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Understanding Nocturnal Ambulatory Blood Pressure in Youth

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UNIVERSITY OF MIAMI

UNDERSTANDING NOCTURNAL AMBULATORY BLOOD PRESSURE IN YOUTH

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In adolescents, nocturnal ambulatory blood pressure (ABP) provides a rich and informative picture of cardiovascular health and risk. This study modeled nocturnal ABP taken during one night using multi-level growth modeling. A combination of demographic, daytime, clinical, and sleep related variables were also included as inter-individual predictors of nighttime blood pressure regulation. Adolescents (N = 252) were obtained from three larger studies addressed cardiovascular risk in youth attending public high schools in Miami-Dade County. Participants included in this study completed a baseline medical screening, initial ABP assessment visit and had at least one ABP reading taken during sleep. Within a multi-level growth model, time since sleep was included as a Level 1 variable to establish the pattern of nocturnal blood pressure decline. Next at Level 2, demographic (i.e. race/ethnicity, gender, SES), daytime (i.e., school attendance, substance use, stress encountered during the day), and clinical (i.e. obesity, metabolic syndrome, fitness, parental/family history of hypertension) variables were added as predictors of both the intercepts and instantaneous rates of blood pressure decline. Lastly, in a smaller sample of participants who completed sleep quality measures (N = 101), sleep related variables (i.e. normality of this sampled sleep, sleep environment, sleep disturbances) were added as predictors of instantaneous rates of blood

pressure decline. While associated with higher blood pressure at the beginning of sleep; identifying as non-Hispanic Black or Other, having a family history of hypertension and reporting higher stress during the day were associated with faster rates of blood pressure decline during sleep. Similarly, while associated with lower intercepts at the beginning of sleep, identifying as non-Hispanic White and as a girl were associated with slower rates of decline during sleep. While specific racial/ethnic identifications, gender, family history of hypertension and greater stress were related to differing average levels of daytime ABP as expected, sleep appeared to act as a recovery period for these blood pressure differences in this sample of adolescents. This study suggests the importance of sleep within adolescent populations at risk for CVD for whom the reduction of blunted nocturnal decline and prevention of target organ damage is targeted.

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CHAPTER 1: INTRODUCTION

A disturbing public health picture has emerged as cardiovascular disease risk factors in youth have increased, especially as these risk factors have implications for later adult cardiovascular health (Cooper et al., 2000). Studies have shown that risk factors such as elevated blood pressure, high low-density lipoprotein (LDL) cholesterol, tobacco use and clinically elevated body mass index (BMI) measured in childhood are related to measures of cardiovascular disease in adulthood, such as carotid artery intima-media thickness (Berenson and the Bogalusa Heart Study Research Group, 2002; Davis, Dawson, Riley,& Lauer, 2001; Raitakari et al., 2003). It should be noted that risk factors measured during adolescence were more predictive of later subclinical cardiovascular disease than cardiovascular risk factors measured in early childhood (Davis et al., 2001; Raitakari et al., 2003). Therefore, with increasing presence of risk factors and their future impact on health, research findings have suggested adolescence as a critical period in the development of cardiovascular disease risk (Karvey et al., 2003).

Blood pressure is an important indicator of cardiovascular risk (Desias, Stockbridge,& Temple, 2006). In youth, current blood pressure trends have made it a focus of study. When comparing average blood pressure levels across decades, Mutner, He, Cultuer, Wildman and Whelton (2004) found small average increases (1.4 mmHg in mean systolic blood pressure, 3.3 mmHg in diastolic blood pressure) in blood pressure among youth. Beyond mean blood pressure levels, current cases of hypertension in youth have been increasingly categorized as essential (a product of genetic, biological and lifestyle factors) as opposed to secondary to another medical disorder, suggesting

growing cardiovascular risk in this population (Vogt, 2001). Increasing levels of risk factors associated with blood pressure have raised concerns as well. For example, growing rates of obesity in youth have cardiovascular health implications as BMI has a positive and incremental relationship with blood pressure, even after controlling for other risk factors (Din-Dziethman, Liu, Beilo,& Shamsa, 2007; Falkner et al., 2006; Loria, Crespo, & Burt, 1996; Moore, Stephens, Wilson, Wilson, & Eichner, 2006; Mutner et al., 2004; Sorof, Lai, Turner, Poffenbarger,& Portman, 2004). Also, elevated blood pressure status has been shown to impact future cardiovascular health as youth with elevated blood pressure are more likely to have the same status in adulthood (Mahoney, Lauer, Lee, & Clarke, 1996; Williams et al., 2002). These studies have shown that understanding blood pressure in adolescence has important implications for both current and later cardiovascular health in elevated risk populations.

Nocturnal Ambulatory Blood Pressure (ABP)

Research has shown nocturnal ABP is often related to clinical outcomes, sometimes more than overall or daytime ABP levels (Kikuyaet al., 2005; Mancia & Parati, 2000). Nocturnal ABP has also been uniquely related to left ventricular mass index (LVMI), sleep apnea, stroke and mortality (Profant & Dimsdale, 1999), though stronger evidence exists for adults (Fagard, Staessen,& Thijs, 1997; Flynn, 2010). Within youth, nocturnal blood pressure levels have occasionally been related to target organ changes. Richey and colleagues found that an increased LVMI was related to higher nighttime systolic blood pressure levels (Richey et al., 2008). Nocturnal ABP has also been identified as related to current cardiovascular functioning in clinical populations. For example, in youth with type I diabetes, Torbjornsdotter, Jaremko, and Berk (2004)

found that higher levels of nocturnal blood pressure were related to having poorer renal function as well as renal morphological changes, which are direct evidence of target organ damage. The relationship between nocturnal ABP and current as well as future health outcomes has supported its clinical utility.

By measuring blood pressure repeatedly and usually over a 24 hour period, researchers and clinicians using ABP monitoring (ABPM) have had the opportunity to create rich pictures of individuals' blood pressure regulation. Often, studies have used more summary-like methods to capture nocturnal blood pressure regulation in youth (Profant & Dimsdale, 1999; Richey et al., 2008; Torbjornsdotter, Jaremko,& Berk, 2004). Exploring nocturnal ABP in greater detail could further the understanding of nocturnal blood pressure regulation. Research in healthy adults using polysomnography showed various patterns within nighttime blood pressure from one hour before sleep until the end of the first REM cycle (Carrington et al., 2005). These researchers found a decline in blood pressure after "light outs", suggesting an anticipatory effect, and an additional decline after the onset of stable sleep, a sharp and seemingly abrupt drop. During sleep, the number of arousals was negatively associated with decline in blood pressure. More specifically, more arousals were associated with quick and fleeting increases in blood pressure. It appears that nighttime blood pressure declines throughout sleep phases, in a roughly linear fashion. While this study provided some information on how blood pressure declines at night, this study modeled nighttime blood pressure in only one hour of sleep in a sample of healthy adults. A review of the literature did not reveal similar studies or typical use of multiple data points to model nocturnal blood pressure change

over time in youth, suggesting the need for additional research on how blood pressure declines in this population.

Definitions and Reliability of Nocturnal ABP

Definitions of nighttime blood pressure have varied somewhat across research studies. Some researchers have used researcher set sleep times to define nocturnal blood pressure, while other researchers have used participant reported sleep times (Dimsdale et al., 2000). Identification of sleep and wake intervals influence what readings are considered nocturnal and consequently affect the research results and generalizability of findings (Sorof & Portman, 2001). Attempting to determine the effects of definitional differences, Dimsdale and colleagues tested the reliability in mean nocturnal blood pressure levels and dipping status across three days using different techniques to determine sleep times (Dimsdale et al., 2000). Specifically, they compared differences between daytime and nighttime mean blood pressure based on clock time (set by the researcher), bedtime (self-reported) and sleep time (verified polysomnographically). While they did not find any one definition to be superior to others in regards to reliability, these researchers recommended defining sleep times in a way that allows for individual variability. In later research, Delaney and colleagues found, that when comparing researcher-set versus self-report sleep times, self-reported sleep times were superior in determining nighttime blood pressure and nocturnal dip (Delaney, Pellizzari, Speiser & Frank, 2009). Overall, research supported the superiority of individual variability as opposed to imposing rigid researcher set sleep times. Using methods that allow for individual variability appear essential in accurately exploring nighttime blood pressure regulation.

In addition to the way that nighttime blood pressure is determined, nocturnal blood pressure regulation has been defined in many ways. Often less than a ten percent decline from average awake to average asleep blood pressure has been used to define nocturnal dipping (Borel et al., 2009; Darcan et al., 2006; Delaney, Pellizzari, Speiser, & Frank, 2009; Dimsdale et al., 2000; Hughes, Kobayashi, & Deichert, 2007; Spurilli et al., 2009; Urbina et al., 2008). It should be noted that patterns of inverted dipping, increased blood pressure at night compared to daytime levels, and double dippers (those with blood pressure declines beyond 20% of their daytime mean levels)are other categorizations of interest in understanding nighttime blood pressure (Hoshide et al., 2002; Manabe et al., 2001). Not being able to accurately categorize different types of dippers has been associated with misclassification. This distinction could be important as an individual who has a smaller decrease in blood pressure during nighttime could be very different from a person who has no decrease, an increase or an overly pronounced decrease in blood pressure during sleep. The way in which dippers and non-dippers have been categorized has the capacity to influence how nocturnal blood pressure regulation is understood.

Establishing reliability is essential for determining the utility of a concept and strengthening its predictive value. Summaries of earlier research have observed reliability estimates for measures of nocturnal blood pressure that typically ranged from .42-.80 (Dimsdale et al., 2000; Sprulli et al., 2009). Schwartz, Grossman, Warren, and Pickering (1997) found correlations ranging from .81-.88 when using maximum likelihood estimates of the mean and variances of differences between awake and sleep ambulatory blood pressure measured across two separate assessments, a marked improvement over

the reliability estimates made using Pearson's correlation in this sample. These authors concluded that statistical techniques that better handled random measurement error led to greater observed correlations across time and consequently improved the reliability of the investigated concept, difference between daytime and nighttime blood pressure. Overall, while research often questions the stability of the categorization of nocturnal dipping across measurements, it has been influenced greatly by the statistical methods used. Findings have suggested that nocturnal blood pressure regulation can be studied reliably, especially with the use of appropriate statistical methods.

ABPM requires interpreting multiple blood pressure measurements over time. The complexity of measuring ABP has made the choice of statistical methods used for interpretation important for validity and generalizability of findings. Growth modeling using maximum likelihood (ML) estimation has been deemed appropriate for data measured multiple times within individuals and has also allowed for testing differences in slopes and intercepts across these individuals observed over time (Hox, 1998; SAS; 2008). Growth modeling has provided a more robust, powerful and realistic estimation of change across multiple observations gathered within persons when compared to the use of Repeated Measures ANOVAs (RM-ANOVAs) (Quene & Van den Bergh, 2004; Hox, 1998). Particular to comparing ABP levels, Schwartz, Grossman, Warren and Pickering (1997) found using ML estimation lead to better measures of variances, especially for nocturnal blood pressure for which less readings are typically taken. Unlike RM-ANOVAS, assumptions of sphericity and homogeneity of variance are less stringent when using ML estimation as variances and covariances are estimated directly from the data. In addition, ML estimation allows for data to be missing at random. Not having to

remove each subject missing an observation allows for greater power in analyses (Quene & Van den Bergh, 2004). ML estimation has given researchers a better equipped tool to deal with the complexity of the multi-level design ABPM interpretation requires.

One must also consider what type of ML estimation would be most appropriate for the specific research design, research questions, and collected data. If a researcher were primarily interested in comparing nested models, Full Information Likelihood estimation (FML) has been recommended. While if a researcher were more interested in commenting on random effects such as variances or covariances, the use of Restricted Likelihood Estimation (REML) would be more appropriate. REML has also been shown to provide more accurate estimates, especially in smaller samples (Hox 1998). The use of REML would be most appropriate given the number and range of ABP readings taken in individuals during sleep.

Predictors of Nocturnal Blood Pressure

Due to inter- as well as intra-individual differences in blood pressure regulation, a number of variables that typically influence ABP readings, moment to moment as well as overall, have often been used to explain differences in ABP. These variables include, but are not limited to: age, BMI, weight, consumption, physical maturity, location, physical fitness, substance use, gender, race/ethnicity and socioeconomic status (SES), many of which are routinely collected using ABPM diaries. Researchers have also used more comprehensive ABPM diaries to evaluate how social, psychosocial and anthropomorphic factors relate to ABP responses in youth (Brady & Matthews, 2006; Chen, Matthews, & Zhou, 2007; Clark & Gochett, 2006; Meininger, Liehr, Chan, Smith,& Mueller, 2004).

The variety of variables that influence ABP has made it necessary to include a comprehensive list of ABP predictors.

Variables Used to Explain Frequently Observed Demographic Differences

Several variables have been suggested as uniquely related to nighttime ABP regulation, inferred from mean nighttime blood pressure levels as well as nocturnal dipping. One predictor often examined in nocturnal blood pressure research has been race. Within the United States, African Americans have been found to exhibit less nocturnal dipping and sometimes higher nocturnal blood pressure when compared to non-Hispanic White Americans. Even with similar average daytime and nighttime blood pressures levels between African Americans and non-Hispanic White Americans, smaller dips have been observed in African Americans (Hughes et al., 2007). These types of differences have not been observed in racial comparisons between White and Black persons outside of the United States, supporting the importance of social and historical context (Jones & Hall, 2006; Profant & Dimsdale, 1999). It appears important to view race as a proxy for social experiences.

Some researchers have begun to include exposure to racism into a biopsychosocial model of cardiovascular disease that suggests racism increases African Americans' exposure to stress, a known risk factor for cardiovascular disease (Clark, Anderson, Clark,& Williams, 1999). This conceptualization is consistent with recent trends to move away from treating race as a purely biological and genetic categorization, but as a historical and social construct (Smedly & Smedly, 2005). This conceptualization also integrates nicely into a body of work that suggests stressors (i.e. high demand and

low control work situations, acute events, chronic stress exposure) have effects on cardiovascular health, morbidity or incidence of negative cardiovascular events (Dimsdale, 2008). In cardiovascular health research, including variables that better capture individual experiences beyond racial identification continues to be warranted.

Measuring characteristics of sleep has been one avenue used to explain racial differences in nighttime blood pressure regulation. For example, researchers have found African Americans displayed poorer sleep quality, a variable which negatively impacted blood pressure regulation (i.e., less time in bed, sleep time asleep, taking longer to fall asleep, more time wake and poorer efficiency) (Hughes et al., 2007; Loredo, Ancoli-Israel,& Dismsale, 2001; Routledge & McFetridge-Durdle, 2007). While poor sleep quality influences nocturnal decline, it does not fully explain differences observed in nighttime blood pressure regulation (Pickering, Shimbo & Hass, 2006). The inclusion of other variables, capturing individual's social, daily and lifetime experiences, remains necessary as they could help more fully explain observed differences in blood pressure.

In addition to sleep quality variables, researchers have looked at social and other environmental explanations for nocturnal blood pressure regulation differences. For example, the relationship among violence exposure, catecholamine excretion and non-dipping patterns in African American adolescents has been explored. Wilson, Kliewer, Teasley, Plybon, and Sica (2000) observed that being a victim of violence was associated with non-dipping status. In addition, boys who reported more indirect violence exposure (i.e., hearing about violence during the day) were more likely to be categorized as non-dippers. In adults, controlling for age, sex, BMI and mean levels of blood pressure did not remove racial differences in dipping (Spuruill et al., 2009). These researchers did find

dipping to be positively associated with education, income, being married, and having social support. The inclusion of these variables accounted for 36% of the effect of race on dipping, suggesting the need to include many social constructs beyond race. While controlling for a variety of factors such as gender, age, ethnicity, BMI, and sleep quality (measured as the number of apneas and hyponeas per hour of sleep), these researchers found that ethnicity as well as SES, as measured by occupation instead of education, were independent predictors of nocturnal dipping (Stepnowsky, Nelesen, DeJardin & Dimsdale, 2004). It appears that life experiences as well as social factors have been related to nighttime blood pressure regulation and explain differences observed between racial groups.

Clinical Factors in Need of Additional Exploration

Researchers have taken different approaches to exploring the relationship between metabolic syndrome criteria and nocturnal blood pressure regulation. With the dangerous cardiovascular risk profile that is developing in youth, studying the metabolic syndrome in this population is needed. The metabolic syndrome is a collection of metabolic factors that, in combination, pose a higher risk for cardiovascular disease/diabetes and related poorer prognosis than these risk factors alone (de Ferranti et al., 2004; Schillaci et al., 2004). Though established in adults, the metabolic syndrome has also been observed and studied within youth. Within youth, the Adult Treatment Panel III (ATP III) definition of having three or more of the following factors qualifies as having the metabolic syndrome: elevated blood pressure (systolic blood pressure $\geq$ 130 mmHg and/or average diastolic blood pressure $\geq$ 85 mmHg),high abdominal obesity (waist circumference over 35 inches for women or 40 inches for men), fasting glucose greater than 100 mg/dL, high

triglycerides levels (over 150 mg/dL) and/or low HDL levels (less than 40 mg/dL for women and under 50 mg/dL for men) has been used. Using this criteria to determine prevalence of metabolic syndrome, national data showed a 2-10% prevalence in non-clinical samples and almost 33% prevalence in youth labeled as overweight or obese (Cook, Weitzman, Auinger, Nguyen, & Dietz, 2003; de Ferranti et al., 2004; Duncan, Li, & Zhou, 2004). The prevalence and clinical implications of the metabolic syndrome have made this clinical syndrome essential in understanding cardiovascular at-risk youth.

While some researchers have explored the relationship between individual metabolic syndrome criteria/factors and nocturnal blood pressure, other researchers have explored the relationship between the presence or absence of the metabolic syndrome and nocturnal blood pressure. When using all metabolic syndrome criteria individually as predictors, most studies have found central obesity and insulin resistance, shown from correlational or indirect relationships, to account for the majority of variance explained in nocturnal blood pressure (Borel et al., 2009; Giladrini et al., 2007; Holl, Palvolvic, Heinze, & Thon, 1999; Lubre et al., 2008;Majane et al., 2007; Pons et al., 2009). Due to the clinical characteristics of the study samples, these studies suggest this relationship may only be observable in the presence of other cardiovascular risk factors such as obesity, diabetes, or clinically elevated HOMA levels. This observation suggested a possible synergistic relationship among cardiovascular risk factors such as obesity measures and glucose intolerance that manifests in blood pressure regulation at night (Lubre et al. 2008). There was less consistent and weaker evidence for relationships between the remaining metabolic syndrome criteria (i.e., low levels of high density lipoproteins (HDL), high triglycerides and elevated blood pressure) and nocturnal blood

pressure (Aguilar, Ostrow, De Luca,& Suarez, 2010; Ben-Dova & Bursztyn, 2009; Diamantopoulos et al., 2006; Giladrini et al., 2008). Regarding elevated blood pressure, having a blunted nocturnal decline has not been observed as a feature of all persons with essential hypertension, despite its relationship to negative cardiovascular prognosis (Ohjuboet al., 2002). It has been difficult to isolate the predictive power of these interrelated factors as noted by researchers exploring ABP in obese and diabetic populations (Eguchi, 2011). Findings have been inconsistent regarding the relationship between the metabolic syndrome and blunted dipping status (Ben-Dova & Bursztyn, 2009;Giladriniet al., 2008; Seiet al., 2007), but have suggested a strong role of insulin resistance and central adiposity in predicting nocturnal blood pressure regulation (Eguchi, 2011). Research has suggested it is theoretically and clinically appropriate to consider these factors together as the metabolic syndrome, not individually.

Other variables used to explain blood pressure differences in youth include measures of health behaviors such as caffeine intake and tobacco use. For example, caffeine is a central nervous stimulant that is associated with many negative physical outcomes (i.e. sinus tachycardia, tachycardia, vasodilatation, agitation, dizziness, insomnia, and restlessness) that are more pronounced in persons with heightened cardiovascular risk (Giardina, Gersh, & Saperia, 2010; Myers, 2004, Mort & Krise. 2008; Nurminen, Niittynen, Korpela & Vapaatalo, 1999). On average, laboratory manipulations using caffeine have found immediate blood pressure increases ranging from 3–15 mm Hg (Mort & Kurse, 2008) and are heavily influenced by baseline caffeine consumption as well as the amount of caffeine being administered (Mullen, Whitehouse, Shine & Toweel, 2011). Though the definitive role of caffeine in increasing blood pressure levels

is complicated by tolerance, research consistently calls for assessment of caffeine use within the context of blood pressure measurement (James, 2004, Mort & Kruse, 2008). To date, most of this research has been conducted in adults, showing a need for additional research on this relationship in youth (Temple, Dewey & Briatico, 2010; Savoca, Evans, Wilson. Harshfield & Ludwig, 2004; Seifert, Schaechter, Hershorin & Lipshultz, 2011).

Tobacco use is another health behavior associated with cardiovascular risk factors and negative cardiovascular outcomes such as coronary artery disease, peripheral artery disease, aortic aneurysm, stroke, hypertension, atherosclerosis, heart failure and arrhythmias, though the exact mechanisms have yet to be definitively determined (Leone, 2011). The effect of smoking on blood pressure has been found to vary by the type and duration of smoking exposure, possibly due to changes to the arterial walls. While overall exposure to passive smoking is associated with increases in blood pressure, decreases in blood pressure have been observed within active smoking (Leone, 2011). It should be noted there was more research on the immediate effects of tobacco on blood pressure in adults or animals, likely due to ethical reasons (Narkiewicz, Van de Borne, Lausberg, Cooley, Winniford, Davison et al., 1998). Though the percentage of adolescents who have tried cigarettes, smoke occasionally or smoke regularly has decreased within the past decade; national surveys suggest that almost one-fifth of adolescents have smoked a cigarette in the past 30 days (CDC, 2011). With the prevalence of smoking in adolescents and its impact on cardiovascular health, tobacco has been identified as a health behavior variable that must be included when attempting to accurately interpret blood pressure differences in youth.

RATIONALE

The superior predictive ability and clinical utility of nocturnal blood pressure has made it an important target for research. ABPM has allowed for rich exploration of blood pressure regulation in youth, a population currently showing an unfavorable cardiovascular risk factor profile. Most research in youth, however, has used means to explore nighttime blood pressure regulation. Modeling nocturnal blood pressure over time provides a more detailed and possibly informative picture of individual youths' nighttime blood pressure regulation. Taking advantage of the multitude of data captured during ABP monitoring, modeling nocturnal ABP over the entire sleep duration would provide a more detailed picture of nocturnal blood pressure regulation in youth, extending findings that employed summary methods (i.e., mean nighttime blood pressure, categorization of blunted nocturnal decline, elevated night time blood pressure status). As with all studies using ABPM, using a theoretically and clinically backed combination of predictors to explain differences in blood pressure regulation would be needed. Addressing related research questions would add to the understanding of what explains differences in nocturnal blood pressure regulation in youth.

Often, research has used mean ABP levels as outcomes within the exploration of nocturnal ABP in youth. Showing how blood pressure declines overnight would provide a richer picture of the nocturnal dipping phenomenon as well as differences in regulation at night. Seeing how specific predictors of nocturnal blood pressure have affected the pattern modeled over time would be a novel approach to understanding blood pressure regulation within youth. Demographic (i.e., gender, race/ethnicity, measure of SES), daytime (i.e., attendance of school, substance use, amount of stress encountered before

sleep), clinical (i.e., measures of obesity, presence of the metabolic syndrome, fitness, family history) and sleep-related (i.e., quality, disturbances) variables have been identified as appropriate to explore as predictors of nocturnal ABP measured over time. Greater information about how blood pressure changes during sleep within youth would extend findings and allow more in-depth analysis of blood pressure regulation patterns.

This project aimed to address two issues that may add to the current understanding of blood pressure regulation in youth. First, the pattern of nocturnal blood pressure within youth will be modeled using Maximum Likelihood estimation. After successfully modeling nocturnal blood pressure in youth, the specific predictive role of demographic (i.e., gender, race/ethnicity, measure of SES), daytime (i.e., attendance of school, substance use, amount of stress encountered before sleep), and clinical (i.e., measures of obesity, presence of the metabolic syndrome, fitness, family history) variables will be analyzed by testing expected differences in the intercept and instantaneous rate of changes due to these predictors. Also, the impact of sleep-related variables on the decline of nocturnal blood pressure will be explored by testing the differences in instantaneous rates of changes in blood pressure due to sleep quality, disturbances, and related environmental considerations.

RESEARCH QUESTIONS

1. What pattern does nocturnal blood pressure take in youth?

2. Does the nocturnal blood pressure pattern's intercept or instantaneous rates of change differ due to demographic factors (i.e., gender, race/ethnicity, SES), daytime factors (i.e., school attendance, substance use, stress encountered during the day), clinically relevant

variables (i.e., obesity, metabolic syndrome, fitness, parental/family history of hypertension), or measures of sleep quality (i.e., normality of this sampled sleep, sleep environment, sleep disturbances)?

HYPOTHESES

Pattern of Nocturnal Blood Pressure

Within individuals, a decline in blood pressure during sleep as compared to daytime blood pressure levels is predicted. This pattern in nocturnal blood pressure will best be modeled using a quadratic, linear and intercept factor.

Demographic factors

Identifying as a girl will be associated with lower blood pressure at t = 0 and larger rates of decline in blood pressure during sleep as compared to boys. Race and ethnicity will be related to blood pressure levels at the beginning of sleep as well as the rates of change of blood pressure during sleep. Identifying as Non-Hispanic Black will be associated with higher blood pressure at t = 0 and smaller rates of decline in blood pressure during sleep when compared to those identifying as Hispanic White. Identifying as non-Hispanic White will be associated with a lower blood pressure at t = 0 and larger rates of decline in blood pressure during sleep when compared to those identifying as Hispanic White. Individuals classified as Other are not expected to show differences in intercepts or instantaneous change when compared to those identifying as Hispanic Whites. SES will be negatively related to blood pressure levels at the beginning of sleep as well as the rates of change of blood pressure during sleep. Higher SES will be associated with lower blood pressure at t = 0 and greater rates of decline in blood

pressure during sleep. The addition of SES will reduce the impact of race/ethnicity on blood pressure. The inclusion of demographic variables will allow for a larger portion of variance within and between persons to be explained when compared to a model without demographic variables, however demographic effects would no longer be significant once daytime, clinical and sleep variables are included.

Daytime factors

Attending school, consuming caffeine, and using tobacco will be positively related to the intercept at t = 0 and smaller rates of decline in blood pressure during sleep. Stress during the day will be positively related to the intercept at t = 0 and smaller rates of decline in blood pressure during sleep. The inclusion of daytime variables will allow for a larger portion of variance within and between persons to be explained when compared to a model with only demographics variables. Daytime variables will no longer continue to be significantly related to intercepts and rates of change once clinical as well as sleep variables are included.

Clinical variables

Waist circumference and/or BMI will be significantly related to blood pressure at t = 0 and rates of decline in blood pressure during sleep. Meeting criteria for the metabolic syndrome and a positive cardiovascular family history will be positively related to blood pressure at t = 0 and negatively related to rates of decline in blood pressure during sleep. Fitness will be negatively related to blood pressure at t = 0 and positively related to rates of decline in blood pressure during sleep. The inclusion of clinical variables will allow for a larger portion of variance within and between persons

to be explained when compared to a model with only demographics and daytime variables. These variables will continue to be significant once sleep variables are included.

Sleep variables

Not having a typical night's sleep, sharing a bed, sharing a room and awakening during the night will be negatively related to rates of change of blood pressure, showing a blunting effect on blood pressure decline over the night. The inclusion of sleep variables will allow for a larger portion of variance in the slope within persons to be explained when compared to a model without sleep quality variables.

CHAPTER 2: METHOD

Participants

Participants were drawn from three larger studies (Cohort 1, 2, and 3)addressing cardiovascular risk in youth. Adolescents were identified as having elevated blood pressure during Miami-Dade county public school tenth grade hypertension screening. All participants engaged in similar baseline assessments that will be detailed further. Cohorts 1, 2 and 3 were comparable at baseline on demographics variables such as race/ethnicity, gender, maternal education, paternal education, and age. Clinical factors such as BMI, waist circumference, caffeine use, fitness level and presence of the metabolic syndrome were also comparable across groups. There were a larger proportion of youth using tobacco in Cohort 1 ($X^2(2) = 6.62$, $p < .05$) [Cohort 1: 10% (n = 98), Cohort 2: 4% (n = 26), Cohort 3: 2%(n = 127)]. In addition, all participants in Cohort 2had a family history of hypertension ($X^2(2) = 7.51$, $p < .05$) [Cohort 1: 78% (n = 97), Cohort 2: 100% (n = 26), Cohort 3: 77% (n = 128)].

To be eligible for inclusion in the present study, a participant needed to complete the baseline medical screening as well as initial ABP assessment visits and have at least one ABP reading taken during sleep. While 308 adolescents completed the baseline medical screening visit and initial ABP assessment visit, only 252 participants had at least one sleep nocturnal blood pressure reading and could be included in analyses; 56 participants were excluded due to having no sleep ABP readings (i.e., removal of monitor, not sleeping, errors for all readings recorded during sleep) or sleeping for more than 15 hours. For persons whose raw data did not indicate which readings were sleep

readings, a standard 10 pm sleep time was used with their reported awake time (n = 9). For persons for whose awake time needs to be estimated, their mean sleep time was calculated from their 7-Day Physical Activity Recall taken at the same time point as the ABP measurement occurred (n = 2). In the final sample, 39% (n = 98) were from Cohort 1, 10% (n = 26) were from Cohort 2, and 51% (n = 128) were from Cohort 3.

Demographics

The final sample consisted of 252 adolescents who averaged 16.1 *(SD* = .7) years of age. The sample was mostly boys (72%). Regarding race/ethnicity, adolescents were able to choose from the following options: Non-Hispanic White, White Hispanic, Black Hispanic, American Indian, African American, Caribbean Black, Asian, or Other. Though aware of the potential information lost in combining different ethnicity and racial groups, the sample sizes available as well as the typical categorization of race/ethnicity in blood pressure literature appeared to support the combination of groups. Research in youth that compares race and ethnicity with cardiovascular measures typically used non-Hispanic White (NHW) participants as the reference group. The demographics of this sample, however, reflected the distribution of race/ethnicity in Miami-Dade. Within those who identified as Hispanic, persons indicated being from a number of Caribbean, Central and South American countries (i.e., Cuba, Costa Rica, Puerto Rico, Peru, Honduras, Nicaragua, Ecuador, Columbia, Brazil, Argentina, Dominican Republic, Venezuela, Bolivia, and Guatemala). Consequently, with Hispanic being the largest ethnic group in this sample and the social nature of racial and ethnic identification, this study used Hispanics (n = 121) as the reference group for race/ethnicity, not non-Hispanic Whites (n = 32). In order to create a group reflecting Hispanic ethnicity, White and Black Hispanic

participants were combined to create a Hispanic group. To create a group reflecting non-Hispanic Black racial categorization, African Americans and Caribbean Blacks were combined to create a non-Hispanic Black (NHB) category (n = 78). Remaining ethnic and racial groups were combined into the Other category (n = 21). Regarding SES, the average years of education for the head of household was 14 (*SD* = 3) in this sample.

Daytime Variables

Regarding the day that ABP was collected, only 47% (n = 118) attended school during ABP collection, with information missing on 7 participants. The majority of the sample used caffeine (84%, n = 210), while a small percentage 6% (n = 14) used tobacco. In addition, participants on average reported being stressed (somewhat stressed or very stressed) at least three times during the day (M = 2.54, *SD* = 4.00). The majority of participants reported being stressed at least once (61%).

Clinical Variables

Out of the total sample, 75% (n=118) of participants had elevated blood pressure as determined at the medical screening visit. Elevated blood pressure was determined by systolic or diastolic blood pressure greater than or equal to the 90th percentile adjusted for height, gender and age (National Institutes of Health: National Heart, Lung and Blood Institute, 1996). In addition, 53% (n = 133) had a positive parental history of hypertension. Using information on maternal and paternal grandparents, it was determined that 80% (n = 200) has a positive family history of hypertension. Regarding measures of obesity, the average BMI of the sample fell in the overweight range (M = 29.0, *SD* = 7.2) and the average waist circumference for the sample was 36.5 inches (*SD*

= 6.9). The average fitness level as measured by VO_2max was 36.41 ml/kg/min (*SD* = 10.38), indicating a poor fitness level. When compared to national fitness measured levels determined by VO^2Max, both boys (*M* = 39.36, *SD* = 9.62) and girls (Girls *M* = 29.14, *SD* = 8.46) had a mean fitness level that fell at or below the 15th percentile for their age and gender (Eisenmann, Laurson, & Welk, 2011). In addition, 17% (n=42) of participants met criteria for the metabolic syndrome determined using the Adult Treatment Panel III criteria (National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Cholesterol in Adults (Adult Treatment Panel III, 2002). Youth were identified as having the metabolic syndrome if they had at least three metabolic syndrome factors: elevated blood pressure (average causal systolic blood pressure > 130 mmHg and/or average baseline causal diastolic blood pressure ≥ 85 mmHg*)*, high abdominal obesity (waist circumference over 35 inches for girls or 40 inches for boys), fasting glucose greater than 100 mg/dL, high triglycerides levels (over 150 mg/dL) and/or low HDL levels (less than 40 mg/dL for girls and under 50 mg/dL for boys).

Sleep Variables

Sleep characteristics were only collected for the Cohort 3. Persons who provided detailed sleep quality data will be referred to as the reduced sample throughout the rest of the study. Of the 101 persons who reported full sleep quality information, 53% reported the night during which ABP was collected was not a typical night sleep (n = 54). Most participants awakened during the night 70% (n = 71). Of those who reported awakening during the night, the majority identifying waking up because of the ABP monitor (67% n = 49). Almost thirty percent of participants reported awakening due to environmental

factors such as noise (14%, n = 10), light (1%, n = 1) and temperature (13%, n = 9). In addition, 18% of those who awoke cited using the bathroom as the reason (n = 13). There were five participants that reported awakening for a reason other than those assessed (7%). It should be noted that there was a smaller percentage of those identifying as non-Hispanic White (8%, n = 8) and smokers (3%, n = 3) in this reduced sample. In addition, the majority of the reduced sample had elevated blood pressure due to the nature of recruitment in Cohort 3 (98%, n = 99). See Tables1 and 2 for descriptive statistics for the full and reduced samples.

Procedures

Potential participants were initially identified for recruitment if they showed elevated blood pressure (systolic or diastolic blood pressure greater than or equal to the 90^{th} percentile adjusted for height, gender and age) during a school based blood pressure screening for 10^{th} graders in Miami Dade County public schools. For those students who were identified as having elevated blood pressure on two separate assessments one month apart, their parents were contacted about further participation in a study addressing cardiovascular risk in youth.

If parents expressed interest in further participation, a home visit was set up to determine eligibility and describe the demands and outline the procedures of the study. At the home visit, written informed consent from parents and informed assent from the adolescents was obtained. It should be noted that participants with any existing health conditions or diagnoses (i.e., asthma, heart murmurs, diabetes, severe allergies, renal disease, secondary hypertension, seizure disorder, developmental disabilities, cancer,

bronchial conditions, ventricular arrhythmias, heart disease, amputation or related birth defect, major psychological disorder, spinal cord injury, or arthritis), blood pressure greater than 160/100 mmHg, over the age of 18, taking hypertensive medications or having residence in the United States under 4 years were excluded. Casual blood pressure was taken using a mercury sphygmomanometer on the right arm. Three blood pressure readings were taken at five, seven and nine minutes following the initial five minutes rest period. In order to get an accurate blood pressure reading of participants at rest, the average of the last two readings were used to calculate clinical blood pressure. In addition, self–report questionnaires were given to the parents to assess medical family history and other family social characteristics. Family medical history was obtained from parent report. At the end of the home visit, participants belonging to Cohort 1 identified a friend to participate in the study with them. After which, contact information for this friend was collected. This was a procedure used to recruit adolescents who could potentially serve as a comparison group without elevated blood pressure during recruitment for Cohort 1. With parental permission, the same home visit procedure was completed. It should be noted that during recruitment for later intervention studies, which included Cohorts 2 and 3, no recruitment for non-elevated blood pressure participants was done. For the present study, data collected during the initial medical screening visit and ambulatory blood pressure assessment visit are included in analyses, allowing for comparison across the three cohorts.

Medical Screening Visit

During the medical screening visit, participants had their casual blood pressure, weight, height (without shoes), waist circumference, and blood collected with commonly

used standardized procedures. Casual blood pressure was collected using the same procedure from the home visit. An average of the final two readings was used to determine the baseline casual blood pressure measure for participants. The weight and height (collected after shoe removal) were used to calculate BMI (kg/m^2). Waist circumference was measured in inches and averaged over two readings. Fasting blood samples, using a catheter inserted in the arm, were taken to measure glucose, insulin, serum cholesterol, triglycerides and lipoproteins. To measure fitness, participants completed a maximal treadmill tests using a Balke (walk-jog) exercise protocol in order to calculate peak VO_2 (volume of oxygen: ml/kg/min). In addition, participants completed a 7-day activity recall during this visit in which they reported bed and wake-up times for the previous seven days.

Ambulatory Blood Pressure Assessment Visit

Each participant was fitted with an ambulatory blood pressure monitor (Accutracker II ambulatory blood pressure monitor). For calibration purposes, readings between the mercury sphygmomanometer and ambulatory blood pressure monitor were compared using a T-connection. Device differences equal to or less than 6 mmHg were viewed as acceptable. After a five minute rest period, calibration readings comparing the Accutracker and the mercury sphygmomanometer readings were taken at five and seven minutes in the supine position. After two separate two-minute rest periods, calibration readings were taken at two and four minutes in the sitting position and then in the standing position. After calibration was within acceptable limits, the T-connection was removed and instructions about how to use, remove and return the monitor were given to the participant. The diary allowed the participant to document his/her environment and

activity level. Only diary data entries that were directly comparable across the three cohorts were included for analyses: location, degree of physical activity, posture, and stress level. Cohort 3 completed a more detailed sleep related diary card in which they reported whether they had a typical night sleep, shared a bed/room, or awoke during sleep. Those who indicated that they awoke during sleep were able to indicate the following reasons: the monitor, light, noise, temperature, using the bathroom, or other. ABP and heart rate readings were taken every 20 minutes during the day and hourly between 10pm and 7 am. The following criteria were used to edit and or exclude questionable ambulatory blood pressure readings: no diary recording of posture or locations, a systolic reading less than 60 mmHg or greater than 260 mmHg, a diastolic reading less than 40 mmHg and greater than 150 mmHg, or a difference between systolic and diastolic reading less than 20 mmHg or greater than 150 mmHg. Using diaries to determine when the participant went to bed and awoke, mean awake and sleep systolic and diastolic blood pressures were calculated. Regarding blunted nocturnal decline, a participant whose difference between average awake and sleep blood pressures was less than ten percent of their average awake blood pressure was classified as having a blunted nocturnal decline. Similar to elevated blood pressure status determined at baseline, elevated nighttime blood pressure was determined if a participants showed an average systolic or diastolic blood pressure greater than or equal to the 90^{th} percentile adjusted for height, gender and age during sleep during ABPM.

Statistical Analyses

Statistical Analysis Systems (SAS) version 9.2 was used to calculate descriptive statistics for demographic, ABPM, daytime, clinical, and sleep variables and conduced

multi-level analyses using REML. Normality and kurtosis were tested using SAS 9.2. Hierarchical linear modeling was used to test the hypotheses outlined in the rationale.

The first research question, *What pattern did nocturnal blood pressure take in youth,* was addressed by using time since sleep to model nocturnal blood pressure decline within Level 1 of the multi-level model (refer to equation 1 below).

$$\text{L1: } Y_{ij} = \pi_{0i} + \pi_{1i}(\text{time} - \text{time went to sleep}_{cwc}) + \pi_{ai}(\text{time} - \text{time went to sleep}_{cwc})^2 + \sigma_{\varepsilon} \quad (1)$$

$$\text{L2: } \pi_{0i} = \gamma_{00} + \zeta_{oi} \quad (2)$$

$$\pi_{1i} = \gamma_{10} + \zeta_{1i} \quad (3)$$

$$\pi_{2i} = \gamma_{20}$$

First, an empty model, one without any predictors, was estimated prior to the addition of time as a Level 1 variable. This model provided the mean nocturnal blood pressure, without considering time since sleep, for all sampled adolescents as well as random error between the participating adolescents. This model served as comparison point for the final model containing both a linear and quadratic time component. These equations above modeled each individual's blood readings taken during sleep as a function of the individual's minutes asleep, measured linearly and quadratically. For additional details of the models estimated at each step of the model building process, refer to Appendix A. Comparisons of residual variances, variances in the intercept, variances in the instantaneous rate of decline and covariances were made as additional variables were added to determine how their inclusion impacted the amount of variance explained.

After successful modeling the pattern of nocturnal blood pressure during sleep using time, inter-individual variables were incrementally added to Level 2 of the multi-level model (see equation 4 & 5 below) to address the following research question:

Do the nocturnal blood pressure pattern's intercept or instantaneous rate of changes differ due to demographic factors (i.e., gender, race/ethnicity, SES), daytime factors (i.e., school attendance, substance use, stress encountered during the day), clinically relevant (i.e., obesity, metabolic syndrome, fitness, family history of hypertension), or measures of sleep quality (i.e., normality of sampled sleep, sleep environment, sleep disturbances)?

L2:

$$\pi_{0i} = \gamma_{00} + \gamma_{01}\,(\text{Girl}) + \gamma_{02}(\text{NHW}) + \gamma_{03}\,(\text{NHB}) + \gamma_{04}\,(\text{Other}) + \gamma_{05}(\text{SES-SES}_{GMC}) + \gamma_{06}\,(\text{schoolday}) + \gamma_{07}\,(\text{caffeine}) + \gamma_{08}(\text{tobacco}) + \gamma_{09}(\text{stress}) + \gamma_{010}(\text{waist-waist}_{GMC}) + \gamma_{011}(\text{BMI-BMI}_{GMC}) + \gamma_{012}(\text{mets}) + \gamma_{013}(\text{fitness-fitness}_{GMC}) + \gamma_{014}(\text{famhyphx}) + \zeta_{oi} \qquad (4)$$

$$\pi_{1i} = \gamma_{10} + \gamma_{11(\text{intercept*slope})} + \gamma_{12}\,(\text{Girl}) + \gamma_{13}(\text{NHW}) + \gamma_{14}\,(\text{NHB}) + \gamma_{15}\,(\text{Other}) + \gamma_{16}(\text{SES-SES}_{GMC}) + \gamma_{17}\,(\text{schoolday}) + \gamma_{18}\,(\text{caffeine}) + \gamma_{19}(\text{tobacco}) + \gamma_{110}(\text{stress}) + \gamma_{111}(\text{waist-waist}_{GMC}) + \gamma_{112}\,(\text{BMI-BMI}_{GMC}) + \gamma_{113}(\text{mets}) + \gamma_{114}(\text{fitness-fitness}_{GMC}) + \gamma_{115}(\text{famhyphx}) + \gamma_{116}\,(\text{ntypicalsleep}) + \gamma_{117}\,(\text{shareoom}) + \gamma_{118}(\text{sharebed}) + \gamma_{119}(\text{awakenmon}) + \gamma_{120}(\text{awakenenivo}) + \gamma_{121}(\text{awakenbath}) + \gamma_{122}(\text{awakenoth}) + \zeta_{oi} \qquad (5)$$

$$\pi_{2i} = \gamma_{20}$$

Demographic, daytime, clinical and then sleep variables were added incrementally at Level 2 of the multi-level model as predictors of the intercept at the

beginning of sleep and the instantaneous rates of decline during sleep. The addition of demographic variables tested whether gender, race/ethnicity, and SES lead to statistically significant differences in blood pressure at t = 0 and instantaneous rates of decline during sleep, controlling for the effects of other demographic variables. Second, the addition of daytime factors tested whether school attendance, tobacco use, nicotine use, and number of times stress was reported were related to differences in blood pressure at t = 0 and instantaneous rates of decline, controlling for other daytime and all demographic variables. Next, clinical variables were introduced as Level 2 variables in order to test if the presence of the metabolic syndrome, family history of hypertension, fitness level, or measures of body size (BMI and waist circumference) were related to observed intercepts and instantaneous rates of changes during sleep, controlling for the effect of other clinical variables, demographic and daytime variables. Refer to Appendix B for a full description of related equations and coefficients. Again, comparisons of residual variances, variances in the intercept, variances in the instantaneous rate of decline and covariances were made as additional variables were added to determine how their inclusion impacted the amount of variance explained.

Analyses regarding the additional role of sleep quality in predicting the pattern of nocturnal blood pressure regulation were conducted in a subset of the original sample, belonging to Cohort 3 and having detailed sleep quality data, referred to as the reduced sample (see Appendix C for all related equations and coefficients). The previously described steps were all repeated exclusively within participants who completed measures of sleep quality from Cohort 3. First, time was successfully included as a Level 1 predictor of nocturnal blood pressure. Then, demographic, daytime and clinical

variables were introduced as Level 2 predictors of the intercept and instantaneous rates of change. Finally, sleep variables were introduced as predictors of instantaneous rates of changes to determine the impact of a typical night's sleep, sharing a room or bed and awakening for particular reasons (i.e., ABP monitor, environmental reasons, bathroom use and other reasons) on rates of decline during sleep (controlling for the impact of demographic, daytime and clinical variables as well as the effect of other sleep related variables). Again, comparisons of residual variances, variances in the intercept, variances in the instantaneous rate of decline and covariances were made as additional variables were added to determine how their inclusion impacted the amount of variance explained within and between individuals.

CHAPTER 3: RESULTS

Descriptive Statistics

Adolescents reported being asleep an average of 8 hours (M = 480 minutes, SD= 142 minutes) and had an average of 10 ABP readings (SD =6) taken during reported sleep. There was no statistically significant difference in duration of sleep during ABPM between boys and girls [$F_{gender}(1, 244) = 1.56$, $p = .21$)]. There were statistically significant differences in duration of sleep based on race/ethnicity, F_{race} (3, 244) = 3.43, p = .02). Tukey-Kramer post hot tests found that non-Hispanic Blacks reported being asleep 14 minutes fewer than those identified as Others (461.11 v 446.81 minutes, p = .03). No other statistically significant differences between race/ethnicity groups were noted.

Using their mean awake and sleep ABP readings, 48% (n = 120) of adolescents were categorized as being systolic blunted nocturnal decliners, 32% (n = 82) as being diastolic blunted nocturnal decliners, and 17% (n = 42) as having elevated blood pressure during sleep determined by their height, gender and age based on 90th percentile. Due to the literature noting racial and ethnic differences in nighttime blood pressure regulation, chi-square tests were run to determine if racial or ethnic identification was related to blunted decline and/or nighttime elevated blood pressure status (see Table 3 for frequencies of blunted nocturnal decline and nighttime elevated blood pressure by gender and racial/ethnic identification). Racial/ethnic identification was not statistically related to whether or not someone's systolic or diastolic blood pressure declined more than ten percent during sleep (Systolic Blunted Decline: X^2 (3) = 4.43, p = .22; Diastolic Blunted

Decline: X^2 (3) = 4.44, p = .23) or was elevated during sleep (X^2 (3) = 5.58, p = .14).There was no statistically significant difference in mean sleep systolic or diastolic blood pressure between boys and girls (Systolic: F(1, 250) = .69, p =.41; Diastolic: F(1,250) = 2.04, p = .15) or among racial/ethnic groups (Systolic: F(3, 248) = 1.96, p =.12; Diastolic: F(3, 248) = 2.11, p = .10) (see Table 4 for means by gender and race/ethnicity for nighttime blood pressure). In this sample, those classified as having daytime elevated blood pressure were not disproportionately classified as having blunted nocturnal blood pressure decline (Systolic Blunted Decline: X^2 (1) = .51,p= .47; Diastolic Blunted Decline: X^2 (1) = 0.32, p = .57). With an interest in understanding blood pressure regulation during sleep when compared to daytime levels, the average awake ABP was used as the intercept at t = 0 for each individual. There was, however, no statistically significant difference between the last awake ABP reading and mean awake ABP for systolic blood pressure (t(251) = -1.30, p = .20). The awake diastolic blood pressure was on average 3 mmHg higher than the last awake ABP reading for participants (t(251) = -4.35, p< .001). See Table 5 for the mean awake, last and sleep ABP readings.

Pattern of Nocturnal Blood Pressure

Systolic Blood Pressure (SBP)

The unconditional means model did not include predictors at either Level 1 or Level 2. This model provided the average systolic ambulatory blood pressure, 118 mmHg, recorded across participants throughout the observed time period (γ_{00}= 118.17, *se* = .81, p< .001; Level 1: Y_{ij} = 118.17). This model accounted for 43% of variance in ambulatory blood pressure that is attributable to inter-individual level (level 2) variables,

which was determined by dividing the measure of level 2 variance, by the sum of both level 1 and level 2 variances ($\zeta_{oi} / (\sigma_{\varepsilon} + \zeta_{oi}$). Modeling blood pressure across sleep in a quadratic manner, the subsequent quadratic model included minutes since sleep and a product of minutes since sleep as predictors at Level 1 [Level 1: Y_{ij}= 126.40 + -.0571(time –time went to $sleep_{cwc}$) + $.00007_i$ (time –time went to $sleep_{cwc})^2$]. For ease of interpretation, blood pressure changes are interpreted by hour. This aforementioned model indicated that the mean systolic blood pressure of individuals at t = 0 for was 126 mmHg. Referring to linear decline, the mean rate of instantaneous change in systolic blood pressure per hour during sleep was -3 mmHg (γ_{10}=-.05705, *se* = .00471, *p*< .001). Referring to quadratic change, the mean changing rate of instantaneous change in systolic blood pressure per hour during sleep of individuals was .25 mmHg (γ_{20}=.00007, *se* = 7.306E-6, *p*< .001). The inclusion of the linear and quadratic time components as level 1 predictors, $\gamma_{10}$and $\gamma_{20,}$ accounted for an additional 13% of the variance in intra-individual variance in systolic nocturnal blood pressure when compared directly to the unconditional means model, which a reduction in $\sigma_{\varepsilon,}$ from 188.58 to 164.31. Greater variability was noted in the variance of the intercept, ζ_{oo}, which increased from 142.92 to 153.46 and remained statistically significant.

To control for the impact of height of an individual's intercept on the rate of blood pressure decline during sleep, a product of the individual's sample centered daytime blood pressure and time since sleep was entered into the final model, $\gamma_{11,}$ at Level 2. This model indicated that the mean systolic blood pressure of individuals at t = 0 was 126 mmHg (γ_{00} =126.47, *se* = 1.0215, *p*< .001). Referring to linear decline, the mean rate of instantaneous change in systolic blood pressure per hour during sleep was -3 mmHg

(γ_{10}=-.05777, *se* = .00482, *p*< .001). Referring to quadratic change, the mean changing rate of instantaneous change in systolic blood pressure per hour during sleep was .27 mmHg (γ_{20}= .000073, *se* = 7.417E-6, *p*< .001). Analyses showed that for each 1 mmHg an individual was above the group mean averaged intercept, one's systolic blood pressure decreased .05 mmHg slower per hour during sleep (γ_{11}=.000829, *se* = .001551, *p*< .001). The magnitudes of the estimated intercept, instantaneous rate of changes, mean changing rate of instantaneous change and amount of residual variance in systolic (12%) blood pressure was attributable to within person differences were similar across models with and without the control variable. The residual variance in nighttime blood pressure, σ_{ε}, slightly increased from 164.31 to 165.25, and remained significant between these two models. The variability within the intercept, ζ_{oo}, decreased with the inclusion of a control variable, reducing from 153.46 to 148.87, and also remained significant. The variability of the instantaneous rates of changes, ζ_{11}, increased slightly from .0003 to .0005, with the inclusion of a control variable. The covariance between the intercept and instantaneous rate of change increased and became significant, -.0576 to -.1377. See Table 6 for full models details including fixed effects, random effects and model fit all models. See Graph 1 for a presentation of the sample average pattern of nocturnal blood pressure decline over the sample average 8 hours of sleep duration.

Diastolic Blood Pressure (DBP)

The unconditional means model for diastolic blood pressure did not include Level 2 or Level 2 predictors. This model provided the average diastolic ambulatory blood pressure, 63 mmHg, recorded across participants throughout the observed time period (γ_{00}= 62.97, *se* = .47, *p*< .001; Level 1: Y_{ij} = 62.97). This model accounted for 27% of

variance in ambulatory blood pressure that is attributable to inter-individual level (level 2) variables, which as determined by dividing the measure of level 2 variance, by the sum of both level 1 and level 2 variances ($\zeta_{oi} / (\sigma_{\varepsilon} + \zeta_{oi}$). Modeling blood pressure across sleep in a quadratic manner, the subsequent quadratic model included minutes since sleep and a product of minutes since sleep as predictors at Level 1 [Level 1: Y_{ij} = 67.58 + -.0381(time –time went to sleep$_{cwc}$) + .00005$_i$ (time –time went to sleep$_{cwc}$)^2]. For ease of interpretation, blood pressure changes are interpreted by hour. This model indicated that the mean diastolic blood pressure of individuals at t = 0 for was 65 mmHg. Referring to linear decline, the mean rate of instantaneous change in diastolic blood pressure per hour during sleep was -2 mmHg (γ_{10}=-.03810, *se* = .0036, *p*< .001). Referring to quadratic change, the mean changing rate of instantaneous change in diastolic blood pressure per hour during sleep of individuals was .19 mmHg (γ_{20} =.000054, *se* = 5.591E-6, *p*< .001). The inclusion of the linear and quadratic time components as level 1 predictors, γ_{10} and $\gamma_{20,}$ accounted for an additional 7% of the variance in intra-individual variance in diastolic nocturnal blood pressure when compared directly to the unconditional means model which a reduction in $\sigma_{\varepsilon,}$ from 114.20 to 105.75. Less variability was noted in variance of the intercept, ζ_{oo}, which decreased from 42.22 to 39.83 and remained statistically significant.

To control for the impact of height of an individual's intercept on the rate of blood pressure decline during sleep, a product of the individual's sample centered daytime blood pressure and time since sleep was entered into the final model, $\gamma_{11,}$ at Level 2. This model indicated that the mean blood pressure of individuals at t = 0 for diastolic blood pressure was 68 mmHg (γ_{00}=67.67, *se* = .6587, *p*< .001). Referring to linear

decline, the mean rate of instantaneous change in diastolic blood pressure per hour during sleep was -3 mmHg (γ_{10}=- .04217, *se* = .003762, *p*< .001). Referring to quadratic change, the mean changing rate of instantaneous change in diastolic blood pressure per hour during sleep was .21 mmHg respectively (γ_{20} = .000058, *se* = 5.77E-6, *p*< .001). Analyses showed that for each 1 mmHg an individual was above the group mean averaged intercept, one's diastolic blood pressure decreased .09 mmHg slower per hour during sleep (γ_{11}=.001499, *se* = .000016, *p*< .001). The magnitudes of the estimated intercept, instantaneous rate of changes, mean changing rate of instantaneous change and amount of residual variance in diastolic (7%) blood pressure was attributable to within person differences were similar across models with and without the control variable. The residual variance in nighttime blood pressure, σ_{ε}, slightly increased from 105.75 to 106.61, and remained significant between these two models. The variability within the intercept, ζ_{oo}, decreased with the inclusion of a control variable, reducing from 39.83 to 36.94 and also remained significant. The variability of the instantaneous rates of changes, ζ_{11}, increased slightly from .0001 to .0002, with the inclusion of a control variable. The covariance between the intercept and instantaneous rate of change increased and became significant, -.0063 to -.0507. See Table 6 for full models details including fixed effects, random effects and model fit all models. See Graph 1 for a presentation of the sample average pattern of nocturnal blood pressure decline over the sample average 8 hours of sleep duration.

The Inclusion of Demographic, Daytime and Clinical Variables

In order to see the impact of including demographic, daytime and clinical variables, these variables were incrementally added into Level 2 of the model.

Demographic variables were entered first. Next, daytime variables were entered into a model including demographic variables. Last, clinical variables were entered into the model. Only the final model containing demographic, daytime and clinical variables are discussed in detail with this results section as observed demographic and clinical effects remained largely significant with the addition of other variables (Refer to Table 7 for details of fixed effects, random effects and model fit with the subsequent addition of demographic, daytime and clinical variables). Comparison of random effects was done using variances and co-variances from the model without inter-individual predictors. For systolic blood pressure, the residual variance in nighttime blood pressure, σ_{ε}, slightly increased from 165.25 to 165.55, with the inclusion of inter-individual variables and remained significant between these two models. For diastolic blood pressure, the residual variance in nighttime blood pressure, σ_{ε}, decreased from 106.61 to 105.24, with the inclusion of inter-individuals variables and remained significant between these two models. See Graphs 2-6 for representations of the expected differences in intercepts and instantaneous rates of nocturnal blood pressure decline for statistically significant demographic, daytime and clinical variables over an eight hour sleep period. Refer to Appendix 2 for a more detailed summary of the equations used in the model building process.

Intercepts

Systolic Blood Pressure

In the final model including demographic, daytime and clinical variables as Level 2 predictors, there were some effects on intercepts due to inter-individual differences.

Identifying as non-Hispanic Black as opposed to Hispanic was related to a higher intercept at t = 0 (γ_{03} = 8.99, *se* = 2.3705, $p < .001$), suggesting non-Hispanic Black youth in this sample had a 9 mmHg higher daytime systolic blood pressure when compared to Hispanic White youth. The number of times an individual reported stress was also significantly related to a higher blood pressure reading at t = 0 (γ_{09}= 0.60, *se* = 0.25, $p<.05$), which suggests daytime blood pressure increased almost 1 mmHg for each time an adolescent reported being stressed during the day. Regarding clinical variables, family history of hypertension was related to a higher systolic blood pressure at t = 0 (γ_{014}=5.66, *se* = 2.49, $p < .05$), meaning those identified as having a positive family history of hypertension had a 6 mmHg higher daytime ambulatory blood pressure when compared to those without a family history of hypertension. It should be noted that inclusion of demographic, daytime and clinical variables explained an additional 14% in the inter-individual variance in the systolic intercept when compared to the model containing solely time. The variability within the intercept, ζ_{oo}, decreased with the inclusion of level 2 variables from 148.87 to 127.33, and also remained significant.

Diastolic Blood Pressure

In the final model including demographic, daytime and clinical variables as Level 2 predictors, there were some effects on intercepts due to inter-individual differences. Identifying as non-Hispanic White as opposed to Hispanic was related to lower diastolic blood pressure at t = 0 (γ_{02}=-4.46, *se* = 1.85, $p < .05$), while identifying as non-Hispanic Black as opposed to Hispanic was related to a higher intercept at t = 0 (γ_{03}=5.18, *se* = 1.45, $p < .001$). Those identifying as non-Hispanic White had a 4 mmHg lower daytime blood pressure and those identifying as non-Hispanic Black had a 5 mmHg higher

daytime blood pressure when compared to Hispanic Whites in this sample. The number of times an individual reported stress was significantly related to a higher blood pressure reading at t = 0 (γ_{09}= 0.35, *se* = 0.15, p<.05), suggesting less than one mmHg increase in average daytime diastolic blood pressure for each reported period of stress. Regarding clinical variables, meeting criteria for the metabolic syndrome was associated with a higher expected diastolic blood pressure of almost 4 mmHg at t = 0 (γ_{012} =3.7954, *se* =1.8595, p< .05). It should be noted that inclusion of demographic, daytime and clinical variables explained an additional 21% of the variance in the diastolic intercept when compared to the model containing solely time as predictors of blood pressure. The variability within the intercept, ζ_{oo}, decreased with the inclusion of level 2 variables from 36.94 to 29.20, and also remained significant.

Instantaneous Rates of Change

Systolic Blood Pressure

On average, those identifying as non-Hispanic Black has faster instantaneous rates of decline per minute during sleep when compared to those identifying as Hispanic Whites, for an average of an average of 1 mmHg per hour of sleep (γ_{14} = -.0157, *se* = .0060, p< .01). Greater stress was also associated with a faster instantaneous rate of blood pressure decline, for an average of .24 mmHg faster decline per hour (γ_{110} = -0.0013, *se* = 0.0006, p< .05). A positive family history of hypertension was also related to a faster instantaneous rate of decline in systolic blood pressure during sleep, for an average of .8 mmHg per hour (γ_{115} = -0.0133, *se* = 0.0061, p< .05). It was observed that following the inclusion of clinical variables, identifying as a girl was no longer related to the

instantaneous rate of change in systolic blood pressure (see Table 7 for these demographic effects on the instantaneous rate of change during sleep before the inclusion of clinical variables). The inclusion of demographic, daytime and clinical variables explained an additional 9% in the inter-individual variance in the instantaneous rate of change of systolic blood pressure compared to the multi-level model including time as the only predictor of blood pressure decline. The variability of the instantaneous rates of changes, ζ_{11}, increased slightly from .0004 to .0005, with the inclusion of Level 2 variables. The covariance between the intercept and instantaneous rate of change decreased from -0.1377 to -0.1081, and remained statistically significant.

Diastolic Blood Pressure

Identifying as non-Hispanic White was associated with a slower instantaneous rate of change per minute of diastolic blood pressure during sleep when compared to Hispanics, for an average of .62 mmHg slower decline per hour (γ_{14} = 0.0104, *se* = 0.0052, $p < .05$). On average, those identifying as non-Hispanic Black declined faster per minute when compared to those identifying as Hispanic, for an average of .63 mmHg faster decline per hour of sleep (γ_{14} = -.0105, se = .0043, $p < .05$). Greater stress was also associated with faster instantaneous rate- of blood pressure decline per minute, for an average of a .07 mmHg faster decline per hour ($\gamma_{11\,0}$ = -0.0011, *se* = 0.0005, $p < .05$). The inclusion of demographic, daytime and clinical variables explained an additional 12% of the variance in the instantaneous rate of change of diastolic blood pressure. The variability of the instantaneous rates of changes, ζ_{11}, increased slightly from .00019 to .0002, with the inclusion of Level 2 variables. The covariance between the intercept and

instantaneous rate of change decreased from -0.5073 to -0.0372, and remained statistically significant.

Inclusion of Sleep Variables in Sample Subset

Since the analyses examining the role of sleep quality data only included a subset of the full sample, analyses were concluded to determine whether the models specified earlier were comparable in the reduced sample. Analyses suggested that while the statistical significance of some variables changed within this sample, the direction and magnitude of the fixed effects was comparable across samples. Closer observation of the standard errors related to the fixed effects that changed suggested that greater standard errors in this smaller sample were responsible for this finding. The final model accounted for an additional 1% and 11% in the inter-individual variance in the rate of instantaneous change of systolic and diastolic blood pressure respectively as compared to the model without sleep quality related variables. For systolic blood pressure, the residual variance in nighttime blood pressure, σ_{ε}, slightly decreased from 200.53 to 200.79, and remained significant between these two models. For diastolic blood pressure, the residual variance in nighttime blood pressure, σ_{ε}, increased from 120.47 to 120.70, and remained significant between these two models. See Table 8 for full models details including fixed effects, random effects and model fit for this model including sleep variables in the reduced sample.

Intercepts

Systolic Blood Pressure

Identifying as non-Hispanic Black as opposed to Hispanic was related to a higher systolic blood pressure at t = 0 (γ_{03} = 11.92, *se* = 4.13, $p < .01$), suggesting non-Hispanic Black youth in this sample had a 12 mmHg higher daytime systolic blood pressure when compared to Hispanic White youth. The variability within the intercept, ζ_{oo}, increased with the inclusion of level 2 variables from 187.83 to 189.05, and also remained significant.

Diastolic Blood Pressure

Identifying as Other as opposed to Hispanic was related to a higher diastolic blood pressure at t = 0, (γ_{04} = 7.92, *se* = 3.85, $p < .05$), suggesting youth identifying as Other in this sample had a 8 mmHg higher daytime diastolic blood pressure when compared to Hispanic White youth. The variability within the intercept, ζ_{oo}, increased with the inclusion of level 2 variables from 36.86 to 36.96, and also remained significant.

Instantaneous Rates of Change

Systolic Blood Pressure

None of the sleep variables was significantly related to rate of instantaneous change in systolic blood pressure per minute during sleep. A positive family history of hypertension was related to a faster instantaneous rate of decline in systolic blood pressure during sleep (γ_{115}=-0.0215, *se* = 0.0105, $p < .05$), suggesting on average of a 1 mmHg faster decline per hour. The variability of the instantaneous rates of changes, ζ_{11},

decreased from .0000645 to .000600, with the inclusion of Level 2 variables. The covariance between the intercept and instantaneous rate of change decreased from -0.1793 to -0.1725, and remained statistically insignificant.

Diastolic Blood Pressure

None of the sleep variables was significantly related to rate of instantaneous change in diastolic blood pressure per minute during sleep. Again, a positive family history of hypertension was related to a faster instantaneous rate of decline in diastolic blood pressure during sleep (γ_{115} =-0.0148, *se* = 0.0069, $p < .05$), suggesting an average of .89 mmHg faster decline per hour. More fixed effects on the instantaneous rates of change were observed in diastolic blood pressure when compared to systolic blood pressure. Identifying as non-Hispanic White was associated with a slower instantaneous rate of change per minute of diastolic blood pressure during sleep when compared to Hispanics (γ_{14} = .0231, *se* = .0106, $p < .05$), while identifying as Other and attending school were both associated with faster decline per minute (γ_{14} = -.0301, *se* = .0116, $p < .01$; γ_{17} = -.0129, *se* = .0056, $p < .05$). On average, when compared to Hispanic Whites, identifying as NHW was associated with a 1.3 mmHg slower decline per hour of sleep and identifying as NHB was associated with an almost 2 mmHg faster decline per hour of sleep. Attending school was associated with an almost 1 mmHg faster decline per hour during sleep. See Graph 6 for representations of the expected differences in intercepts and instantaneous rates of nocturnal blood pressure decline for statistically significant demographic, daytime and clinical variables, unique to this reduced sample, over an eight hour sleep period. The variability of the instantaneous rates of changes, ζ_{11}, decreased from .000205 to .000182, with the inclusion of Level 2 variables. The covariance

between the intercept and instantaneous rate of change decreased from -0.0537 to -0.0494, and remained statistically insignificant.

CHAPTER 4: DISCUSSION

This study explored the complex nature of predictors of nocturnal blood pressure decline in adolescents. While research hypotheses anticipated differences in rate of blood pressure decline due to demographic, daytime and clinical factors, the direction of most observed differences was contrary to hypotheses. In this sample, faster rate of decline was observed in those identifying as non-Hispanic Black or Other, having a family history of hypertension and reporting higher stress during the day; all predictors that were associated with higher blood pressure intercept at the beginning of sleep. Also, slower rate of decline in girls and those identifying as non-Hispanic White were observed within the sample; identifications associated with lower blood pressure intercept at the beginning of sleep. It appeared that while particular racial/ethnic identifications, gender, family history of hypertension and greater stress were related to differing average levels of daytime ABP as expected, sleep appeared to act as a recovery period for these differences. In addition, these observed demographic findings were fairly stable with the inclusion of additional variables hypothesized to reduce these effects such as daytime activity or clinical variables (e.g., daytime stress, obesity, metabolic syndrome, and/or family history of hypertension). With the inclusion of sleep quality variables, however, only family history continued to be related to systolic nocturnal blood pressure regulation. However, identifying as Other or non-Hispanic White, attending school, and family history were related to rates of decline in diastolic blood pressure during sleep. These variables' significant effects on the pattern of nocturnal decline as well as their changes in significance suggested what combinations of variables were most important in

explaining differences in systolic and diastolic nocturnal blood pressure regulation among youth.

Modeling the Pattern of Nocturnal Decline

By successfully modeling the nocturnal blood pressure decline based on time using daytime average ABP levels as the intercept, this study showed the plausibility of modeling nocturnal blood pressure decline in such a manner. This was a useful method to explore patterns of nocturnal blood pressure regulation for two reasons. First, with research emphasizing the importance of understanding nocturnal decline, the ability to capture both awake and sleep ABP in one model was necessary. Second, the inclusion of time in modeling the decline of nocturnal blood pressure accounted for an additional 7-13% of the variance within an individual's blood pressure during sleep, showing the added value in modeling the decline over time. Most studies examining nighttime blood pressure regulation in youth predicted the probability of having blunted nocturnal decline/nighttime hypertension or mean levels of nighttime blood pressure (Profant & Dimsdale, 1999). The use of summary methods has been shown to introduce error into measurement, especially when the number of observations varies across individuals, a typical characteristic of ABPM (Kamarck, Schwarts, Janicki, Shiffman,& Raynor, 2003).By more directly modeling the decline in blood pressure during sleep as compared to daytime levels within individuals, this study departed from the typical assumption of different group patterns in nighttime blood pressure based strictly on mean levels (Profant & Dimsdale, 1999). Next, the use of actual reported sleep and awake times allowed for a more accurate picture of individual blood pressure regulation during sleep. Within this sample, there was great individual variability in the times adolescents reported going to

sleep and waking up as well as the amount of time spent asleep. If standard times for awake or sleep were used for all participants, many ABP measurements would have been wrongly classified. This study took a more direct and accurate approach to understanding the phenomena of nocturnal blood pressure decline in youth.

Importance of the Sleep as a Recovery Period

Sleep has been observed as a period during which blood pressure should decrease compared to daytime levels, resulting from a decrease in activity as well as change in posture (Carrington et al., 2005). While differences in daytime blood pressure were observed due to gender, race/ethnicity, stress and family history, differences in blood pressure were eliminated within 8 to 10 hours of sleep. While eight hours of sleep was sufficient to eliminate most observed differences during the day, this sample's average amount of sleep was below the recommended 9-10 hours for adolescents (Millman, 2005). Surveys of sleep in adolescents suggest that sleep deprivation is common in adolescents (Dahl, 2008; Kotagal &Pianosi, 2006; Roberts, Roberts & Duong, 2009). Most research on cardiovascular risk and disturbed sleep patterns in youth has been done in clinical samples with sleep apnea, respiratory issues or renal dysfunction (Daniels, 2001; Ievers-Landis & Redline, 2007; Triggle, 2008). A review of the literature showed most research on the role of sleep deprivation in youth with severe medical problems, not just cardiovascular at risk profiles.

Predictors of Intercepts and Rates of Nocturnal Decline

With a sample of youth who were at risk for cardiovascular disease (i.e., identified as having elevated blood pressure, higher rates of obesity, poor fitness,

minority), differences reflecting heightened risk (i.e., poorer nocturnal decline) were expected. This sample consisted of a large portion of adolescents with primary hypertension, as participants were excluded for having secondary hypertension. Research has suggested that blunted nocturnal decline is a marker of secondary, not primary hypertension, (Seeman, Palyzoma, Dusek & Janda, 2005) which was consistent with the observation in this sample that those with elevated blood pressure were not more likely to be blunted decliners. While attempting to identify what differences existed among a diverse sample of youth, results highlighted the need to look for the causes of secondary hypertension in those with nocturnal dip and sustained nighttime hypertension (Seeman et al., 2005). The recovery-like effect of sleep observed in this sample suggested that, while reflecting elevated cardiovascular risk, the impact of heightened daytime ABP levels could be reduced with sufficient sleep. While it appears that demographic, daytime and clinical variables may alert health care professionals and researchers to those at risk for elevated blood pressure; the risk of poor nighttime blood pressure regulation could not be identified using the same factors.

The recovery-like findings during sleep related to race/ethnicity, gender, stress and family history of hypertension are novel. After eight hours of reported sleep (or the average of amount of time adolescents in this sample reporting being asleep), differences in rate of decline due to identifying as non-Hispanic White/non-Hispanic Black compared to Hispanic and having a positive family history of hypertension reached the average differences observed at the intercept related to these variables. This observation suggested these differences were eliminated after 8 hours of sleep. While after 9-10 hours of sleep (or the recommended length of sleep for adolescents), the blood pressure

differences due to stress were within 1 mmHg of the average differences observed at the intercept. One might have expected that these differences were simply products of how nighttime blood pressure regulation was defined in this sample (i.e., modeled using time with daytime blood pressure as the intercept). The overwhelming amount of non-significant mean sleep blood pressure differences by gender and race/ethnicity in this sample suggested that observed differences in rates of instantaneous change during sleep were more likely to truly reflect the recovery-like properties of sleep in this elevated risk sample from known risk factors of poor nighttime blood pressure regulation.

Demographics

While it was possible to interpret the slower decline in girls as evidence of blunted decline, this observed difference in rate of decline appeared to act as a recovery effect for the higher daytime blood pressure observed in boys in the sample. By the end of the average of amount of time adolescents in this sample reported being asleep, this gender difference in systolic rate of decline during sleep reached the average difference observed at the intercept. Measured in adolescence, it appeared that gender differences in daytime blood pressure were still being corrected during sleep, meaning that differences in daytime systolic blood pressure regulation have not yet generalized to nighttime blood pressure. The finding within this study showed that observed gender differences were no longer significantly related to the rate of systolic blood pressure decline after the inclusion of clinical variables during sleep. This study's finding was consistent with research supporting that differences in gender and systolic blood pressure regulation are observed in adult population (Pimenta & Oparil, 2008; Routledge & McFetridge-Durdle, 2007). Researchers had found that clinical variables such as BMI did not reduce gender

differences in mean daytime blood pressure levels measured longitudinally, not individual blood pressure regulation over the night course (Wang et al., 2006). Novel findings from this study suggest that within primary elevated blood pressure youth, gender differences in blood pressure regulation at this age might have been better accounted for by differences in other factors impacting nocturnal blood pressure regulation.

A number of interesting racial/ethnic differences were observed. While it was possible to interpret the slower decline in non-Hispanic Whites as evidence of blunted decline, this observed expected difference in rate of decline appeared to act as a recovery effect for the higher daytime blood pressure observed in Hispanics compared to non-Hispanic Whites. By the end of the average of amount of time adolescents in this sample reported being asleep, this racial/ethnic difference in diastolic rate of decline during sleep reached the average difference observed at the intercept. Contrary to most studies, this study did not support a higher prevalence of blunted nocturnal blood pressure decline in minority youth with elevated cardiovascular risk (Minor, Wofford, & Jones, 2008; Routledge & McFetridge-Durdle, 2007). While previous research suggested poorer nighttime blood pressure regulation in non-Hispanic Black youth, researchers have noted that these group differences in blood pressure levels increase as cohorts age (Wang et al., 2006).Most racial and ethnic differences in rate of decline during sleep reached the average differences observed at the intercept after the average amount of time adolescents in this sample reporting being asleep. However, for non-Hispanic Blacks, diastolic blood pressure differences did not fully recover until 9 hours of sleep. These observations have suggested that sleep served as a recovery period for racial/ethnic differences in daytime

blood pressure levels within these adolescents and highlighted the importance of how blood pressure regulation is measured. With methods that better account for the variability among and within individuals, this study's findings showed that blunted nocturnal decline was not related to racial/ethnic group membership in a sample of cardiovascular at risk youth. Due to the novel nature of using Hispanics as the reference group in blood pressure comparisons, it should be noted that similar effect sizes related to identifying as Other/non-Hispanic Black were noted when non-Hispanic White was used as the reference group in preliminary analyses. Also, while the research literature strongly supported a higher prevalence of non-dipping blood pressure in those diagnosed with essential hypertension who are non-Hispanic Black, there has been much less literature on youth from racial/ethnic groups such as those identifying as Hispanic/Latino, Asian-American, Native Americans or multi-racial (Routledge & McFetridge-Durdle, 2007). This study explored nighttime blood pressure regulation in a sample that reflected greater diversity within Hispanics/Latinos sampled. This study also did not exclude less frequently studied racial/ethnic groups such as Asian Americans, Native Americans, or Caribbean Blacks. In addition to findings that racial/ethnic identification was not yet related to increased risk for blunted nocturnal decline in adolescents with elevated cardiovascular risk levels, this study highlighted the need to consider variability within and between individuals when exploring nocturnal blood pressure decline and provided information on racial/ethnic groups often excluded from the typical Black and White blood pressure comparisons.

The measure of SES in this sample was not significantly related to the rate of blood pressure decline during sleep. In this study, the highest year of education for the

identified head of household was used as an individual level measure of SES. In studies that have observed SES' moderation effects in nighttime blood pressure regulation in racially diverse adolescents, measures of SES were used that reflected neighborhood levels of SES (i.e. neighborhood income, low/high SES neighborhood). For example, blunting effects of unfair treatment and anger on nocturnal decline were only observed in non-Hispanic Black adolescents from low SES neighborhoods (Beatty & Matthews, 2009; McGrath, Matthews,& Grady, 2006). In addition to how SES was measured, the diverse representation of ethnic/racial groups in the sample may explain why SES was not related to ABP in this sample. Previous research has shown SES not be related to blood pressure levels in diverse Hispanic youth (i.e., Puerto Rican, Cuban and Mexican American) (Loria, Crespo, & Burt, 1996). This current study supported the findings that individual level measured SES in racially/ethnically diverse adolescents was not significantly predictive of nighttime blood pressure regulation.

Daytime Variables

Consistent with the literature in adolescents, more reported stress was related to higher daytime ABP (Brady & Matthews, 2006). In the present study, attending school and reporting more stress were related to a faster decline during sleep. The effect of school attendance was only seen in the reduced sample, which contained higher prevalence of youth with elevated blood pressure. That difference in findings between the full and reduced sample suggested the possibility of different predictors of nocturnal decline for elevated and non-elevated youth. While the significance of school attendance on nocturnal decline was consistent with findings that work stress in adults with essential hypertension is related to nocturnal decline, school attendance was associated with a

faster rate of decline during sleep, not a blunted decline as often observed in adults (Routledge & McFetridge-Durdl, 2007). Similar to observed demographic differences, by the end of eight hours of reported sleep (or the average of amount of time adolescents in this sample reporting being asleep), differences related to increased stressor school attendance in the rate of decline during sleep were within 1 mmHg of the average differences observed at the intercept. Again, it appeared that sleep served as a recovery period for differences in daytime blood pressure levels due to stress or school attendance within these adolescents. This observation was consistent with findings that racial and ethnically diverse youth who displayed higher daytime blood pressure related to where they lived showed greater systolic and diastolic nocturnal decline (Harshfield, Wilson, Treiber,& Alpert, 2002). Overall, these current findings suggested that while addressing stress or other daytime activities should be an important target for understanding elevated daytime blood pressure, these variables' impact on nocturnal blood pressure did not appear to be detrimental given sufficient hours of sleep.

Neither tobacco nor caffeine use was related to higher daytime blood pressure or rates of decline during sleep. Tobacco and caffeine have been routinely used as control variables since use of these substances is known to be associated to daytime or 24-hour blood pressure levels (Pilote et al., 2007; Savoca et al., 2005; Urbina et al., 2008). Not finding a relationship between substance use and daytime blood pressure may be due to study specific assessment as well as the nature of the relationships between these substances and blood pressure (James, 2004; Mort & Kruse, 2008; Leone, 2011). Regarding assessment, previous studies finding relationships used more detailed assessments of substance use (i.e., the specific amount of caffeine in foods consumed)

(Savoca et al., 2005). The more consistent relationships between caffeine and blood pressure have been found in laboratory settings (Seifert, Schaechter, Hershorin & Lipshultz, 2011; Temple, Dewey & Briatico, 2010), which is more reflective of the immediate effects of caffeine of body. Laboratory studies, though likely a product of increased internal validity, have be increasing the likelihood of finding effects of caffeine on blood pressure by their design and overestimating the impact caffeine, alone, has on cardiovascular health and risk factors (Myers, 2004). In addition, this study's measure of caffeine consumption was not intended to be a measure of acute or average use, rather a means to control for the impact of consumption of caffeine only on the day of ABP assessment. Related, youth were strongly discouraged from caffeine consumption on the day of assessment within this study. Results of this study can be interpreted within the context of the controlling for, not explaining, the role of caffeine in nighttime blood pressure regulation in youth.

In addition, cardiovascular health related concerns about caffeine and tobacco may indeed rest in the possible greater negative consequences of substance intake in cardiac at risk populations and the negative impact of increased caffeine use on other health behaviors often co-morbid with cardiac issues such as unhealthy eating choices and greater intake of sugar and calorie heavy sodas (Bonita, Mandarano, Shuta & Vinson, 2007; Savoca, Evans, Wilson, Harshfield & Ludwig, 2004; Seifert, Schaechter, Hershorin & Lipshultz, 2011;). Also, due to the relationship between caffeine and tobacco use and negative outcomes in those at elevated cardiovascular risk, caffeine and tobacco consumption should continue to be monitored and assessed in elevated risk populations (James 2004; Campbell et al., 2008). They have been identified as important concepts to

include when understanding this cardiovascular at risk group, regardless of their measurable impact on blood pressure regulation measured once. Due to the relationship between substance use and sleep hygiene, the inclusion of such variables may have more clinical use in promoting healthy sleep than in differentiating nocturnal decline patterns in youth (Kotagal & Pianosi, 2006). While no significant statically relationships were found directly between substance use and blood pressure in this sample, the elevated cardiovascular risk level of our sample made the inclusion of these variables essential to interpreting results and excluding other potential causes of differences among the sample.

Clinical Variables

With many participants already having elevated blood pressure, there may have been little variability in the impact of clinical risk factors related to elevated blood pressure. The finding that poorer nighttime blood pressure regulation was not related to obesity was consistent with findings from Lubre and colleagues (2001), who observed similar decreases in blood pressure during sleep in both obese and non-obese youth. Often, the observed incremental relationship between obesity and blood pressure has measured elevated blood pressure risk, supporting obesity as a target for primary prevention of developing additional cardiovascular disease risk factors (i.e. elevated blood pressure during the day or night), not necessarily risk stratification for youth already showing elevated blood pressure (Din-Dziethamet al., 2007; Goran, Ball, & Crus, 2003; Gilardiniet al., 2008; Pons et al., 2008; Sorof & Daniel, 2002). With the adult cutoff for hypertension being used as a criterion for the metabolic syndrome, the relationship between higher diastolic daytime levels and the presence of the metabolic syndrome could simply have been a reflection of the definition used in analyses. In this

sample however, metabolic syndrome was not related to the rate of decline in blood pressure during sleep, adding to the literature that the metabolic syndrome might be related to daytime and nighttime blood pressure regulation differently (Ben-Dov & Bursztyn, 2009). Most research looking at the relationship between nighttime blood pressure regulation and the presence of the metabolic syndrome, not just its individual components, has been done in adults as literature in youth has focused on the individual components of the metabolic syndrome when exploring differences in nocturnal blood pressure (Aguilar, Ostrow, De Luca,& Suarez, 2010; Ben-Dov & Bursztyn, 2009; Giladrini et al., 2007; Holl et al., 1999; Lubre et al. 2008; Pons et al. 2008). This study observed that the metabolic syndrome and obesity were not related to nocturnal blood pressure decline in adolescents with a high proportion of elevated cardiovascular risk due to obesity and elevated blood pressure.

Family history of hypertension has been widely accepted as a risk factor for elevated blood pressure in youth (Morgenstern & Sinaiko, 2008; Wang et al., 2006). Similar to effects of demographic factors, the faster decline for those with a positive family history of hypertension appeared to suggest recovery from higher daytime values in youth with this risk factor. This observation was consistent with findings that in youth identified as having a positive family history of essential hypertension those who displayed higher daytime blood pressure had greater systolic and diastolic nocturnal decline at night (Harshfield et al., 2002). The findings in this study gave additional support to the observation that while a risk factor for higher daytime levels, in adolescents, family history was not necessarily a marker for blunted nocturnal decline.

Fitness was not found to be related to measures of blood pressure in this sample, likely due to the poor average fitness level characterizing this clinical sample. The unique relationship between fitness and blood pressure, even after controlling for anthropomorphic measures, has been shown in mostly non-clinical samples with a range of fitness levels (Fraser, Phillips, & Harris, 1983; Nielsen & Andersen, 2003). Studies that have looked at the predictive value of fitness on later cardiovascular health have found weaker relationships for adolescent fitness levels, but rather seem to identify changes in fitness levels as predict of later cardiovascular health (Hasselstrom, Hansen, Froberg & Andersen, 2002). The inclusion of fitness as a predictor of blood pressure regulation was essential to interpreting results, but may not be ideal for identifying fitness's unique contributions given likely restriction of range issues in this sample.

Sleep Variables

The included sleep quality variables did not significantly predict differences in blood pressure regulation during sleep, but did improve the overall explanation of the decline in blood pressure during sleep. Studies that have found a significant impact of sleep quality on blood pressure in youth have looked at cardiovascular risk as measured by becoming hypertensive or being pre-hypertensive (measured by clinical and/or non-sleep ABPs), not the individual picture of nighttime blood pressure regulation (Javaheri, Strofer-Isser, Rosen,& Redline, 2008).Non-significant findings in this current study were consistent with the observation that sleep quality and nighttime activity have allowed for a more accurate picture of nighttime blood pressure regulation, but did not always significantly influence nocturnal blood pressure regulation as disturbances in sleep appear to have minimal effects on overall nighttime blood pressure regulation (Carrington et al.,

2005; Routledge & McFetridge-Durdl, 2007). For example, studies have found that the effects of sleep disturbances related to urination and other types of awakening during sleep were reduced when more accurate sleep patterns were assessed (Routledge & McFetridge-Durdl, 2007). The current study highlighted the importance of assessing sleep quality for a more accurate assessment of nighttime blood pressure regulation.

Differences in Systolic and Diastolic Blood Pressure Regulation

While identifying as non-Hispanic Black and stress were related to both systolic and diastolic ABP regulation, family history and identifying as non-Hispanic White acted differently. Family history was only related to systolic blood pressure regulation during sleep. Racial/ethnic differences in nighttime blood pressure regulation were noted between non-Hispanic White and Hispanic participants for diastolic blood pressure only. Since research stated that systolic and diastolic blood pressure relate differently to cardiac dysfunction (Adams, 2008), these differences in regulation could have suggested differential risk factors in systolic and diastolic dysfunction. Systolic dysfunction has been related to decreased cardiac output as well as higher ventricular filling pressure leading to inadequate blood supply to organs, potential impairment in sodium excretion and pulmonary complications (Adams, 2008). Research has shown diastolic dysfunction has been observed as related to renal dysfunction and vascular abnormalities (Adams, 2008). Consequently, family history may be related to nighttime blood pressure regulation due to cardiac output, sodium excretion and the pulmonary system, while identifying as non Hispanic White as opposed to Hispanic may be more associated with vascular function in adolescents. While mostly recovery like sleep properties were observed in this current study, this sample consisted of a portion of youth with blunted

nocturnal decline and nighttime elevated blood pressure, suggesting the identification of demographic and clinical variables significantly and uniquely related to nocturnal decline. Interestingly, while those youth meeting criteria for the metabolic syndrome had higher daytime diastolic blood pressure, no significant effect of the metabolic syndrome on the rate of diastolic blood pressure decline was noted. This effect was the only higher intercept effect not accompanied by a corresponding faster rate of decline during sleep. This finding could point towards a relationship between presence of the metabolic syndrome in youth and diastolic dysfunction and is consistent with the relationship between diastolic dysfunction and renal issues.

Limitations

Although this study added to the literature that aims to understand nighttime blood pressure regulation in youth, there were some limitations. The use of self-reported sleep times was an improvement over using standard researcher set times for sleep and awakening, but self-report sleep times were still prone to error. In addition, because some participants who did not report approximate sleep and awake times, some individual's times were estimated from summary data about their typical sleep patterns over one week. It should also be noted that due to the use of data from three different cohorts, different ABP diary cards were given to participants leading to the availability of detailed sleep quality and environment data in only 41% of the full sample. Regarding how demographic variables were captured, a more comprehensive or multi-dimensional assessment of racial/ethnic identification and SES was warranted. With racial/ethnic differences in the intercepts remaining after the inclusion of daytime, clinical and sleep variables, it appeared that additional variance in racial and ethnic group membership

could be explained. The inclusion of other psychosocial variables would help reduce differences unexplained by the included demographic, daytime and clinical variables. Specific to experiences related to group membership/identification and the study of cardiovascular blood pressure responses, some researchers have begun to include measures of racial identity that have explained additional variance (Beatty & Matthews, 2009; Clark, Cobb, Hopkins, & Smith, 2006). In addition, the measure of SES used in this study was entered at Level 2. It may have been more informative to use a Level 3 measure of SES that took neighborhood, school and environmental level variables into account that would not be captured by parental level of education, especially given the racial/ethnic diversity of this sample. Also, in the assessment of stress, the type of stressors was not reported, halting further explaining of how and what stress was related to blood pressure regulation in youth. Regarding substance use, dichotomous assessment of use lacked the variability in use possibly needed to see impact on blood pressure regulation. While the variables included did address proposed research questions, there was room for improved assessment of the constructs measured in this sample.

Future Directions

Future directions point towards a more in-depth assessment of nocturnal blood pressure decline in adolescents as well as its relationship to other cardiovascular outcomes. Researchers have used polysomnography to determine sleep periods more accurately than using self-reported sleep times, a step that would reduce the error introduced by self-report data. A more comprehensive approach to assessing race/ethnicity as well as SES should be taken, which would include psychosocial or societal level measures that more fully capture the constructs of race/ethnicity and SES.

For race/ethnicity, including measures related to perception, coping, discrimination, commitment to, exploration of or constitution of one's racial/ethnic identification have been areas identified that accomplish this goal (Phinney & Ong, 2007; Scottman, Sellers, & Nyuyen, 2008). For SES, the inclusion of neighborhood level measures of SES and environment should occur. Also, including measures of chronic stress as well as the type of stressors encountered by individuals would improve the ability to explore the relationship between stress and blood pressure regulation. The use of ecological momentary assessment could be useful in obtaining a fuller picture of how stress, psychosocial variables or substance use were experienced during the day and leading to a more accurate picture of how these constructs relate to blood pressure regulation (Shiffman, Stone, & Hufford, 2008; Stone & Shiffman, 1994). Since ABPM often involved diary use, the use of ecological momentary assessment of psychological constructs and substance use would naturally fit into existing protocols.

With more evidence for a relationship between day-night blood pressure differences and LVM in youth needed (Fagard, Staessen, & Thijs, 1997; Flynn, 2010), the next step for a project exploring nocturnal decline in youth would be to relate observations to that marker of cardiovascular disease and dysfunction. Some research has suggested the nocturnal blood pressures as well as nocturnal blood decline are better predictors of target organ damage than daytime ABP, but findings are not consistent or well established in youth (Fargard et al., 1995; Flynn, 2010). Leading towards greater generalizability of research findings, there would be added value in exploring these research questions in a sample that better reflects the diversity of youth in the United States who need cardiovascular intervention (Brady, Fivush, Flynn, & Parekh, 2008;

Stabouli et al., 2009).Determining predictors of target organ damage in a diverse sample at elevated cardiovascular risk who still appear to recover from elevated blood pressure levels during the day could be useful in identifying what aspects of ABP regulation are related to clinical cardiovascular outcomes. This type of information would greatly inform clinicians and researchers aiming to improve cardiovascular health in youth.

Clinical Implications

The clinical implications of this study would suggest the importance of sleep within adolescent populations at risk for CVD for whom the reduction of blunted nocturnal decline and prevention of target organ damage is targeted. Due to the restorative nature of sleep in youth with elevated blood pressure, the findings of this study suggest that health care providers concerned with cardiovascular risk begin to assess sleep patterns and promote sleep hygiene within at risk populations. While there is additional research necessary to firmly establish the relationship between nocturnal decline and LVM in youth, the current study strongly supported the need for adequate sleep to diminish the impact of elevated systolic and diastolic blood pressure in youth.

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Table 1. *Descriptive Percentages of the Full and Subset with Detailed Sleep Quality Data Samples*

	Sample	
	Full	Subset with Sleep Quality Data
Boys	72	74
Hispanic	48	52
Non-Hispanic White	13	8
Non-Hispanic Black	31	31
Other	8	9
Attending School	48	45
Caffeine Use	84	85
Tobacco Use	6	3
Metabolic Syndrome	17	14
Positive Family history of Hypertension	80	78
Elevated Blood Pressure	75	98
Systolic Blunted nocturnal decline	48	44
Diastolic Blunted nocturnal decline	32	37
Nighttime elevated blood pressure	17	21

Note: Full Sample N = 252. Subset with Sleep Quality Data N = 101

Table 2. *Means of the Full Sample and Subset with Detailed Sleep Quality Data*

	Sample	
	Full	Subset with Sleep Quality Data
Highest Year of Education of Household Head	14.3 (2.9)	14.1 (3.2)
Number of times stress reported during the day	2.5 (4.0)	2.9 (4.1)
Waist circumference (inches)	36.5 (6.9)	35.5 (6.4)
BMI (kg/m^2)	29.0 (7.2)	27.7(6.6)
Fitness (ml/kg/min)	36.4 (10.4)	37.7 (9.1)
SBP during sleep (mmHg)	116.5 (13.7)	116.6 (15.2)
DBP during sleep (mmHg)	61.3 (7.8)	61.9 (8.1)
Average Awake SBP (mmHg)	129.7(11.5)	130.3 (12.5)
Average Awake DBP (mmHg)	71.7 (6.9)	72.1 (6.1)
Last Awake SBP reading (mmHg)	128.5 (19.1)	126.9 (21.3)
Last Awake DBP reading (mmHg)	68.4 (12.7)	68.5 (13.3)
No. of readings taken during sleep	10.19 (5.57)	10.49 (6.19)

Table 3. *Frequencies of Nocturnal Blunted Decline and Nighttime Elevated Blood Pressure by Gender and Race/Ethnicity Groups*

		Blunted Nocturnal Systolic BP Decline	Blunted Nocturnal Diastolic BP Decline	Nighttime Elevated BP Status
Gender	Boys	45% (n = 81)	31% (n = 57)	15% (n = 28)
	Girls	55% (n = 39)	35% (n = 25)	19% (n = 14)
Race/Ethnicity	Non-Hispanic Whites	50% (n = 16)	22% (n = 7)	6% (n = 2)
	Hispanics	46% (n = 56)	36% (n =44)	16% (n = 20)
	Non-Hispanic Blacks	54% (n = 42)	35% (n = 27)	23% (n = 18)
	Others	29% (n = 6)	19% (n = 4)	10% (n = 2)

Table 4.*Systolic and Diastolic BP Means during Sleep by Gender and Race/Ethnicity Groups*

		Systolic Sleep BP M (*SD*)	Diastolic Sleep BP M (*SD*)
Gender	Boys	117 (*13*)	61 (*7*)
	Girls	115 (*15*)	62 (*9*)
Race/Ethnicity	Non-Hispanic Whites	116 (*13*)	59 (*6*)
	Hispanics	116 (*13*)	61 (*7*)
	Non-Hispanic Blacks	119 (*15*)	63 (*9*)
	Others	112 (*12*)	60 (*8*)

Table 5. *Mean Differences between Daytime ABP measures*

	Last Awake BP	Average Awake BP	*t*	df
Systolic	128.5 (19.1)	129.7 (11.5)	-4.4***	25
Diastolic	68.4 (12.7)	71.7 (6.9)	-1.3	251

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

Standard Deviations appear in parentheses below

Table 6. *Modeling the Pattern of Nighttime ABP: Parameter Estimates, (Standard Errors), Random Effects and Model-Data Fit*

	Models of nighttime blood pressure with only Level 1 predictors					
	Systolic			Diastolic		
Parameter	Unconditional means	Quadratic	Quadratic with control	Unconditional means	Quadratic	Quadratic with control
	Fixed Effects					
γ_{00}	118.17 (.81)***	126.40 (1.03)***	126.47 (1.02)***	62.97 (.47)***	67.58 (.66) ***	67.67 (.66)***
γ_{10}		-.0571 (.0047)***	-.0578 (.0048)***		-.0381 (.0036)***	-.0422 (.0038)***
γ_{11}			.0008 (.0002)***			.0015 (.0002)***
γ_{20}		.00007 (7.31E-6)***	.00007 (7.47E-6)***		.00005 (5.59E-6)***	.00006 (5.77E-6)***
	Variance Components					
σ^2	188.58 (5.54)***	164.31 (5.00)***	165.25 (5.05)***	114.20 (3.35)***	105.75 (3.21)***	106.61 (3.25)***
τ_{00}	142.92 (14.81)***	153.46 (19.04)***	148.87 (18.730)***	42.220 (5.003)***	39.832 (6.989)***	36.94 (6.79)***
T_{11}		.0003 (.00008)***	.0005 (.0001)***		.0001 (.00004)**	.00019 (.00005)***
τ_{01}		-.0576 (.0300)	-.1377 (.0394)***		-.0063 (.0129)	-.05073 (.0163)**
	Model Fit					
Deviance	21258.6	21073.0	21072.6	19825.5	19739.5	19702.9
#par	3	7	8	3	7	8
AIC	21262.6	21081.0	21080.6	19829.5	19747.5	19710.9
BIC	21269.7	21095.1	21094.7	19836.6	19761.6	19725.0

Note. * $p < .05$; ** $p < .01$; *** $p < .001$. Standard Deviations appear in parentheses below

Table 7. *Models Adding Demographic, Daytime and Clinical Variables at Level 2: Parameter Estimates, (Standard Errors), Random Effects and Model-Data Fit*

Models of nighttime blood pressure with only Level 1 predictors						
	Systolic			Diastolic		
Parameter	Including Demographic	Including Daytime	Including Clinical	Including Demographic	Including Daytime	Including Clinical
	Fixed Effects					
γ_{00}	125.50 (1.47)***	122.78 (2.95)***	116.40 (3.65)***	66.50 (0.92)***	64.19 (1.81)***	60.94 (2.26)
γ_{01}	-3.13 (2.14)	-3.22 (2.17)	-0.11 (2.79)	0.83 (1.30)	0.81 (1.32)	2.26 (1.70)
γ_{02}	-3.26 (2.96)	-3.22 (2.95)	-4.16 (3.06)	-3.90 (1.78)*	-4.10 (1.77)*	-4.46 (1.85)*
γ_{03}	6.72 (2.18)**	7.90 (2.25)***	8.99 (2.37)***	3.59 (1.33)**	4.14 (1.38)**	5.18 (1.45)
γ_{04}	1.07 (3.51)	0.62 (3.68)	1.05 (3.79)	3.04 (2.12)	2.19 (2.22)	2.12 (2.30)
γ_{05}	0.30 (0.33)	0.37 (0.34)	0.38 (0.35)	0.20 (0.20)	0.26 (0.20)	0.25 (0.21)
γ_{06}		-1.56 (1.95)	-1.82 (2.00)		-0.54 (1.18)	-0.38 (1.21)
γ_{07}		2.39 (2.65)	2.69 (2.72)		2.47 (1.61)	2.79 (1.66)
γ_{08}		-2.25 (3.95)	-3.82 (3.95)		-3.64 (2.36)	-4.49 (2.38)
γ_{09}		0.61 (0.25)*	0.60 (0.25)*		0.32 (0.15)*	0.35 (0.15)*
γ_{010}			0.37 (0.42)			-0.002 (0.257)
γ_{011}			-0.04			0.03

			(0.38)			(0.23)
γ_{012}			4.56			3.80
			(3.04)			(1.86)*
γ_{013}			0.24			0.15
			(0.13)			(0.08)
γ_{014}			5.66			1.99
			(2.49)*			(1.53)
γ_{10}	-0.0567	-0.0545	-0.0376	-0.0387	-0.0317	-0.0210
	(0.0055)***	(0.0084)***	(0.0104)***	(0.0042)***	(0.0062)***	(0.0076)***
γ_{11}	0.0010	0.0009	0.0010	0.0016	0.0017	0.0018
	(0.0002)***	(0.0002)***	(0.0002)***	(0.0002)***	(0.0002)***	(0.0002)***
γ_{12}	0.0120	0.0117	0.0098	-0.0001	0.0001	-0.0018
	(0.0051)*	(0.0051)*	(0.0066)	(0.0036)	(0.0036)	(0.0047)
γ_{13}	0.0116	0.0117	0.0129	0.0073	0.0090	0.0104
	(0.0069)	(0.0068)	(0.0073)	(0.0049)	(0.0049)	(0.0052)*
γ_{14}	-0.01190	-0.01424	-0.0157	-0.0083	-0.0083	-0.0105
	(0.0054)*	(0.0055)*	(0.0060)**	(0.0038)*	(0.0040)*	(0.0043)*
γ_{15}	-0.0061	-0.0038	-0.0031	-0.0134	-0.0118	-0.0106
	(0.0083)	(0.0085)	(0.0089)	(0.0058)*	(0.0060)*	(0.0062)
γ_{16}	-0.0009	-0.0010	-0.0007	-0.0006	-0.0007	-0.0007
	(0.0008)	(0.0008)	(0.0008)	(0.0005)	(0.0006)	(0.0006)
γ_{17}		0.0023	0.0016		-0.0037	-0.0055
		(0.0044)	(0.0046)		(0.0032)	(0.0033)
γ_{18}		-0.0010	-0.0021		-0.0041	-0.0052
		(0.0062)	(0.0065)		(0.0045)	(0.0047)
γ_{19}		-0.0032	-0.00343		0.0041	0.0054
		(0.0088)	(0.009140)		(0.0063)	(0.0065)
γ_{110}		-0.0012	-0.00128		-0.0010	-0.0011
		(0.0006)*	(0.000639)*		(0.0004)*	(0.0005)*
γ_{111}			0.000515			0.0002
			(0.001017)			(0.0007)

γ_{112}			-0.00031			0.00012
			(0.00091)			(0.0006)
γ_{113}			-0.0095			-0.0080
			(0.0076)			(0.0054)
γ_{114}			-0.0002			-0.0002
			(0.0003)			(0.0002)
γ_{115}			-0.01325			-0.0070
			(0.0061)*			(0.0043)
γ_{20}	0.00007	0.00007	0.00007	0.00006	0.0001	0.00005
	(7.54E-6)***	(7.61E-6)***	(7.80E-6)***	(5.81E-6)***	(5.87E-6)***	(5.99E-6)***
	Variance Components					
σ^2	165.51	165.11	165.55	106.48	105.58	105.24
	(5.09)***	(5.21)***	(5.34)***	(3.26)**	(3.31)***	(3.38)***
τ_{00}	140.54	133.56	127.33	32.83	30.52	29.20
	(18.35)***	(18.50)***	(18.57)***	(6.54)***	(6.60)***	(6.65)***
T_{11}	0.0005	0.0004	.0004	0.0002	0.0002	.0002
	(0.0001)***	(0.0001)***	(.0001)***	(0.0001)***	(0.0001)***	(.0001)***
τ_{01}	-0.1339	-0.1106	-.1081	-0.0449	-0.0413	-.03719
	(0.0394)***	(0.0388)**	(.0395)**	(0.0159)**	(0.0160)***	(.0162)*
	Model Fit					
Deviance	20858.4	19838.7	19036.6	19502.7	18545.1	17803.9
#par	18	26	36	18	26	36
AIC	20866.4	19846.7	19044.6	19510.7	18553.1	17811.9
BIC	20880.4	19860.6	19058.2	19524.8	18567.0	17825.5

Note. * $p < .05$; ** $p < .01$; *** $p < .001$. Standard Deviations appear in parentheses below

Table 8. *Adding Sleep Variables at Level 2: Parameter Estimates, (Standard Errors), Random Effects and Model-Data Fit*

Parameter	Systolic Model 5	Diastolic Model 5
	Fixed Effects	
γ_{00}	116.33	58.82
	(6.78)***	(3.93)***
γ_{01}	3.100	4.47
	(5.37)	(3.12)
γ_{02}	-5.61	-7.01
	(6.81)	(3.89)
γ_{03}	11.92	4.17
	(4.13)**	(2.36)
γ_{04}	8.16	7.92
	(6.62)	(3.85)*
γ_{05}	0.54	0.12
	(0.57)	(0.32)
γ_{06}	-2.00	1.92
	(3.87)	(2.18)
γ_{07}	3.04	2.92
	(5.21)	(2.98)
γ_{08}	-2.20	-1.25
	(10.35)	(5.78)
γ_{09}	0.19	0.32
	(0.46)	(0.26)
γ_{010}	0.87	-0.05
	(0.82)	(0.47)
γ_{011}	-0.462	0.05
	(0.76)	(0.43)
γ_{012}	2.60	3.62
	(6.21)	(3.55)

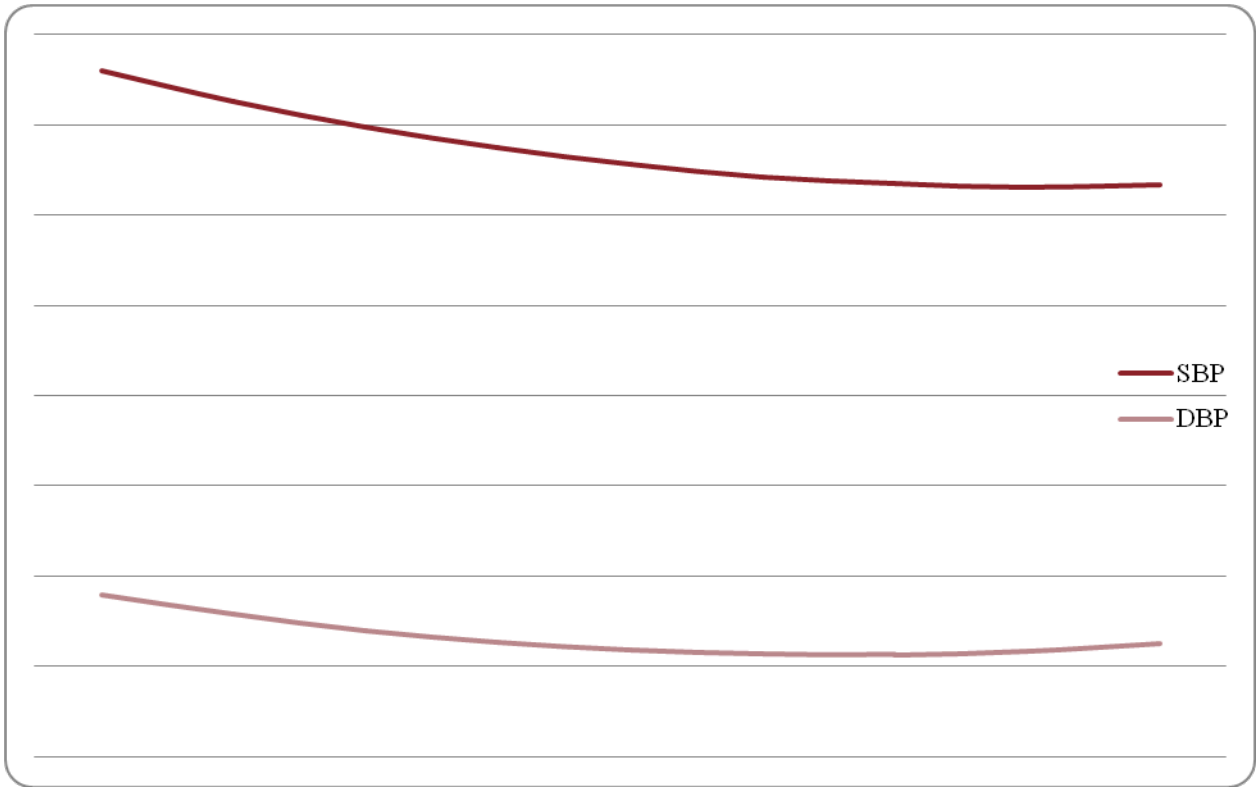
γ_{013}	0.24	0.24
	(0.31)	(0.18)
γ_{014}	6.16	4.21
	(4.40)	(2.51)
γ_{10}	-0.0340	-0.0009
	(0.0198)	(0.0135)
γ_{11}	0.0007	0.0018
	(0.0003)*	(0.0004)***
γ_{12}	0.01200	-0.0051
	(0.0139)	(0.0091)
γ_{13}	0.03021	0.0231
	(0.0163)	(0.0106)*
γ_{14}	-0.0162	-0.0082
	(0.0110)	(0.0071)
γ_{15}	-0.01804	-0.0301
	(0.0175)	(0.0116)**
γ_{16}	-0.0017	-0.0015
	(0.0014)	(0.0009)
γ_{17}	0.0031	-0.0129
	(0.0083)	(0.0056)*
γ_{18}	-0.0115	-0.0124
	(0.0129)	(0.0084)
γ_{19}	-0.0052	-0.0087
	(0.0221)	(0.0139)
γ_{110}	-0.0001	-0.0013
	(0.0012)	(0.0008)
γ_{111}	-0.0007	0.0001
	(0.0021)	(0.0014)
γ_{112}	0.000602	0.0002
	0.0018	(0.0012)
γ_{113}	(0.0076)	-0.00470

γ_{113}	(0.0076)	-0.00470
	0.0153	(0.0101)
γ_{114}	-4.61E-6	-0.0001
	0.0008	(0.0005)
γ_{115}	-0.0215	-0.0148
	(0.0105)*	(0.0069)*
γ_{116}	-0.0034	-0.0020
	(0.0074)	(0.0041)
γ_{117}	0.0101	0.0060
	(0.0075)	(0.0041)
γ_{118}	-0.0148	0.0003
	(0.0137)	(0.0076)
γ_{119}	-0.0063	-0.0051
	(0.0084)	(0.0047)
γ_{120}	0.0008	-0.0015
	(0.0090)	(0.0050)
γ_{121}	0.0048	-0.0008
	(0.0100)	(0.0056)
γ_{122}	0.0087	-0.0008
	(0.0170)	(0.0056)
γ_{20}	0.00001	0.00005
	(0.0000)***	(8.89E-6)***
	Variance Components	
σ^2	200.79	120.70
	(9.56)***	(5.68)***
τ_{00}	189.05	36.956
	(40.96)***	(12.94)**
T_{11}	0.0006	0.000182
	(0.0003)**	(0.0001)*
τ_{01}	-0.1725	-0.0494
	(0.0965)	(0.0308)

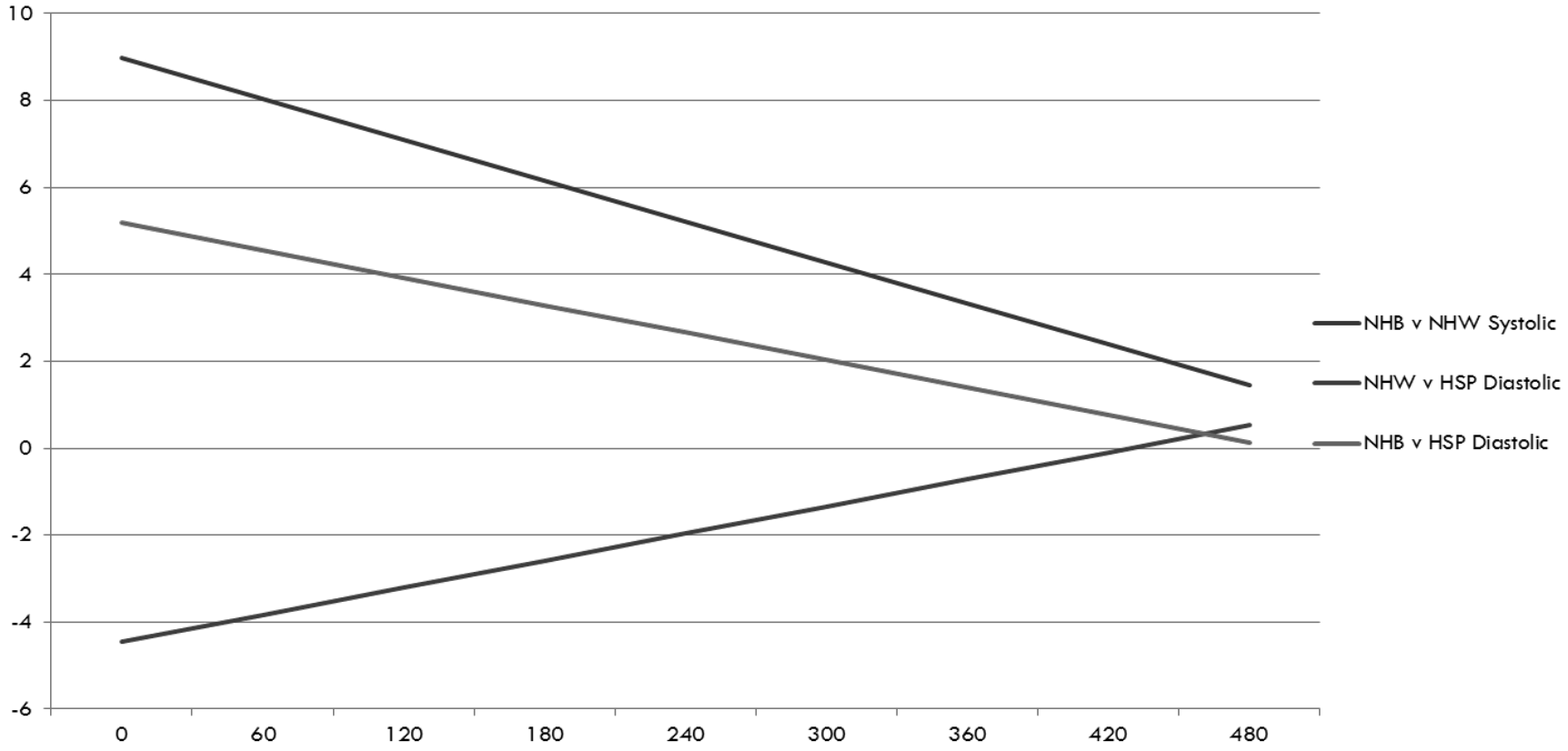
Deviance	9020.2	8404.7
#par	43	43
AIC	9028.2	8412.7
BIC	9038.7	8423.1

Note. * $p < .05$; ** $p < .01$; *** $p < .001$. Standard Deviations appear in parentheses below

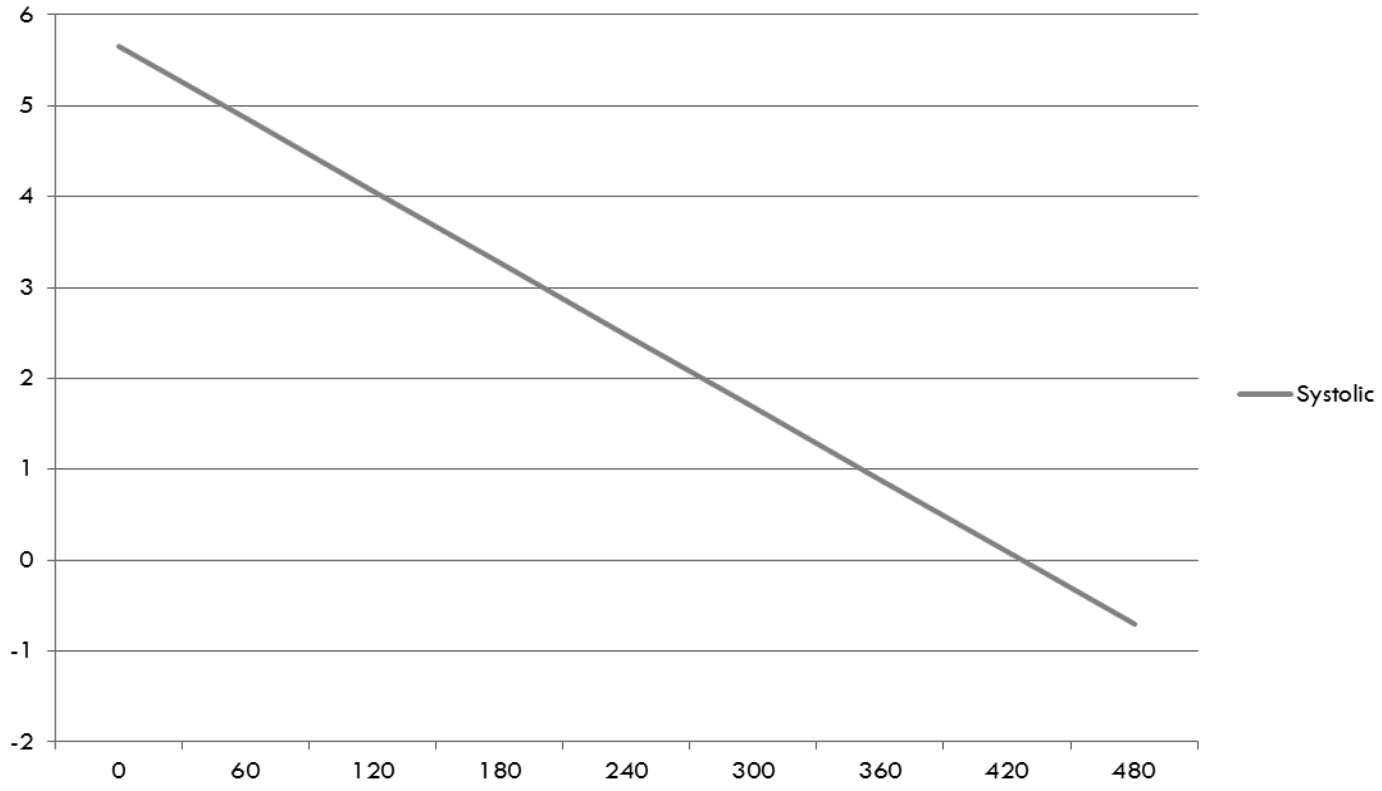
Graph 1. Pattern of Nocturnal Decline in Systolic and Diastolic Blood Pressure Measured across Sample's Average Sleep Duration (480 minutes) with Time Added as Level 1 Predictor



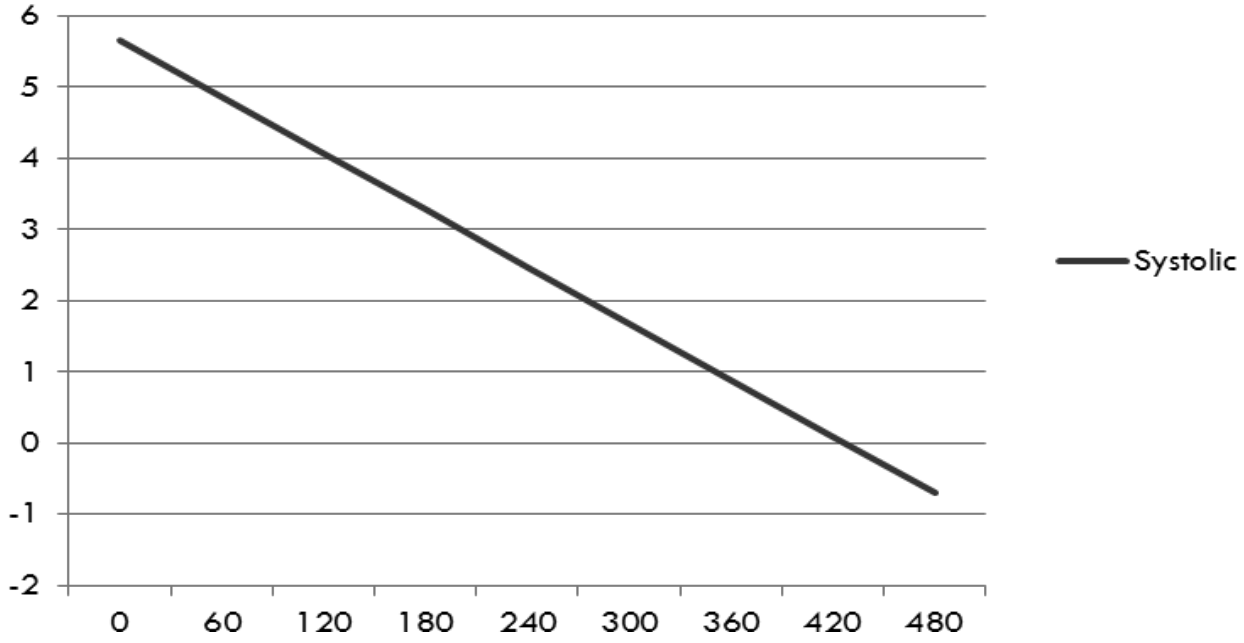
Graph 2. Statistically Significant Expected Differences in Intercepts and Instantaneous Rates of Nocturnal Blood Pressure (mmHg) Decline due to Racial/Ethnic Group Identification over Eight Hours (480 minutes) of Sleep



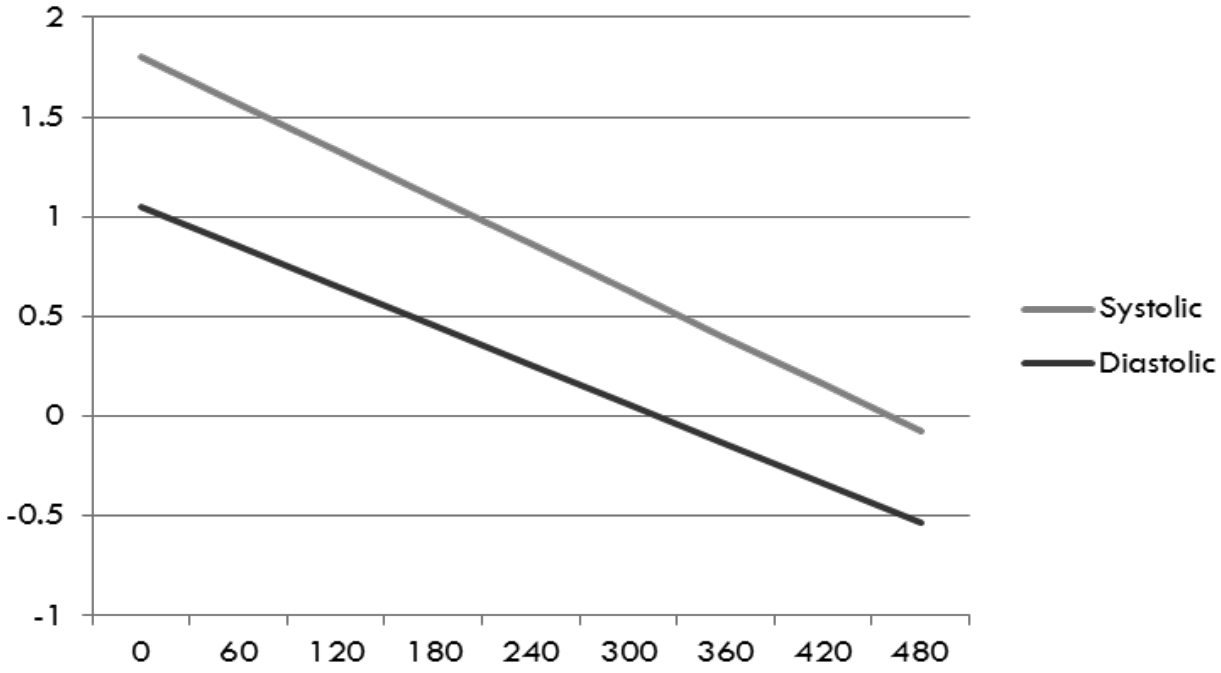
Graph 3. Statistically Significant Expected Differences in Intercepts and Instantaneous Rates of Nocturnal Systolic Blood Pressure (mmHg) Decline due to Gender Identification over Eight Hours (480 minutes) of Sleep



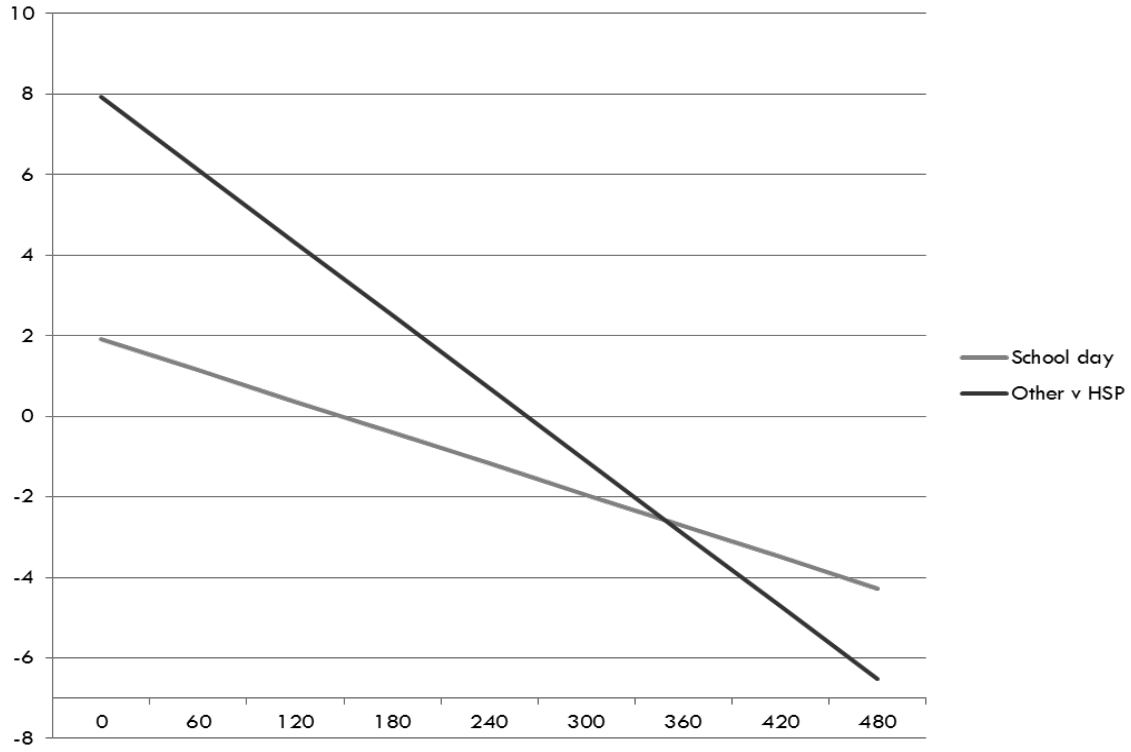
Graph 4. Statistically Significant Expected Differences in Intercepts and Instantaneous Rates of Nocturnal Systolic Blood Pressure (mmHg) Decline due to Positive Family History of Hypertension over Eight Hours (480 minutes) of Sleep



Graph 5. Statistically Significant Expected Differences in Intercepts and Instantaneous Rates of Nocturnal Systolic and Diastolic Blood Pressure (mmHg) Decline due to Reporting Sample Averaged Periods of Stress over Eight Hours (480 minutes) of Sleep



Graph 6. Statistically Significant Expected Differences in Intercepts and Instantaneous Rates of Nocturnal Diastolic Blood Pressure (mmHg) Decline due Racial/Ethnic Identification and School Attendance within the Sample for whom Sleep Quality Characteristics were Collected over Eight Hours (480 minutes) of Sleep



Appendices

Appendix A. Equations specified to determine what pattern nocturnal blood pressure takes in youth

Modeling mean nocturnal blood pressure in youth (Unconditional Means)

Level 1:

$$Y_{ij} = \pi_{0i} + \sigma_{\varepsilon} \quad (1)$$

Level 2:

$$\pi_{0i} = \gamma_{00} + \zeta_{oi} \quad (2)$$

Y_{ij} = each individual blood pressure reading taken during sleep

π_{0i} = the mean blood pressure measured across sleep, time invariant

σ_{ε} = random variance within individual reading when time is held constant

γ_{00} = expected value of mean nocturnal blood pressure for all individuals

ζ_{oi} = random error between individuals when time is held constant

Adding linear and quadratic time components (centering based on beginning of sleep time)

Level 1:

$$Y_{ij} = \pi_{0i} + \pi_{1i}(\text{time} - \text{time went to sleep}_{cwc}) + \pi_{ai}(\text{time} - \text{time went to sleep}_{cwc})^2 + \sigma_{\varepsilon} \quad (6)$$

Level 2:

$$\pi_{0i} = \gamma_{00} + \zeta_{oi} \quad (7)$$

$$\pi_{1i} = \gamma_{10} + \zeta_{1i} \quad (8)$$

$$\pi_{2i} = \gamma_{20} \quad (9)$$

Y_{ij} = each individual blood pressure reading taken during sleep

π_{0i} = expected individual's blood pressure reading at t =0 within sleep

π_{1i} =expected instantaneous change at t =0 in an individual's blood pressure per minute during sleep

π_{2i} = expected rate of change of instantaneous change in an individual's blood pressure per minute during sleep

σ_{ε} = random variance within individual reading when blood pressure is measured quadratically over the nighttime period (variance unexplained by intercept and linear change)

γ_{00} =mean intercept of nighttime blood pressure of individuals

ζ_{oi} = random error mean intercept of nighttime blood pressure of individuals

γ_{10} =mean rate of instantaneous change in blood pressure per minute during sleep of individuals

ζ_{1i} = random error mean rate of instantaneous change in blood pressure per minute during sleep of individuals

γ_{20} =mean changing rate of instantaneous change in blood pressure per minute during sleep of individuals

Adding a sample centered daytime average blood pressure and time since sleep product to account for differences in rate of decline based on intercept

Level 1:

$$Y_{ij} = \pi_{0i} + \pi_{1i}\,(\text{time} - \text{time went to sleep}_{cwc}) + \pi_{ai}\,(\text{time} - \text{time went to sleep}_{cwc})^2 + \sigma_{\varepsilon} \quad (10)$$

Level 2:

$$\pi_{0i} = \gamma_{00} + \zeta_{oi} \quad (11)$$

$$\pi_{1i} = \gamma_{10} + \gamma_{11} + \zeta_{1i} \quad (12)$$

$$\pi_{2i} = \gamma_{20}$$

Y_{ij} = each individual blood pressure reading taken during sleep

π_{0i} = expected individual's blood pressure reading at t =0 within sleep

π_{1i} =expected instantaneous change at t =0 in an individual's blood pressure per minute during sleep

π_{2i} = expected rate of change of instantaneous change in an individual's blood pressure per minute during sleep

σ_{ε} = random variance within individual reading when blood pressure is measured quadratically over the nighttime period (variance unexplained by intercept and linear change)

γ_{00} =mean intercept of nighttime blood pressure of individuals

ζ_{oi} = random error mean intercept of nighttime blood pressure of individuals

γ_{10} =mean rate of instantaneous change in blood pressure per minute during sleep of individuals

γ_{11} = rate of instantaneous change in blood pressure per minute during sleep of individuals related to their sample centered mean daytime ambulatory blood pressure

ζ_{1i} = random error mean rate of instantaneous change in blood pressure per minute during sleep of individuals

γ_{20} =mean changing rate of instantaneous change in blood pressure per minute during sleep of individuals

Appendix B. Equations specified to determine what how demographic, daytime and clinical variables impact the pattern of blood pressure during sleep

Adding demographic variables and determine how much they reduce inter-individual variability

Girl= identifying as a girl as compared to a boy

NHW= identifying at non- Hispanic White as opposed to Hispanic

NHB= identifying as Non-Hispanic Black as opposed to Hispanic

Other =identifying as a race/ethnicity other than Hispanic, Non-Hispanic Black, Non-Hispanic White

SES = number of years of education the head of the household has compared to the sample average

Level 2:

$$\pi_{0i} = \gamma_{00} + \gamma_{01}\,(\text{Girl}) + \gamma_{02}(\text{NHW}) + \gamma_{03}\,(\text{NHB}) + \gamma_{04}\,(\text{Other}) + \gamma_{05}(\text{SES-SES}_{GMC}) + \zeta_{oi} \quad (13)$$

γ_{00} = expected blood pressure reading at t =0 within sleep for an male Hispanic individual of average SES

γ_{01} = expected difference in the blood pressure reading at t = 0 due to identifying as a girl as opposed to a boy, controlling for the effects of racial/ethnic identification and SES

γ_{02} = expected difference in the blood pressure reading at t = 0 due to identifying asnon-Hispanic White as opposed to Hispanic, controlling for the effects of gender, other racial/ethnic identifications and SES

γ_{03} = expected difference in the blood pressure reading at t = 0 due to identifying as Non-Hispanic Black as opposed to Hispanic, controlling for the effects of gender, other racial/ethnic identifications and SES

γ_{04} = expected difference in the blood pressure reading at t = 0 due to identifying as Other as opposed to Hispanic, controlling for the effects of gender, other racial/ethnic identifications and SES

$\gamma_{05=}$ expected difference in the blood pressure reading at t = 0 due to one unit change in mean SES, controlling for the effects of gender and racial/ethnic identification

$\pi_{1i} = \gamma_{10} + \gamma_{11(intercept*slope)+} \gamma_{12}$ (Girl) + γ_{13}(NHW)+ γ_{14} (NHB) + γ_{15} (Other) + γ_{16} (SES-SES_{GMC}) + ζ_{oi} (14)

γ_{10} = expected instantaneous change at t =0 per minute during sleep for a male, Hispanic individual of average SES

$\gamma_{11=}$ expected difference in instantaneous change at t =0 per minute during sleep for an individual due to one unit change in mean centered daytime ABP controlling for gender, racial/ethnic identification and SES

γ_{12} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as a girl as opposed to a boy, controlling racial/ethnic identification and SES

γ_{13} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as non-Hispanic White as opposed to Hispanic, controlling for gender, other racial/ethnic identifications and SES

γ_{14} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as non-Hispanic Black as opposed to Hispanic, controlling for gender, other racial/ethnic identifications and SES

γ_{15} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as a race/ethnicity other than Hispanic, non-Hispanic Black or Non-Hispanic White, controlling for gender and SES

γ_{16} = expected difference in instantaneous change at t =0 per minute during sleep for due to one unit change in mean SES, controlling for the effects of gender and racial/ethnic identification

$\pi_{2i} = \gamma_{20}$

γ_{20} = expected rate of change of instantaneous change in an individual blood pressure per minute during sleep

Adding in the daytime factors and determining how much they reduce between person variability

Schoolday = attended school on day of daytime ABP collection

Caffeine = indicated participant consumed caffeine prior the collection of this nocturnal blood pressure

Tobacco = indicated participant consumed tobacco prior the collection of this nocturnal blood pressure

Stress = number of times participant endorsed being stressed during the daytime ABP collection

Level 2:

$$\pi_{0i} = \gamma_{00} + \gamma_{01}\,(\text{Girl}) + \gamma_{02}(\text{NHW}) + \gamma_{03}\,(\text{NHB}) + \gamma_{04}\,(\text{Other}) + \gamma_{05}(\text{SES-SES}_{GMC}) + \gamma_{06}\,(\text{schoolday}) + \gamma_{07}\,(\text{caffeine}) + \gamma_{08}(\text{tobacco}) + \gamma_{09}(\text{stress}) + \zeta_{oi} \quad (15)$$

γ_{00} = expected blood pressure reading at t =0 within sleep for an male Hispanic individual who is of average SES, who did not attend school, did not consume caffeine or tobacco and reported no stress during the day.

γ_{01} = expected difference in the blood pressure reading at t = 0 due to identifying as a girl as opposed to a boy, controlling for the effects of racial/ethnic identification, SES, school attendance, substance use and stress

γ_{02} = expected difference in the blood pressure reading at t = 0 due to identifying as non-Hispanic White as opposed to Hispanic, controlling for the effects of gender, other racial/ethnic identification, school attendance, substance use and stress

γ_{03} = expected difference in the blood pressure reading at t = 0 due to identifying as Non-Hispanic Black as opposed to Hispanic, controlling for the effects of gender, other racial/ethnic identifications, school attendance, substance use and stress

γ_{04} = expected difference in the blood pressure reading at t = 0 due to identifying as Other as opposed to Hispanic, controlling for the effects of gender, other racial/ethnic identifications, school attendance , substance use and stress

$\gamma_{05=}$ expected difference in the blood pressure reading at t = 0 due to one unit change in mean SES, controlling for the effects of gender, racial/ethnic identification, school attendance, substance use and stress

γ_{06} = expected difference in the blood pressure reading at t = 0 due to attending school, controlling for the effects all demographic factors, substance use and stress

γ_{07} = expected difference in the blood pressure reading at t = 0 due to caffeine consumption, controlling for the effects all demographic factors, school attendance, tobacco use and stress

γ_{08} = expected difference in the blood pressure reading at t = 0 due to caffeine consumption, controlling for the effects all demographic factors, school attendance, tobacco use and stress

γ_{09} = expected difference in the blood pressure reading at t = 0 due to one unit change in number of times stress was reported during the day, controlling for the effects all demographic factors, school attendance and substance use

$\pi_{1i} = \gamma_{10} + \gamma_{11(intercept*slope)+} \gamma_{12}$ (Girl) + γ_{13}(NHW)+ γ_{14} (NHB) + γ_{15} (Other)+ γ_{16}(SES-SES_{GMC}) + γ_{17} (schoolday) + γ_{18} (caffeine) + γ_{19}(tobacco) + γ_{110}(stress) + ζ_{oi} (16)

γ_{10} = expected instantaneous change at t =0 per minute during sleep for a male, Hispanic individual with average SES's blood pressure who had did not attend school, did not consume caffeine or tobacco and reporting no stress during the day

$\gamma_{11=}$ expected difference in instantaneous change at t =0 per minute during sleep for an individual due to one unit change in mean centered daytime ABP blood pressure controlling for demographics, school attendance, substance use and stress

γ_{12} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as a girl as opposed to a boy, controlling racial/ethnic identification, SES, school attendance, substance use and stress

γ_{13} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as non-Hispanic White as opposed to Hispanic, controlling for gender, other racial/ethnic identifications, SES, school attendance, substance use and stress

γ_{14} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as non-Hispanic Black as opposed to Hispanic, controlling for gender, other racial/ethnic identifications, SES, school attendance, substance use and stress

γ_{15} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as a race/ethnicity other than Hispanic, non-Hispanic Black or Non-Hispanic White, controlling for gender, SES , school attendance, substance use and stress

γ_{16} = expected difference in instantaneous change at t =0 per minute during sleep for due to one unit change in mean SES, controlling for the effects of gender, racial/ethnic identification, school attendance, substance use and stress

γ_{17} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to attending school, controlling for the effects of all demographic factors, substance use and stress

γ_{18} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to caffeine consumption, controlling for the effects of all demographic variables, attendance of school, tobacco use and stress

γ_{19} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to tobacco use to a one unit increase in stress exposure during the day controlling for the effects of all demographic variables, attendance of school, consumption of caffeine, and stress

γ_{110} = expected difference in instantaneous change at t =0 per minute during sleep due to one unit change in number of times stress was reported during the day, controlling for the effects all demographic factors, school attendance and substance use

$\pi_{2i} = \gamma_{20}$

γ_{20} = expected rate of change of instantaneous change in an individual blood pressure per minute during sleep

Adding in the clinical factors and determining how much they reduce between person variability

Waist = sample centered waist circumference

BMI = sample centered BMI

mets = indicates if person meets the criteria for the metabolic syndrome

fitness = V02Max

famhyphx = having a positive family history of hypertension

Level 2:

$$\pi_{0i} = \gamma_{00} + \gamma_{01}\,(\text{Girl}) + \gamma_{02}(\text{NHW}) + \gamma_{03}\,(\text{NHB}) + \gamma_{04}\,(\text{Other}) + \gamma_{05}(\text{SES-SES}_{GMC}) + \gamma_{06}\,(\text{schoolday}) + \gamma_{07}\,(\text{caffeine}) + \gamma_{08}(\text{tobacco}) + \gamma_{09}(\text{stress}) + \gamma_{010}(\text{waist-waist}_{GMC}) + \gamma_{011}\,(\text{BMI-BMI}_{GMC}) + \gamma_{012}(\text{mets}) + \gamma_{013}(\text{fitness-fitness}_{GMC}) + \gamma_{014}(\text{famhyphx}) + \zeta_{oi} \qquad (17)$$

γ_{00} = expected blood pressure reading at t =0 within sleep for an male Hispanic individual who is of average SES, who did not attend school, did not consume caffeine or tobacco, reported no stress during the day, which a sample average waist circumference and BMI,

does not meet criteria for the metabolic syndrome, has a sample average fitness level and no family history of hypertension.

γ_{01} = expected difference in the blood pressure reading at t = 0 due to identifying as a girl as opposed to a boy, controlling for the effects of racial/ethnic identification, SES, school attendance, substance use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{02} = expected difference in the blood pressure reading at t = 0 due to identifying as non-Hispanic White as opposed to Hispanic, controlling for the effects of gender, other racial/ethnic identification, school attendance, substance use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{03} = expected difference in the blood pressure reading at t = 0 due to identifying as Non-Hispanic Black as opposed to Hispanic, controlling for the effects of gender, other racial/ethnic identifications, school attendance , substance use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{04} = expected difference in the blood pressure reading at t = 0 due to identifying as Other as opposed to Hispanic, controlling for the effects of gender, other racial/ethnic identifications, school attendance, substance use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

$\gamma_{05=}$ expected difference in the blood pressure reading at t = 0 due to one unit change in mean SES, controlling for the effects of gender, racial/ethnic identification, school attendance, substance use, stress, metabolic syndrome, fitness and family history of hypertension

γ_{06} = expected difference in the blood pressure reading at t = 0 due to attending school, controlling for the effects all demographic factors, substance use, stress, metabolic syndrome, fitness and family history of hypertension

γ_{07} = expected difference in the blood pressure reading at t = 0 due to caffeine consumption, controlling for the effects all demographic factors, school attendance, tobacco use, stress, obesity metabolic syndrome, fitness and family history of hypertension

γ_{08} = expected difference in the blood pressure reading at t = 0 due to caffeine consumption, controlling for the effects all demographic factors, school attendance, tobacco use, stress, obesity metabolic syndrome, fitness and family history of hypertension

γ_{09} = expected difference in the blood pressure reading at t = 0 due to one unit change in number of times stress was reported during the day, controlling for the effects all demographic factors, school attendance, substance use, obesity metabolic syndrome, fitness and family history of hypertension

γ_{010} = expected difference in the blood pressure reading at t = 0 due to one unit change waist circumference (sample centered), controlling for the effects all demographic factors, school attendance, substance use, stress, BMI, metabolic syndrome, fitness and family history of hypertension

γ_{011} = expected difference in the blood pressure reading at t = 0 due to one unit change in BMI (sample centered), controlling for the effects all demographic factors, school attendance, substance use, stress and waist circumference, metabolic syndrome, fitness and family history of hypertension

γ_{012} = expected difference in the blood pressure reading at t = 0 due to meeting criteria for the metabolic syndrome, controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, syndrome, fitness and family history of hypertension

γ_{013} = expected difference in the blood pressure reading at t = 0 due to one unit change in fitness (sample centered), controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, metabolic syndrome, and family history of hypertension

γ_{014} = expected difference in the blood pressure reading at t = 0 due to having a positive family history of hypertension, controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, metabolic syndrome and fitness

$$\pi_{1i} = \gamma_{10} + \gamma_{11(intercept*slope)+} \gamma_{12}\,(Girl) + \gamma_{13}(NHW) + \gamma_{14}\,(NHB\,) + \gamma_{15}\,(Other) + \gamma_{16}(SES\text{-}SES_{GMC}) + \gamma_{17}(schoolday) + \gamma_{18}\,(caffeine) + \gamma_{19}(tobacco) + \gamma_{110}(stress) + \gamma_{111}(waist\text{-}waist_{GMC}) + \gamma_{112}\,(BMI\text{-}BMI_{GMC}) + \gamma_{113}(mets) + \gamma_{114}(fitness\text{-}fitness_{GMC}) + \gamma_{115}(famhyphx) + \zeta_{oi} \quad (18)$$

γ_{10} = expected instantaneous change at t =0 per minute during sleep for a male, Hispanic individual with average SES's blood pressure who had did not attend school, did not consume caffeine or tobacco, reporting no stress during the day, and an average waist circumference and BMI, does not meet criteria for the metabolic syndrome, has a sample average fitness level and no family history of hypertension.

$\gamma_{11=}$ expected difference in instantaneous change at t =0 per minute during sleep for an individual due to one unit change in mean centered daytime ABP blood pressure

controlling for demographics, school attendance, substance use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{12} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as a girl as opposed to a boy, controlling racial/ethnic identification, SES, school attendance, substance use ,stress, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{13} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as non-Hispanic White as opposed to Hispanic, controlling for gender, other racial/ethnic identifications, SES, school attendance, substance use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{14} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as non-Hispanic Black as opposed to Hispanic, controlling for gender, other racial/ethnic identifications, SES, school attendance, substance use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{15} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as a race/ethnicity other than Hispanic, non-Hispanic Black or Non-Hispanic White, controlling for gender, SES , school attendance, substance use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{16} = expected difference in instantaneous change at t =0 per minute during sleep for due to one unit change in mean SES, controlling for the effects of gender, racial/ethnic identification, school attendance, substance use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{17} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to attending school, controlling for the effects of all demographic factors, substance use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{18} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to caffeine consumption, controlling for the effects of all demographic variables, attendance of school, tobacco use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{19} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to tobacco use during the day controlling for the effects of all demographic and sleep quality variables, attendance of school, stress, consumption of caffeine, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{110} = expected difference in instantaneous change at t =0 per minute during sleep due to one unit change in number of times stress was reported during the day, controlling for the effects all demographic factors, school attendance, substance use, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{111} = expected difference in instantaneous change at t =0 per minute during sleep due to one unit change in sample centered waist circumference, controlling for the effects all demographic factors, school attendance, substance use, sample centered BMI, metabolic syndrome, fitness and family history of hypertension

γ_{112} = expected difference in instantaneous change at t =0 per minute during sleep due to one unit change in sample centered BMI, controlling for the effects all demographic factors, school attendance, substance use, sample centered waist circumference, metabolic syndrome, fitness and family history of hypertension

γ_{113} =expected difference in instantaneous change at t =0 per minute during sleep due to meeting criteria for the metabolic syndrome, controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, syndrome, fitness and family history of hypertension

γ_{114} = expected difference in instantaneous change at t =0 per minute during sleep due to one unit change in fitness (sample centered), controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, metabolic syndrome, and family history of hypertension

γ_{115} = expected difference in instantaneous change at t =0 per minute during sleep due to having a positive family history of hypertension, controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, metabolic syndrome and fitness

$\pi_{2i} = \gamma_{20}$

γ_{20} = expected rate of change of instantaneous change in an individual blood pressure per minute during sleep

Appendix C. Equations specified to the impact of sleep quality variables on the pattern of blood pressure during sleep

Adding sleep variables for those with more detailed sleep quality information

Ntypicalsleep = whether an individual described the sleep as non-typical

shareroom = whether the individual has roommate during sleep

sharebed = whether the individual has bedmate during sleep

awakenmon = whether the individual awakened during the night due to the ABP monitor

awakenenvi = whether the individual awakened during the night due to environmental factors such as noise, light or temperature

awakenbath = whether the individual awakened during the night to use the bathroom

awakenoth = whether the individual awakened during the night for a reason other than the monitor, environment or bathroom

Level 2:

$\pi_{0i} = \gamma_{00} + \gamma_{01}$ (Girl) + γ_{02}(NHW)+ γ_{03} (NHB) + γ_{04} (Other)+ γ_{05}(SES-SES_{GMC}) + γ_{06} (schoolday) + γ_{07} (caffeine) + γ_{08}(tobacco) + γ_{09}(stress) + γ_{010}(waist-$waist_{GMC}$) + γ_{011} (BMI-BMI_{GMC}) + γ_{012}(mets) + γ_{013}(fitness-$fitness_{GMC}$) + γ_{014}(famhyphx) + ζ_{oi} (19)

γ_{00} = expected blood pressure reading at t =0 within sleep for an male Hispanic individual who is of average SES, who did not attend school, did not consume caffeine or tobacco, reported no stress during the day, which a sample average waist circumference and BMI, does not meet criteria for the metabolic syndrome, has a sample average fitness level and no family history of hypertension.

γ_{01} = expected difference in the blood pressure reading at t = 0 due to identifying as a girl as opposed to a boy, controlling for the effects of racial/ethnic identification, SES, school attendance, substance use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{02} = expected difference in the blood pressure reading at t = 0 due to identifying as non-Hispanic White as opposed to Hispanic, controlling for the effects of gender, other racial/ethnic identification, school attendance, substance use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{03} = expected difference in the blood pressure reading at t = 0 due to identifying as Non-Hispanic Black as opposed to Hispanic, controlling for the effects of gender, other

racial/ethnic identifications, school attendance , substance use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

γ_{04} = expected difference in the blood pressure reading at t = 0 due to identifying as Other as opposed to Hispanic, controlling for the effects of gender, other racial/ethnic identifications, school attendance, substance use, stress, obesity, metabolic syndrome, fitness and family history of hypertension

$\gamma_{05=}$ expected difference in the blood pressure reading at t = 0 due to one unit change in mean SES, controlling for the effects of gender, racial/ethnic identification, school attendance, substance use, stress, metabolic syndrome, fitness and family history of hypertension

γ_{06} = expected difference in the blood pressure reading at t = 0 due to attending school, controlling for the effects all demographic factors, substance use, stress, metabolic syndrome, fitness and family history of hypertension

γ_{07} = expected difference in the blood pressure reading at t = 0 due to caffeine consumption, controlling for the effects all demographic factors, school attendance, tobacco use, stress, obesity metabolic syndrome, fitness and family history of hypertension

γ_{08} = expected difference in the blood pressure reading at t = 0 due to caffeine consumption, controlling for the effects all demographic factors, school attendance, tobacco use, stress, obesity metabolic syndrome, fitness and family history of hypertension

γ_{09} = expected difference in the blood pressure reading at t = 0 due to one unit change in number of times stress was reported during the day, controlling for the effects all demographic factors, school attendance, substance use, obesity metabolic syndrome, fitness and family history of hypertension

γ_{010} = expected difference in the blood pressure reading at t = 0 due to one unit change waist circumference (sample centered), controlling for the effects all demographic factors, school attendance, substance use, stress, BMI, metabolic syndrome, fitness and family history of hypertension

γ_{011} = expected difference in the blood pressure reading at t = 0 due to one unit change in BMI (sample centered), controlling for the effects all demographic factors, school attendance, substance use, stress and waist circumference, metabolic syndrome, fitness and family history of hypertension

γ_{012} = expected difference in the blood pressure reading at t = 0 due to meeting criteria for the metabolic syndrome, controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, syndrome, fitness and family history of hypertension

γ_{013} = expected difference in the blood pressure reading at t = 0 due to one unit change in fitness (sample centered), controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, metabolic syndrome, and family history of hypertension

γ_{014} = expected difference in the blood pressure reading at t = 0 due to having a positive family history of hypertension, controlling for the effects all demographic factors, daytime variables, obesity, fitness and the metabolic syndrome

$\pi_{1i} = \gamma_{10} + \gamma_{11(intercept*slope)+} \gamma_{12}$ (Girl) + γ_{13}(NHW)+ γ_{14} (NHB) + γ_{15} (Other)+ γ_{16}(SES-SES_{GMC}) + γ_{17} (schoolday) + γ_{18} (caffeine) + γ_{19}(tobacco) + γ_{110}(stress) + γ_{111}(waist-$waist_{GMC}$) + γ_{112} (BMI-BMI_{GMC}) + γ_{113}(mets) + γ_{114}(fitness-$fitness_{GMC}$) + γ_{115}(famhyphx) + γ_{116} (ntypicalsleep) + γ_{117} (shareoom) + γ_{118}(sharebed) +γ_{119}(awakenmon) + γ_{120}(awakenenivo) + γ_{121}(awakenbath) + γ_{122}(awakenoth) ζ_{oi} (20)

γ_{10} = expected instantaneous change at t =0 per minute during sleep for a male, Hispanic individual with average SES's blood pressure who had did not attend school, did not consume caffeine or tobacco, reporting no stress during the day, and an average waist circumference and BMI, does not meet criteria for the metabolic syndrome, has a sample average fitness level, no family history of hypertension, had a typical night sleep, does not share a room or bed and did not awaken during the night

$\gamma_{11=}$ expected difference in instantaneous change at t =0 per minute during sleep for an individual due to one unit change in mean centered daytime ABP blood pressure controlling for demographics, school attendance, substance use, stress, obesity, metabolic syndrome, fitness, family history of hypertension and sleep characteristics

γ_{12} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as a girl as opposed to a boy, controlling racial/ethnic identification, SES, school attendance , substance use, stress, obesity, metabolic syndrome, fitness, family history of hypertension and sleep characteristics

γ_{13} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as non-Hispanic White as opposed to Hispanic, controlling for gender, other racial/ethnic identifications, SES, school attendance, substance use, stress, obesity, metabolic syndrome, fitness, family history of hypertension and sleep characteristics

γ_{14} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as non-Hispanic Black as opposed to Hispanic, controlling for gender, other racial/ethnic identifications, SES, school attendance, substance use, stress, obesity, metabolic syndrome, fitness, family history of hypertension and sleep characteristics

γ_{15} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to identifying as a race/ethnicity other than Hispanic, non-Hispanic Black or Non-Hispanic White, controlling for gender, SES , school attendance, substance use, stress, obesity, metabolic syndrome, fitness, family history of hypertension and sleep characteristics

γ_{16} = expected difference in instantaneous change at t =0 per minute during sleep for due to one unit change in mean SES, controlling for the effects of gender, racial/ethnic identification, school attendance, substance use, stress, obesity, metabolic syndrome, fitness, family history of hypertension and sleep characteristics

γ_{17} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to attending school, controlling for the effects of all demographic factors, substance use, stress, obesity, metabolic syndrome, fitness, family history of hypertension and sleep characteristics

γ_{18} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to caffeine consumption, controlling for the effects of all demographic variables, attendance of school, tobacco use, stress, obesity, metabolic syndrome, fitness, family history of hypertension and sleep characteristics

γ_{19} = expected difference in instantaneous change at t =0 per minute during sleep for an individual due to tobacco use to a one unit increase in stress exposure during the day controlling for the effects of all demographic variables, attendance of school, consumption of caffeine, obesity, metabolic syndrome, fitness, family history of hypertension and sleep characteristics

γ_{111} = expected difference in instantaneous change at t =0 per minute during sleep due to one unit change in number of times stress was reported during the day, controlling for the effects all demographic factors, school attendance, substance use, obesity, metabolic syndrome, fitness, family history of hypertension and sleep characteristics

γ_{111} = expected difference in instantaneous change at t =0 per minute during sleep due to one unit change in sample centered waist circumference, controlling for the effects all demographic factors, school attendance, substance use, sample centered BMI, metabolic syndrome, fitness, family history of hypertension and sleep characteristics

γ_{112} = expected difference in instantaneous change at t =0 per minute during sleep due to one unit change in sample centered BMI, controlling for the effects all demographic factors, school attendance, substance use, sample centered waist circumference, metabolic syndrome, fitness, family history of hypertension and sleep characteristics

γ_{113} =expected difference in instantaneous change at t =0 per minute during sleep due to meeting criteria for the metabolic syndrome, controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, syndrome, fitness, family history of hypertension and sleep characteristics

γ_{114} = expected difference in instantaneous change at t =0 per minute during sleep due to one unit change in fitness (sample centered), controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, metabolic syndrome, family history of hypertension and sleep characteristics

γ_{115} = expected difference in instantaneous change at t =0 per minute during sleep due to having a positive family history of hypertension, controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, metabolic syndrome, fitness and sleep characteristics

γ_{116} = expected difference in instantaneous change at t =0 per minute during sleep due to having a non-typical night sleep, controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, metabolic syndrome, fitness, family history of hypertension, sharing a bed or room during sleep and awakening during the night

γ_{117} = expected difference in instantaneous change at t =0 per minute during sleep due to sharing a room during sleep for the effects all demographic factors, school attendance, substance use, stress, obesity, metabolic syndrome, fitness, family history of hypertension, having a non-typical night sleep, sharing a bed, and awakening

γ_{118} = expected difference in instantaneous change at t =0 per minute during sleep due to sharing a bed during sleep for the effects all demographic factors, school attendance, substance use, stress, obesity, metabolic syndrome, fitness, family history of hypertension, having a non-typical night sleep, sharing a room, and awakening

γ_{119} = expected difference in instantaneous change at t =0 per minute during sleep due to awakening during the night due to the monitor, controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, metabolic syndrome, fitness, family history of hypertension, having a non-typical night sleep, sharing a room/bed and awakening for other reasons

γ_{120} = expected difference in instantaneous change at t =0 per minute during sleep due to awakening during the night due to something in the environment, controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, metabolic syndrome, fitness, family history of hypertension, having a non-typical night sleep, sharing a room/bed, and awakening for other reasons

γ_{121} = expected difference in instantaneous change at t =0 per minute during sleep due to awakening during the night due to using the bathroom, controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, metabolic syndrome, fitness, family history of hypertension, having a non-typical night sleep, sharing a room/bed, and awakening for other reasons

γ_{122} = expected difference in instantaneous change at t =0 per minute during sleep due to awakening during the night due to a reason other than the monitor, environment and bathroom, controlling for the effects all demographic factors, school attendance, substance use, stress, obesity, metabolic syndrome, fitness, family history of hypertension, having a non-typical night sleep, sharing a room/bed, and awakening for other reasons

$\pi_{2i} = \gamma_{20}$

γ_{20} = expected rate of change of instantaneous change in an individual blood pressure per minute during sleep