WASO that significantly changed the strength of the relationship.[37] The Johnson-Neyman technique generates a set of values of the moderator for which the relationship between the independent and dependent variables becomes significant or vice versa.[39] This procedure allows us to obtain a cutoff point of the moderator at which the effect of the independent variable on the dependent variable is statistically significant ($p = 0.050$). As a result, the relationship between cfPWV and

systolic BPV became stronger when sleep efficiency was lower than 84% and when WASO was higher than 67 minutes. We did not find other sleep variables to have moderation effects on the relationship between cfPWV and systolic BPV. We graphically depicted the information in Figures 2 and 3.

4. Discussion

In this study, we found that objectively measured impaired sleep was associated with arterial stiffness and BPV, respectively, independent of the common CVD risk factors and intake of sleep medication. Namely, low sleep efficiency and high WASO were associated with increased arterial stiffness, whereas low sleep efficiency was associated with elevated BPV over 24 hours. We additionally identified that both sleep efficiency (< 84%) and WASO (> 67 minutes) served as moderators that strengthen the positive relationship between arterial stiffness and BPV. This study is innovative and has important merits, as it demonstrates a potential mechanism of impaired sleep interacting with subclinical CVDs to augment adverse cardiovascular outcomes.

4.1. Sleep and Arterial Stiffness

Past investigations on sleep and arterial stiffness were mostly centered on patients diagnosed with OSA using PSG.[40] A recent study analyzed pooled data obtained from six OSA studies in 362 patients who underwent PSG studies. The results showed that OSA severity, measured by apnea–hypopnea index, oxygen desaturation index, and sleep time with oxygen saturation less than 90%, had close associations with arterial stiffness.[41] Buchner and colleagues[42] reported that OSA was related to increased augmentation index and carotid-radial PWV, after controlling for hypertension, age, gender, body mass index, and antihypertensive medications. Although a substantial body of literature has

established OSA to be an independent risk factor of arterial stiffness, there is scarce evidence on how sleep characteristics beyond OSA are related to this subclinical CVD.

Notably, only two published peer-reviewed studies have investigated the relationship between actigraphy-based objective sleep and arterial stiffness. In a longitudinal study in which 306 patients with CVD risk factors were divided into four groups based on levels of brachial-ankle PWV progression, researchers found that the presence of low sleep quality was a significant covariate to predict the group with the greatest PWV progression (≥ 20%) when compared to the group without progression (odds ratio = 3.62, confidence interval 1.04–12.55, $p = 0.04$).[43] Yet, a secondary analysis of the Multi-Ethnic Study of Atherosclerosis[44] (N = 908, mean age 68.4 years) showed no relationship between actigraphy-measured sleep quality and aortic PWV estimated by magnetic resonance imaging.[44] While those previous studies focused on individuals of advanced age, our study suggested that healthy middle-aged adults who experience impaired sleep are also vulnerable to arterial stiffness. In particular, our study was among the few that used cfPWV to quantify arterial stiffness. Because the thoracic and abdominal aorta contributes most to the arterial buffering function, cfPWV—which measures along the aorto-iliac pathway—has prognostic superiority over other parameters of arterial stiffness, such as baPWV and femoral-ankle PWV.[28,29] Although the underlying mechanisms of sleep dynamics and arterial stiffness have not been completely understood, it is possible that impaired sleep may lead to the dysregulation of inflammatory responses (e.g., increased levels of interleukin-6 and C-reactive protein), which reduce endothelial function and subsequently lead to arterial stiffness.[45,46] Evidence has also linked sleep problems to increased insulin resistance, altered

hypothalamic-pituitary-adrenal system activity, and increased sympathetic activity, all of which may partially explain the process of arterial stiffness progression.[13]

4.2. Sleep and Blood Pressure Variability

Impaired sleep, defined as an insufficient amount or poor quality, has been shown to increase BP, but its influence on BPV is still unknown. The work by Aggarwal and colleagues[47] showed that mild sleep disturbances—e.g., self-reported poor sleep quality, long sleep onset, and insomnia—had associations with increased SBP and vascular inflammation among women, even in the absence of sleep deprivation. In another study where 300 participants underwent ambulatory BP monitoring and actigraphy assessments over two days, individuals with low sleep efficiency exhibited increased daytime SBP, nighttime BP, as well as average BP during the next day.[48] Both findings highlighted the indispensable role of sleep quality on BP regulation; meanwhile, they could provide insight into our study results. The reduction in sleep quality may alter the autonomic function as well as the circadian rhythm. This pathophysiological change can consequently lead to sympathetic overactivity and vagal withdrawal, resulting in BP elevation and wide fluctuation.[49–52]

Thus far, little evidence is available in regard to the relationship between sleep characteristics and BPV outcomes. Pengo and colleagues[53] included a cohort of patients diagnosed with chronic kidney disease in their study. The researchers observed that patients who experienced moderate or severe insomnia exhibited significantly increased nocturnal systolic BPV, as calculated by standard deviation. Compared to their study which evaluated sleep subjectively, our study advanced this evidence by showing that actigraphy-based sleep efficiency, an important parameter recommended to quantify

sleep quality,[54] had a clinical significance in implicating BPV. Current knowledge on the effect of objective sleep outcomes on BPV has been predominantly established with the use of polysomnographic technology. For example, in a group of 384 patients who underwent PSG evaluation, researchers noted that individuals who were newly diagnosed with OSA had higher 24-hour systolic BPV than those without OSA.[55] Martynowicz et al.[56] also found that patients with severe OSA had increased nocturnal systolic and diastolic BPV in comparison to those with mild-to-moderate OSA. Our study is innovative because the utilization of actigraphy provides new information about sleep characteristics and BPV, with the focus on a broader study population. More importantly, the relationship between low sleep efficiency and 24-hour BPV observed in our study implied that the impact of poor sleep would sustain over the short term, which goes beyond our current knowledge of its effect on nocturnal BP fluctuations. We believe that this finding is particularly informative as we identified a new trajectory of habitually impaired sleep contributing to CVD risks through excessive BPV.

4.3. Arterial Stiffness and Blood Pressure Variability

We provide additional evidence that healthy, middle-aged individuals with increased arterial stiffness might be susceptible to great BPV, independent of office MAP and HR. Our results support the hypothesis that arterial stiffness could be a potential risk factor of BPV.[57] One principal mechanism which can explain this relationship is the altered baroreflex sensitivity (BRS).[58] In normal conditions, the baroreceptors located in the aortic arch and the carotid sinus detect a change in the BP level and immediately send input signals to control centers in the brain stem, which consequently modulate autonomic outflow to reach BP homeostasis.[59,60] Recent epidemiological data have

suggested that arterial stiffness accounts for reduced BRS;[58,61] such impairment in vascular compliance can limit baroreceptors' capacity to buffer BP fluctuations and, hence, increase BPV.[23,58] Conversely, more existing evidence in the literature tends to favor the hypothesis of arterial stiffness as a consequence of excessive BPV. For example, the findings from a large sample of 3,000 patients who were treated or untreated for hypertension suggested that 24-hour systolic BPV was an independent determinant of increased cfPWV.[23] Another study also identified that 24-hour BPV was associated with an increased risk of vascular target organ damage, as measured by cfPWV, among 344 hypertensive patients.[62] Indeed, a popular debate exists regarding the cause/effect relationship between the two subclinical CVDs.[63] Although the relationship between arterial stiffness and 24-hour BPV could be bi-directional,[23] our data were only able to support arterial stiffness as a risk factor of BPV, not vice versa. Given the fact that current findings on this topic are mainly derived from cross-sectional studies, longitudinal investigations are necessary to unravel which subclinical CVD precedes first.

4.4. Sleep Moderates the Relationship between Arterial Stiffness and Blood Pressure Variability

Another finding in our study that deserves discussion is that either low sleep efficiency or high WASO plays a moderating role in the relationship between arterial stiffness and BPV. That is, individuals with increased arterial stiffness exhibit a greater likelihood of having excessive BPV when they experience poor sleep quality. This finding implies that sleep not only directly impacts on the cardiovascular function but also accelerates CVD development by intensifying the risk of subclinical CVDs. Thus,

the influence of arterial stiffness on 24-hour BPV becomes stronger among those with impaired sleep. Additionally, as individuals with impaired sleep may experience a series of sleep problems (e.g., abnormal sleep duration, increased sleep onset, high WASO, early awakening, and daytime sleepiness),[64] we posit that the joined adverse effect may make individuals even more vulnerable to increased arterial stiffness and short-term BPV. Based on the evidence presented above, conducting early sleep assessments may be critical to identify high-risk populations—to then be followed by targeted sleep interventions to prevent negative cardiovascular consequences. More importantly, because middle-aged healthy adults without overt CVD have demonstrated susceptibility to subclinical CVD when they have impaired sleep, this group may benefit from sleep hygiene promotion and stratification for CVD risks through subclinical measures (e.g., arterial stiffness and BPV) in order to lower their CVD burden in elderly life.

4.5. Limitations

Our study provided an in-depth understanding of the inter-relationships between sleep, arterial stiffness, and BPV; yet, some limitations of the present study must be noted. First, as discussed above, we were unable to infer any causal relationships between sleep, arterial stiffness, and BPV due to the nature of this study design. Future studies should consider employing longitudinal designs to explore the temporal sequence of arterial stiffness and 24-hour BPV. Second, because our participants were limited to two racial groups from a single southeast state, it might be difficult to generalize the findings to other populations. This limitation can be mitigated by having a larger, more diverse sample to replicate the study results. Third, the length and timing of sleep assessment may affect our study results. We acknowledge that two consecutive nights of actigraphy

recording may not sufficiently represent participants' natural sleep pattern. The weekday-weekend variations in sleep schedule should also be taken into account when interpreting the sleep results. Future investigations are recommended to conduct at least seven consecutive nights of sleep assessment to provide an accurate prediction of sleep outcomes.[65,66] Fourth, although wrist actigraphy is a feasible and sensitive measure, this tool tends to overestimate sleep duration while underestimate sleep disturbances in certain study populations, such as individuals who have insomnia or lack of motion (e.g., advanced age or being sedentary).[19] Thus, assessing sleep characteristics with PSG may help confirm the study findings. Fifth, the effect of ambulatory BP monitoring may cause sleep arousal at night. The increase in nocturnal awakenings due to intermittent cuff inflation could directly impact WASO and sleep efficiency. To validate the moderating roles of sleep quality in arterial stiffness and BPV, we suggest researchers initiating ambulatory BP monitoring after sleep evaluation to reduce the confounding effect. Finally, we did not collection information about the history of sleep disorders (e.g., OSA) from participants. Considering that OSA is an important risk factor of arterial stiffness and BPV, future studies that investigate the relationship between sleep and subclinical CVDs should consider screening for sleep disorders.

5. Conclusions

Taken together, impaired sleep is a prominent risk factor of arterial stiffness and 24-hour BPV. Increased arterial stiffness has an association with great 24-hour BPV, and this positive relationship can be intensified under the influence of poor sleep quality, namely low sleep efficiency and high WASO. Future studies are recommended to overcome the limitations of the present study—such as small sample size and cross-

sectional study design—to replicate and confirm our findings. Routine sleep assessment and improving sleep quality should warrant the attention of clinical health providers to improve cardiovascular outcomes in this frequently overlooked middle-aged adult population.

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Tables

Table 1. Demographic and clinical characteristics of study sample

	Sample characteristics (N = 78)
Age, year	47.04 ± 8.99
Male sex	37 (47.4%)
Race	
Caucasians	40 (51.3%)
Asian Americans	38 (48.7%)
BMI, kg/m^2	25.37 ± 4.73
Smoking history	
Never smoked	54 (69.2%)
Lifetime smoking history	24 (30.8%)
Sleep medication	12 (15.4%)
Office BP, mmHg	
SBP	121.55 ± 15.51
DBP	76.89 ± 9.67
MAP	91.77 ± 11.10
Office HR, bpm	64.89 ± 9.39
Sleep onset, min	3.60 ± 5.69
Sleep duration, h	6.55 ± 1.30
Sleep efficiency, %	83.57 ± 7.48
Number of awakenings	22.91 ± 7.69
WASO, min	70.78 ± 32.44
cfPWV, m/s	6.97 ± 1.08
24-h ambulatory BP, mmHg	
SBP	116.86 ± 13.15
DBP	73.99 ± 8.48
MAP	88.23 ± 9.10
24-h ambulatory HR, bpm	73.58 ± 8.60
Systolic BPV	8.94 ± 2.31
Diastolic BPV	7.11 ± 1.67

Note: All data are presented as either mean ± SD or N (%).

Abbreviations: BMI = body mass index; BPV = blood pressure variability; cfPWV = carotid-femoral pulse wave velocity; DBP = diastolic blood pressure; HR = heart rate; MAP = mean arterial pressure; SBP = systolic blood pressure; WASO = wake after sleep onset

Table 2. Linear regressions of each of the sleep measures on cfPWV and systolic BPV

	cfPWV		Systolic BPV	
	β (SE)	p	β (SE)	p
Sleep onset, h	0.04 (0.89)	0.968	-2.08 (2.56)	0.420
Sleep duration, h	0.02 (0.07)	0.794	-0.26 (0.20)	0.211
Sleep efficiency, %	-0.03 (0.01)	0.029	-0.08 (0.03)	0.018
Number of awakenings	0.01 (0.01)	0.190	0.02 (0.03)	0.578
WASO, min	0.01 (0.00)	0.014	0.01 (0.01)	0.056

Note: All regression models were adjusted for age, sex, race, BMI, smoking history, sleep medication, office MAP, and office HR.

Abbreviations: β = beta coefficient; BPV = blood pressure variability; cfPWV = carotid-femoral pulse wave velocity; SE = standard error; WASO = wake after sleep onset

Table 3. Moderation analysis of sleep efficiency on the relationship between cfPWV and systolic BPV

	β (SE)	p
Model 1		
cfPWV, m/s	0.93 (0.33)	0.007
Model 2		
cfPWV, m/s	0.77 (0.34)	0.026
Sleep efficiency, %	-0.06 (0.03)	0.074
Model 3		
cfPWV, m/s	4.39 (1.84)	0.020
Sleep efficiency, %	2.67 (1.67)	0.115
cfPWV x sleep efficiency	-0.04 (0.02)	0.050

Note: In Model 1, PWV was the main predictor and systolic BPV was the outcome of interest. In Model 2, sleep efficiency was added to Model 1. In Model 3, the interaction term of cfPWV and sleep efficiency was added to Model 2. All regression analyses were adjusted for age, sex, race, BMI, smoking history, sleep medication, office MAP, and office HR.

Abbreviations: β = beta coefficient; BPV = blood pressure variability; cfPWV = carotid-femoral pulse wave velocity; SE = standard error

Table 4. Moderation analysis of WASO on the relationship between cfPWV and systolic BPV

	β (SE)	p
Model 1		
cfPWV, m/s	0.93 (0.33)	0.007
Model 2		
cfPWV, m/s	0.81 (0.35)	0.023
WASO, min	0.01 (0.01)	0.218
Model 3		
cfPWV, m/s	-0.10 (0.52)	0.845
WASO, min	-4.46 (2.24)	0.051
cfPWV x WASO	0.01 (0.01)	0.026

Note: In Model 1, PWV was the main predictor and systolic BPV was the outcome of interest. In Model 2, WASO was added to Model 1. In Model 3, the interaction term of cfPWV and WASO was added to Model 2. All regression analyses were adjusted for age, sex, race, BMI, smoking history, sleep medication, office MAP, and office HR.

Abbreviations: β = beta coefficient; BPV = blood pressure variability; cfPWV = carotid-femoral pulse wave velocity; SE = standard error; WASO = wake after sleep onset

Figures

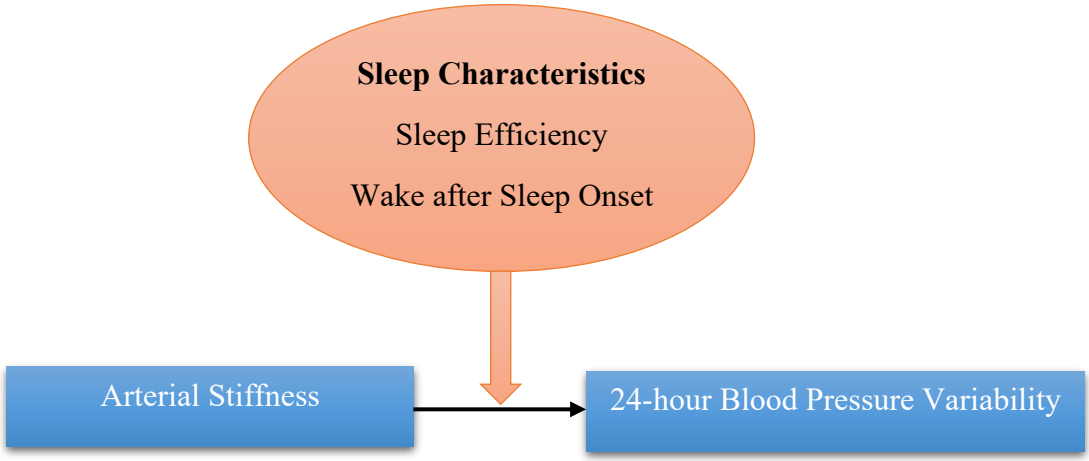

Figure 1. Conceptual Framework. Sleep characteristics, including sleep efficiency and wake after sleep onset, moderate the relationship between arterial stiffness and 24-hour blood pressure variability.

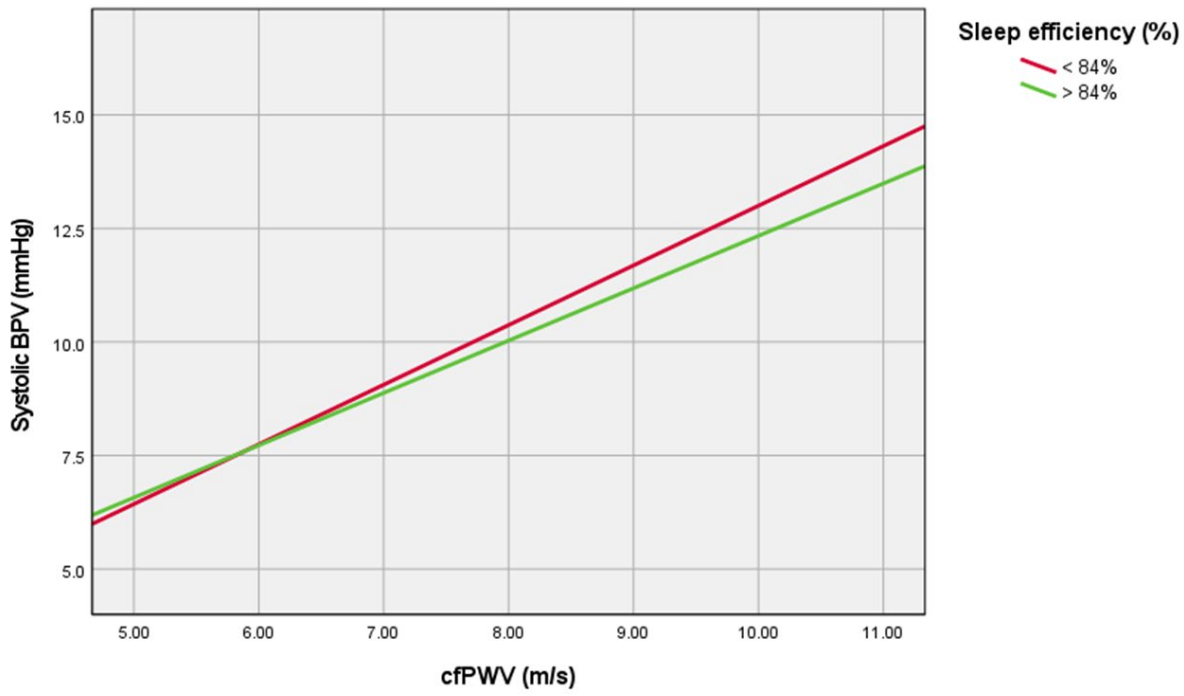

Figure 2. Relationship between cfPWV and systolic BPV by levels of sleep efficiency. Individuals with higher cfPWV were more likely to have increased systolic BPV if their sleep efficiency was less than 84%.

Abbreviations: BPV = blood pressure variability; cfPWV = carotid-femoral pulse wave velocity

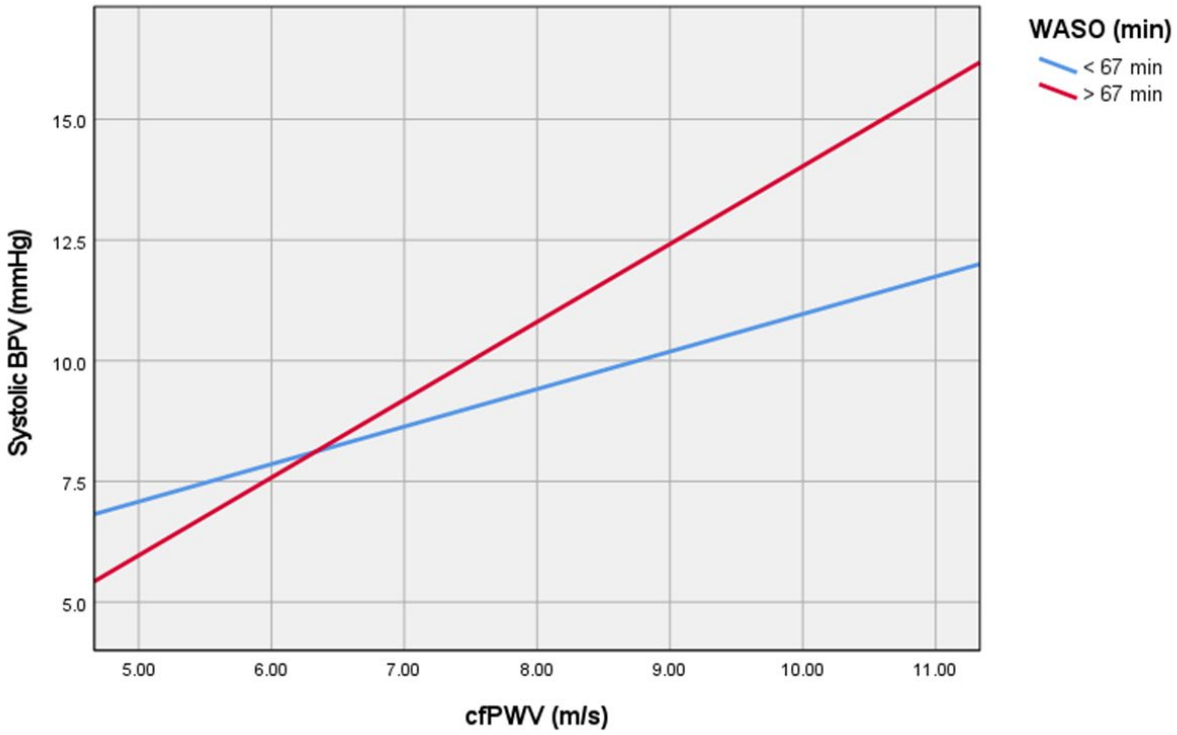

Figure 3. Relationship between cfPWV and systolic BPV by levels of WASO. Individuals with higher cfPWV were more likely to have increased systolic BPV if their WASO was greater than 67 minutes.

Abbreviations: BPV = blood pressure variability; cfPWV = carotid-femoral pulse wave velocity; WASO = wake after sleep onset

Chapter 6: Discussion and Conclusions

This dissertation project studied two populations at high-risk for negative cardiovascular outcomes – IPV female survivors and Chinese Americans. Chapter 6 presents a summary of key study findings, strengths and limitations, and nursing implications.

Summary of Study Findings and Implications

First, the dissertation reviewed current literature on cardiovascular health in women with a history of IPV. A total of 19 articles were included for data analysis and synthesis. Although the overall findings revealed a mixed relationship, abused women were more likely to smoke and use alcohol, experience cardiovascular symptoms, and have long-term cardiovascular complications than non-abused women. The evidence suggested that IPV as a stressor could modify women's lifestyle behaviors and trigger a cascade of biological changes that led to potential CVD risks. The review highlighted the importance of evaluating coping skills among IPV female survivors. Employing a total risk approach to estimate this group's overall cardiovascular outcomes should be considered in clinical practice.

Next, the dissertation focused on cardiovascular risks in another understudied population— Chinese Americans. A cross-sectional study was conducted to assess racial differences in behavioral factors (sleep and physical activity) and psycho-physiological factors (psychological stress and fatigue) between Chinese Americans and non-Hispanic whites. The study sample consisted of 41 Chinese Americans and 46 non-Hispanic whites between 35 and 64 years old. The findings showed that Chinese Americans had poorer objective sleep outcomes, performed lower levels of physical activity, and reported

higher psychological stress and fatigue than their white counterparts, after controlling for the covariates. The study additionally demonstrated that both race and poor perceived sleep quality were independent indicators of high psychological stress and fatigue. These results showed significant health disparities among Chinese Americans. In particular, the adverse behavioral and psychophysiological health outcomes may put this minority group at high risks for future CVD development. This study highlighted the need to investigate social and cultural determinants of cardiovascular health in Chinese Americans. Healthcare providers are encouraged to identify Chinese Americans' special health needs and potential barriers to optimal cardiovascular health, which will be crucial in guiding culturally appropriate interventions for this minority group.

The fact that the minority group experienced poor sleep, which then impacted psycho-physiological health ignited the interest in evaluating the relationship among sleep, arterial stiffness (measured by cfPWV), and 24-hour BPV (measured by an ABPM). The sample included a total of 78 healthy Chinese Americans and non-Hispanic whites between 35 and 64 years old. The results demonstrated that lower sleep efficiency was independently associated with higher cfPWV and higher systolic BPV, while WASO was independently associated with higher cfPWV only. Additional investigations revealed a positive relationship between cfPWV and systolic BPV, and this relationship was moderated by low sleep efficiency (< 84%) and high WASO (> 67 minutes), respectively. These findings illustrated a unique pathophysiological mechanism of the relationship between impaired sleep and subclinical cardiovascular markers. The evidence underscored that improving sleep outcomes could be an approach to reducing cardiovascular risks.

Strengths and Limitations

This cross-sectional study has two important merits. It was the first to comprehensively investigate cardiovascular health in an overlooked ethnic minority population. Considering that Asian American subgroups have marked differences in CVD risks, this study specifically focused on Chinese Americans and revealed their negative behavioral, as well as psycho-physiological health factors that were closely associated with CVD. This study provided essential information about cardiovascular health disparities in Chinese Americans and implied the necessity to implement measures to improve health outcomes in minority populations. Additionally, this study was the first to examine objective sleep characteristics and two important subclinical cardiovascular markers. Subclinical CVD commonly precedes the occurrence of adverse events by years to decades. The moderation effect of impaired sleep on the link between those two subclinical cardiovascular markers showed that promoting sleep health could be an effective strategy for reducing future CVD events. By addressing sleep problems in high-risk populations, these findings could transform current clinical practice from CVD treatment to prevention.

Despite the strengths, several limitations should be noted. First, the results may require careful interpretation due to the small sample coming from a single southeastern state. A future study with a larger, more diverse, and fully powered sample is needed to confirm the study findings and produce generalizable results. Second, due to the cross-sectional nature of this study, the causal relationships between sleep, arterial stiffness, and BPV cannot be inferred. Researchers should consider employing longitudinal designs to explore the temporal sequence of arterial stiffness and BPV, as well as their

associations with sleep. Third, the study assessed sleep characteristics over two consecutive nights with an actigraph. This short period of recording may not sufficiently represent the participants' natural sleep patterns. In addition, the quality of sleep assessment could be affected by the concurrent ambulatory BP monitoring on the second night of evaluation. Therefore, conducting a sleep evaluation over five to seven consecutive nights may provide more accurate data.

Conclusions

In conclusion, this dissertation study showed that IPV female survivors and Chinese Americans were both at potential risks for CVD. Nurses are in a critical position to detect the health outcomes of these vulnerable and minority populations. For IPV female survivors, the evaluation of their coping skills and mental distress can be incorporated into routine clinical assessment. Focus can be placed on identifying high-risk situations that most likely trigger unhealthy lifestyle behaviors and then encouraging these women to adopt effective coping strategies. For Chinese Americans, researchers should consider exploring potential factors that underlie their cardiovascular health disparities. Implementing culturally sensitive interventions may promote health behaviors and reduce psycho-physiological burdens in this minority group. For the general middle-aged adult populations, nurses can take a leading role in CVD prevention by conducting routine sleep assessments and encouraging optimal sleep hygiene practice.

Appendices

Appendix A. English Version of Study Questionnaires

Demographic Questionnaire

1. Date of birth: _____ (month/year)
2. Sex: _____ Male _____ Female
3. How long have you lived in the United States? _____ (Specify in years)
4. What is your highest education?
   _____ Less than middle school degree
   _____ Middle school degree
   _____ High school degree or equivalent (e.g., GED)
   _____ Associate degree/some college
   _____ Bachelor's degree
   _____ Master's degree
   _____ Doctoral degree or professional degree

5. Which income group does your household fall under?
   _____ Under $29,999
   _____ $30,000 - $49,999
   _____ $50,000 - $74,999
   _____ $75,000 - $99,999
   _____ $100,000 - $149,999
   _____ $150,000 - $199,999
   _____ $200,000 or more

6. What is your smoking status?
   _____ Current smoker
   _____ Former smoker
   _____ Never smoked

7. Have you been diagnosed with any cardiovascular diseases (e.g., high blood pressure, diabetes, high cholesterol, etc)?
   _____ Yes, please specify:
   _____ No

8. Have you been taking any medications in the past year?
   _____ Yes, please list:
   _____ No

Pittsburgh Sleep Quality Index

The following questions relate to your usual sleep habits **during the past month only**. Your answers should indicate the most accurate reply for the **majority of days and nights** in the past month. Please answer all questions. During the past month,

1. When have you usually gone to bed? _____
2. How long has it taken you to fall asleep each night? _____ (in minutes)
3. When have you usually gotten up in the morning? _____
4. How many hours of actual sleep do you get at night? (This may be different than the number of hours you spend in bed) _____

5.During the past month, how often have you had trouble sleeping because you…	0	<1 time/week	1-2 times/week	3 times + /week
a. Cannot get to sleep within 30 minutes				
b. Wake up in the middle of the night or early morning				
c. Have to get up to use the bathroom				
d. Cannot breathe comfortably				
e. Cough or snore loudly				
f. Feel too cold				
g. Feel too hot				
h. Have bad dreams				
i. Have pain				
j. Other reason(s), please describe, including how often you have had trouble sleeping because of this reason(s):				
6. During the past month, how often have you taken medicine (prescribed or "over the counter") to help you sleep?				
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?				
8. During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?	No problem at all	Only a very slight problem	Somewhat of a problem	A very big problem
9. During the past month, how would you rate your sleep quality overall?	Very good	Fairly good	Fairly bad	Very bad

Global Physical Activity Questionnaire

Physical Activity		
I am going to ask you about the time you spend doing different types of physical activity in a typical week. **Please answer these questions even if you do not consider yourself to be a physically active person.** Think first about the time you spend doing work. Think of **work** as the things that you have to do such as paid or unpaid work, study/training, household chores, seeking employment. In answering the following questions **'vigorous-intensity activities'** are activities that require hard physical effort and cause large increases in breathing or heart rate, **'moderate-intensity activities'** are activities that require moderate physical effort and cause small increases in breathing or heart rate.		
Questions	Response	Code
Activity at work		
1. Does your work involve vigorous-intensity activity that causes large increases in breathing or heart rate like *[carrying or lifting heavy loads, digging or construction work*] for at least 10 minutes continuously?	Yes 1 No 2 *If No, go to P4*	P1
2. In a typical week, on how many days do you do vigorous-intensity activities as part of your work?	Number of days: ⌊__⌋	P2
3. How much time do you spend doing vigorous-intensity activities at work on a typical day	Hours: minutes ⌊__⌋ : ⌊__⌋ hrs mins	P3
4. Does your work involve moderate-intensity activity that causes small increases in breathing or heart rate such as brisk walking *[or carrying light loads*] for at least 10 minutes continuously?	Yes 1 No 2 *If No, go to P7*	P4
5. In a typical week, on how many days do you do moderate-intensity activities as part of your work?	Number of days: ⌊__⌋	P5
6. How much time do you spend doing moderate-intensity activities at work on a typical day?	Hours: minutes ⌊__⌋ : ⌊__⌋ hrs mins	P6
Travel to and from places		
The next questions exclude the physical activities at work that you have already mentioned. Now I would like to ask you about the usual way you travel to and from places. For example, to work, for shopping, to market, to place of worship.		
7. Do you walk or use a bicycle (*pedal cycle*) for at least 10 minutes continuously to get to and from places?	Yes 1 No 2 *If No, go to P10*	P7
8. In a typical week, on how many days do you walk or bicycle for at least 10 minutes continuously to get to and from places?	Number of days: ⌊__⌋	P8
9. How much time do you spend walking or bicycling for travel on a typical day?	Hours: minutes ⌊__⌋ : ⌊__⌋ hrs mins	P9

Recreational activities		
The next questions exclude the work and transport activities that you have already mentioned. Now I would like to ask you about sports, fitness and recreational activities (leisure).		
10. Do you do any vigorous-intensity sports, fitness or recreational (*leisure*) activities that cause large increases in breathing or heart rate like [*running or football,]* for at least 10 minutes continuously?	Yes 1 No 2 *If No, go to P13*	P10
11. In a typical week, on how many days do you do vigorous-intensity sports, fitness or recreational (*leisure*) activities?	Number of days: ⌊__⌋	P11
12. How much time do you spend doing vigorous-intensity sports, fitness or recreational activities on a typical day?	Hours : minutes ⌊__⌋⌊__⌋ : ⌊__⌋⌊__⌋ hrs mins	P12
13. Do you do any moderate-intensity sports, fitness or recreational *(leisure*) activities that causes a small increase in breathing or heart rate such as brisk walking (*cycling, swimming, volleyball*) for at least 10 minutes continuously?	Yes 1 No 2 *If No, go to P16*	P13
14. In a typical week, on how many days do you do moderate-intensity sports, fitness or recreational (*leisure*) activities?	Number of days: ⌊__⌋	P14
15. How much time do you spend doing moderate-intensity sports, fitness or recreational (*leisure*) activities on a typical day?	Hours : minutes ⌊__⌋⌊__⌋ : ⌊__⌋⌊__⌋ hrs mins	P15
Sedentary behavior		
The following question is about sitting or reclining at work, at home, getting to and from places, or with friends including time spent [sitting at a desk, sitting with friends, travelling in car, bus, train, reading, playing cards or watching television], but do not include time spent sleeping.		
16. How much time do you usually spend sitting or reclining on a typical day?	Hours : minutes ⌊__⌋⌊__⌋ : ⌊__⌋⌊__⌋ hrs mins	P16

Perceived Stress Scale

The questions in this scale ask you about your feelings and thoughts during the last month. In each case, you will be asked to indicate by circling how often you felt or thought a

#		0 Never	1 Almost Never	2 Sometimes	3 Fairly Often	4 Very Often
1	In the last month, how often have you been upset because of something that happened unexpectedly?					
2	In the last month, how often have you felt that you were unable to control the important things in your life?					
3	In the last month, how often have you felt nervous and "stressed"?					
4	In the last month, how often have you felt confident about your ability to handle your personal problems?					
5	In the last month, how often have you felt that things were going your way?					
6	In the last month, how often have you found that you could not cope with all the things that you had to do?					
7	In the last month, how often have you been able to control irritations in your life?					
8	In the last month, how often have you felt that you were on top of things?					
9	In the last month, how often have you been angered because of things that were outside of your control?					
10	In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?					

Fatigue Symptom Inventory

For each of the following, circle the <u>one number</u> that best indicates how that item applies to you.

1. Rate your level of fatigue on the day you felt **most** fatigued during the past week:

0	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
Not at all fatigued										**As fatigued as I could be**

2. Rate your level of fatigue on the day you felt **least** fatigued during the past week:

0	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
Not at all fatigued										**As fatigued as I could be**

3. Rate your level of fatigue on the **average** during the past week:

0	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
Not at all fatigued										**As fatigued as I could be**

4. Rate your level of fatigue **right now**:

0	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
Not at all fatigued										**As fatigued as I could be**

5. Rate how much, in the past week, fatigue interfered with your **general level of activity**:

0	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
No interference										**Extreme interference**

6. Rate how much, in the past week, fatigue interfered with your **ability to bathe and dress yourself**:

0	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
No interference										**Extreme interference**

7. Rate how much, in the past week, fatigue interfered with your **normal work activity (includes both work outside the home and housework)**:

0 1 2 3 4 5 6 7 8 9 10

No interference **Extreme interference**

8. Rate how much, in the past week, fatigue interfered with your **ability to concentrate**:

0 1 2 3 4 5 6 7 8 9 10

No interference **Extreme interference**

9. Rate how much, in the past week, fatigue interfered with your **relations with other people**:

0 1 2 3 4 5 6 7 8 9 10

No interference **Extreme interference**

10. Rate how much, in the past week, fatigue interfered with your **enjoyment of life**:

0 1 2 3 4 5 6 7 8 9 10

No interference **Extreme interference**

11. Rate how much, in the past week, fatigue interfered with your **mood**:

0 1 2 3 4 5 6 7 8 9 10

No interference **Extreme interference**

12. Indicate **how many days**, in the past week, you felt fatigued for any part of the day:

0 1 2 3 4 5 6 7

Days **Days**

13. Rate **how much of the day**, on average, you felt fatigued in the past week:

0 1 2 3 4 5 6 7 8 9 10

None of the day **The entire day**

14. Indicate which of the following best describes the **daily pattern** of your fatigue in the past week:

0	*1*	*2*	*3*	4
Not at all fatigued	**Worse in the morning**	**Worse in the afternoon**	**Worse in the evening**	**No consistent daily pattern of fatigue**

Sleep Log

	Day 1	Day 2
What time did you turn off the lights and get into bed?		
How long did it take you to fall asleep?		
How many times did you wake up, not counting your final awakening?		
What time was your final awakening?		
What time did you get out of bed for the day?		
How would you rate the quality of your sleep?	Very poor Poor Fair Good Very good	Very poor Poor Fair Good Very good

Appendix B. Chinese Version of Study Questionnaires

基本调查问卷

1. 出生时间： _____ (月/年)
2. 性别： 男 _____ 女 _____
3. 您居住在美国多长时间：_____ (请精确到年)
4. 最高教育程度
   _____ 初中以下　　_____ 初中
   _____ 高中　　_____ 大专
   _____ 大学　　_____ 研究生
   _____ 博士
5. 工作状态
   _____ 全职　　_____ 半职
   _____ 无工作　　_____ 自由职业
   _____ 学生　　_____ 退休
6. 您是否吸烟?
   _____ 目前吸烟
   _____ 曾经吸烟
   _____ 从不吸烟
7. 您有心血管疾病史吗？(例如高血压，糖尿病等)
   _____ 有，请列出：
   _____ 无
8. 您在最近一年内有服用任何药物吗？
   _____ 有，请列出：
   _____ 无

匹兹堡睡眠质量指数

下面一些问题是关于您最近 1 个月的睡眠情况，请选择回填写最符合您近 1 个月实际情况的答案

1. **过去一个月内**，通常**晚上**几点上床? _____
2. **过去一个月内**，**每天晚上需要花多**长时间入睡?（用分钟表示） _____
3. **过去一个月内**，通常早上几点起床?_____
4. **过去一个月内**，**晚上**实际睡眠几个小时? (**这可能与您卧**床时间不一样)

_____

5. **过去一个月内**，**因**以下原因您有多少次存在睡眠困扰：				
a.无法在 30 **分**钟内入睡	0	< 1 **次/周**	1-2 **次/周**	≥ 3 **次/周**
b. **夜**间易醒/早醒	0	< 1 **次/周**	1-2 **次/周**	≥ 3 **次/周**
c. 夜间去厕所	0	< 1 **次/周**	1-2 **次/周**	≥ 3 **次/周**
d. **呼吸不**畅	0	< 1 **次/周**	1-2 **次/周**	≥ 3 **次/周**
e. **咳嗽或**大声打鼾	0	< 1 **次/周**	1-2 **次/周**	≥ 3 **次/周**
f. 因**感**觉冷影响睡眠	0	< 1 **次/周**	1-2 **次/周**	≥ 3 **次/周**
g. 因**感**觉热影响睡眠	0	< 1 **次/周**	1-2 **次/周**	≥ 3 **次/周**
h. **做**恶梦	0	< 1 **次/周**	1-2 **次/周**	≥ 3 **次/周**
i. **疼痛不适**	0	< 1 **次/周**	1-2 **次/周**	≥ 3 **次/周**
j. **其它**原因，**请说明**: _____	0	< 1 **次/周**	1-2 **次/周**	≥ 3 **次/周**
6.**过去一个月内**，您有多少次服用药物帮助睡眠	0	< 1 **次/周**	1-2 **次/周**	≥ 3 **次/周**
7. **过去一个月内，在开**车，用餐，日常社交活动时，有多少次觉得难以保持**清醒状**态	0	< 1 **次/周**	1-2 **次/周**	≥ 3 **次/周**
8. **过去一个月内**，要保持足够的热情去完成事情对您有多困难	完全没有	轻微/少许	一般	极其明显
9. **过去一个月内**，您如何评价自己的睡眠质量	很**好**	较**好**	较**差**	很**差**

体力活动问卷

核心内容：体力活动

工作时的体力活动

下面我要询问你通常每周做各类体力活动所花费的时间。请回答下列问题 (即使你认为自己不经常参加体力活动) 。 首先是工作中的体力活动。**工作**是指你必须完成的有酬或无酬工作，学习/培训，或家务。**关于剧烈活动**，是指高负荷的体力活动并引起呼吸心跳显著增加。**中等强度**的活动是指一定负荷的体力活动并引起呼吸心跳轻度增加。 请借助我为您提供的图片回答下列问题

1	你的工作需要做**剧烈活动**以致引起呼吸和心跳显著增加 [如搬运或举重物、挖掘或建筑工作] 时间至少持续 10 分钟吗？	是 1 否 2 *若为否，跳转至* ***P4***	P1
		工作中每天花多长时间做剧烈运动 _____天	P2
		工作中每天花多长时间做剧烈运动 _____ : _____ 小时 分钟	P3
2	你的工作需要做引起呼吸和心跳轻度增加的**中等强度**活动如快步走[或搬运较轻的物品] 时间至少持续 10 分钟吗？	是 1 否 2 *若为否，跳转至* ***P7***	P4
		工作中每周花多长时间做中等强度运动 _____天	P5
		工作中每天花多长时间做中等强度运动 ____ : ____ 小时 分钟	P6

交通时的体力活动

以下问题不包括上述工作时的体力活动。现在我要询问你通常的交通方式。例如，去上班、去购物、去市场等

3	你每周去某个地方时需要步行或骑自行车至少持续 10 分钟以上吗？	是 1 否 2 *若为否，跳转至* ***P10***	P7
		每周有几天会花在步行或骑自行车上？ _____天	P8
		每天有多长时间花在步行或骑自行车上？ ____ : ____ 小时 分钟	P9

娱乐性体力活动

以下问题不包括上述的工作和交通过程中的体力活动。现在我询问你有关运动、健身和娱乐性体力活动（休闲）的问题

4	你每周进行引起你呼吸和心跳显著增加的**剧烈**的运动、健身和娱乐性（休闲）体力活动并至少持续 10 分钟以上吗？	是 1 否 2 *若为否，转跳至* ***P13***	P10
		每周有几天做剧烈运动？ _____天	P11
		每天有多长时间做剧烈运动？ _____ : _____ 小时 分钟	P12
5	你进行引起你呼吸和心跳轻度增加的**中等强度**的运动、健身和娱乐性体力活动（休闲），如快步走（骑自行车、游泳、排球）至少持续 10 分钟或以上吗？	是 1 否 2 *若为否，跳转至* ***P16***	P13
		每周有几天做中等强度运动？ _____天	P14
		每天有多长时间做中等强度运动？ _____ : _____ 小时 分钟	P15
久坐习惯 以下问题是**关**于工作时、在家里、交通过程中、**会**朋友时坐着或**靠**着所花费的时间。 包括坐在**桌**前，**与**朋友一起坐着，乘坐轿车、公共汽车、火车，阅读，打扑克或看电视。**但不包括睡觉的时间**。			
6	你通常每天有多少时间坐着或靠着？	小时：分钟 _____ : ____ 小时 分钟	P16

压力知觉量表

我们想要询问最近一个月中您个人的感受和想法。请您对如下每一个陈述指出您的感受的频率。虽然有些问题看起来相似，实则有差异。所以请您尽量以快速、不假思索的方式填答。也不要去过多考虑每一项陈述背后的涵意，以确保真实反应您近期的压力状况。

#	请回想**最近一个月以来**，发生下列各状况的频率	0 从不	1 偶尔	2 有时	3 经常	4 总是
1	对某些突然发生的事情感到不安					
2	感觉无法控制自己生活中重要的事					
3	感到紧张不安和压力					
4	对自己处理私人事情的能力很有信心					
5	感到事情顺心如意					
6	发现自己无法处理所有必须要做的事					
7	有办法控制生活中恼人的事情					
8	觉得自己是驾驭事情的主人					
9	常生气，因为很多事情的发生是超出自己所能控制范围内的					
10	常感到困难的事情堆积如山，自己无法克服它们					

疲劳调查问卷

我们有时感觉疲乏或劳累，请根据下列陈述选择最恰当的一个数字

1. 近一周来，你感到**最疲劳**那一天的疲劳程度

   0 1 2 3 4 5 6 7 8 9 10

   (0 = 一点也不疲劳；10 = 极度疲劳)

2. 近一周来，你感到**最不疲劳**那一天的疲劳程度

   0 1 2 3 4 5 6 7 8 9 10

   (0 = 一点也不疲劳；10 = 极度疲劳)

3. 近一周你疲劳的**平均程度**?

   0 1 2 3 4 5 6 7 8 9 10

   (0 = 一点也不疲劳；10 = 极度疲劳)

4. 你**此时此刻**的疲劳程度

   0 1 2 3 4 5 6 7 8 9 10

   (0 = 一点也不疲劳；10 = 极度疲劳)

5. 近一周来，疲劳对你**日常活动**的影响程度

   0 1 2 3 4 5 6 7 8 9 10

   (0 = 一点也没影响；10 = 影响极大)

6. 近一周来，疲劳对你自己**洗浴，穿衣能力**的影响程度

   0 1 2 3 4 5 6 7 8 9 10

   (0 = 一点也没影响；10 = 影响极大)

7. 近一周来，疲劳对你**工作活动**的影响程度，包括户外工作和做家务

   0 1 2 3 4 5 6 7 8 9 10

   (0 = 一点也没影响；10 = 影响极大)

8. 近一周来，疲劳对你**集中力**的影响程度

   0 1 2 3 4 5 6 7 8 9 10

   (0 = 一点也没影响；10 = 影响极大)

9. 近一周来，疲劳对你**人际关系**的影响程度

   0 1 2 3 4 5 6 7 8 9 10

   (0 = 一点也没影响；10 = 影响极大)

10. 近一周来，疲劳对你**享受生活**的影响程度

    0 1 2 3 4 5 6 7 8 9 10

    (0 = 一点也没影响；10 = 影响极大)

11. 近一周来，疲劳对你**情绪**的影响程度

    0 1 2 3 4 5 6 7 8 9 10

    (0 = 一点也没影响；10 = 影响极大)

12. 近一周来，你有几天感到疲劳

0 1 2 3 4 5 6 7

(0 = 一天都没有；7 = 七天都有)

13. 一天之中，你大概有多长时间感觉疲劳

0 1 2 3 4 5 6 7 8 9 10

(0 = 没有疲劳；7 = 一整天疲劳)

14. 近一周来，请选择一项恰当描述你疲劳的时间

0	1	2	3	4
没有疲劳	早上最疲劳	下午最疲劳	晚上最疲劳	每天疲劳的时间无规律

睡眠日记

	第一天	第二天
您晚上熄灯睡觉的时间?		
您躺下多长时间才入睡?		
您入睡后夜间醒来的次数?		
您最终醒来的时间?		
您早上几点起床?		
请您给自己的睡眠质量打分	非常差 不好 一般 好 非常好	非常差 不好 一般 好 非常好