Associations Between Blood Pressure and Sleep: A Comprehensive Examination of Pediatric Blood Pressure Measurement and Objective Sleep Dimensions

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ABSTRACT

Associations between blood pressure and sleep: A comprehensive examination of pediatric blood pressure measurement and objective sleep dimensions

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Hypertension is a significant public health concern in both adult and pediatric populations. With increasing prevalence of hypertension, there is growing emphasis on identifying risk factors for high blood pressure. Poor sleep has emerged as a significant predictor of hypertensive status. In particular, short sleep duration has been widely studied as a risk factor for cardiovascular disease, including hypertension. There is evidence that other dimensions of sleep, such as sleep disruption or timing, may influence blood pressure, but relatively few studies examine these associations. The objective of the present dissertation was to examine associations between blood pressure and specific sleep dimensions. Study 1 consisted of a scoping review of pediatric ambulatory blood pressure monitoring (ABPM) measurement practices and meta-analysis of diurnal variation in pediatric blood pressure. Studies showed moderate fidelity to current measurement standards, but there was significant variability in data reporting. Youth showed pronounced diurnal variation in blood pressure, supported by significant differences in day and night blood pressure. Study 2 was a meta-analysis examining associations between casual blood pressure (i.e., in-office recording) and the following sleep dimensions in children and adults: sleep duration, awakenings, sleep efficiency, sleep irregularity, and sleep stages. Higher blood pressure was associated with shorter sleep duration, more frequent awakenings, and poorer overall sleep. There was evidence of changes across age, with older samples showing stronger associations between blood pressure and sleep. Study 3 examined associations between casual

blood pressure and objectively measured sleep dimensions (i.e., actigraphy, polysomnography) in a community sample of children. Higher blood pressure was significantly associated with shorter sleep duration, later bedtime, more frequent arousals (i.e., sleep disruption), and less time spent in restorative sleep stages. Overall, findings from this dissertation indicate that blood pressure is associated with multiple dimensions of sleep from early in the lifespan. Further research is needed to examine how these associations progress over time in order to develop effective prevention and interventions for hypertension.

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Contribution of Authors

This thesis consisted of three manuscripts. The contributions of each co-author are outlined below:

Study 1: Rowe, H., Noel, N. A. O., Paradis, G., Zappitelli, M., & McGrath, J. J. Twenty-four-hour ambulatory blood pressure monitoring in children and adolescents: Scoping review of fidelity to measurement guidelines & meta-analysis of pooled pediatric ABPM data.

Hillary Rowe conducted systematic literature searches, reviewed previous research studies, conducted statistical analysis, and wrote the draft of the manuscript. J. McGrath supervised the literature review, meta-analytic procedures, and contributed to editing the manuscript. N. Noel coded studies for psychometric validation of the extraction of data. G. Paradis and M. Zappitelli assisted with study conceptualization and provided guidance on blood pressure measurement.

Study 2: Rowe, H., & McGrath, J. J. Associations between casual blood pressure and objective sleep dimensions: A meta-analysis.

H. Rowe conducted systematic literature searches, reviewed previous research studies, conducted statistical analysis, and wrote the draft of the manuscript. J. McGrath supervised the literature review, meta-analytic procedures, assisted with article coding, and contributed to editing the manuscript.

Study 3: Rowe, H., & McGrath, J. J. Associations between casual blood pressure and objective sleep in a community sample of youth.

H. Rowe formulated research questions and study objectives, cleaned data and conducted statistical analysis, and wrote the draft of the manuscript. J. McGrath supervised data collection, assisted with study formulation, provided input on analyses, and edited the manuscript.

All authors reviewed the final manuscript and approved the contents.

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GENERAL INTRODUCTION

The present research program is a comprehensive investigation of the associations between blood pressure and different sleep dimensions. The dissertation will begin with a general introduction to provide an overview of background information in the following areas: epidemiology of hypertension, physiological mechanisms linking blood pressure and sleep, assessment of blood pressure in sleep research, and associations between blood pressure and sleep dimensions. I conducted three interrelated studies. Study 1 included a scoping review of measurement practices in ambulatory blood pressure monitoring (ABPM) and meta-analysis of diurnal variation in pediatric blood pressure. Study 2 was a meta-analysis of associations between blood pressure and sleep dimensions in children and adults. Study 3 examined blood pressure and objectively measured sleep dimensions in a community sample of healthy children. The manuscripts will be presented in sequence, with transition sections between studies to explain context and implications. Explanations of key terms and nuances (e.g., casual vs. resting blood pressure) will be provided throughout the sections. Finally, I will conclude with a general discussion to connect findings and provide future directions for this work.

Epidemiology of Hypertension

Hypertension (i.e., chronically elevated blood pressure) is a major public health concern. (Angell et al., 2015; Fisher & Curfman et al., 2018; Mittall & Singh, 2010). Along with increasing obesity rates, hypertension has experienced a dramatic global increase (Nguyen & Lau, 2012). Over 30% of adults worldwide meet diagnostic criteria for hypertension, representing an estimated 1.4 billion people affected across 90 countries (Mills et al., 2016). Hypertension was previously considered a rare diagnosis in children, but its incidence has paralleled rising childhood obesity rates (Flynn, 2013). A meta-analysis of 47 pediatric studies

indicated that the prevalence of hypertension (i.e., systolic or diastolic blood pressure greater than or equal to 95th percentile by age, sex, and height) increased from 1.26% between 1990 and 1999 to 6.02% between 2010 and 2014 (Song et al., 2019). Pediatric hypertension has been identified as an area of concern by experts in the field as childhood blood pressure is a strong predictor of blood pressure status in adulthood. Childhood blood pressure tends to "track" into adulthood, indicating that children tend to maintain the same relative blood pressure percentile over time (Li et al., 2009). As such, children with higher blood pressure are at higher risk of hypertension and cardiovascular disease long-term. Further, there is evidence that adverse effects of elevated blood pressure are already visible in childhood, including signs of cardiac structural changes and target organ damage (Flynn et al., 2022). Youth rarely experience death or severe cardiovascular disease (e.g., heart attack, stroke) resulting from hypertension, but the likelihood of developing these conditions increases if childhood hypertension is not detected and treated (Falkner et al., 2010).

Hypertension has been named as the leading preventable cause of cardiovascular disease and premature death worldwide (Mills et al., 2020). There is large body of research evaluating potential risk factors and targets for prevention and intervention strategies. Poor sleep has emerged as a strong predictor of elevated blood pressure and hypertension among adults. Insufficient quantity and quality of sleep are increasingly common across nations, with up to one-third of adults reporting poor sleep (Grander, 2019). Trends in sleep have been primarily assessed in population-based samples reporting subjective sleep quality, sleep duration, and sleep problems or disorders (Garland et al., 2018; Rowshan Ravan et al., 2010). Sleep duration, in particular, has decreased significantly in recent decades (Caraballo et al., 2022; Keyes et al., 2015; Sheehan et al., 2019). Poor sleep has been linked to greater all-cause mortality and

increased risk for cardiometabolic health conditions, including hypertension, metabolic syndrome, cerebrovascular disease, and obesity (Bixler, 2009; Fernandez-Mendoza et al., 2017). Increased mortality associated with insufficient sleep has been attributed to the critical roles of sleep in regulating cardiovascular functions, hormone release, autonomic functioning, and glucose homeostasis (Bixler, 2009).

Physiological Mechanisms Linking Blood Pressure and Sleep

Daily blood pressure patterns are closely linked to the 24-hour sleep-wake cycle (Smolensky et al., 2007). Blood pressure normally follows a distinct diurnal rhythm characterized by a nocturnal decrease (i.e., blood pressure dipping) and morning peak. In healthy adults, nocturnal dipping is defined as a decrease in blood pressure of 10 to 20% during the night; in contrast, a decrease of less than 10% indicates a lack of dipping (i.e., non-dipping; Loredo et al., 2004). Nocturnal blood pressure and dipping status have been proposed as strong predictors of hypertensive status in adults as blood pressure is continuously elevated throughout the 24-hour period (Yano & Kario, 2012).

Blood pressure variation is regulated by many of the same 24-hour circadian rhythms and environmental factors that influence sleep and other biological functions. Circadian rhythms are regulated by the suprachiasmatic nucleus (SCN) of the brain and are driven by clock genes. The central circadian clock consists of feedback loops in which transcription factors CLOCK and BMAL1 receive feedback from PER1/2/3 and CRY1/2 transcription and translation (Wang et al., 2023). These processes occur even without external input but are also entrained by environmental cues and behaviour. Timing of light exposure, caloric intake, physical activity, and body position can alter circadian rhythms. As such, circadian rhythms can become desynchronized when behaviour (i.e., short sleep duration, irregular sleep timing) does not

coincide with endogenous circadian processes (Scheer et al., 2009). Circadian misalignment has been linked to poor cardiovascular health, including hypertension, through the disruption of autonomic, metabolic, and endocrine functioning (Morris et al., 2016).

Assessment of Blood Pressure in Sleep Research

Blood pressure has traditionally been measured in office and laboratory settings, which is commonly referred to as "casual blood pressure", or "resting blood pressure" when the measurement is taken following a baseline period. (Note that, despite its name, "resting" blood pressure has nothing to do with sleep.) Recent clinical guidelines suggest that blood pressure should be measured repeatedly across a 24-hour period using ambulatory blood pressure monitoring (ABPM; Flynn et al., 2022). This is relevant for sleep research, as variations in blood pressure typically co-occur with sleep-wake patterns. There has been a shift toward emphasizing ABPM and nocturnal blood pressure data as they provide information on daily patterns, rather than isolated single measurement recordings.

Despite the advantages, there are limitations and methodological challenges associated with ABPM. First, 24-hour blood pressure recording is far more invasive and time-consuming than casual blood pressure recording. The individual must continuously wear the monitor for at least 24 hours, during which the cuff inflates at approximately 20- to 60-minute intervals to take readings (Flynn et al., 2022). There is evidence that ABPM can disrupt sleep, which may be problematic when conducting research on sleep and blood pressure (Gaffey et al., 2021). Additionally, there is significantly less existing ABPM data compared to casual blood pressure, particularly in children. Normative data for pediatric ABPM are based on two homogeneous samples, while casual blood pressure data are available from large, representative samples from diverse geographic regions (Wühl, et al., 2002; Yip et al., 2014

Blood pressure is most commonly evaluated using measurements taken by trained professionals in office, clinic, or laboratory settings; again, these recordings are called casual, office, or resting blood pressure. Casual blood pressure is distinct from nighttime or nocturnal blood pressure or blood pressure dipping. Comparisons of casual blood pressure and ABPM have yielded inconsistent results; some studies indicate significant discrepancies between the methods, while others show comparable results (Koch et al., 2000; Nishibata et al., 1995). There have been concerns that casual blood pressure is less accurate than ABPM in diagnosing hypertension, as it cannot capture blood pressure dipping, nocturnal hypertension, or masked hypertension (i.e., normal blood pressure in office but elevated blood pressure out of clinic; Flynn et al., 2022; Pickering et al., 2007). Nevertheless, casual blood pressure remains the most common method of identifying elevated blood pressure. Casual blood pressure measurement is easily accessible compared to ABPM, and most hypertension diagnoses are made using office recordings. With the ubiquity of casual blood pressure measurement, it is important to evaluate whether it is associated with sleep and other potential risk factors for hypertension. Given that blood pressure fluctuates across time and settings, it is important to understand how different types of measurement may be associated with sleep dimensions.

Association Between Blood Pressure and Sleep Dimensions

Sleep duration (i.e., the amount of time between sleep onset and final awakening) is the most common sleep dimension in research due to its straightforward measurement. There is considerable evidence that short sleep duration is associated with higher blood pressure. Both self-reported and objectively measured short sleep have been linked to increased risk for hypertension in adults and children (Bathgate & Fernandez-Mendoza, 2018). Experimental studies have also shown that partial sleep restriction (e.g., restricting sleep to 4-6 hours per night,

compared to 8 hours) is directly associated with higher blood pressure (Banks & Dinges, 2007; Lusardi et al., 1999). It is thought that short sleep influences blood pressure through disrupted autonomic balance, increased adiposity and metabolic changes, and disrupted circadian rhythmicity (Morris et al., 2016). Specifically, shorter sleep is associated with reduced parasympathetic activity and shift toward sympathetic dominance, leading to higher heart rate, greater orthostatic reactivity, and lower high-frequency heart rate variability during sleep and after awakening (Banks & Dinges, 2007; Reichenberger et al., 2023). Shorter sleep duration may also mean that less time is spent in restorative physiological processes, such as parasympathetic dominance and deeper sleep stages (i.e., stage N3 or slow-wave sleep).

There is evidence that other sleep dimensions may play important roles in the regulation of blood pressure patterns. For example, irregularity of sleep duration (e.g., sleeping for different amounts of time from night to night) and sleep timing (e.g., going to bed at different times) have shown relations with higher blood pressure. Sleep patterns are closely tied to the 24-hour blood pressure profile, and changes in sleep often align with alterations in blood pressure patterns – *both* blood pressure during the *day* and blood pressure at *night*. Irregular sleep patterns can lead to misalignment of the circadian rhythms that regulate blood pressure and other cardiovascular processes, increasing risk for hypertension over time (Scheer et al., 2009). Circadian misalignment has been associated with several risk factors for hypertension, including blunted blood pressure dipping and elevated nocturnal blood pressure (Morris et al., 2016). Additionally, studies on sleep fragmentation (i.e., sleep disruption by nocturnal awakenings) indicate that more frequent sleep disruptions and arousals (i.e., brief 3 to 15-second increases in brain activity) are associated with hypertension as the individual experiences spikes in blood pressure each time they wake throughout the night (Dean et al., 2015; Ren et al., 2022). The proportion of time

spent in specific sleep stages has also been associated with blood pressure. Sleep is composed of four stages (stage N1, N2, N3 and rapid eye movement or REM; Carskadon & Dement, 2005). Stages N1 through N3 are known as non-REM sleep; non-REM sleep comprises about 75% of sleep and reflects increasingly deeper sleeps. Stage N3, or slow-wave-sleep (SWS), is the deepest, most restorative stage of sleep. REM is not considered to be restorative sleep and is characterized by muscle atonia, rapid eye movements, and increased brain activity. Experimental studies indicate that reduced stage N3 sleep is associated with increased sympathetic activity and reduced parasympathetic activity (Tasali et al., 2008; Zhong et al., 2005). Consequently, reduced time spent in N3 sleep leads to higher blood pressure and heart rate during a greater proportion of the sleep period. Greater sleep fragmentation may contribute to this finding, as frequent awakenings may prevent progression into deeper, more restorative stages of sleep.

Current Program of Research

The aim of the current program of research was to comprehensively examine the associations between blood pressure and various sleep dimensions. There is increasing evidence that sleep is associated with blood pressure, and that poor sleep is a significant risk factor for hypertension. Three complementary studies were conducted to evaluate blood pressure measurement practices, summarize and synthesize the literature on blood pressure and sleep, and comprehensively examine associations between blood pressure and various sleep dimensions. **Study 1** was a scoping review of measurement practices and fidelity to published guidelines in pediatric ABPM research. As part of this study, a meta-analysis was conducted exploring diurnal variation and the role of methodological and demographic effect modifiers on day and night blood pressure in youth. **Study 2** consisted of a meta-analysis examining the associations between casual blood pressure and sleep assessed using objective sleep measures. Focus was on

including multiple sleep dimensions (i.e., sleep duration, sleep efficiency, awakenings, irregularity, sleep stages) and studies from childhood into middle adulthood. **Study 3** built on the previous studies to examine the associations between sleep and casual blood pressure in a community sample of children and adolescents. Multiple sleep dimensions were assessed using objective sleep measures (i.e., actigraphy and polysomnography) to ensure comprehensive sleep measurement.

Study 1:

Twenty-Four Hour Ambulatory Blood Pressure Monitoring in Children and Adolescents: Scoping Review of Fidelity to Consensus Measurement Guidelines & Meta-Analysis of Pooled Pediatric ABPM Data

Hillary Rowe, MA, Neressa A. O. Noel, BSc RPSGT, Gilles Paradis, MD, Michael Zappitelli, MD & Jennifer J. McGrath, PhD MPH

Introduction

Pediatric hypertension is a growing public health concern. Although previously rare, the increased incidence of diagnosed hypertension in children parallels rising childhood obesity rates (Flynn et al., 2014). Between 2000 and 2015, the global prevalence of hypertension more than doubled in youth aged 6 to 19 years, increasing from 2-3% to 5-8% (Song et al., 2019). Precursors to cardiovascular disease emerge early in the life course (Berenson et al., 1998; Mahoney et al., 1991). Childhood blood pressure "tracks" into adulthood (tracking refers to maintaining the same relative position within age- and sex- groups over time), placing hypertensive youth at greater risk for adverse long-term cardiovascular outcomes (Chen & Wang, 2008; Li et al., 2009). Persistently high blood pressure from childhood and adolescence is linked to the development of target-organ damage with alterations in carotid intima-media thickness and left ventricular hypertrophy, as well as premature mortality (Yang et al., 2020). Further, adults with a history of resolved childhood hypertension do not show the same risks, highlighting the importance of accurate measurement, diagnosis, and treatment.

Blood pressure is routinely measured using office, or casual, blood pressure recordings with the auscultatory method (Jones et al., 2003). The American Heart Association's Ambulatory Blood Pressure Monitoring in Children and Adolescents 2022 Update recommend its use to confirm the diagnosis of hypertension in youth (Flynn et al., 2022). A key advantage of ABPM over clinic measurements is the ability to assess nocturnal blood pressure and 24hr variability (Mancia, 2023). Blood pressure follows a distinct diurnal rhythm characterized by a nocturnal decrease of 10 to 20% (i.e., dipping) and morning surge. Lack of blood pressure dipping is associated with greater risk of cardiovascular disease, cerebrovascular disease, and target-organ damage due to sustained elevation of blood pressure at night (Cuspidi et al., 2004; Otsuka et al.,

1996). High rates of non-dipping are observed in adults with chronic kidney failure, sleep disorders, autonomic dysfunction, and endocrine disorders, among other health conditions (Cuspidi et al., 1999; Loredo et al., 2004; Mitsnefes et al., 2003; Okamoto et al., 2009). Nocturnal dipping and absolute values during sleep are more robust predictors of cardiovascular events than wake blood pressure (Mancia, 2023). Circadian timing is closely linked to 24hr blood pressure variability and has emerged as a significant risk factor in the development of cardiovascular disease, highlighting the importance of examining diurnal variation in ABPM (Morris et al., 2016; Thosar et al., 2018).

ABPM has been used in child research and clinical settings for several decades, but the first youth-specific ambulatory measurement recommendations were only published in 2008 (Urbina et al., 2008). In its Global Hypertension Practice Guidelines, the International Society of Hypertension noted a recent "flurry of updated evidence-based guidelines" across countries, most of which recommend the use of ABPM and specify measurement guidelines for children and adolescents (Unger et al., 2020). Prior to this, identification of hypertension in youth was primarily based on casual (i.e., office) blood pressure data. Comparison of recent pediatric guidelines are outlined in Table 1. These guidelines outline largely consistently measurement recommendations for using ABPM in youth related to selection of validated devices, recording specifications (duration, frequency), data reporting, and interpretation. It remains unknown the extent to which there is fidelity with these measurement guidelines when evaluating blood pressure in youth.

Age-specific percentile curves of normative casual blood pressure were reported as early as the First Task Force Report on Blood Pressure Control in Children (Blumenthal et al., 1977). Yet, normative data for pediatric ABPM remain strikingly absent. Despite repeated criticisms of

the lack of reliable ABPM reference data (Flynn et al., 2022), many cohort studies using ABPM in children and adolescents (e.g., Pearce & O'Sullivan, 2006; So et al., 2016), and calls for a cumulative pediatric ABPM database repository (Sorof & Portman, 2001), data from only two studies are predominantly cited (cf. Lurbe et al., 2016). The most referenced normative values for ABPM in children and adolescents were recorded three decades ago (1993-1996) by the German Working Group on Pediatric Hypertension (Wühl et al., 2002). The only other commonly cited source for age- and sex-specific 24-hour blood pressure percentile distributions was conducted with Chinese children and adolescents in Hong Kong (Yip et al., 2014).The generalizability of these data has been questioned due to their homogeneous samples and limited sampling characteristics (e.g., no children <120cm in height, healthy students with no history of diseases known to affect BP; Flynn et al., 2022; Macumber, 2017; Patel & Daniels, 2019). Relatedly, the sample used by Wühl and colleagues showed low age-related variation in diastolic blood pressure, which has added to challenges in establishing appropriate thresholds. The American Heart Association outlined gaps in pediatric ABPM emphasizing the lack of normative data and the need for comparing ABPM values with data from large pediatric populations (Flynn et al., 2022).

The present study aimed to empirically evaluate the extant literature on ABPM in community samples of children and adolescents. Our objectives were (i) to systematically review fidelity with current measurement guidelines, (ii) to compile pediatric ABPM data across studies, (iii) to evaluate the effects of modifiers on pediatric blood pressure, and (iv) to evaluate diurnal variation in pediatric ABPM.

Method

Systematic literature search

An electronic literature search was conducted using EBSCO's Academic Search Complete database (MEDLINE, Psychology and Behavioral Sciences Collection) for articles published from January 1970 to October 2023. The complete Boolean expression for the search keywords (e.g., "ambulatory blood pressure" AND [child* OR adolescen* OR youth OR pediatric OR paediatric]) is presented in Figure 1. Title and abstract screening yielded 118 initial articles. Ascendancy and descendancy approaches were used to identify an additional 34 articles, yielding 152 potential articles for full-text review.

Article inclusion criteria and selection

Inclusion criteria required ≥24-hour ABPM in children or adolescents recruited from clinics (e.g., routine pediatrician visits), community, or school settings. Age was restricted to 5 to ≤18 years (mean age of sample). Only English-language publications were reviewed. Exclusion criteria included recruitment from hospital, inpatient, or specialized medical clinics combined with diagnosed condition (e.g., chronic kidney disease, diabetes, previous organ transplant), case studies, clinical guidelines, or literature reviews. Of the 152 full-text reviewed articles, 73 met inclusion criteria and reported relevant data. To address the issue of multiplicity, cohort names, recruitment dates, study location, and authorship were screened to ensure only non-redundant data were included. After distilling these 73 articles, there were 41 pediatric cohorts represented by unique information presented across the articles. Forty-one articles were included in the systematic fidelity review and meta-analysis (see Table 1). The completed literature search strategy in accordance with PRISMA guidelines is presented in Figure 1.

Data extraction

Sample Characteristics. Sample size, demographics (e.g., country / geographic region, age, sex/gender, ethnoracial group), anthropometrics (e.g., height, weight, BMI), recruitment

setting (i.e., school, community), and family history of blood pressure problems (i.e., reported parental/grandparent history) were extracted from each study. Few studies reported BMI Z-scores or percentiles; only BMI (kg/m^2) was coded. Ethnoracial group was coded; only percent Non-white/Caucasian participants could be derived and harmonized across studies due to limited reporting. Socioeconomic status, sleep timing and duration, smoking, caffeine intake, waist circumference, and pubertal stage were coded preliminarily, but ultimately could not be included due to minimal reporting.

ABPM Measurement Guidelines. Measurement variables aligned with existing ABPM pediatric guidelines (see Table 2) were coded to evaluate measurement fidelity. Coded variables included device information (method: oscillometric / auscultatory; model: validated / non-validated, e.g., SpaceLabs 90207); recording specifications (duration: length in hours; sampling frequency: day, night; intervals: fixed / random); definition of day versus night period (sleep wake diary / pre-defined periods, e.g., 8:00 to 22:00); and, data quality (minimum number of recordings; percent successful recordings; use of artifacts-outlier cutoffs). Explicit mention of these recording specifications was required to meet the criterion of compliant.

Blood Pressure Values. Systolic and diastolic ABPM means and standard deviations were coded for 24hr, day, night, and noctural dipping (harmonized definition: [day BP – night BP)/day BP] x 100). If mean and standard deviation data were not reported, other metrics were coded (e.g., median, standard error, IQ1 25^{th} percentile, IQ3 75^{th} percentile) and mathematically harmonized (cf., Wan et al., 2014). Other dipping and blood pressure load values were reported but could not be aggregated due to disparate formulas used (e.g., night:day ratio; percent change).

Two additional blood pressure metrics were coded given the objective to yield pooled pediatric data: (i) Casual BP means were extracted when available. Casual BP was defined as daytime recordings in a clinic, office, or laboratory; home recordings were not included. (ii) Diurnal variation was estimated on an exploratory basis. Hedges' *g* effect size was calculated to yield the magnitude of the group-level difference between day versus night BP. (This is to be distinguished from individual-level nocturnal dipping, which cannot be calculated from group means.) Hedges' *g* (standardized mean difference with pooled standard deviation) appropriately corrects for biases due to small or unequal sample sizes. Hedges' *g* is interpreted similarly to a Cohen's *d*, which is suitable for population-representative data.[31] Thus, a Hedges' g of -1.0 would provide evidence of diurnal variation such that a cohort's blood pressure would be one standard deviation unit lower at night compared to the day.

Coding Reliability. Over 40 coding decisions were required for each article. Articles were coded by three reviewers (HR, SM, JM). Inter-rater reliability with a fourth independent reviewer (NN) was excellent (Intraclass Correlation Coefficient, *ICC*=.99); and intra-rater reliability after a 9-month delay was excellent (*ICC*=.99). Two reviewers (JM, HR) traced the ancestry of cohorts to streamline overlapping articles. When multiple articles reported data for the same cohort, the article with the largest sample size reporting information for the coded variable of interest was retained. Data were coded such that participants were only represented once in any given meta-analytic model.

Analytic Strategy

Analyses were performed using SPSS (Version 29), R (version 4.2.1; packages metafor, meta, ggplot2), and MetaWin (version 2). Fidelity with measurement guidelines was tabulated to yield rates of compliance for each consensus guideline. Meta-regression was performed using

mean ABPM values from all studies to estimate inverse-variance-weighted pooled statistical summaries and 95% confidence intervals. Mixed-effects models with random effects were conducted for each recording period (day, night, 24hr, nocturnal dipping) and other metrics (casual BP, diurnal variation). Random effects meta-analytic models (REML estimator) were selected as they allow for the possibility of natural variation among effect sizes and are recommended over fixed effect models when there is evidence of heterogeneity (Borenstein et al., 2010; Hedges & Vevea, 1998). Cochran's Q_T, Higgins & Thompson's I^2 statistic (Higgins & Thompson, 2022), and tau were used to estimate heterogeneity. To evaluate potential publication bias, Orwin's method, Trim-and-Fill plots, and Egger's test were calculated; results were largely consistent and Egger's test p value is presented for parsimony.

Effect modifiers were tested using moderator analyses to examine *a priori* possible sources of heterogeneity. Model stratification by sex, age, and height was precluded due to limited reporting of original data; instead, these variables at the cohort-level were considered as effect modifiers. Moderators included sample characteristics (age, sex, height, BMI, race, geographic region, family history, recruitment setting) and measurement variables (device model, measurement method, definition of day / night intervals, duration of recording, recording frequency). Random-effect meta-regression analyses were used to test continuous (i.e., β slope, Q_E) and categorical (i.e., Q_M) moderator variables. One moderator analysis used fixed effect meta-analytic models (i.e., measurement method: oscillometric or auscultatory), as coded data represent all possible options. Forest plots were used to compile pooled pediatric cohort data.

Sensitivity analyses were conducted using Bayesian modeling approaches (R brms package) as well as estimating bootstrapped 95% confidence intervals (R metafor package, boot

library) for all models. All sensitivity estimates were robust, yielding negligible differences (<0.10 mmHg) from original estimates, and are not presented for parsimony.

Results

Descriptive characteristics are summarized in Table 3. There were a total of 9,535 participants aged 7.5 to 17.6 years (M_{age}=12.98, SD=2.61) among the 41 pediatric cohorts. Males and females were generally equally represented among the articles reporting sex (M=51.88% male, SD=11.31; k=36); none reported percent non-binary nor distinguished sex from gender. Samples included 67.67% non-White participants (SD=26.50; k=21). Most cohorts were recruited from schools (70.73%) across North America (43.9%), Europe (26.8%), Asia (19.5%), South America (4.9%), and Oceania (New Zealand, 2.4%).

Guideline Fidelity

Ambulatory blood pressure was predominantly measured using the oscillometric method of measurement (73.2%). On average, studies reported day intervals from 7:10AM to 21:56PM, with night intervals from 22:33PM to 6:34AM. Recordings were taken every 22.23 minutes during the day and 38.99 minutes at night. Recording specifications are presented in Table 3. In terms of fidelity to measurement guidelines (see Table 4), about half of the studies reported cuff placement on the non-dominant arm (61.0%) and use of an appropriately-sized cuff (51.2%). Almost all studies reported recording duration of 24 hours (95.1%) and sufficient recording frequency during the day and night (87.8%). Most studies reported separate day (90.2%) and night (87.8%) blood pressure; fewer studies reported 24-hour blood pressure (61.0%). Blood pressure dipping and blood pressure load were rarely reported, at 12.2% and 17.1% of studies, respectively. The day and night periods were defined using individual-level sleep patterns (i.e., timing unique to each participant using sleep diary logs) in 31.7% of the studies, while an

additional 17.1% applied ubiquitous transition times for all participants between the day and night periods to ensure separation. Few studies met guideline recommendations on minimum number of recordings (40 recordings over 24 hours; 9.76%) or minimum percentage of successful recordings (70% successful; 14.6%). Similarly, only 19.5% of studies described data handling procedures or exclusion decisions for blood pressure values outside the normal range.

Compiled Pediatric ABPM Data

Pooled cohort data are provided in Table 5. See Appendix A for forest plots. Twenty-four-hour means were 114.96 mmHg for systolic and 66.20 mmHg for diastolic blood pressure. Daytime ambulatory blood pressure means were 117.46 mmHg systolic and 69.93 mmHg diastolic. Nighttime means were 105.03 mmHg systolic and 57.83 mmHg diastolic. Among the five studies that reported blood pressure dipping, mean blood pressure was lower at night than during the day for both systolic (10.01% lower) and diastolic blood pressure (16.81% lower). Exploration of diurnal variation at the group-level revealed cohorts had significantly lower blood pressure at night for both systolic (Hedges' g=-1.38) and diastolic blood pressure (Hedges' g=-1.97; n.b., Hedges' g=-1.00 would indicate a one standard deviation unit lower blood pressure at night than during waking). All meta-analytic models yielded statistically significant Q values and large I^2 values, indicating that the pooled cohort data were heterogeneous and further moderator analyses were warranted.

Effect Modifiers

Meta-regression models with continuous effect modifiers are presented in Table 6 (Appendix B). Older age was significantly associated with higher 24-hour (see Figure 2), day, and night systolic blood pressure and nocturnal diastolic dipping. Age was a robust predictor of between-study heterogeneity (see R^2 in Table 6), especially for nocturnal diastolic dipping

(98.2% heterogeneity explained). Greater proportion of non-White participants was associated with decreased systolic diurnal variation. Taller height was significantly associated with higher 24-hour, day, and night systolic blood pressure, while larger weight was significantly associated with higher day and night systolic blood pressure; height and weight were not associated with any diastolic blood pressure variables. Higher BMI was significantly associated with higher day and night systolic and diastolic blood pressure. Higher casual SBP was significantly associated with higher 24-hour systolic blood pressure, while higher casual DBP was significantly associated with lower night systolic blood pressure.

Among the ABPM recording specifications, later end time of the day interval and later start time of the night interval were significantly associated with greater nocturnal diastolic dipping and accounted for all between-study heterogeneity. Later end time of the night interval was significantly associated with lower 24-hour, day, and night systolic blood pressure, higher nocturnal systolic dipping, and lower 24-hour diastolic blood pressure; these associations were not found for the start time of the day interval. More frequent sampling during the day was significantly associated with lower diastolic diurnal variation.

Meta-analytic models with categorical effect modifiers are presented in Table 7 (see Appendix C). Region was a significant moderator for 24-hour and night systolic blood pressure and nocturnal diastolic dipping. Cohorts conducted in North America had higher 24-hour systolic blood pressure than cohorts in Europe and Asia, and higher night systolic blood pressure than those in South America, Europe, and Asia. Cohorts in Asia had higher nocturnal diastolic dipping than cohorts in South America. For all ABPM systolic and diastolic variables, oscillometric devices yielded lower blood pressure values than ausculatory devices; however, I^2

and significant Egger test *p* values imply these findings should be interpreted with caution due to substantial heterogeneity.

Discussion

The primary objectives of this comprehensive research synthesis were to systematically examine fidelity to measurement guidelines, to compile pooled pediatric ABPM data, to consider effect modifiers of pediatric blood pressure, and to evaluate diurnal variation in ABPM.

Measurement Fidelity with Guidelines

Overall, ABPM measurement across the pediatric cohorts was only moderately compliant with current consensus guidelines. Concordance was most evident for recommendations on use of validated devices, recording specifications (≥24hr duration; sampling frequency), and reporting of day and night blood pressure. The oscillometric method of measurement was most commonly used. Readings were primarily taken at fixed intervals every 20-30 minutes during the day and 30-60 minutes at night. Most devices have automated settings for the sampling interval (although users can manually override interval setting). Fidelity with data reporting was less consistent. Most studies reported day and night blood pressure separately as recommended by current guidelines (Flynn et al., 2022). An additional 17.1% of the cohorts discarded transition periods of approximately two hours at the start and end of the night interval. The decision to eliminate transition periods can reduce the risk of inadvertently including sleeping time within the day interval; however, this lack of precision interferes with accurate identification of the night interval and creates data loss. Reporting of data quality was similarly low. Most studies did not report the minimum number or percentage of successful readings or methods of handling extreme outliers. This makes interpretation of the results difficult, as we cannot ascertain the quality of the data used in the analyses. Data quality was evaluated based on the most recent

published measurement standards, which may be seen as a limitation because many articles were published before the guidelines. However, most of the recommendations are similar across time points and the greater issue is the consistent lack of reporting on data quality in both recent and older studies.

Most cohorts selected validated measurement devices and programmed them to collect data at an appropriate sampling frequency for sufficient duration. However, there was significant room for improvement in user decision-making. The unmet guideline recommendations were primarily those that require the user to make decisions on data partitioning, data cleaning, and data reporting, as well as more individualized measurement practices (e.g., cuff sizing, sleep diaries). Many of the variables requiring user-based decisions seemed to influence blood pressure values, especially the type of blood pressure measurement (see below) and timing of day and night intervals. This raises concerns since expert recommendations are inconsistently applied in these areas, and many studies do not disclose their pertinent measurement decisions. It is quite likely that this may be due to space limitations in articles; however, to advance the field, it is important to ensure that these specifications are included. One suggestion would be to report this technical information using article supplements or adopt the use of a standardized statement indicating the chosen measurement guideline and the number of criteria met. Implementing reporting standards would promote better adherence to measurement guidelines and improve understanding of the data. This is particularly important since clinical decision-making and practice recommendations are informed by research. More detailed information on measurement decisions and data quality would allow for greater accuracy in measurement and identification of pediatric hypertension.

Several methodological variables were associated with blood pressure values and dipping. Studies using the oscillometric method showed consistently lower blood pressure and greater dipping and diurnal variation than those using auscultatory devices. Both measurement approaches are widely used and approved for use in children, but findings comparing the methods are mixed (Flynn et al., 2022; Duncombe et al., 2017; Liu et al., 2015). Ultimately, the device model should be considered when interpreting ABPM data as the measurement approach can affect the blood pressure values. This is particularly important when evaluating auscultatory data or harmonizing data across cohorts, as the existing normative values for casual blood pressure used the oscillometric method (Wühl et al., 2002; Yip et al., 2014). There may be value in conducting further validation studies to determine if consistent ABPM values can be derived with both measurement methods. In the meantime, perhaps an arithmetic conversion factor could be devised to transform values from one type of device into the other; this would be particularly helpful when compiling data from multiple cohorts. This may also facilitate comparison between ABPM and casual blood pressure values. Our findings showed that daytime ABPM values were somewhat higher than casual blood pressure. This has been observed in past pediatric studies, but it will be important to clarify the relation between ambulatory and casual blood pressure due to the prevalence of white coat hypertension (i.e., higher blood pressure in office) in youth (Koch et al., 2000; Krmar et al., 2015; Sorof & Portman, 2000).

Pooled Pediatric Cohort Data

The second objective of this study was to compile pediatric data for ambulatory blood pressure. To date, only two studies are used as quasi-normative ambulatory blood pressure data in pediatric populations (Wühl et al., 2002; Yip et al., 2014). These studies are the only available reference values for pediatric ABPM and are widely used despite criticisms of their

generalizability. We pooled data from 41 cohorts with over 9,500 children and adolescents to yield mean and standard deviation reference values for 24-hour, day, night, nocturnal dipping, and diurnal variation systolic and diastolic ABPM. Limited information in the literature precludes our ability to create normative reference values (i.e., reported data not stratified by age, sex, height; distributions rarely reported). Experts in the field of pediatric hypertension have called for larger population-representative pediatric cohort studies as the two studies used for current normative values lack diversity and generalizability across populations. However, there is an immediate opportunity for the timely creation of a data repository using previously recorded ABPM individual-level data that could be curated to develop normative age-, sex-, and height-specific percentile curves. Sorof and Portman (2001) acknowledged the importance of creating a database of pediatric ABPM data, but to our knowledge, this has not yet been curated. A pediatric repository would be an important contribution to the field and could fundamentally transform the way in which ABPM data are used and interpreted. It is important to note that for the current review, we intentionally selected broad community- and school-based studies and excluded medical and high-risk populations. Ultimately, it would be ideal to compile all available data to characterize pediatric ABPM data across healthy and clinical populations.

Blood Pressure Effect Modifiers

Findings from the meta-regression suggested that several variables influence pediatric blood pressure values and diurnal variation. Consistent with existing research, greater age, height, weight, and BMI were associated with higher ambulatory blood pressure (Falkner et al., 2006; Modesti et al., 1994; Regnault et al., 2014). Age and body size are known correlates of casual blood pressure in youth and these findings support the presence of this association in ambulatory blood pressure. Blood pressure percentiles for both casual and ambulatory blood

pressure are generally age-, sex- and height-specific as these factors are strongly correlated to blood pressure. Our findings indicated that age, height, and weight were associated with systolic blood pressure but not with diastolic blood pressure. This may be related to concerns about DBP assessment with ABPM. Most ABPM devices use the oscillometric method, which is less accurate for DBP, and the current ABPM reference values show minimal variability in DBP across age and height (Flynn et al., 2022; Liu et al., 2015; Wühl et al., 2002).

Additionally, geographic region was significantly associated with blood pressure, with North American studies reporting significantly higher blood pressure. This may be related to childhood obesity rates or larger populations of certain ethnic groups (Sorof et al., 2004). Interestingly, at the cohort-level, sex (percent male) and ethnoracial status (percent non-White) were not linked to any of the blood pressure variables. Previous studies indicate that sex differences in blood pressure are present from a young age (Dasgupta et al., 2006; Syme et al., 2009); however, due to the data available, we could not examine group differences between males and females. Of note, gender was not reported in any studies. Future studies should examine sex- and gender-based differences in pediatric ABPM since women are underrepresented in cardiovascular research and often have worse cardiovascular outcomes than men (Wenger, 2003; Woodward, 2019). There is evidence of racial differences in pediatric casual blood pressure, mainly in American samples (Brady et al., 2010; Rosner et al., 2009). The present review included studies from many countries with different ethnic compositions and conceptualizations of ethnicity, which may explain the lack of effect.

Diurnal Variation

Although most cohorts reported day and night means, only 12.2% provided data on blood pressure dipping. Since almost all studies derived day and night blood pressure, these data could

have been easily used to calculate dipping; although, many software programs do not automatically provide these data. The lack of reported day and night means precluded the evaluation of diurnal rhythms for many cohorts. Timing of the day / night intervals was established using set thresholds by the majority of studies; only 31.7% of cohorts used participants' sleep and wake patterns recorded in diaries to derive precisely timed intervals. This is concerning as the day and night intervals seemed to influence blood pressure values; dipping was lower in studies with later day end times and higher in studies with later night end time. Later night end time was also associated with lower systolic blood pressure. Due to the limited data, it is unclear if this finding reflects the effect of sleep timing on blood pressure. However, it is evident that the timing of the day and night intervals affects the blood pressure values, and ubiquitous cohort-wide day and night intervals should not be implemented without considering the potential impact of this methodological decision to discard data. More research is needed to compare the effects of using individual sleep timing versus fixed day and night intervals to derive day and night blood pressure. This is particularly important when working with pediatric data as sleep patterns change dramatically across development. Younger children require more sleep and often nap, while adolescents are typically phase-delayed and naturally prefer later sleep and wake times (Crowley et al., 2007; Gellermann et al., 1997). Using cohort-wide cut-off points does not capture individual development and variation in sleep patterns and is likely less accurate than recording participants' own sleep timing.

Dipping was positively associated with age, which is consistent with existing literature. Blood pressure dipping is present from childhood but seems to increase as adolescents mature into an adult blood pressure profile (Gellermann et al., 1997; Modesti et al., 1994). Dipping was also associated with day and night interval timing; later day interval end, and night start and end

times, were linked to higher dipping. This indicates that the definition of the day and night periods significantly affect the level of dipping observed. This finding likely reflects the use of transition periods and suggests that dipping is more evident when the day and night intervals are clearly separated. It is important to interpret these results with caution, however, given the low number of studies that reported dipping. It should also be noted that diurnal variation was an exploratory variable that used group differences between day and night blood pressure. The diurnal effects may have been more notable if more studies reported dipping rather than day and night means. Our analyses were limited considerably by the data reported in the literature; many studies used variables that could not be harmonized across studies (e.g., dipping load, thresholds). Nevertheless, our findings provide sufficient evidence that diurnal variation is present in pediatric blood pressure and should be investigated further with more comprehensive ABPM data. Future studies should consider how to best assess circadian variation in blood pressure; this would require advances in ABPM technology to allow measurement over several days combined with other circadian markers (e.g., core body temperature, dim light melatonin onset).

Additionally, relatively simple changes in data reporting and analysis could yield valuable information on other aspects of pediatric blood pressure beyond 24-hour means. For example, our results demonstrated a large, significant difference between day and night means of both systolic and diastolic blood pressure. This suggests that children experience substantial nocturnal reduction in blood pressure and provides promising evidence that blood pressure dipping is present early in life. However, to make inferences about typical dipping during childhood, it is necessary to calculate and report nightly dipping, rather than providing only group means for day and night blood pressure. Dipping is an important marker of cardiovascular

functioning that is closely linked to sleep patterns and many other biological functions. Blunted dipping is often observed in populations with disrupted circadian rhythms such as shift workers and those with sleep disorders (Lanfranchi et al., 2009; Yamasaki et al., 1998). Furthermore, non-dipping appears to have distinct risks beyond the effects of hypertension and is associated with increased risk for cardiovascular disease even in the absence of hypertension (Birkenhäger et al., 2007). The non-dipping blood pressure profile is not well understood, partly due to lack of reproducibility and consistency among studies (Stolarz et al., 2002). This perhaps underscores the need for improved reporting standards and adherence to measurement protocols. Open access data could increase availability for secondary analyses to examine blood pressure dipping. Finally, more advanced methods could also be used to retain the continuity of the data rather than comparing individual day and night means. Techniques such as cosinor analyses are preferred for circadian variation as they permit evaluation of rhythmicity (Jarczok et al., 2019).

Conclusion

Our findings represent an important contribution to the expanding field of blood pressure research as previous reviews and meta-analyses largely focused on adults or pediatric clinical samples. There is clearly a large body of research examining 24-hour blood pressure in youth, but many studies do not follow current clinical guidelines for recording and using ABPM data. This is particularly evident in measurement decision-making and data reporting and supports the need for improved reporting standards and transparency. Furthermore, normative ABPM values are lacking, and most studies cite normative data from two studies with limited generalizability. More data (reporting) on blood pressure are needed across diverse age ranges, regions, and racial groups to ensure that guidelines and normative values accurately reflect the populations in which they are used. Perhaps more importantly, there are clear opportunities for improvement in our

approach to pediatric ABPM. There are considerable existing data that could be used to create an ABPM data repository. This would then allow for the development of more representative reference values, as well as secondary analyses to examine relevant variables (e.g., dipping, diurnal variation) that are typically missing from pediatric ABPM studies. Standards should be introduced for reporting data and disclosing measurement decisions; this would facilitate the comparison and harmonization of data from different cohorts. Additional validation studies would further help in understanding the impact of methodological decision-making on ABPM data.

Table 1. *Summary of 41 Included Studies*.

Reference	Country	Sample size	Mean age (years)	Recruitment setting
Alpay et al., (2009)[52]	Turkey	179	10.80	School
Ayyavoo et al., (2014)[53]	New Zealand	85	8.71	Hospital
Barnes, (2016)[54]	United States	40	16.10	School
Barnes et al., (2004)[55]	United States	73	12.31	School
Barnes et al., (2012)[56]	United States	170	15,65	School
Berenson et al., (1993)[57]	United States	57	15.96	Not reported
Correia-Costa et al., (2016)[58]	Portugal	315	8.77	Hospital
Davis et al., (1996)[59]	United States	34	16.50	School
Egger et al., (1987)[60]	Switzerland	43	13.50	Not reported
Ewart et al., (2011)[61]	United States	167	14.00	School
Ewart & Jorgensen, (2004)[62]	United States	97	14.40	School
Gregoski et al., (2012)[63]	United States	162	15.07	School
Harshfield et al., (1994)[64]	United States	300	13.46	Community
Iturzaeta et al., (2018)[65]	Argentina	110	8.70	Hospital
Jefferies et al., (2003)[66]	New Zealand	44	7.50	Community
Li et al., (2009)[67]	United States	102	13.60	Hospital
Li et al., (2005)[68]	China	252	13.70	School
Lurbe et al., (2001)[69]	Spain	630	9.90	Hospital
Malbora et al., (2010)[70]	Turkey	79	13.89	School
Martikainen et al., (2011)[71]	Finland	231	8.15	Hospital
Meininger et al., (2004)[72]	United States	371	13.50	School
Meininger et al., (1998)[73]	United States	41	Not reported	School
Mezick et al., (2012)[74]	United States	246	15.70	School
Modesti et al., (1994)[39]	Italy	62	10.32	Hospital
Pearce & O'Sullivan, (2006)[75]	United Kingdom	937	11.33	School
Portman et al., (1991)[76]	United States	99	10.00	School
Rahiala et al., (2002)[77]	Finland	50	12.00	Hospital
Räikkönen & Matthews, (2008)[78]	United States	201	14.50	School
Reichert et al., (1995)[79]	Germany	352	11.45	School
Savoca et al., (2005)[80]	United States	82	17.00	School

Schusterova et al., (2013)[81]	Slovakia	44	13.50	Hospital
Sheveleva et al., (2018)[82]	Russia	190	14.60	Not reported
So et al., (2016)[83]	Hong Kong	1,385	12.88	School
Stergiou et al., (2010)[84]	Greece	82	13.07	School
Toker et al., (2015)[85]	Turkey	60	15.00	Hospital
Wang et al., (2006)[86]	United States	663	14.34	School
Wilson et al., (1988)[87]	United States	178	16.17	Community
Wilson et al., (2002)[88]	United States	56	13.30	School
Wing et al., (2010)[89]	Hong Kong	297	10.40	School
Yu et al., (2016)[90]	Hong Kong	38	12.20	School
Zhu et al., (2008)[91]	United States	972	17.60	Community, school

Table 2. *Comparison of Ambulatory Blood Pressure Monitoring Guidelines for Children and Adolescents*.

	Hypertension Canada[92]	**American Heart Association & American Academy of Pediatrics[9,93]**	**European Society of Hypertension[10,94,95]**
Device & Placement			
Device	Validated in children	Validated to ANSI/AAMI/ISO standards	Validated to AAMI/ISO standards for children (www.stridebp.org)
Arm	Non-dominant	Non-dominant	Non-dominant
Cuff Size	Selected based on length (cover 80-100% of arm circumference) and width (40% of arm circumference)	Appropriate size	Selected based on length (cover 80-100% of arm circumference) and width (40% of arm circumference)
Recording Specifications			
Sampling Frequency	Day: every 15-20 min Night: every 20-30 min	Wake: every 15-20 min Sleep: every 20-30 min	Every 15-30 min; At least 70% obtained every 30 minutes
Duration	≥24 hours	≥24 hours (18-20 hrs acceptable if captures sleep)	n/a
Day / Night Intervals			
Wake & Sleep Timing	Sleep/wake time diary	Sleep/wake time diary	Bedtime record
Interval Designation	Use reported sleep time (Can use set intervals if day/night transition time discarded)	n/a	Use reported sleep time (Can use set intervals if day/night transition time discarded)
Data Reported			
BP values	SBP, DBP by Day, Night, 24hr (Mean)	SBP, DBP by Day, Night, 24hr (Mean)	SBP, DBP by Day, Night, 24hr (Mean)
Dipping	([mean awake BP-mean sleep BP]/mean awake BP] ×100)	([mean awake BP-mean sleep BP]/mean awake BP] ×100)	Nocturnal decline (%)
BP load	Percentage readings >95th%ile ABPM	Percentage readings >threshold value	n/a
Data Quality			
Min. Readings	40-50 readings in 24hr ≥1 per hour (including sleep)	40-50 readings ≥1 per hour (including sleep)	20 readings day; 7 readings night ≥2 per hour day; ≥1 per hour night
Min. Successful Readings	n/a	70%	70%
Outliers & Artifacts	Discard values outside range: SBP 60-220 mmHg, DPB 35-120 mmHg	Discard values outside range: SBP 60-220 mmHg, DBP 35-120 mmHg Values considerably outside normal range for age	n/a
Use & Interpretation	Health professional with specialty training in ABPM device application for children & adolescents	Health professional with specialty training in ABPM device application & ABPM data interpretation for children & adolescents Compare values with sex- & height-specific ABP data from large pediatric populations	n/a

Note: [92]Rabi et al., 2020. [9]Flynn et al., 2022;[93]Flynn et al., 2017; [10]Mancia et al., 2023; [94]Parati et al., 2014; [95]Lurbe et al., 2016. ANSI=American National Standards Institute; AAMI=Association for the Advancement of Medical Instrumentation; ISO=International Organization for Standardization.

Table 3. *Descriptive Characteristics of 41 Pediatric Cohorts*

Variable		k*	N	Min.	Max.	M(SD) or [%]
Cohort Characteristics						
Age (years)		40	9,535	7.50	17.60	12.98 (2.61)
Sex (% male)		36	8,954	37.05	100.00	51.88 (11.31)
Race	(% non-white)	21	4,845	15.91	100.00	67.67 (26.50)
Geographical Region	North America	18	4,355	-	-	[43.9%]
	South America	2	122	-	-	[4.88%]
	Europe	11	1,979	-	-	[26.83%]
	Asia	8	3,022	-	-	[19.51%]
	Oceania	1	57	-	-	[2.44%]
Height (cm)		22	5,361	131.11	170.00	155.55 (10.91)
Weight (kg)		20	4,682	33.90	111.64	53.95 (16.26)
Body Mass Index (BMI; kg/m^2)		21	5,720	16.55	38.65	21.63 (4.86)
Family History Hypertension (%)		9	2,953	9.70	88.18	53.26 (23.91)
Recruitment Setting	Clinic	8	1,143	-	-	[19.51%]
	School	29	7,048	-	-	[70.73%]
	Community	2	1,287	-	-	[4.88%]
	No information	1	57	-	-	[2.55%]
ABPM Recording Specifications						
ABPM Device Type	Oscillometric	30	7,889	-	-	[73.17%]
	Auscultatory	8	1,558	-	-	[19.51%]
	No information	2	88	-	-	[4.88%]
ABPM Recording Duration (hr)		39	9,492	24.00	48.00	24.69 (4.06)
Day Interval (hh:mm)	Start	23	5,806	6:00	8:00	7:10 (0:47)
	End	23	5,806	17:59	24:00	21:56 (1:07)
Night Interval (hh:mm)	Start	23	5,908	18:00	24:00	22:33 (1:29)
	End	23	5,908	5:00	8:00	6:34 (0:47)
ABPM Sampling Frequency (min)	Day	37	9,118	7.50	30.00	22.23 (6.34)

	Night	37	9,118	7.50	60.00	38.99 (16.74)

Note. *k* = number of articles; *N* = total number of participants; *M* = mean; *SD* = standard deviation; - = no information. *Abbreviation *k* to designate number of articles is accurate as information for each cohort was extracted from a single article for any given coded variable.

Table 4. *Fidelity to Consensus Measurement Guidelines*.

	k	Compliant (%)
Device & Placement		
Validated device	31	75.6%
Non-dominant Arm	25	61.0%
Appropriate Cuff Size	21	51.2%
Recording Specifications		
Sufficient Sampling Frequency	36	87.8%
Recording Duration ≥ 24h	39	95.1%
Day / Night Intervals		
Sleep / Wake Diary Use	13	31.7%
Set Transition Time Discarded	7	17.1%
Data Reported		
24h Blood Pressure	25	61.0%
Day Blood Pressure	37	90.2%
Night Blood Pressure	36	87.8%
BP Dipping	5	12.2%
BP Load	7	17.1%
Data Quality		
Sufficient Readings (≥40 per 24hr)	4	9.76%
Adequate Successful Readings (≥70%)	6	14.6%
Removal of values outside normal range	8	19.5%

Table 5. *Meta-Analytic Models: Pooled Cohort Data*.

Pooled Cohort Values	k	N	Estimate*	95% CI	Q_M	p†	τ	I^2	Egger
Systolic Blood Pressure									
24h ABPM	25	6715	114.96	(112.81, 117.10)	1665.26	<.001	5.39	99.1%	.133
Day ABPM	37	8476	117.46	(115.79, 119.13)	1912.54	<.001	5.09	98.2%	.577
Night ABPM	36	8105	105.03	(103.53, 106.52)	1598.45	<.001	4.48	97.9%	.262
Noctural Dipping (Individual)	5	1577	10.01	(9.19, 10.82)	46.37	<.001	0.87	92.9%	.062
Diurnal Variation (Group)	36	8105	-1.38	(-1.52, -1.24)	598.76	<.001	.407	93.4%	.194
Diastolic Blood Pressure									
24h ABPM‡	24	6681	66.20	(65.06, 67.33)	1186.36	<.001	2.79	98.1%	.247
Day ABPM‡	36	8442	69.93	(69.16, 70.71)	761.42	<.001	2.29	96.9%	.052
Night ABPM‡	35	8071	57.83	(56.88, 58.77)	974.41	<.001	2.78	98.5%	.024
Noctural Dipping (Individual)§	4	605	16.81	(15.90, 17.71)	17.51	.001	.79	79.1%	.166

Diurnal Variation (Group)	36	8105	-1.97	(-2.17, -1.18)	818.25	<.001	.59	96.2%	.684

Note.

*Pooled estimates are unique to blood pressure value; all estimates are mean (mmHg), except diurnal variation which is Hedges' *g*.

†Significance value of heterogeneity statistic Q_M.

‡Pooled estimate excludes extreme outlier (Davis et al., 1996).

§Pooled estimate excludes extreme outlier (Zhu et al., 2008).

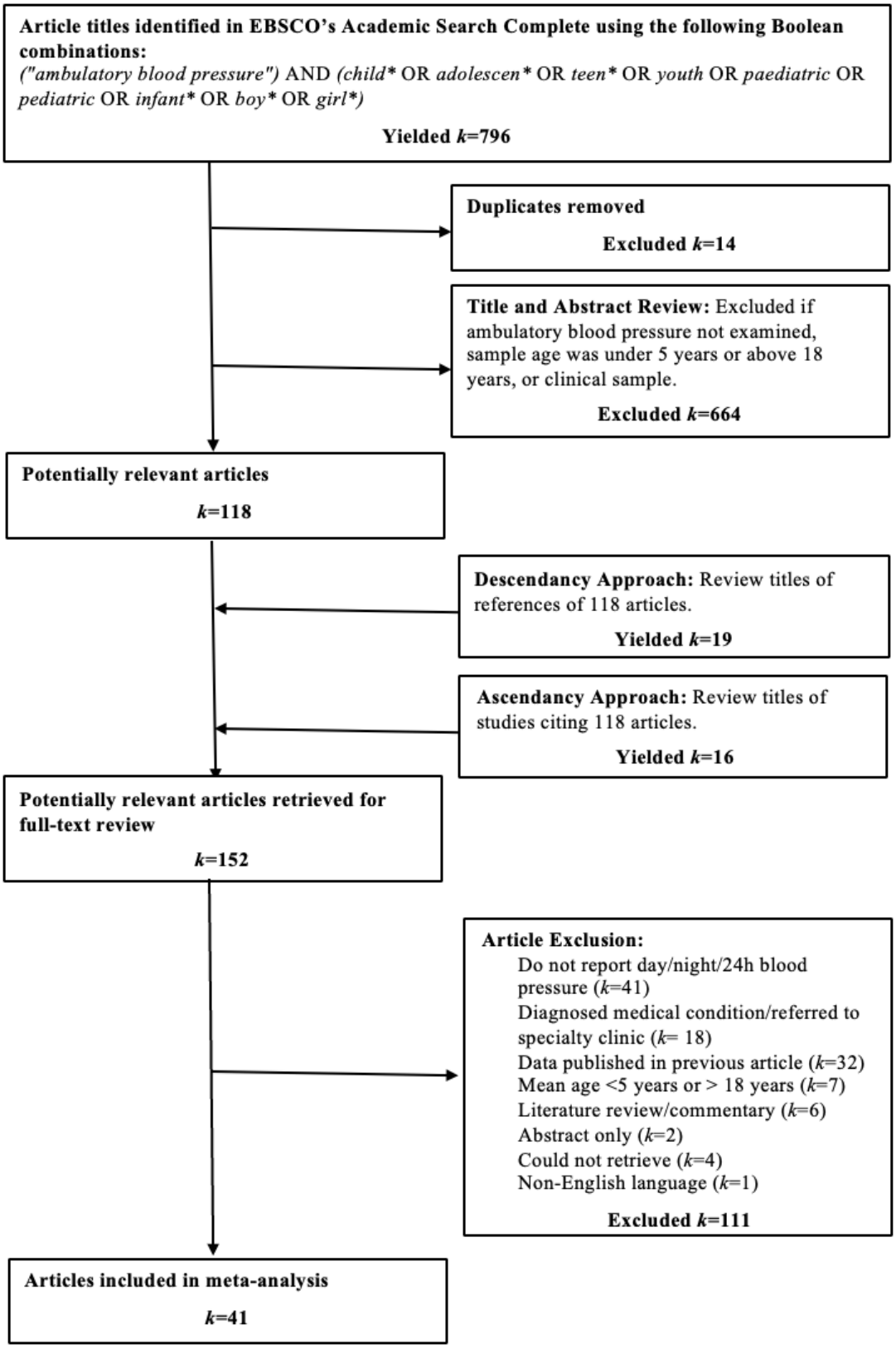

Figure 1. Flowchart for article identification and inclusion in meta-analysis.

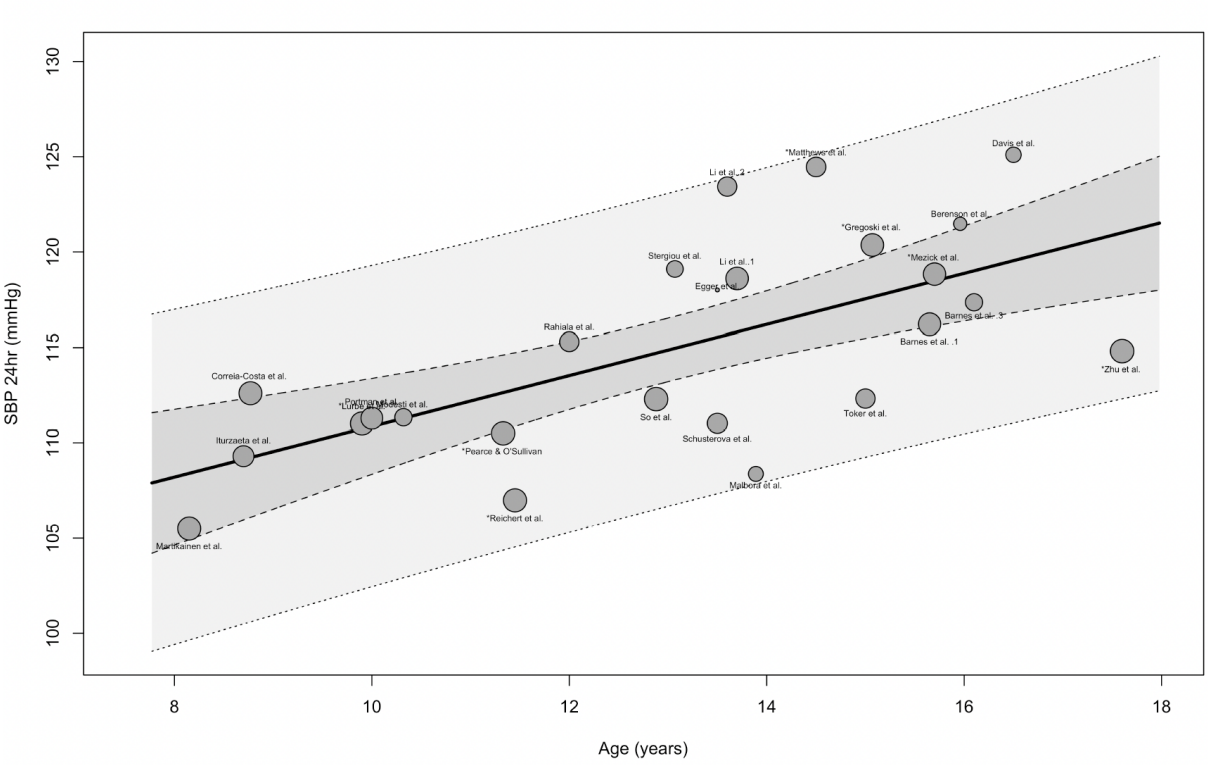

Figure 2. 24-hour systolic blood pressure by age.

TRANSITION FROM STUDY 1 TO STUDY 2:

With the overarching objective to examine the relation between sleep and blood pressure in children and adolescents for my program of research, the goal of Study 1 was to conduct a literature review of youths' blood pressure during wake and sleep. The original goal of Study 1 was to consider blood pressure variation during day and night; however, it quickly became evident that these periods are not systematically defined and many different methodological decisions are made by researchers. As such, Study 1 evolved to more closely examine ambulatory blood pressure monitoring (ABPM) practices within pediatric blood pressure research. I initially compiled a comparison of ABPM measurement guidelines published by Hypertension Canada (Rabi et al., 2020), the American Heart Association and American Academy of Pediatrics (Flynn et al., 2017; Flynn et al., 2022), and the European Society of Hypertension (Lurbe et al., 2016; Mancia et al., 2023; Parati et al., 2014). The guidelines were largely overlapping, but a few noted distinctions were found in standards for defining day and night intervals, data reporting, and data quality, Thus, the first goal of Study 1 was to consider fidelity with these measurement guidelines.

In addition to measurement issues, review of the literature revealed challenges in the interpretation of pediatric ABPM due to limited available data. I found that relatively few studies reported separate day and night blood pressure, and even fewer reported blood pressure dipping. Furthermore, the only available reference data for pediatric ABPM consist of two studies with limited generalizability. This led to the second objective of Study 1, in which I extracted data from over 40 cohorts to aggregate pediatric ABPM values for 24-hour, day, night, nocturnal dipping, and diurnal variation. I then conducted a meta-analysis to examine differences in day and night blood pressure in this subset of articles. The findings showed that youth have clear

diurnal variation in ABPM, and that differences in day and night blood pressure are moderated by demographic and methodological variables. Study 1 was an important step in evaluating the state of blood pressure research, compiling pediatric ABPM data, and providing evidence of diurnal variation in pediatric blood pressure. However, it became clear that issues in data reporting and methodological standards created challenges in interpreting pediatric ABPM data. More data are needed on measurement practices, as well as across age ranges, countries, and racial and ethnic groups to create normative values that are representative and generalizable across populations. Without consistent measurement standards and representative reference values, there is limited ability to extend this research to examine pediatric ABPM in relation to other variables and risk factors. It has been consistently reported by experts in the field that pediatric ABPM data are lacking compared to data available for daytime in-office blood pressure values (i.e., casual blood pressure; Flynn et al., 2022). Despite recommendations to use ABPM when possible, casual blood pressure recording remains the predominant method of assessing blood pressure in youth.

There has been growing focus on pediatric blood pressure measurement due to rising prevalence of hypertension in youth. The identification of early risk factors is thus critical, since childhood blood pressure is a known predictor of hypertensive status in adulthood (Li et al., 2009). Given the close associations between blood pressure and sleep, and clear diurnal variation in blood pressure, poor sleep has emerged as potential target in identifying risk for hypertension. Previous studies suggest that short sleep duration predicts high blood pressure in youth. Most research has investigated the role of sleep duration, but sleep is a multidimensional construct that can be assessed in many ways. There is evidence that other components of sleep may influence blood pressure, including nighttime awakenings, sleep efficiency, irregularity in sleep patterns,

and time spent in different sleep stages. There is little research examining these relations in children, and it is unclear how the associations between blood pressure and sleep might progress over time.

The goals of Study 1 in this program of research were to evaluate fidelity to measurement guidelines in the pediatric blood pressure literature, and to conduct a meta-analysis of day-night differences in pediatric ambulatory blood pressure. The findings indicated that youth exhibit diurnal variation in blood pressure, but there was significant inconsistency in measurement and poor availability of data across the literature. To better evaluate associations between pediatric blood pressure and sleep, it was decided that the second study of my dissertation would focus on casual (i.e., in-office) blood pressure recording, as this is the most common method of blood pressure measurement and is used in most pediatric sleep research. In Study 2, I aimed to address these gaps in the literature by summarizing and synthesizing associations between casual blood pressure and a range of sleep dimensions from childhood into middle adulthood. The roles of potential effect modifiers (e.g., age, sex, race) in these associations were examined.

Study 2:

Associations between casual blood pressure and objective sleep dimensions: A meta-analysis

Hillary Rowe, MA & Jennifer J. McGrath, PhD MPH

Introduction

Poor sleep is increasingly recognized as a significant risk factor for cardiovascular disease. Sleep plays a critical role in many biological and physiological processes; and, sleep disruption is associated with autonomic dysfunction, metabolic changes, insulin resistance, and circadian misalignment, among other health concerns (Cuspidi et al., 2020; Medic et al., 2017). Associations have been observed between sleep and blood pressure, with poor and insufficient sleep generally linked to higher blood pressure and greater risk for hypertension (St-Onge et al., 2016). The mechanisms underlying this association remain uncertain, but it has been proposed that poor sleep disrupts sympathovagal balance, increases inflammation and endothelial dysfunction, and heightens risk for obesity by altering leptin and ghrelin levels (Cuspidi et al., 2020).

Blood pressure follows a distinct 24-hour rhythm that tends to coincide with rest-wake patterns (Thosar et al., 2018). Blood pressure typically decreases by 10 to 20% during sleep (i.e., blood pressure dipping), rises sharply upon awakening, and remains high throughout the day (Smolensky et al., 2007). Nighttime blood pressure and blood pressure dipping have been more strongly associated with cardiovascular risk than daytime casual blood pressure (i.e., in-office recording, a.k.a. casual blood pressure; Thomas et al., 2020). The "non-dipping" blood pressure profile is associated with higher sleep disturbances and lower nighttime plasma melatonin concentration, supporting the role of sleep and circadian rhythms in blood pressure patterns (Mansoor, 2002; Zeman et al., 2005).

Associations between daytime blood pressure and sleep are evident as well. For example, experimental studies indicate that restriction and extension of sleep duration impact daytime blood pressure values (Stock et al., 2020). Although experts recommend 24-hour ambulatory

blood pressure monitoring (ABPM) when diagnosing hypertension, most blood pressure measurement is performed during the day in an office setting (Stergiou et al., 2021; Whelton et al., 2021). Casual blood pressure (i.e., in-office recording) is a routine component of healthcare and often the first step in diagnosis and treatment of elevated blood pressure; as such, it is important to understand the links between sleep and daytime casual blood pressure. This is particularly true in children, as ABPM use is not as prevalent as in adults.

To date, most research has examined the link between sleep duration and blood pressure. Short sleep duration is an established health concern among Western populations, and ease of measurement allows for straightforward evaluation of sleep. Previous systematic reviews and meta-analyses have identified associations between blood pressure and sleep duration. Among adults, several meta-analyses have shown that both short and long sleep duration are risk factors for hypertension (Guo et al., 2013; Wang et al., 2015). Meta-analyses examining other sleep dimensions (e.g., awakenings, sleep efficiency, sleep stages) are less common, and most rely on subjective measures of sleep (e.g., self-reported sleep quality; Lo et al., 2018). Among pediatric research, reviews suggest that this relation may begin early as sleep duration has been associated with blood pressure in youth, but studies are mostly limited to sleep duration and predominantly focus on specific clinical populations (e.g., sleep apnea). Ultimately, associations between sleep and blood pressure have been inconsistently reported and further research has been recommended using standardized measurement methods and objective sleep measures (Fobian et al., 2018; Matthews & Pantesco, 2016).

Sleep is a complex, multidimensional physiological process. Sleep duration is an important predictor of health outcomes, but other aspects of sleep are likely equally important in maintaining optimal health. Sleep has been shown to impact different health outcomes depending

on the dimension measured (Jarrin et al., 2013). For example, studies suggest that the continuity of sleep may have an important role in the association between sleep and blood pressure. More disruptions and arousals from sleep have been associated with higher blood pressure and hypertension (Dean et al., 2015; Ren et al., 2022). Similarly, high irregularity in sleep timing and sleep patterns have been shown to increase risk for elevated blood pressure (Scott et al., 2023). Reviews of the literature suggest further research with more comprehensive sleep measurement to fully capture the relation between sleep and blood pressure, including sleep continuity, irregularity, and timing (Fobian et al., 2018; Matthews & Pantesco 2016).

The aim of the present study was to empirically examine the association between objectively measured sleep and casual blood pressure in children and adults. Specifically, a meta-analysis was conducted to evaluate the magnitude of the association between specific sleep dimensions (e.g., sleep duration, awakenings, sleep efficiency, irregularity, sleep stages) and casual daytime systolic and diastolic blood pressure. Further, the roles of demographic (e.g., age, sex, race, geographic region) and methodological (e.g., sleep measurement method) moderators were evaluated.

Method

Systematic literature search

The literature search was performed using EBSCO Academic Search Complete (MEDLINE, Psychology and Behavioral Sciences Collection, SocIndex). The search period was from January 1970 to June 2024. The complete Boolean expression for the search terms is presented in Figure 2. Title and abstract screening produced 120 initial articles. Ascendancy and descendancy methods yielded 79 further studies. Full-text review was conducted on the final 199 articles. The protocol was registered with the international prospective register of systematic

reviews (PROSPERO) in accordance with PRISMA guidelines (PROSPERO CRD42021268983).

Article inclusion criteria and selection

To be included in the meta-analysis, studies were required to meet the following criteria: sleep measured using actigraphy and/or PSG; reported data on at least one sleep dimension (duration, sleep efficiency, onset latency, awakenings, irregularity and/or sleep stages); reported associations between sleep and casual systolic and/or diastolic blood pressure (e.g., correlation, group differences). Age was restricted to <65 years of age (mean age of sample) given the higher prevalence of comorbid health conditions in older adulthood. The following exclusion criteria were applied: treatment studies without baseline data; specific clinical populations unrelated to blood pressure and/or sleep (e.g., pregnancy, cancer, severe congenital disorders); use of specific medical treatments (e.g., actively undergoing chemotherapy; inpatient settings); non-English language; case studies; abstracts; reviews or meta-analyses; clinical guidelines.

Of the studies selected for full-text review, 30 fulfilled all inclusion and exclusion criteria. Cohort names, recruitment dates, sites, and author lists were reviewed to ensure that data were not represented more than once in the dataset. After screening for redundancy, 31 cohorts from 30 articles were included in the meta-analysis. A summary of included studies is presented in Table 1.

Data extraction

Sample characteristics. Sample size, demographics (e.g., country, age, sex, race/ethnicity), anthropometrics and health status (e.g., BMI, hypertensive status), and recruitment setting were extracted from each article. Race was coded as percentage of non-White participants; coding of specific racial/ethnic groups was not possible due to lack of reporting.

Socioeconomic status, educational level, gender, height, and weight were initially coded but could not be included in the final analyses given limited availability of data reported in articles.

Sleep measurement. Method of objective sleep measurement (i.e., actigraphy and/or PSG) was coded for each study. The duration of sleep recording was coded as the number of nights measured. Sleep data for the following harmonized dimensions were extracted: sleep duration (i.e., time between bedtime and final awakening), awakenings (i.e., number of awakenings; wake after sleep onset in minutes; arousals), sleep efficiency (i.e., percentage of time in bed spent sleeping), irregularity (irregularity in sleep duration; sleep regularity; interdaily stability; intradaily variability, social jetlag), and sleep stages (percentage of time spent in stage N1, N2, N3, REM sleep). All sleep variables were also compiled into an overall sleep variable representing poor sleep; this included all sleep dimensions above as well as less commonly reported sleep dimensions (e.g., sleep timing). The advantage of this overall sleep variable permitted us to include more studies that examined sleep and blood pressure (i.e., studies with less commonly reported metrics would need to be excluded as there would be insufficient studies to warrant separate analyses; e.g., bedtime). Effect sizes for awakenings and irregularity were reverse coded so that associations could be interpreted in the same direction as other sleep dimensions (i.e., poorer sleep was associated with higher blood pressure).

Casual blood pressure. Means were coded for casual blood pressure values for systolic and diastolic blood pressure. Casual blood pressure was defined as daytime blood pressure recordings performed in an office, clinic, hospital, or laboratory setting. Recordings using ambulatory blood pressure monitoring (ABPM) were not included.

Coding reliability. Approximately 30 coding decisions were made per study. Data were extracted by a single rater (HR) who coded all articles in consultation with another rater (SM).

Inter-rater reliability (intraclass correlation coefficient, *ICC*=.99) and intra-rater reliability after an 11-month delay (*ICC*=.99) were excellent.

Statistical analysis

Effect size calculation. Effect size calculations were based on published procedures (Cooper & Hedges, 1994). Fisher's *Z'* was selected as the common effect size metric across articles for the following reasons: most results were presented as continuous associations between sleep dimensions and blood pressure, and almost all test statistics can be converted into Fisher's *Z'* (Rosenthal, 1991). Fisher's Z' values range from $-\infty$ to $+\infty$ and can be interpreted similarly to a correlation. Fisher's variance stabilizing transformation was used to convert bivariate correlations (Cooper, 1998). Unstandardized regression coefficients were converted to *t*-test statistics; *t*-test statistics were then converted into *r* and then into Fisher's *Z* values (Cooper & Hedges, 1994). Dichotomized outcomes (e.g., odds ratio) were used to calculate Cohen's *d* and then converted into Fisher's *Z'* (Chinn, 2000). Means and standard deviations were used to calculate Hedges' *g*, which was then transformed into Fisher's *Z'*. If no statistical values and no information regarding significance were provided, the result was not included in analyses. There are known concerns with combining effect sizes derived from continuous associations and group differences; however, this was deemed the most prudent option to harmonize the data given the few studies that reported group differences. Further, sensitivity analyses were conducted to examine whether the reported data statistic moderated the findings. As described above, the direction of Fisher's Z' was coded uniformly to ensure that positive values reflected higher blood pressure in relation to poorer sleep. Effect sizes were coded for all available and relevant data within each study. Because many articles reported associations between blood pressure and multiple sleep dimensions, there are multiple effect sizes presented for each article. Only one

effect size per sleep dimension was calculated for each participant cohort to prevent inclusion of redundant data in analyses.

Analytic strategy. Analyses were performed using SPSS (Version 29). Meta-regression was performed using mean effect sizes from all studies to estimate inverse-variance-weighted pooled statistical summaries and 95% confidence intervals. Mixed-effects models with random effects were conducted for each sleep dimension (overall, sleep duration, awakenings, sleep efficiency, irregularity, sleep stages). Random effects meta-analytic models (REML estimator) were used as they permit natural variation among effect sizes and are recommended over fixed effect models when there is evidence of heterogeneity (Hedges & Vevea, 1998; Higgins & Thompson, 2002). Cochran's Q_T, Higgins & Thompson's I^2 statistic, and tau were used to estimate heterogeneity.

Effect modifiers were tested using moderator analyses to evaluate possible causes of heterogeneity among samples. Moderators tested were identified *a priori* and registered with PROSPERO (PROSPERO CRD42021268983). Moderators included sample characteristics (age, sex, race, geographic region, recruitment settings) and methodological variables (sleep measurement method). Random-effect meta-regression analyses were used to test continuous (i.e., β slope, Q_E) and categorical (i.e., Q_M) moderator variables. Forest plots were used to depict summaries of overall effect sizes. Effect modifiers were not examined for sleep stages due to the low number of studies.

Results

Descriptive characteristics are presented in Table 3. There were a total of 56,248 participants across cohorts. Studies were evenly divided between pediatric (48.4%) and adult (51.6%) samples. Samples were 54.65% female on average (SD=16.51, k=28). Among studies

reporting race, approximately half of participants were non-White (*M*=50.28%, *SD*=32.03, *k*=18). Cohorts were predominantly North American (61.3%) and recruited from communities (61.3%). Sleep was most commonly measured using actigraphy (80.6%) for an average of 7.5 nights (*SD*=2.62); 19.4% of studies used single-night PSG recording.

Meta-analytic models revealed that overall poorer sleep was significantly associated with higher casual diastolic blood pressure (*Z'*=-.082; see Table 4). Shorter sleep duration was significantly associated with higher casual systolic (*Z'*=-.127) and diastolic (*Z'*=-.093) blood pressure. More awakenings were associated with higher systolic (*Z'*=-.048) and diastolic (*Z'*=-.042) blood pressure. Meta-analytic models had statistically significant *Q* values and large I^2 values, suggesting that the data were heterogeneous and further moderator analyses were needed (see Discussion). Meta-analytic models are shown in Table 4). See supplementary data for forest plots.

Meta-regression models with continuous effect modifiers are presented in Table 5. Age significantly moderated the association between systolic and diastolic blood pressure and overall sleep. In other words, the relation between poorer overall sleep and higher blood pressure was stronger as the sample age increased. Percentage of non-White participants significantly moderated the association between diastolic blood pressure and irregularity, with stronger associations observed as the proportion of non-White participants increased. Meaning, poorer sleep was more strongly associated with higher blood pressure among cohorts with more non-White participants. No significant moderator effects were found for sleep duration, sleep efficiency, or sleep stages. Sex did not influence the associations between blood pressure with any sleep dimensions.

Meta-analytic models were conducted for categorical effect modifiers. Region significantly moderated the association between blood pressure and sleep. Studies conducted in North America and Oceania consistently showed stronger associations between blood pressure and overall sleep compared to other regions. Compared to PSG, studies using actigraphy showed stronger associations between blood pressure and overall sleep and sleep duration. However, PSG-measured awakenings were more strongly associated with blood pressure than actigraphy-derived awakenings. As expected, studies reporting continuous associations showed stronger relations between sleep duration and blood pressure.

Discussion

The aim of this study was to systematically review and synthesize the existing literature on the associations between objectively measured sleep and casual systolic and diastolic blood pressure. We conducted a meta-analysis examining the relations between various sleep dimensions and casual blood pressure in pediatric and adult samples. Demographic and methodological variables were explored as potential effect modifiers of these associations. Sleep dimensions were associated with casual blood pressure, including overall poor sleep, sleep duration, awakenings, and irregularity. These associations were moderated by age, race, and geographic region.

Overall, findings were consistent with past research in that significant associations were observed between sleep and casual blood pressure. Across the 31 cohorts and over 55,000 participants, we found that poorer sleep was generally associated with higher blood pressure. Shorter sleep duration and higher frequency and duration of nighttime awakenings were both linked to higher systolic and diastolic blood pressure. Insufficient sleep duration is thought to increase risk for hypertension through several physiological mechanisms. Experimental studies

suggest that sleep restriction leads to higher blood pressure by disrupting sympathovagal balance, increasing inflammation, and interfering with endothelial function (Bock et al., 2022). Short sleep duration may also indirectly affect blood pressure through disruption of glucose homeostasis and changes in leptin and grehlin levels, increasing risk of obesity and thus hypertension (Cuspidi et al., 2020). Similarly, research suggests that increased awakenings, sleep fragmentation, and arousals are linked to higher blood pressure. Beyond the effects of reduced sleep duration, increased awakenings are thought to interfere with the restorative functions of sleep (Brian et al., 1987). Blood pressure is normally lower during sleep, but nighttime awakenings cause surges of sympathetic activity, resulting in increased blood pressure (Pickering, 1990). Frequent awakenings may disrupt typical nighttime blood pressure patterns, increasing risk for hypertension over time (Cheng et al., 2015).

No associations were observed between sleep efficiency and casual blood pressure. There is limited research examining its relation to casual blood pressure, but past findings indicate that sleep efficiency is inconsistently associated with 24-hour ABPM (Al Haddad et al., 2023; Degaute et al., 1991). Given that sleep efficiency reflects the proportion of time in bed that is spent sleeping, it is possible that this aspect of sleep was better represented by the degree of awakenings during sleep. There are also challenges in quantifying sleep efficiency, including capturing wake time with intention to get out of bed (vs. snooze) as well as inconsistency in the denominator used in calculation (i.e., ratio of total sleep time to time in bed; Reed & Sacco, 2016). This may make it difficult to compare and harmonize sleep efficiency values across studies, potentially masking any associations with casual blood pressure. Similarly, no associations were found between sleep stages and casual blood pressure. This is inconsistent with past research, as higher stage N1 sleep and lower stage N3 sleep have been linked to elevated

blood pressure (Fung et al., 2011). However, this can likely be attributed to the small number of studies and variable data reported.

Moderator analyses revealed a significant effect of age. Poorer overall sleep was more strongly linked to higher blood pressure in cohorts with older participants. This was not observed in other sleep dimensions, likely due to limited number of studies. Past research has yielded inconsistent findings on the role of age in sleep and blood pressure (Daugherty et al., 2020; Grandner et al., 2018). Both hypertension and poor sleep are more prevalent with age, and people may become more vulnerable to the impact of insufficient sleep over time. Additionally, poor sleep may be an early risk factor for the later development of hypertension since childhood blood pressure tracks into adulthood (Li et al., 2009). To our knowledge, there are no prospective studies linking childhood sleep with hypertension in adulthood; however, childhood obstructive sleep apnea independently predicts later development of hypertension (Chan et al., 2020). Future research is recommended to focus on associations between blood pressure and sleep across the lifespan, as children with poorer sleep may be at increased risk of developing hypertension in adulthood. Investigating the emergence of this association earlier in life will help in prevention and early identification of elevated blood pressure in at-risk youth.

Studies conducted in North America showed stronger associations between blood pressure and poor sleep, shorter sleep duration, and awakenings than other geographic regions. This may be partly explained by the higher proportion of North American studies included in the meta-analysis. Additionally, there are likely demographic differences across samples from different regions. Among North American studies, there was a greater emphasis on examining blood pressure in specific populations (e.g., racial and ethnic groups, lower socioeconomic status) known to be at higher risk for hypertension and other cardiovascular conditions (Cundiff

et al., 2015; Lackland, 2014) This is supported by the moderating effect of race on sleep and blood pressure; our findings indicated that studies with a higher percentage of non-White participants had stronger associations between diastolic blood pressure and irregularity of sleep habits. Potential explanations for this effect include biological factors (e.g., salt sensitivity) as well as socioeconomic disparities, access to healthcare and healthy food, and exposure to racism and acculturative stress (Abrahamowicz et al., 2023; Brondolo et al., 2011; Steffen et al., 2006).

Strengths and Limitations

The present study had several strengths and limitations that warrant discussion. First, this study was one of few meta-analyses to comprehensively summarize and synthesize data on the associations between casual blood pressure and sleep. We included data from a range of sleep dimensions measured using actigraphy and PSG to ensure that the multidimensional nature of sleep was adequately represented. Data from children and adults were coded to permit evaluation of these associations over time; this is a significant strength as past meta-analyses are separated by age, and it is unclear when these associations emerge.

Analyses were partly limited by the available data. Sleep dimensions were inconsistently reported and defined across studies, resulting in significant variability. Due to the limited data, variables were harmonized into larger sleep dimensions (e.g., awakenings, irregularity, sleep stages) to resolve differences in measurement and definition to the best extent possible. With further research, there may be more sufficient data to examine each sleep dimension independently and with greater consistency in measurement.

Conclusion

To our knowledge, this is the first meta-analysis to evaluate the association between multiple objectively measured sleep dimensions and casual blood pressure. There is a large and

robust body of literature examining the link between blood pressure and sleep duration, but few studies have examined sleep as a multidimensional construct. Our findings indicate that sleep dimensions such as awakenings and irregularity are significantly related to casual blood pressure, and that these effects may vary depending on geographic region and method of measuring sleep. Further research is needed with more comprehensive sleep assessment and inclusion of objective sleep measurement. Additionally, future research should consider the role of age in the associations between sleep and blood pressure. Previous research primarily consists of cross-sectional studies in middle-aged and older adults because hypertension and related conditions become more prevalent with age. Inclusion of wider age ranges and longitudinal, prospective designs will be important in determining whether causal associations between sleep and blood pressure change across the lifespan.

Table 1. *Summary of 31 Cohorts*.

Year	Author	N	Country	Age	Recruitment setting	Sleep measure	Objective sleep variables	BP measure
2019	Abbott et al.	2,156	USA	18-64 *M*=47.0	Population-based; community	Wrist Actigraphy (7 days)	Sleep duration, sleep midpoint, SJL	Casual SBP & DBP
2021	Al-Rashed et al.	60	Kuwait	*M*=31.87	Community	Wrist Actigraphy (7 days)	Sleep duration, WASO	Casual SBP & DBP
2023	Beunders et al.	894	Netherlands	*M*=11.7	Population-based; community	Wrist Actigraphy (9 days)	Sleep duration, sleep efficiency, awakenings, WASO, SJL, IV, IS	Casual SBP & DBP
2008	Bixler et al.	700	USA	5-12 *M*=9.25	School	1 night in-lab PSG	Sleep efficiency, % REM	Casual SBP & DBP
2022	Bizzotto Trude et al.	470	USA	*M*=12.2	Community	Wrist Actigraphy (7 days)	Sleep duration, WASO, sleep efficiency	Casual SBP & DBP
2022	Confortin et al.	1,268	Brazil	18-19 *M*=18.13	Population-based community	Wrist actigraphy (7 days)	Sleep duration	Casual SBP & DBP
2022	Culver et al.	44	USA	18-39; *M*=22.8	College/university	Wrist Actigraphy (7 days)	Sleep duration	Casual SBP & DBP
2018	Feliciano et al.	829	USA	*M*=13.2	Population-based; doctor's office	Wrist Actigraphy (7-10 days)	Sleep duration, sleep efficiency, WASO, sleep duration variability	Casual SBP
2017	Fernandez-Mendoza et al.	1,344	USA	*M*=48.8	Population-based; community	1 night in-lab PSG	Sleep duration	Casual SBP & DBP
2021	Hoopes et al.	52	USA	18-25 *M*=20.0	College/university	Wrist Actigraphy (14 days)	IS, IV	Casual SBP & DBP
2020	Huang et al.	4,756	UK	46-68 *M*=47.0	Populated-based; community	Thigh actigraphy (7 days)	Sleep duration	Casual SBP
2008	Javaheri et al	238	USA	13-16 *M*=13.7	Population-based; community	3+ days wrist actigraphy	Sleep duration, sleep efficiency	Casual SBP & DBP
2018	Javaheri et al.	1,850	USA	*M*=59	Population-based; community	1 night in-home PSG	% N3, % REM	Casual SBP & DBP
2022	Katamreddy et al.	388	USA	*M*=54.56	Population-based; community	Wrist actigraphy (7 days)	Sleep duration	Casual SBP & DBP

2022	Kelly et al.	57	Ireland	*M*=54.09	Hospital	Wrist actigraphy (6-10 days)	Sleep timing variability, sleep duration variability, IS, IV, SJL	Casual SBP & DBP
2009	Knutson et al.	578	USA	33-45	Population-based; community	Wrist actigraphy (3 days)	Sleep duration,	Casual SBP & DBP
2022	Leppanen et al.	485	Finland	6-8 *M*=7.6	School	Chest actigraphy (4 days)	Sleep duration	Casual SBP & DBP
2017	Massar et al.	59	Singapore	22-33 *M*=22.83	College/university	Wrist actigraphy (7 days)	Sleep duration, sleep efficiency	Casual SBP & DBP
2022	Matre et al.	19	Norway	*M*=40.9	Workplace	Wrist actigraphy (14 days)	Sleep duration	Casual SBP & DBP
2021 Child	Matricciani et al.	1,043	Australia	*M*=12.0	Populated-based; community	Wrist actigraphy (4+ days)	Sleep duration, sleep efficiency, sleep duration variability	Casual SBP & DBP
2021 Adult	Matricciani et al.	1,337	Australia	*M*=44.0	Populated-based; community	Wrist actigraphy (4+ days)	Sleep duration, sleep efficiency, sleep duration variability	Casual SBP & DBP
2019	McMahon et al.	390	USA	21-35 *M*=27.6	Populated-based; community	Armband actigraphy (6-10 days)	Sleep duration, WASO, sleep efficiency, SJL	Casual SBP & DBP
2019	Mi et al.	125	USA	8-17 *M*=12.4	Community	Wrist actigraphy (14 days)	Sleep duration, bedtime	Casual SBP & DBP
2023	Morales-Ghinaglia et al.	303	USA	*M*=16.2	Community	Wrist actigraphy (7 days); 1 night PSG	Sleep midpoint, sleep regularity	Casual SBP & DBP
2021	Nikbakhtian et al.	88,026	UK	43-79 *M*=61.43	Populated-based; community	Wrist actigraphy (7 days)	Bedtime	Casual SBP & DBP
2015	Quante et al.	464	USA	5-9.9 *M*=7.0	Hospital clinics, community	1 night in-lab PSG	N1, N2, N3, REM	Casual SBP & DBP
2004	Reade et al.	90	USA	4.2-18.8 *M*=10.7	Hospital	1 night in-lab PSG	Arousals	Casual SBP & DBP
2020	Rognaldsdottir et al.	252	Iceland	*M*=15.8	School	Wrist actigraphy (7 days)	Sleep duration, sleep duration variability, WASO	Casual SBP & DBP

2011	Sung et al.	133	USA	*M*=13.2	Hospital clinic	Wrist actigraphy (7 days)	Sleep duration	Casual SBP & DBP
2021	Thosar et al.	30	USA	*M*=50.8	Community	1 night in-lab PSG	Sleep efficiency	Casual SBP & DBP
2024	Troxel et al.	142	USA	12-16 *M*=14.0	Community	Wrist actigraphy (7 days)	Sleep duration, sleep efficiency	Casual SBP & DBP

Table 2. *Summary of sleep dimensions included in meta-analysis*.

Sleep Dimension	Measure	Definitions
Sleep Duration	Actigraphy PSG	Time between bedtime and final awakenings; reflects time spent sleeping.
Awakenings	Actigraphy PSG	Number of Awakenings: Total number of discrete awakenings between onset and offset of sleep. Wake after sleep onset (WASO): Total number of minutes awake between onset and offset of sleep. Arousals: Frequency of brief (3-15 seconds) shifts to higher-frequency EEG activity; reflects sleep disruption.
Sleep Efficiency	Actigraphy PSG	Percentage of time spent sleeping between onset and offset of sleep.
Variability	Actigraphy PSG	Variability in sleep duration: irregularity of sleep duration across multiple days. Variability in timing: irregularity of bedtime, wake time, and timing across multiple days. Interdaily stability (IS): degree to which rest-activity patterns resemble each other across individual days. Intradaily variability (IV): magnitude of hour-to-hour transitions between rest and activity within individual days. Social jetlag: difference in sleep timing between weekdays and weekends.
Sleep Stages	PSG	% N1: percentage of sleep spent in stage N1 sleep. %N2: percentage of sleep spent in stage N2 sleep. %N3: percentage of sleep spent in stage N3 sleep (slow-wave sleep; SWS). %REM: percentage of sleep spent in REM sleep.

Table 3. *Descriptive Characteristics of 31 Cohorts*.

Variable		k	N	Min.	Max.	M(SD) or [%]
Age (years)		30	55,670	7.00	61.43	27.34 (18.06)
Age range	Child/Teen (0-18)	15	7,377			48.40
	Adult (19+)	16	48,871			51.60
Sex (% female)		28	55,670	0.00	100.00	54.65 (16.51)
Race	(%Non-white)	18	9,815	12.20	100.00	50.28 (32.03)
Geographical Region	North America	19	9,506			61.30
	Europe	7	42,975			22.60
	Asia	2	119			6.50
	Oceania	2	2,380			6.50
	South America	1	1,268			3.20
Body Mass Index (BMI; kg/m^2)		18	49,726	19.00	37.07	26.76 (4.01)
Elevated blood pressure or hypertension (%)		8	46,142	1.60	59.49	31.11 (19.59)
Sample Recruitment	Clinic/Hospital/Doctor's office	5	1,520			16.10
	School	3	1,437			9.70
	College/University	3	155			9.70
	Community	19	53,117			61.30
	Other	1	19			9.70
Sleep Measurement	Actigraphy	25	51,823			80.60
	PSG	6	4,425			19.40
Sleep Recording Duration (nights)	Actigraphy	22	46,439	3.00	14.00	7.50 (2.62)
	PSG	6	4,425	1.00	1.00	1.00 (0.00)

Note. *k* = number of cohorts; *N* = total number of participants; *M* = mean; *SD* = standard deviation.

Table 4. *Meta-analysis of 31 cohorts*.

Sleep dimension	k	Estimate	95% CI	Q_M	p*	τ	I^2	Egger
Systolic Blood Pressure								
Overall poor sleep	62	-.051	(-.105, .004)	1,614.43	<.001	.04	99.0%	
Sleep duration	21	-.127	(-.202, -.052)	213.15	<.001	.02	88.0%	
Awakenings	8	.048	(.003, .093)	12.88	.07	.00	33.0%	
Sleep efficiency	10	-.066	(-.159, .027)	56.49	<.001	.02	91.0%	
Irregularity	17	.021	(-.151, .193)	1,350.27	<.001	.12	98.0%	
Sleep stages	6	.006	(-.171, .184)	102.04	<.001	.05	96.0%	
Diastolic Blood Pressure								
Overall poor sleep	60	-.082	(-.131, -.033)	1,047.39	<.001	.03	94.0%	
Sleep duration	19	-.093	(-.151, -.035)	89.36	<.001	.01	74.0%	
Awakenings	7	.042	(.002, .082)	5.82	.44	.00	0.0%	
Sleep efficiency	9	-.078	(-.175, .019)	52.28	<.001	.02	91.0%	
Irregularity	17	.118	(-.036, .272)	1,406.00	<.001	.10	98.0%	
Sleep stages	6	.012	(-.109, .133)	48.10	<.001	.02	90.0%	

Note. *Significance value of heterogeneity statistic Q_M.

Table 5. *Meta-Regression Models: Continuous Effect Modifiers.*

Age (years)	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ**
Systolic Blood Pressure										
Overall poor sleep	65	75.375	-.008	.003	.009*	4,293.10	<.001	54.6	98.5%	.135
Sleep duration	19	13,588	-.006	.003	.069	163.99	<.001	14.6	89.6%	.025
Awakenings	8	3,279	-.002	.005	.764	12.85	.045	.00	53.3%	.003
Sleep efficiency	10	5,773	-.005	.003	.063	48.46	<.001	.00	83.5	.01
Irregularity	17	8,936	-.001	.006	.863	1,146.72	<.001	1.9	98.7	.178
Sleep stages	6	4,194	.002	.005	.665	97.78	<.001	.00	95.9	.052
Diastolic Blood Pressure										
Overall poor sleep	60	67,999	-.007	.002	.001*	1,730.45	<.001	19.7	96.6%	.060
Sleep duration	17	8,003	-.002	.003	.506	88.33	<.001	.00	83.0%	.017
Awakenings	7	2,450	-.002	.004	.681	5.64	.342	.00	11.4%	.00
Sleep efficiency	9	4,944	-.005	.003	.115	47.96	<.001	.00	85.4%	.012
Irregularity	17	8,936	.008	.005	.173	956.45	<.001	21.7	98.4	.148
Sleep stages	6	4,194	.002	.003	.554	44.16	<.001	.00	90.9	.022
Sex (% Female)	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ**
Systolic Blood Pressure										
Overall poor sleep	63	70,014	-.003	.005	.571	10,135.30	<.001	.00	99.4%	.307
Sleep duration	17	13,424	-.003	.002	.133	123.94	<.001	26.6	87.9%	.021
Awakenings	8	3,279	-.002	.002	.313	11.36	.078	.00	47.2%	.002
Sleep efficiency	10	5,773	.002	.002	.255	54.72	<.001	.00	85.4%	.011

Irregularity	17	8,936	.014	.012	.255	1,307.09	<.001	.00	98.9%	.202
Sleep stages	6	4,194	.033	.058	.603	95.10	<.001	.00	95.8%	.050
Diastolic Blood Pressure										
Overall poor sleep	59	67,999	-.001	.002	.580	2,322.82	<.001	.00	96.5%	.052
Sleep duration	16	12,595	.000	.002	.828	70.71	<.001	.00	80.2%	.013
Awakenings	7	2,450	-.001	.001	.539	5.41	.368	.00	7.6%	.00
Sleep efficiency	9	4,944	.003	.002	.219	47.84	<.001	.00	85.4%	.012
Irregularity	17	8,936	.010	.012	.415	1,338.47	<.001	.00	98.9	.207
Sleep stages	6	4,194	.029	.039	.493	42.41	<.001	.00	90.6	.021
Race (% Non-White)	**k**	**N**	**B**	**SE**	**p^***	**Q_E**	**$p^\dagger$**	**$R^{2\ddagger}$**	**I^2**	**τ**
Systolic Blood Pressure										
Overall poor sleep	36	19,492	-.001	.003	.838	2,956.28	<.001	6.1	98.8%	.203
Sleep duration	11	5,175	.000	.001	.567	11.31	.255	.00	20.4%	.001
Awakenings	4	1,779	-.002	.002	.456	6.82	.033	.00	70.7%	.007
Sleep efficiency	7	3,393	.002	.002	.477	51.99	<.001	.00	90.4%	.026
Irregularity	4	3,471	.007	.009	.555	833.26	<.001	.00	99.8%	.479
Sleep stages	6	4,194	-.004	.005	.493	87.99	<.001	.00	95.5%	.047
Diastolic Blood Pressure										
Overall poor sleep	32	17,709	-.001	.002	.544	1,501.82	<.001	13.7	98.0%	.123
Sleep duration	10	4,213	.000	.001	.609	9.99	.266	.00	19.9%	.001
Awakenings	3	950	-.001	.003	.718	5.12	.024	.00	80.5%	.029
Sleep efficiency	6	2,564	.002	.002	.331	30.02	<.001	.00	86.7%	.023

Irregularity	4	3,471	.009	.008	.399	637.59	<.001	3.0	99.7%	.366
Sleep stages	6	4,194	-.003	.004	.406	39.01	<.001	.00	89.7%	.020

Note. *Significance value of B (slope); $p < .05$ indicates slope is significantly different than 0.

†Significance value of Q_E (Residual Heterogeneity); $p < .05$ indicates heterogeneity not explained by predictor is significant.

‡R^2 indicates percentage of between-study heterogeneity explained by predictor.

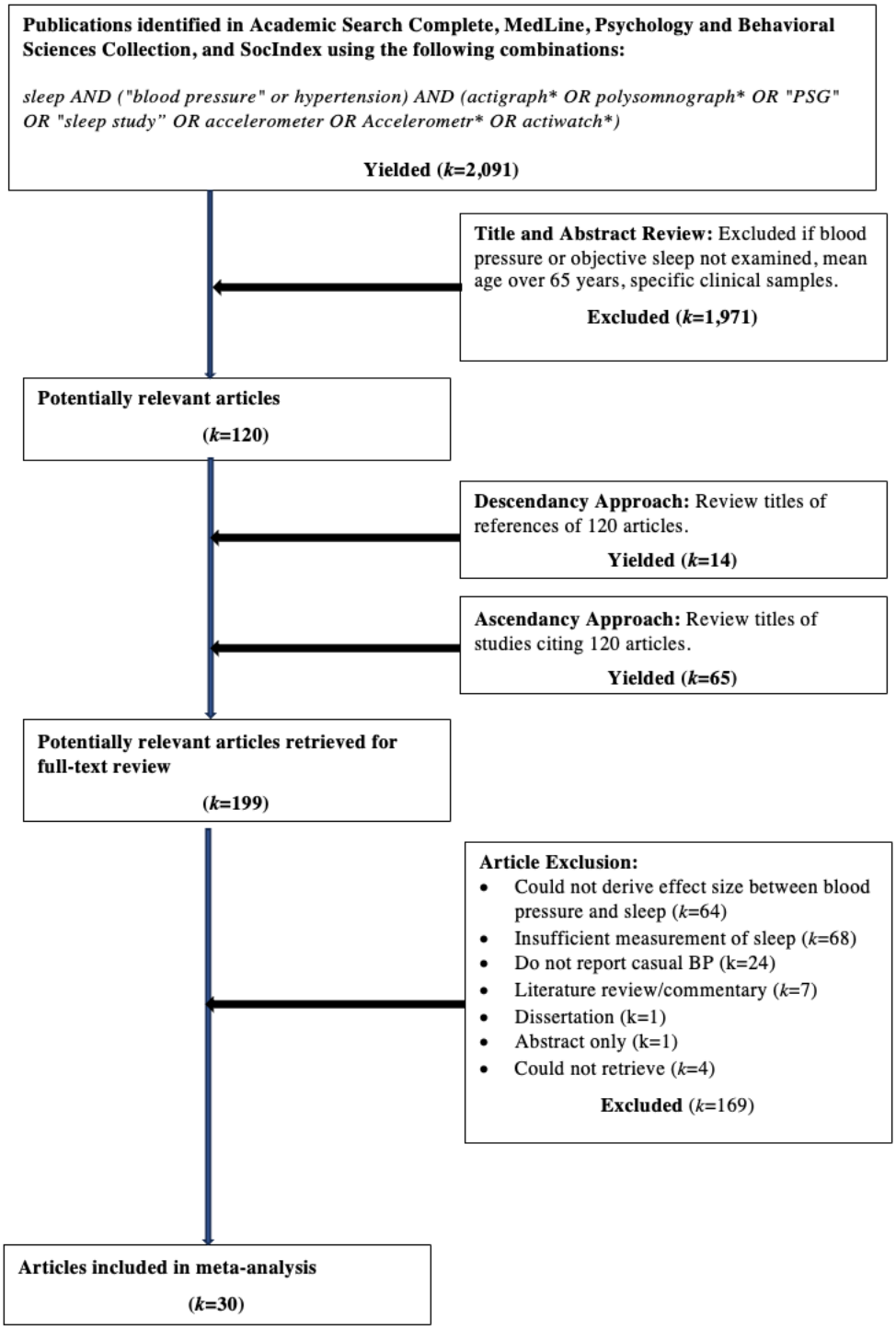

Figure 1. Flowchart for article identification and inclusion in meta-analysis.

TRANSITION FROM STUDY 2 TO STUDY 3

In Study 2, I conducted a meta-analysis examining the associations between casual blood pressure and a number of different sleep dimensions (sleep duration, nighttime awakenings, sleep efficiency, irregularity of sleep patterns, sleep stages). The aim was to explore associations between blood pressure and diverse, objectively measured sleep dimensions, as sleep duration has been heavily emphasized in the literature. Studies were included from childhood through middle adulthood to examine the relations across ages and to consider when this association may emerge. I found that sleep was significantly associated with blood pressure; shorter sleep duration, more awakenings, and poorer overall sleep were linked to higher blood pressure. Interestingly, the association with poor sleep was stronger with age, indicating that sleep may influence blood pressure differently across age groups. There is minimal research examining possible developmental influences, and there is a clear need for more pediatric research using objective sleep measurement and multiple sleep dimensions. The existing research does not fully capture the associations between pediatric blood pressure and sleep, preventing full understanding of potential impacts on blood pressure and hypertension risk.

Studies 1 and 2 provide evidence that blood pressure is associated with sleep and diurnal variation, but there are significant limitations in methodology and data reporting across research in this area. My Study 3 set out to address several of these methodology gaps as part of an original data collection project. I examined the associations between casual blood pressure and a wide range of sleep dimensions in a large community sample of youth. Sleep was assessed using objective sleep measures (actigraphy and polysomnography) and included multiple dimensions to depict sleep as thoroughly as possible. It was hypothesized that youth with poorer sleep (i.e.,

shorter sleep duration, more awakenings, later sleep timing, less time in deep sleep) would show higher blood pressure. Rather than using raw blood pressure data, blood pressure was presented using *Z*-scores, which reflected each child's position relative to the mean blood pressure value for their age, sex, and height. *Z*-scores were chosen to facilitate comparison between youth of different ages and sizes. The moderating roles of various demographic characteristics were also considered, such as age, sex, and race. Analyses also controlled for pubertal status and parental education. My objective was to provide an exhaustive investigation of associations between sleep and blood pressure, while also considering developmental and participant characteristics relevant to pediatric blood pressure.

Study 3:

Associations between casual blood pressure and objective sleep in a community sample of youth.

Hillary Rowe, MA & Jennifer J. McGrath, PhD MPH

Introduction

Sleep has been increasingly linked to cardiovascular functioning and disease, including blood pressure and hypertension. Blood pressure patterns are closely tied to the sleep-wake cycle, and a large body of research suggests that poor sleep is associated with increased risk for hypertension. Specifically, insufficient quantity of sleep has been widely studied as a risk factor in the development of high blood pressure in adults (Wang et al., 2012). Shorter sleep duration has been linked to higher blood pressure among adults in cross-sectional and longitudinal studies (Gottlieb et al., 2006; Knutson et al., 2009; Wang et al., 2012). Furthermore, acute sleep deprivation has been experimentally shown to increase blood pressure in healthy and hypertensive adults (Franzen et al., 2011; Lusardi et al., 1999). Autonomic dysfunction has been identified as a likely contributing factor as short sleep duration is linked to reduced parasympathetic activity and shift to sympathetic dominance, including higher heart rate and orthostatic reactivity and lower high-frequency heart rate variability (Cuspidi et al., 2020; Mancia & Grassi, 2014). These changes have been associated with the development and progression of hypertension. Other potential pathogenic mechanisms include hormonal and metabolic changes such as decreased leptin and increased grehlin levels (i.e., appetite regulating hormones), disrupted glucose homeostasis, increased cortisol levels, and low-grade inflammation (Aggarwal et al., 2018; Byberg et al., 2012; Spiegel et al., 2004a; Spiegel et al., 2004b). Research also suggests that early cardiovascular changes are present in children with poor or insufficient sleep, but it is unclear how this association develops over time (Del Rosso et al., 2020; Peach et al., 2015).

Sleep is a multidimensional construct that cannot be described solely in terms of amount of time spent sleeping. Sleep duration is the most commonly studied sleep variable, largely due

to ease of measurement, and is an important predictor of blood pressure. Other sleep dimensions have been shown to impact health and physiological processes differently, and there is value in including multiple measures of sleep to more accurately depict its relationship with blood pressure (Jarrin et al., 2013). For example, limited studies have examined the role of circadian timing and sleep irregularity (i.e., inconsistency of sleep timing and/or duration), but these have yielded mixed findings (Culver et al., 2022; Feliciano et al., 2019; Parise et al., 2023). When sleep is disrupted or inconsistent, the circadian rhythms controlling blood pressure, sleep, and many other physiological processes can become misaligned, increasing risk for adverse health consequences over time (Scheer et al., 2009). Night shift workers have shown alterations in blood pressure rhythm (e.g., decreased nocturnal dipping), suggesting a potential role of sleep timing (i.e., bedtime, waketime) or irregularity (i.e., variation or inconsistency in sleep timing; Suwazono et al., 2008; Yamasaki et al., 1998). Sleep fragmentation (i.e., frequent awakenings or micro-arousals) has also been related to blood pressure; awakenings and arousals from sleep have been experimentally shown to raise nighttime blood pressure and are associated higher risk of hypertension (Carrington & Trinder, 2008; Morrell et al., 2000). Polysomnography (PSG) studies have also shown that sleep stages may influence blood pressure as blood pressure is higher among those with less time spent in restorative N3 sleep (i.e., slow-wave sleep; Javaheri et al., 2018; Morrell et al., 2000). Both sleep fragmentation and lower proportion of N3 sleep are thought to increase blood pressure through elevated sympathetic activity. In addition to quantified sleep, there is some evidence that poor subjective sleep quality is associated with higher blood pressure (Lo et al., 2018; Yang et al., 2021). Overall, there is considerable research indicating that multiple sleep dimensions contribute to risk for hypertension; yet, these have rarely been examined concurrently in the same sample.

Most research has been conducted in adult samples, likely because risk for hypertension increases with age (Kjeldsen, 2018). Rates of pediatric hypertension are increasing, but there remain knowledge gaps in prevention, detection, and treatment. Sleep has been proposed as a risk factor for high blood pressure in youth, as well as a potential target for prevention and intervention initiatives (Hardy & Urbina, 2021). Short sleep duration and poor sleep quality have been linked to elevated blood pressure in youth (Fobian et al., 2018; Javaheri et al., 2008; Sparano et al., 2019); however, most studies have limited sleep assessment (i.e., single variable; self- and parent-report only) or include only high-risk groups (e.g., sleep-disordered breathing, chronic kidney disease). Research is largely limited to sleep duration, with limited knowledge on awakenings, onset latency, timing, or sleep stages. More evidence is needed in community-based or healthy samples as childhood blood pressure tracks into adulthood, and hypertension is often asymptomatic in otherwise healthy children (Falkner et al., 2010; Li et al., 2009).

The goal of the present study was to examine the associations between daytime casual blood pressure (a.k.a., office blood pressure) and sleep in a community sample of children and adolescents. Our aim was to assess sleep comprehensively using objective methods (i.e., PSG and actigraphy) to allow examination of a wide range of quantitative sleep dimensions. We hypothesized that poorer sleep across sleep dimensions would be associated with higher blood pressure in children and adolescents. Specifically, as observed in past research, we predicted that shorter sleep duration, later sleep timing, greater awakenings and arousals, and reduced time in stage N3 sleep would be associated with higher systolic and diastolic blood pressure.

Method

Study Sample

Youth (*N*=307) aged 9 to 17 years and their parents participated in the larger ongoing Healthy Heart Project, Montreal, Quebec. Participants were recruited using flyers and bookmarks distributed in the surrounding community and schools. Youth were excluded if they had severe psychopathology or neurodevelopmental disorders that would interfere with participation (e.g., active psychosis, severe intellectual disability) or used medications that impact cardiovascular or endocrine functioning. Informed consent and assent were obtained before beginning the study. The study was approved by the Concordia University Research Ethics Board (#10000088).

Procedure

Youth and their parents participated in two visits to the laboratory. During the first visit, participants and parents completed questionnaires assessing demographic information (e.g., sex, age, parental education, household income), sleep behaviours, and health status (e.g., puberty, family history of cardiovascular disease). They were provided with a wrist actigraph and home PSG monitor and instructed to complete a daily sleep diary between visits. Participants returned the wearable monitors and sleep diary two weeks later and were compensated for their time.

Laboratory Measures

Height and weight measurements were taken by a pair of trained research assistants while youth were dressed in light clothing. Height was measured using a standard stadiometer with shoes off. Weight was measured using a bioelectric impedance scale (Tanita Body Composition Analyzer BF-350). Age- and sex-specific BMI Z-scores were calculated using growth charts from the U.S. Centers for Disease Control and Prevention (Centers for Disease Control and Prevention, 2000). Waist circumference was measured using a measuring tape at the narrowest circumference of the body between the lower rib cage and iliac crest. Height and waist circumference were measured twice to the nearest 0.2cm. A third measurement was taken if the

two recordings differed by more than 0.5cm; the mean of the two closest measures was used for analyses. Youth reported their pubertal status using sex-specific schematic drawings of pubic hair growth (adrenarche) and breast or male genital growth (gonadarche) corresponding to Tanner stages 1 to 5 of pubertal development (Golding, Pembrey, & Jones, 2001; Marshall & Tanner, 1969, 1970). Parents reported on family history of high blood pressure and cardiovascular disease.

Casual Blood Pressure Measurement

Office blood pressure was measured using an automated blood pressure monitor (IBS SD700a; inflation pressure 150mmHg, deflation rate 6 mmHg/s). Youth were fitted with an appropriately sized cuff (pediatric or adult) over the brachial artery of the non-dominant arm. Before recording, the automated monitor was calibrated with a manual sphygmomanometer to verify accuracy. After 20 minutes of acclimatization (i.e., sitting and quietly completing questionnaires) and a 5-minute resting period, blood pressure was measured twice two minutes apart in a seated position. Measurement was repeated up to two times if readings differed by more than 4 mmHg. Systolic (SBP) and diastolic blood pressure (DBP) were reported as the mean of the final two readings. Blood pressure measurement was performed in accordance with clinical guidelines (National High Blood Pressure Education Working Group, 2004). Systolic and diastolic blood pressure percentiles were derived from sex-, age-, and height-based norms and then used to calculate SBP and DBP Z-scores (National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents, 2004) Participants were classified as having normal, elevated, or hypertensive blood pressure based on current measurement standards (Flynn et al., 2017; Rowe et al., 2022).

Sleep Measurement

Participants wore a wrist actigraph (Philips Respironics Actiwatch 2) on their non-dominant wrist for up to 14 days (M= 11.9 nights, SD=2.7). They kept a daily sleep diary (based on the Consensus Sleep Diary; Carney et al., 2012) during this time. Actigraphy data were manually inspected and scored using a validated protocol to identify lights on/lights off (i.e., rest) intervals (McGrath et al., 2018). Data were analyzed using Actiware (Version 6) and averaged across the 14-day period. There were minimal data missing (5.4% nights missing). Variables were averaged across the total number of nights recorded. Data were analyzed separately by total nights, weekends, and school night; results were largely similar and only total nights data are presented for parsimony.

Children completed a one-night at-home sleep study with ambulatory PSG (Grass® TREA). At bedtime participants placed: EEG electrodes (Ambu® Blue Sensor) on the forehead (FP1, FP2, active electrodes), left and right mastoids (A1, A2, referent electrodes), and left clavicle (ground electrode); EOG electrodes on the left and right temple; an EMG electrode on the chin; and three ECG electrodes (ClearTrace 2®) in a Lead II configuration. PSG data were scored by a registered polysomnographic technologist using the Rechtstaffen and Kales protocol (Rechtstaffen, 1968) and American Academy of Sleep Medicine guidelines (Iber, 2007). Data missingness was modest (3.8% hardware syncing error; 11.6% poor signal quality).

Actigraphy and PSG data were used to derive the following sleep variables. See Table 1 for more detailed information.

Timing. Bedtime and wake time were defined as the onset (i.e., "lights out") and offset (i.e., "lights on") of the rest interval. Sleep midpoint was calculated as the median time between time of sleep onset and final awakening. Sleep midpoint reflects circadian timing and is associated with chronotype and dim light melatonin onset (Crowley et al., 2007; Martin &

Eastman, 2002). Sleep timing (e.g., bedtime, wake time) was not evaluated for PSG given the single night of recording.

Sleep duration. Duration was calculated as the total number of minutes between sleep onset and the final awakening measured by actigraphy.

Awakenings. The number of awakenings was calculated as the total number of awakenings within the sleep interval. Wake after sleep onset (WASO) was calculated as total duration of awakenings (in minutes) between sleep onset and offset. The arousal index was measured as the total number of arousals divided by the total sleep time (i.e., number of minutes between sleep onset and final awakening) measured using PSG. Arousals are brief (3 to 15 seconds) shifts to higher-frequency EEG activity preceded by at least 10 seconds of sleep (Iber, 2007). The arousal index reflects sleep disruption, with a higher score indicating greater disruption.

Sleep onset latency. Latency was calculated as the number of minutes between lights out and sleep onset. Sleep onset latency reflects the time spent trying to fall asleep and was defined as the total time (in minutes) between the beginning of the rest interval and the beginning of the sleep interval.

Sleep efficiency. Sleep efficiency was computed as the percentage of time spent sleeping within the rest interval (lights out and lights on).

Sleep stages. Sleep stages were reported as the percentage of time within the sleep interval spent in stage N1, N2, N3, and REM sleep. Sleep is divided into non-REM (NREM; stages N1, N2, N3) and REM sleep (Carley & Farabi, 2016; Carskadon & Dement, 2005). Sleep begins with stage N1 (i.e., light sleep) and progresses through the deeper NREM stages before entering REM sleep. Stage N3 (i.e., slow-wave sleep; SWS) is the deepest, most restorative stage

of sleep. REM sleep is characterized by higher EEG activity, low muscle tone, and bursts of rapid eye movement.

Sleep quality. Youth rated their overall sleep quality on a scale from 1 to 10, with higher scores indicating better sleep. Sleep quality is commonly assessed using subjective ratings (Dewald et al., 2010). Single-item ratings are common in sleep research and are considered a valid method of assessing sleep quality (Cappelleri et al., 2009).

Statistical Analysis

Analyses were performed using SPSS version 29.0. Data were inspected for normality and outliers using scatterplots, frequency histograms, and descriptive statistics. Descriptive statistics (means, standard deviations, skewness, kurtosis) were examined for all variables. Group differences in sleep dimensions and systolic and diastolic blood pressure Z-scores were conducted for sex, race (White, Black, Asian, or mixed/other), and family history of elevated blood pressure using one-way ANOVA and independent t-tests. Linear regression analyses were conducted with sleep dimensions predicting systolic and diastolic blood pressure Z-scores. All analyses controlled for pubertal status (i.e., gonadarche) and parental education as these variables are known to influence blood pressure values. Sensitivity analyses showed minimal differences when controlling for gonadarche compared to adrenarche; gonadarche was reported to avoid redundancy. Gonadarche is linked to physiological changes that may be associated with blood pressure, including increased secretion of gonadal hormones and higher adiposity (Aghaee et al., 2022; Pereira et al., 2021; Wójcik et al., 2023). The effects of age, sex, and height were accounted for through the use of Z-scores for blood pressure.

Results

Descriptive statistics. Participant characteristics are presented in Table 2. Participants were 63.2% white (7.8% Asian, 5.2% Black, 3.6% Latin American, 2.3% Middle Eastern/North African, 1.3% Indigenous, 16.6% mixed race or other) and 43.6% female. Youth had a mean age of 12.74 years (*SD*=2.05) and were approximately mid-puberty, reflected by mean Tanner stage ratings of 2.72 (*SD*=1.63; gonadarche) and 2.93 (*SD*=1.60; adrenarche). On average, children had normal BMI (mean percentile = 56.01, *SD*=25.30). Most parents had completed postsecondary education (*M*=16.46 years of education, *SD*=3.14). About half of youth had a reported family history of elevated blood pressure (46.6%) or cardiovascular disease (46.9%). Children had a mean systolic blood pressure of 100.87mmHg (*SD*=12.77) and mean diastolic blood pressure of 59.58mmHg (*SD*=9.81). Most participants (88.3%) had blood pressure in the normal range, but mean percentiles indicated that SBP (mean percentile = 28.36%^{ile}, *SD*=26.49) and DBP (mean percentile = 38.25%^{ile}, *SD*=25.30) were below average compared to the general population (Xi et al., 2016).

Group comparisons. SBP Z-scores were comparable across sexes, while boys showed lower DBP Z-scores than girls. This indicates that boys had lower DBP *Z*-scores relative to age-, sex-, and height-based normative values. There were no differences in blood pressure *Z*-scores across racial groups or by family history of elevated blood pressure. Girls were found to spend more time in stage N2 and stage N3 sleep than boys. Boys had greater WASO than girls. Group differences were found for race in actigraphy-derived wake time and number of awakenings and self-reported sleep quality. Children of mixed race or other racial status had later wake times than other racial groups. Black and Asian youth had more awakenings than other racial groups. Black youth reported the lowest sleep quality, while children of mixed race or other racial group reported the highest sleep quality. Youth with a family history of elevated blood pressure showed

later actigraphy-measured bedtime, wake time, and midpoint but lower WASO than youth without a family history of elevated blood pressure. Results from group comparisons are presented in Tables 4, 5, and 6 (Appendix D).

Sleep dimensions. We tested the hypothesis that poorer sleep (shorter sleep duration, later sleep timing, greater awakenings / arousals, reduced time in stage N3 sleep) would be associated with higher systolic and diastolic blood pressure. Shorter sleep duration was associated with higher SBP Z-score (*B*=-.004, *t*=-2.264, *p*=.025). Later bedtime was associated with higher DBP Z-score (*B*=.00003, *t*=2.067, *p*=.041). No significant associations were observed for wake time or midpoint with blood pressure. (Note these sleep timing metrics were derived from actigraphy.) For awakenings / arousals, youth with more actigraphy-measured nocturnal awakenings had lower DBP Z-scores (*B*=-.052, *t*=-2.156, *p*=.033); WASO was unrelated to blood pressure for both actigraphy- and PSG-derived measures. Higher PSG-derived arousal index (*B*=.037, *t*=-2.719, *p*=.007) was associated with higher DBP Z-score. For sleep stages, greater percentage of time spent in N1 sleep (*B*=.092, *t*=3.764, *p*<.001), and lower percentage of time in N3 sleep (*B*=-.026, *t*=-2.009, *p*=.046), were associated with higher DBP Z-score. No associations were found for onset latency, sleep efficiency, or reported sleep quality with blood pressure. See Table 3 for full results.

Discussion

Past research supports the association between poorer sleep and elevated daytime blood pressure (Fobian et al., 2018). The present study examined associations between actigraphy- and PSG-measured sleep and casual blood pressure in a community sample of youth. Youth were primarily healthy with SBP and DBP values within the normal range. The proportion of youth with elevated blood pressure was aligned with previous estimates, but mean blood pressure

values were notably lower than casual blood pressure values reported in comparable samples (Barba et al., 2014; Dong et al., Neuhauser et al., 2011; Sharma et al., 2018; Xi et al., 2016).

Blood pressure Z-scores were largely comparable across sex, race, and family history of elevated blood pressure. Boys had lower mean DBP Z-scores than girls. This differs from most past research, which indicates that boys are more likely to have higher blood pressure than girls (Dasgupta et al., 2006; Syme et al., 2009: Wang et al., 2006). However, both male and female youth in our sample had mean blood pressure within the healthy range. Girls spent more time in stage N2 sleep compared to boys. There is minimal research on sex differences in sleep stages in youth, but this finding has been observed in adults (Ohayon et al., 2004). Differences were observed across racial groups in sleep dimensions. Of note, children from visible minority groups showed poorer sleep for certain dimensions. Black youth reported the lowest subjective sleep quality, and Black and Asian youth showed more actigraphy-measured awakenings than other racial groups. Racial disparities in sleep have been widely documented in the United States, with Black youth at particularly high risk of poorer sleep (El-Sheikh et al., 2016; Guglielmo et al., 2018). Youth with a family history of elevated blood pressure showed later sleep timing and lower WASO, compared to children without a positive family history. Past studies have shown that family history of high blood pressure is a risk factor for hypertension in the child, but its relation with sleep is less clear (Pacienca et al., 2013; Wang et al., 2008). It should be noted that there were minimal group differences across sex, race, and family history of elevated blood pressure, and the observed differences were found in sleep dimensions with no association to blood pressure values. Instead, these differences may indicate areas of potential risk. It is uncertain which factors contribute to the emerging associations between sleep and blood pressure

in youth, and demographic and family variables may be an area of focus in future research for prevention targets.

Our findings indicated that shorter sleep duration was significantly associated with higher SBP. Sleep duration has been widely linked to blood pressure, and insufficient sleep is acknowledged as a significant risk factor for hypertension. These findings provide further support that the quantity of sleep plays an important role in regulating cardiovascular functions in healthy youth. Short sleep duration is often discussed as a cardiovascular risk factor in the context of full-time employment, shift work, and age-related sleep changes, but increasing research shows the health risks of insufficient sleep in youth. Up to a third of children and adolescents report sleeping less than the recommended amount, representing a potential target for preventing hypertension (Patte et al., 2017; Sousa-Sá et al., 2021).

Later bedtime was associated with higher DBP. Past research has shown that children and adolescents with later bedtimes are at increased risk of developing hypertension, beyond the effects of shorter sleep duration (Jansen et al., 2020; Matricciani et al., 2021; Mi et al., 2019; Wang et al., 2023). This relation is primarily attributed to misalignment of the circadian rhythms that regulate blood pressure patterns. Circadian rhythms are partly entrained through environmental and behavioural cues such as light exposure, physical activity level, and caloric intake. Changes in timing of sleep can lead to desynchronization of the circadian rhythms, which has been associated with increased risk for hypertension and cardiovascular disease. The impact of circadian misalignment may be compounded in youth as adolescents experience changes in sleep pattern with the onset of puberty. Adolescents often have later bed and wake times due to delayed circadian timing (i.e., sleep phase delay) combined with a reduction in sleep pressure (i.e., homeostatic drive to fall asleep; Crowley et al., 2006). It is unclear how these

developmental changes in sleep timing might influence blood pressure and risk for hypertension, and more research is needed to examine the role of circadian timing specifically in pediatric blood pressure. We did not find any associations between blood pressure and wake time or midpoint; future research could incorporate sleep dimensions that capture daily changes in sleep pattern (e.g., interdaily stability; intradaily variability; relative amplitude; Hoopes et al., 2021; ren et al., 1999).

Our findings were more mixed for the sleep dimension of awakenings / arousals. Significant associations were observed between arousal index and DBP Z-score. Higher frequency of arousals during the night was associated with higher DBP. This is consistent with findings showing that arousal index and sleep fragmentation are linked to higher blood pressure and risk for hypertension (Dean et al., 2015; Ekstedt et al., 2004; Ren et al., 2022). Research suggests that significant fragmentation reduces the restorative properties of sleep. This finding has been noted even without reduced sleep time or significant nighttime awakenings; disruptions in the continuity of sleep have been associated with greater daytime sleepiness and impairment even when the total sleep time is adequate (Brian, 1987; Veauthier et al., 2013). Interestingly, no associations were found between WASO and blood pressure in our sample, while greater number of awakenings measured with actigraphy was associated with lower DBP Z-scores. Since micro-arousals are brief increases in EEG activity rather than true awakenings, this may explain why they influence blood pressure differently. It might be expected that awakenings would be more strongly associated with high blood pressure as the individual would spend more time awake but is possible that greater frequency of brief arousals increases risk for elevated blood pressure even without full awakenings during the night. This result may also reflect differences in measurement, as PSG was recorded for one night compared to 14 days of actigraphy recording.

The specific roles of arousals and awakenings in predicting blood pressure are unclear due to lack of consistency in definition across studies, so further research is recommended to replicate these findings (Stepanski et al., 2002).

Sleep onset latency and sleep efficiency were not associated with blood pressure. Along with the lack of association with WASO, this may suggest that time spent awake in bed has less influence on blood pressure than other aspects of sleep timing or continuity. Few previous studies have included sleep onset latency, but past research indicates that poorer PSG-measured sleep efficiency is associated with higher blood pressure (Javaheri et al., 2008). This discrepancy in results may be related to measurement, as there is significant variability in how sleep efficiency is defined across studies (Reed & Sacco, 2016). Sleep efficiency is often considered to be a measure of sleep quality, and our sample similarly did not show any associations between self-reported sleep quality and blood pressure. Adult studies report greater likelihood of hypertension associated with poor sleep quality, but pediatric data are limited (Lo et al., 2018). Associations with cardiovascular outcomes appear to be stronger with objective sleep measurement in youth, and possibly due to reduced accuracy from parent-report measures or using self-report measures in younger children (Matthews & Pantesco, 2016).

Youth with a higher percentage of N1 and lower percentage of N3 sleep were more likely to have higher DBP. Consistent with these findings, past studies suggest that adults with hypertension spend more time in N1 (i.e., light sleep) and less time in N3 (i.e., SWS sleep) relative to normotensive samples (Kanklerska et al., 2023; Liao et al., 2016). In particular, time spent in SWS is a significant predictor of hypertensive status (Fung et al., 2011; Javaheri et al., 2018; Matthews et al., 2014). Autonomic nervous system activity likely plays a key role in the relation between SWS and cardiovascular functioning. In healthy youth and adults, blood

pressure and heart rate typically decrease during SWS and increase to above waking levels in REM sleep (Silvani, 2008; Somers et al., 1993; Trinder et al., 2001). During NREM sleep, autonomic control is characterized by reduced sympathetic activity and greater parasympathetic dominance; sympathetic activity is predominant in REM sleep (Tobaldini et al., 2014). Experimental studies show that sleep restriction and reduced SWS sleep are associated with a shift in sympathovagal balance toward sympathetic dominance and parasympathetic withdrawal (Tasali et al., 2008; Zhong et al., 2005). Youth with increased N1 sleep and decreased N3 sleep thus spend more of the sleep period with higher blood pressure and heart rate. Higher nocturnal blood pressure has been identified as a risk factor for hypertension and may even be more predictive of cardiovascular outcomes than daytime blood pressure (Hansen et al., 2010; Tsioufis et al., 2011).

DBP was associated with several different sleep dimensions across actigraphy- and PSG-measured sleep, while SBP was only related to actigraphy-derived sleep duration. Specifically, higher DBP was associated with later bedtime, higher arousal index, more time spent in stage N1 sleep, and less time spent in stage N3 sleep. These findings indicate that poorer sleep is more strongly linked to higher DBP values, while the relation with SBP is less pronounced. Although SBP and DBP values are typically closely related, they may have different effects on various health outcomes. For example, SBP is generally considered to be a better predictor of risk for cardiovascular and cerebrovascular disease, but some research suggests that DBP is more strongly related to cardiovascular functioning (Cai et al., 2016; Dai et al., 2020). The individual roles of SBP and DBP are unclear, but they may differ in their associations to sleep. Additionally, it must be noted that our sample had lower than average SBP and DBP values, which may explain the relative lack of associations with sleep. Most youth had blood pressure

values within a limited range and there may not have been sufficient variability to observe associations between the variables. This could be related to high parental education level in our sample. Pediatric studies often recruit participants with lower parental education or socioeconomic status as these are risk factors for poor sleep and hypertension, and associations may be more visible in these samples (Bizzotto Trude et al., 2023).

This was one of the first studies to examine associations between pediatric casual blood pressure and objectively measured sleep dimensions. We found that higher blood pressure was associated with a range of sleep dimensions, including sleep duration, bedtime, arousals, and sleep stages. Sleep duration has been the primary target of most pediatric blood pressure research, but these findings emphasize the need for thorough sleep measurement and inclusion of distinct sleep dimensions. This is especially important in child and adolescent research, as pediatric blood pressure has lagged behind adult research. Adult practices have often been adapted for youth without sufficient consideration of developmental factors, such as growth, puberty, and changes in sleep pattern. Consequently, there remain significant gaps in our understanding of early risk factors and the developmental course of hypertension. The current study represents a first step in comprehensively evaluating the association between pediatric sleep and blood pressure. More research is needed to extend these findings and further examine the roles of various sleep dimensions and circadian timing.

Strengths and Limitations

The present study had several methodological limitations that warrant discussion. First, PSG data were derived from a single night of at-home recording. PSG is the gold standard of sleep assessment but is known to produce a "first night effect" (Miettinen et al., 2018). One night of data does not fully represent an individual's sleep patterns, and sleep may appear poorer as the

wearer adjusts to the device. Future studies should incorporate multiple nights of PSG when possible; however, ambulatory PSG is not practical for long-term sleep measurement given the invasiveness and short battery life. Further, current hardware and data storage limitations make multi-night ambulatory PSG recordings nearly technically impossible. Second, participants were predominantly healthy youth recruited from the community. Our sample had unusually low SBP and DBP percentiles, which may have masked associations between the variables. This may be related to the high parental education level, as most parents had completed a university degree. Parental education is a marker of socioeconomic status and is associated with blood pressure in youth (Kwok et al., 2016). Intentionally recruiting specific clinical and at-risk populations (e.g., obesity) along with healthy children could provide a wider range of blood pressure values and potentially reveal more prominent relationships with sleep. This work could also be extended by including ambulatory blood pressure monitoring (ABPM) to capture 24-hour blood pressure in combination with sleep assessment. There is very limited research on ABPM and sleep in healthy children, so this may be the next step in understanding the development of blood pressure patterns and hypertension from childhood.

Despite some limitations, this is one of few studies to include both actigraphy and PSG in investigating associations between children's blood pressure and sleep. Sleep was assessed across 14 days of actigraphy and one night of PSG, yielding information about youths' daily sleep patterns as well as single-night sleep architecture in a naturalistic setting. Comprehensive sleep assessment allowed examination of multiple sleep dimensions, including sleep timing, sleep duration, onset latency, sleep disruptions, sleep efficiency, and sleep stages. Research in this area has focused on sleep duration, but our findings support the potential role of other sleep dimensions in regulating blood pressure. These include sleep fragmentation (i.e., awakenings,

arousals) and sleep stages, which may be associated with autonomic functioning and risk for elevated blood pressure. Additionally, there is need for more investigation of the role of sleep timing and irregularity of sleep (i.e., within-person variation in sleep). Blood pressure levels are closely related to sleep-wake patterns, and disruption in circadian timing is a known predictor of hypertension and cardiovascular disease (Morris et al., 2016). This may be particularly relevant in youth as adolescents are biologically predisposed to experience delayed sleep phases (i.e., naturally sleeping and waking later) and are thus already at increased risk for circadian misalignment (Crowley et al., 2007; Crowley et al., 2014). Future studies should explore the impact of sleep timing, irregularity, fragmentation, and sleep stages in healthy and high-risk youth using comprehensive, objective sleep measurement. Understanding the effects of sleep variables beyond sleep duration will be critical in identifying early risk factors and targets for hypertension prevention.

Table 1. *Definition of sleep dimensions*.

	Actigraphy	PSG
Timing	Bedtime Onset of rest interval ("lights out") Wake time Offset of rest interval ("lights on") Midpoint Median time between bedtime and wake time	N/A
Duration	Number of minutes between onset of sleep interval and final awakening	N/A
Latency	Number of minutes between onset of rest interval ("lights out") and onset of sleep interval	N/A
Awakenings	Number of awakenings Total number of discrete awakenings (contiguous epochs scored as wake separated by ≥1epoch scored as sleep) within the sleep interval WASO Total duration of awakenings in minutes	Number of awakenings Total number of discrete awakenings (contiguous epochs scored as wake separated by ≥1epoch scored as sleep) within the sleep interval WASO Total duration of awakenings in minutes Arousal Index Total number of arousals divided by the total sleep time (i.e., number of minutes between sleep onset and final awakening)
Efficiency	N/A	Percentage of time spent asleep between the sleep onset and final awakening
Sleep stages	N/A	% time in N1 Percentage of sleep interval spent in stage N1 sleep % time in N2 Percentage of sleep interval spent in stage N2 sleep % time in N3 Percentage of sleep interval spent in stage N3 sleep; known as slow-wave sleep

		% time in REM Percentage of sleep interval spent in REM sleep.

Table 2. *Descriptive characteristics of children and adolescents (N=307).*

Variable	***M (SD)***	***N (%)***
Age (years)	12.74 (2.05)	
Sex (% female)		134 (43.6)
Race/ethnicity (%)		
White		194 (63.2)
Asian		24 (7.8)
Black		16 (5.2)
Latin American		11 (3.6)
Middle Eastern/North African		7 (2.3)
Indigenous		4 (1.3)
Mixed race/other		51 (16.6)
Parental education (years)	16.46 (3.14)	
Body mass index (BMI) percentile	56.01 (28.93)	
Gonadarche (1-5)	2.72 (1.63)	
Adrenarche (1-5)	2.93 (1.60)	
Family history of high BP (%yes)		143 (46.6)
Family history of CVD (%yes)		144 (46.9)
Blood Pressure		
SBP (mmHg)	100.87 (12.77)	
DBP (mmHg)	59.58 (9.81)	
SBP percentile	28.66 (26.49)	
DBP percentile	38.25 (25.30)	
Hypertensive status		
Normal		271 (88.3)
Elevated		12 (3.9)
Hypertension		16 (5.4)

Table 3. *Associations between casual blood pressure and sleep dimensions*.

	B	Unstd. *SE*	*β*	*t*	*p*
Systolic Blood Pressure					
Actigraphy					
Bedtime	.00001	.000	.058	.684	.495
Wake time	-.00002	.000	-.081	-.934	.352
Midpoint	.0000002	.000	-.001	-.007	.994
Duration	-.004	.002	-.193	-2.264	.025*
Sleep onset latency	.003	.010	.026	.301	.764
Number of awakenings	-.031	.027	-.099	-1.143	.255
WASO	.000	.004	-.009	-.106	.916
PSG					
Sleep onset latency	-.003	.004	-.067	-.902	.368
Number of awakenings	.004	.011	.031	.409	.683
WASO	.004	.003	.094	1.224	.223
Efficiency	.001	.001	.013	.173	.863
Arousal index	-.002	.017	-.008	-.111	.912
% time in N1	.060	.032	.143	1.900	.059
% time in N2	.008	.007	.086	1.147	.253
% time in N3	-.007	.007	-.068	-.893	.373
% time in REM	-.026	.014	-.187	-1.849	.068
Self-report sleep quality	-.083	.141	-.076	-.589	.558
Diastolic Blood Pressure					
Actigraphy					
Bedtime	.00003	.000	.177	2.067	.041
Wake time	.00002	.000	.098	1.117	.266
Midpoint	.00003	.000	.152	1.754	.082
Duration	-.003	.002	-.172	-1.972	.051
Sleep onset latency	-.006	.009	-.064	-.723	.471
Number of awakenings	-.052	.024	-.188	-2.156	.033*
WASO	-.00007	.002	-.001	-.008	.994
PSG					
Sleep onset latency	.002	.003	.038	.515	.607
Number of awakenings	.009	.008	.082	1.070	.286
WASO	.001	.003	.025	.320	.749
Efficiency	-.007	.006	-.090	-1.439	.152
Arousal index	.037	.013	.197	2.719	.007*
% time in N1	.092	.024	.278	3.764	<.001*
% time in N2	.007	.006	.094	1.242	.216
% time in N3	-.012	.006	-.151	-2.009	.046*
% time in REM	-.022	.012	-.180	-1.852	.067
Self-report sleep quality	-.027	.135	-.026	-.199	.843

Note. *p<.05. All analyses controlled for parent education and pubertal status (gonadarche).

TRANSITION TO GENERAL DISCUSSION

The purpose of Study 3 was to investigate associations between specific sleep dimensions and casual blood pressure in children and adolescents. A wide range of sleep dimensions were derived from objective measurements to accurately reflect the complex nature of sleep. Relevant demographic and participant variables (e.g., age, sex, height, pubertal status) were considered in analyses given their potential role in pediatric blood pressure.

Broadly, findings showed that poorer sleep was significantly associated with higher blood pressure. The associations between sleep and blood pressure were fairly consistent with those observed in Study 2; higher blood pressure was linked to shorter sleep duration and more frequent sleep disruptions (i.e., arousals or awakenings). Further, Study 3 showed that higher blood pressure was seen in youth with later bedtimes, less time spent in deep sleep (i.e., less stage N3 sleep, more stage N1 sleep), and poorer overall sleep. Of note, I collected information on timing and sleep stages for the meta-analysis in Study 2, but there were minimal data available and high variability in how sleep dimensions were defined. The present findings are thus an important contribution in this area as they provide further evidence of how different sleep dimensions are related to pediatric blood pressure.

Across all three studies in this dissertation, there was evidence that demographic, health-related, and participant characteristics may affect associations between blood pressure and sleep. In Study 3, differences in blood pressure values and sleep dimensions were observed for sex, race, and presence of family history of hypertension. These variables did not appear to influence associations between blood pressure and sleep, but may reflect areas of interest for future research. As we improve our understanding of relations between pediatric blood pressure and

sleep, it will be critical to identify potential moderating factors as these could guide development of prevention and treatment of hypertension.

GENERAL DISCUSSION

My doctoral research program aimed to examine the association between sleep and blood pressure in children and adolescents. Poor sleep is a known predictor of high blood pressure and hypertension. Research has mainly focused on short sleep duration as a health risk factor, but there is growing evidence that other dimensions of sleep contribute to the relation between sleep and blood pressure. Identification of early risk factors and signs of hypertension have become increasingly important with rising prevalence rates, but there is a need for more research on the development of these associations earlier in life. The broad goal of my dissertation was to explore blood pressure measurement in youth and its relations with objective, multidimensional measures of sleep.

Study 1 examined 24-hour ABPM through a scoping review of measurement practices and meta-analysis comparing day and night blood pressure in youth. Although ABPM is considered the gold-standard method of assessing blood pressure, measurement practices were often inconsistent with consensus recommendations. Studies showed only moderate fidelity with published guidelines, and there was a clear lack of reporting on methodological decision-making. Many studies only reported 24-hour means, preventing evaluation of blood pressure dipping and diurnal variation. This is problematic, as the meta-analysis indicated that youth have significant differences in day and night blood pressure. These findings suggest that blood pressure dipping is present from early in life, but most studies do not optimize their data reporting to allow for investigation of this effect across samples. It was evident that although youth showed diurnal variation in blood pressure, it is difficult to draw conclusions about the relations between ABPM and sleep with the available data.

Study 2 built on the findings from Study 1 to examine associations between sleep and casual (i.e., office) blood pressure. With the limitations of 24-hour blood pressure research, there remained questions regarding the association between blood pressure and sleep in youth. Thus, Study 2 provided a meta-analytic summary of existing literature examining relations between sleep and casual blood pressure. Overall, results showed that poorer sleep was associated with higher blood pressure. This was observed across sleep duration, awakenings, and overall poor sleep, suggesting that multiple dimensions of sleep may play a role in blood pressure. The associations were stronger with age, meaning that the risk associated with poor sleep may become greater over time. These findings highlighted the importance of comprehensively assessing sleep when studying associations with blood pressure, particularly in children given the relative lack of pediatric research.

Study 3 aimed to address remaining knowledge gaps by exploring associations between diverse sleep dimensions and casual blood pressure in a large community cohort of youth. Sleep was measured objectively using actigraphy and PSG to accurately capture multiple aspects of sleep. Consistent with Study 1 and 2, we found that higher blood pressure was related to poorer sleep across several dimensions, including sleep duration, percentage of stage N1 and N3 sleep, and frequency of arousals. These associations were present in a generally healthy, community sample of children and adolescents, suggesting that sleep may begin to influence blood pressure before the emergence of symptomatic disease or adverse effects. Future studies are needed to understand the role of different sleep dimensions and how they may be differentially associated with blood pressure in children.

Conclusions and Implications

These three complementary studies provide a comprehensive understanding of pediatric blood pressure measurement and its associations with various sleep dimensions. Compared with adult research, there is a relative lack of understanding of the potential impact of poor sleep in youth. Pediatric blood pressure research has often lagged behind adult research due to the view of hypertension as an adult health condition; this has led to adult measurement practices and knowledge of physiological processes being applied to children's blood pressure without thorough investigation of relevant developmental factors (Falkner et al., 2023; Macumber, 2017). The present studies represent some of the first research to thoroughly examine pediatric blood pressure and sleep while considering child-specific factors and measurement challenges.

The reviews conducted in Studies 1 and 2 clearly showed that there are remaining gaps in our knowledge of children's blood pressure and sleep. Measurement practices are often inconsistent with best practices, and researchers rarely assess sleep thoroughly when recording blood pressure data. This was seen across both ABPM and casual blood pressure research, suggesting that both areas would benefit from changes to improve measurement and data reporting standards. This would allow optimal use of the data collected and provide a more complete understanding of factors influencing children's blood pressure. Currently, it is challenging to summarize and compare data across studies due to significant variability in how blood pressure and sleep dimensions are measured and defined. These studies were an initial step in compiling and harmonizing existing data, but there is a need for more work to develop practices and data reporting standards. This is particularly important as research guides the development of clinical guidelines and intervention programs, and improved accuracy is needed for effective treatment and prevention initiatives.

Our findings consistently demonstrated that the relations between blood pressure and sleep are present from early in the lifespan. Furthermore, we provided evidence that blood pressure is linked to multiple sleep dimensions and circadian processes. A significant strength of the current program of research is the comprehensive sleep measurement across studies; multiple sleep dimensions and objective measures were included to ensure that sleep was fully depicted as a multidimensional construct. Findings from the meta-analysis in Study 2 were consistent with original data in Study 3, in that multiple sleep dimensions were linked to casual blood pressure, rather than sleep duration alone. More research is needed to investigate these associations using prospective, longitudinal designs to evaluate how relations may change over time. My dissertation included only cross-sectional data, preventing any inferences about causality and further supporting the need for longitudinal and experimental data. There is also a need for more research examining developmental factors, such as puberty and age-related changes in sleep patterns, more closely. Our findings indicate that sleep may become more strongly associated with blood pressure with age, and it will be important to identify factors contributing to this effect. Childhood and adolescence are critical timepoints for preventing later hypertension and cardiovascular disease, and understanding the progression of risk will allow for more targeted interventions.

Finally, there was evidence across my three studies of a potential role of circadian timing in blood pressure. Study 1 showed that children experience diurnal variation in blood pressure (i.e., differences between day and night ABPM). This is supported by Studies 2 and 3, which suggested that sleep timing and irregularity influence daytime casual blood pressure. In the literature, sleep patterns (e.g., timing, irregularity) are often used as markers of circadian timing and are considered potential risk factors for circadian misalignment; however, these are not the

most accurate measures of circadian timing. Future studies could consider measuring established biomarkers of circadian timing to examine whether these are linked to associations between blood pressure and sleep dimensions. Examples include core body temperature, dim light melatonin onset (i.e., onset time of melatonin secretion) and salivary cortisol levels (Kennaway, 2023; Shimada et al., 1995; Waterhouse et al., 2005). The present studies focused on breadth of sleep assessment and thus could not include detailed examination of every dimension of sleep and circadian timing, but this warrants further examination in pediatric samples.

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Appendix A: Forest plots for meta-analytic models in Study 1.

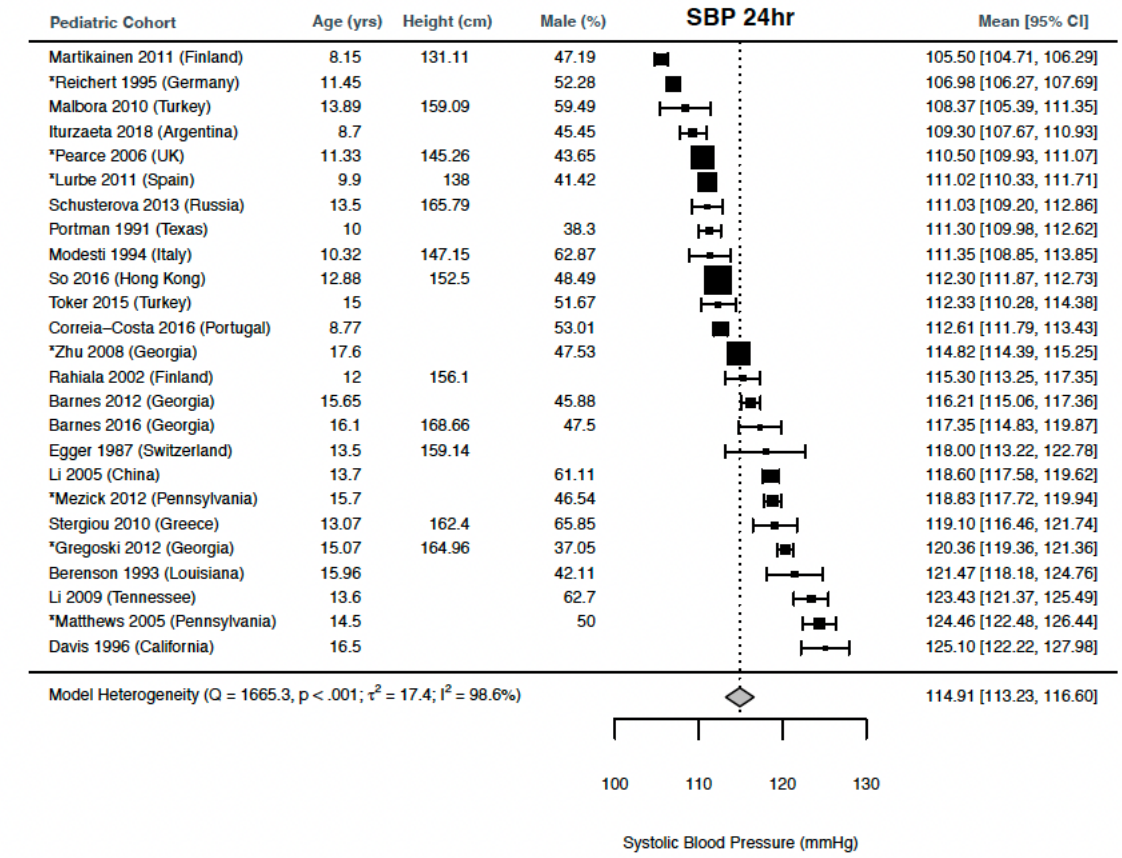

Figure 3. Hedges' *g* effect sizes for 24-hour systolic blood pressure.

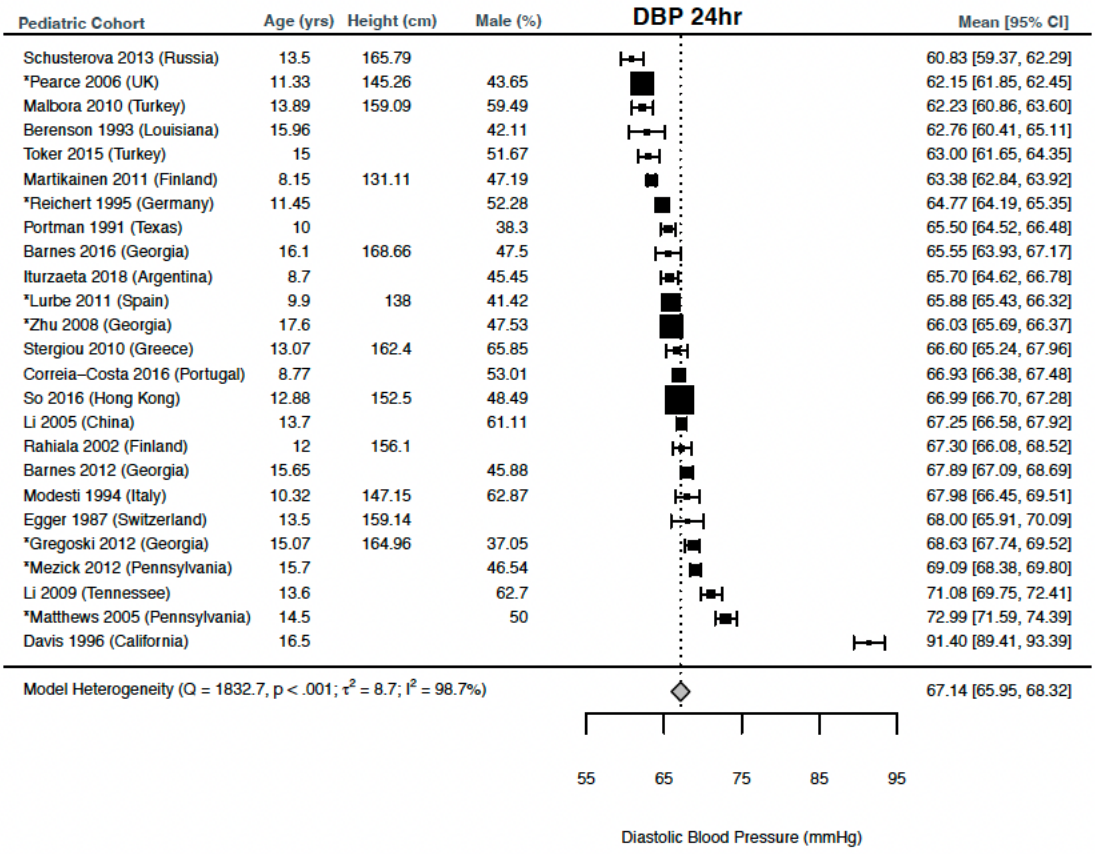

Figure 4. Hedges' *g* effect sizes for 24-hour diastolic blood pressure.

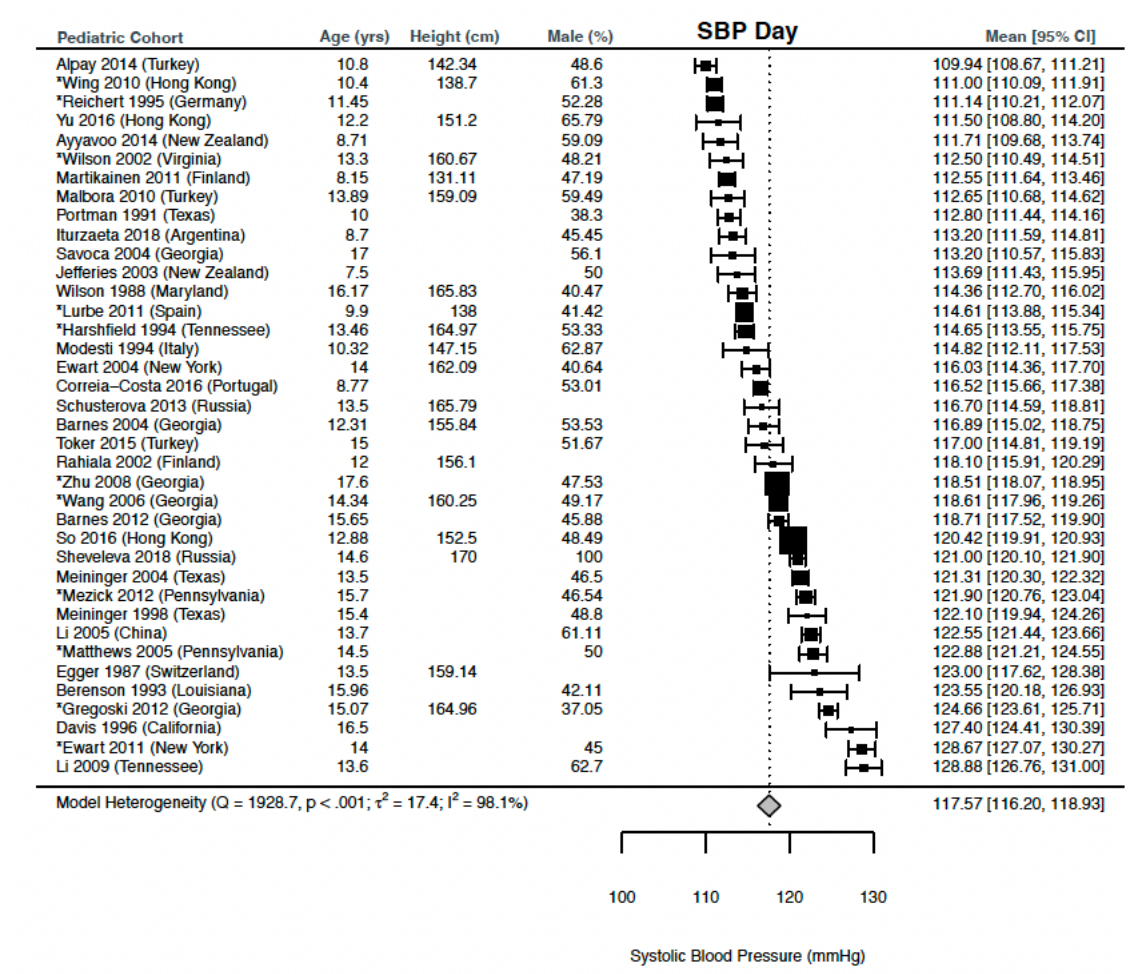

Figure 5. Hedges' *g* effect sizes for daytime systolic blood pressure.

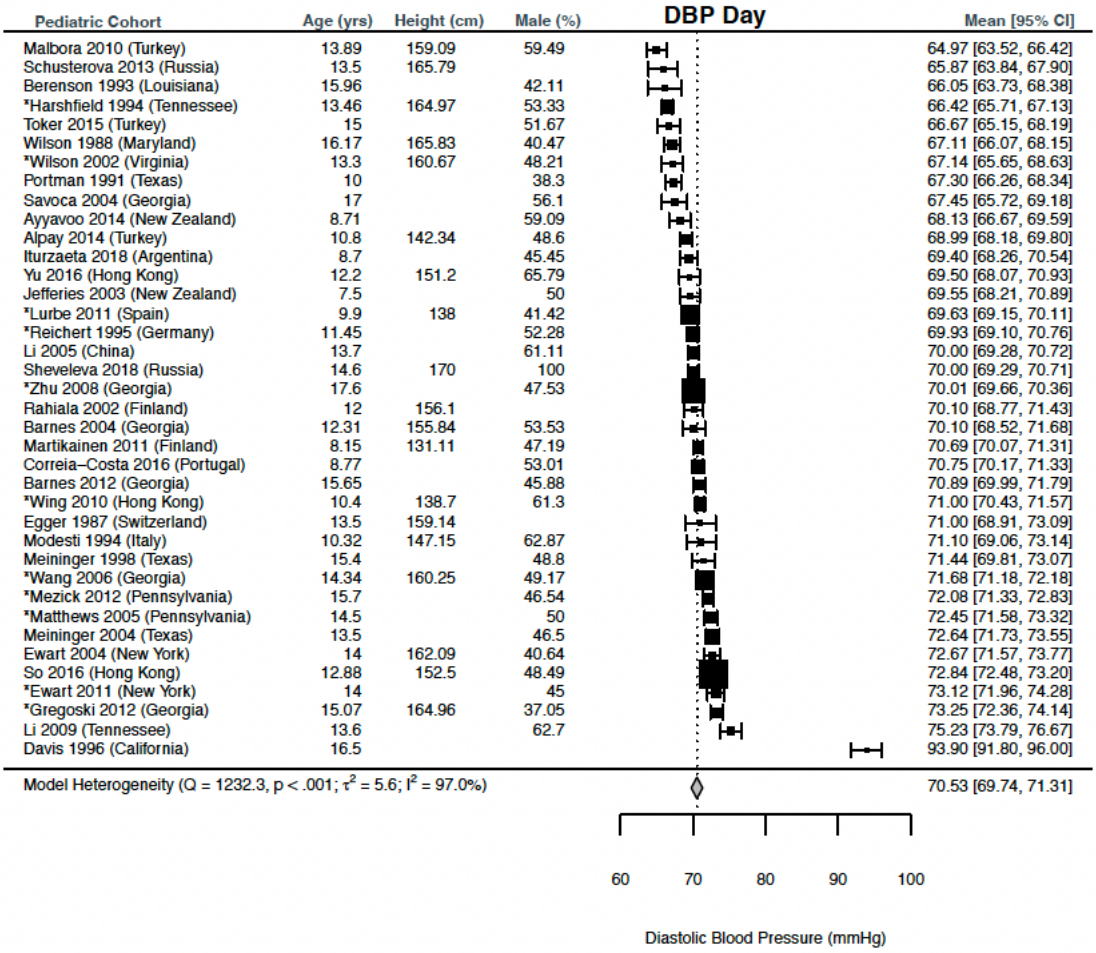

Figure 6. Hedges' *g* effect sizes for daytime diastolic blood pressure.

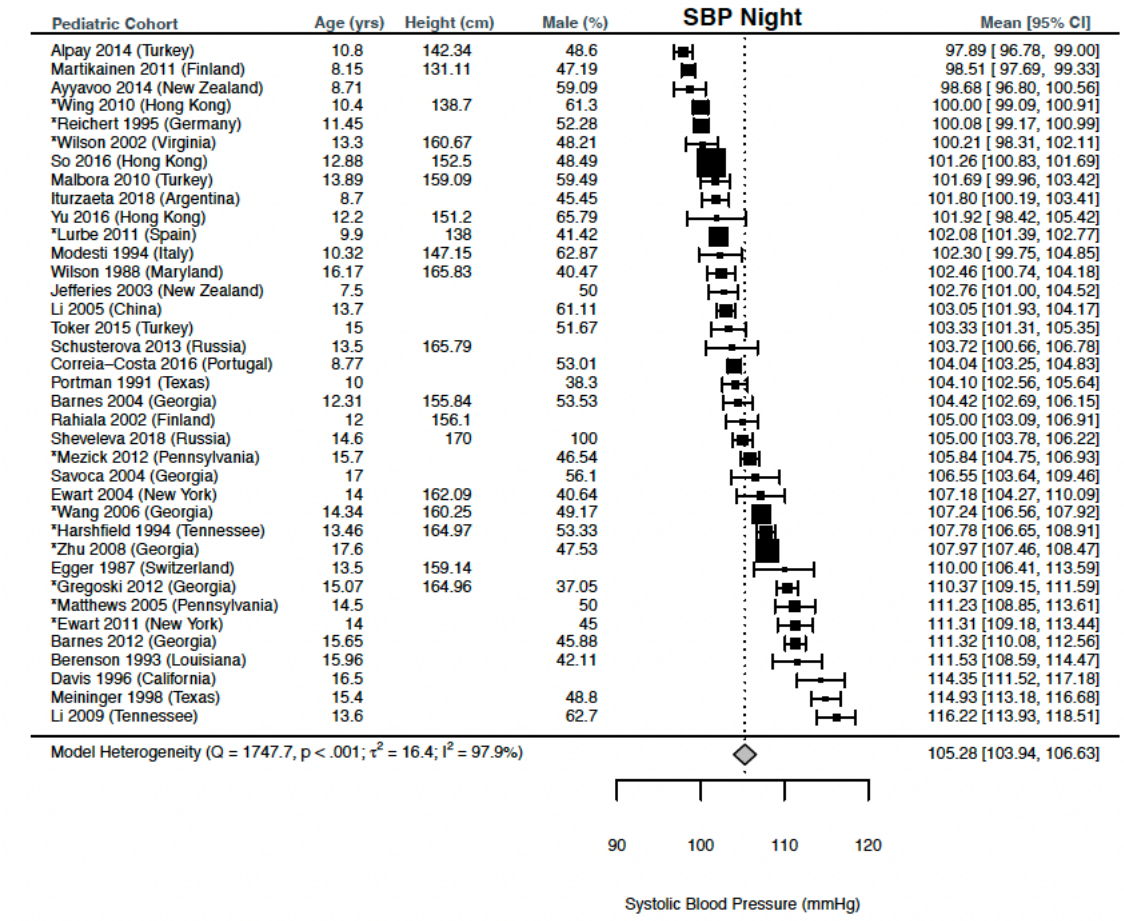

Figure 7. Hedges' *g* effect sizes for night systolic blood pressure.

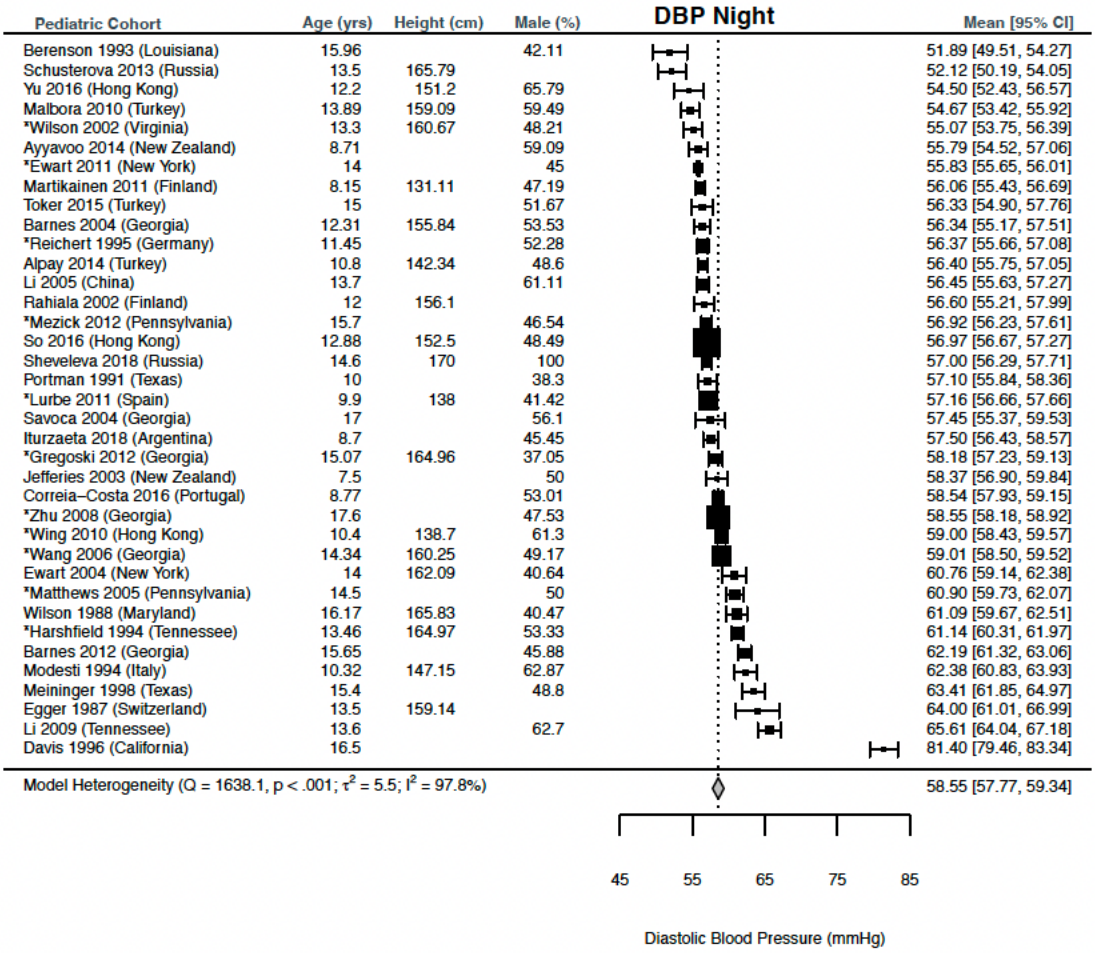

Figure 8. Hedges' *g* effect sizes for night diastolic blood pressure.

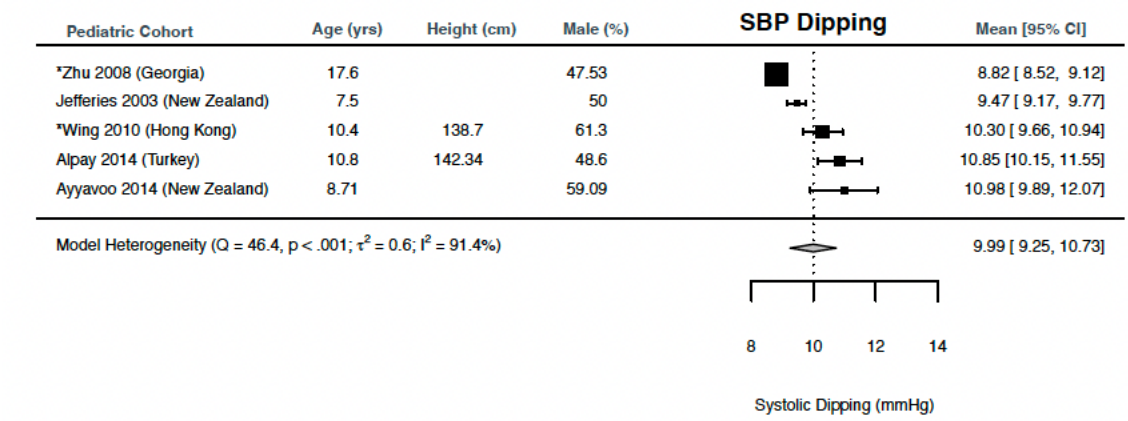

Figure 9. Hedges' *g* effect sizes for systolic blood pressure dipping.

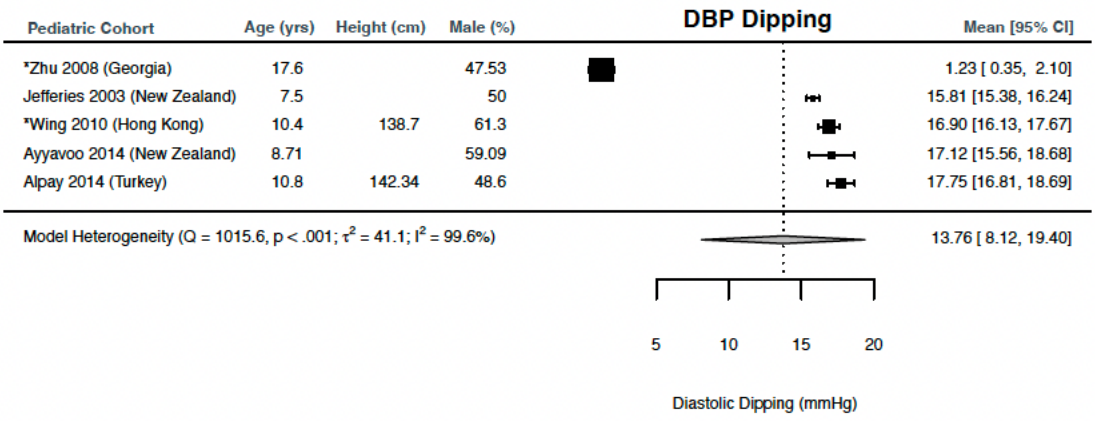

Figure 10. Hedges' *g* effect sizes for diastolic blood pressure dipping.

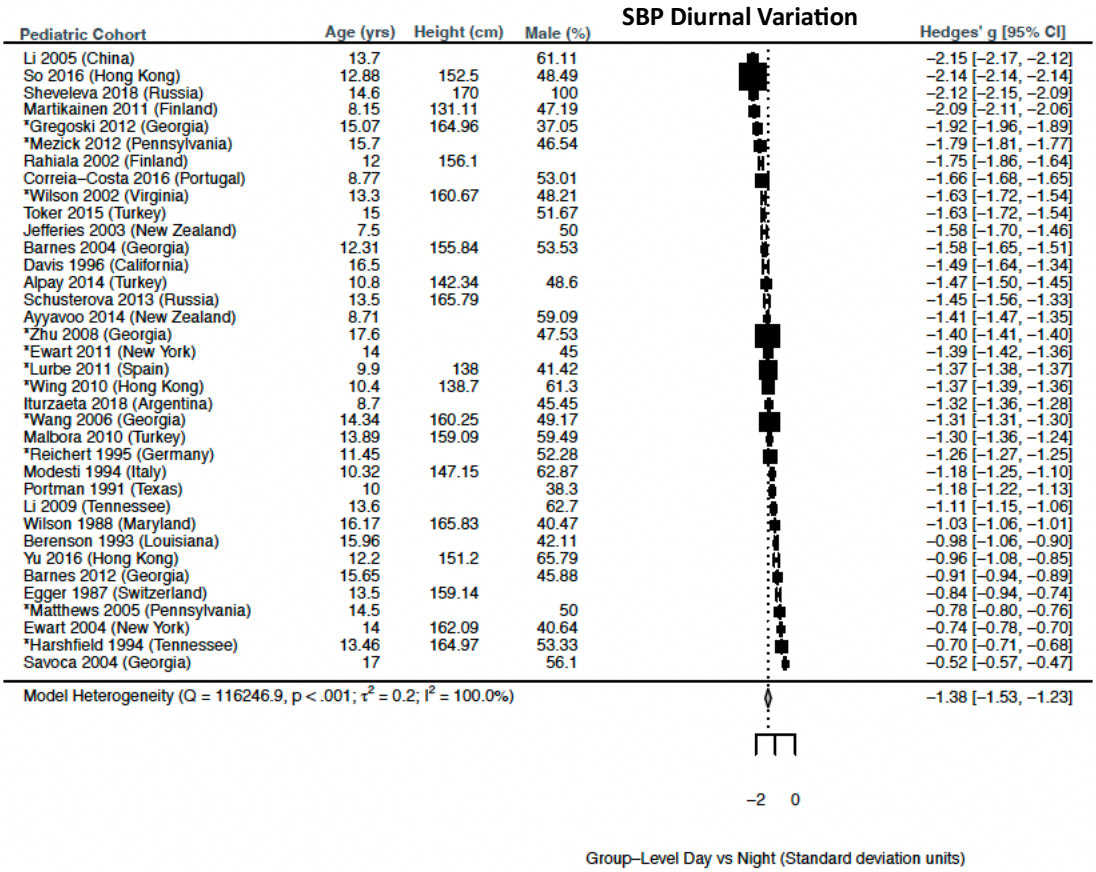

Figure 11. Hedges' *g* effect sizes for diurnal variation in systolic blood pressure.

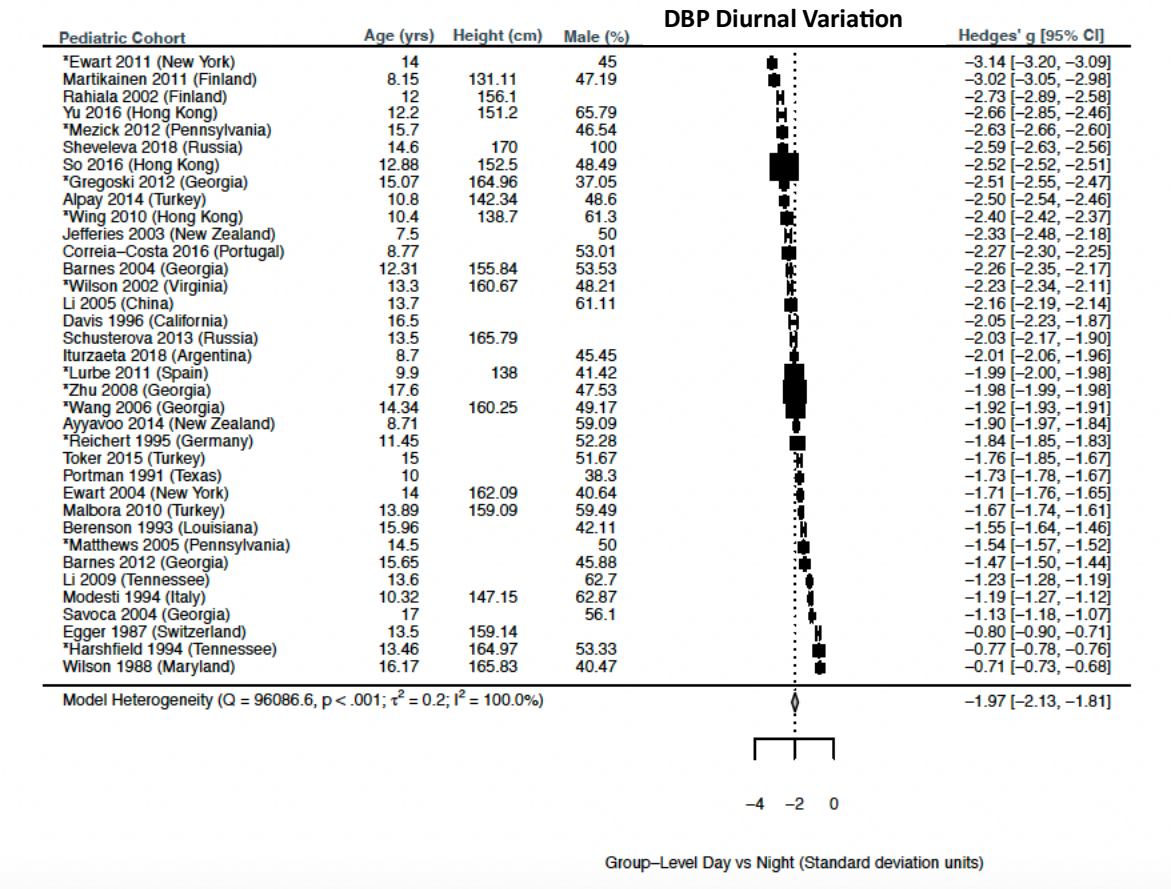

Figure 12. Hedges' *g* effect sizes for diurnal variation in systolic blood pressure.

Appendix B: Meta-regression models for Study 1.

Table 6. *Meta-Regression Models for Continuous Effect Modifiers*.

Age (yrs)	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ**	**Egger**
Systolic Blood Pressure											
24h ABPM	25	6715	**1.334**	**.32**	**<.001**	964.16	<.001	**42.0%**	98.4%	4.10	.153
Day ABPM	37	8476	**1.031**	**.28**	**<.001**	1375.75	<.001	**27.1%**	98.1%	4.34	.574
Night ABPM	36	8105	**1.032**	**.23**	**<.001**	843.66	<.001	**37.2%**	97.2%	3.55	.097
Noctural Dipping (Individual)‖	5	1577	-0.131	.11	.233	29.758	<.001	8.1%	87.0%	0.84	.000
Diurnal Variation (Group)	36	8105	0.037	.03	.174	581.08	<.001	2.7%	93.0%	0.40	.535
Diastolic Blood Pressure											
24h ABPM§	24	6681	0.174	.23	.443	1041.85	<.001	0.0%	98.5%	2.81	.861
Day ABPM§	36	8442	0.000	.16	.999	760.16	<.001	0.0%	96.9%	2.33	.024
Night ABPM§	35	8071	0.098	.19	.600	968.19	<.001	0.0%	98.5%	2.82	.819
Noctural Dipping (Individual)‖	4	605	**0.481**	**.13**	**<.001**	1.96	.375	**98.2%**	4.0%	0.105	.290
Diurnal Variation (Group)	36	8105	0.066	.04	.076	766.13	<.001	6.3%	95.8%	0.57	.655
Sex (% Male)	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ**	**Egger**
Systolic Blood Pressure											
24h ABPM	21	6544	0.071	.15	.637	1571.96	<.001	0.0%	99.3%	5.46	.157
Day ABPM	33	8305	-0.001	.08	.987	1860.33	<.001	0.0%	98.8%	5.12	.748
Night ABPM	32	7934	-0.042	.07	.549	1525.96	<.001	0.0%	98.5%	4.43	.124
Noctural Dipping (Individual)‖	5	1577	0.078	.07	.273	26.632	<.001	6.5%	92.9%	.84	.022
Diurnal Variation (Group)	32	7934	-0.008	.01	.240	578.09	<.001	1.7%	94.3%	.41	.364
Diastolic Blood Pressure											

24h ABPM[§]	21	6544	0.060	.08	.427	1010.91	<.001	0.0%	98.7%	2.74	.947
Day ABPM[§]	33	8305	-0.006	.04	.872	734.63	<.001	0.0%	97.2%	2.34	.038
Night ABPM[§]	32	7934	0.002	.04	.956	901.66	<.001	0.0%	98.4%	2.63	.854
Noctural Dipping (Individual)[‖]	4	605	.012	.10	.902	15.03	.001	0.0%	82.9%	0.98	.357
Diurnal Variation (Group)	32	7934	-0.005	.01	.619	778.90	<.001	0.0%	96.5%	.59	.627
Ethnoracial Group (% Non-White)	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ**	**Egger**
Systolic Blood Pressure											
24h ABPM	9	3462	-0.010	.06	.868	480.02	<.001	0.0%	98.5%	4.35	.006
Day ABPM	20	5929	0.041	.05	.372	1076.64	<.001	0.0%	98.8%	5.25	.962
Night ABPM	19	5558	-0.005	.04	.891	773.84	<.001	0.0%	98.5%	4.59	.121
Noctural Dipping (Individual)[‖]	4	1492	0.016	.01	.239	31.284	<.001	11.8%	93.3%	.80	.000
Diurnal Variation (Group)	19	5558	**-0.008**	**.004**	**.045**	297.09	<.001	16.9%	94.6%	.43	.974
Diastolic Blood Pressure											
24h ABPM[§]	8	3428	-0.031	.03	.348	153.02	<.001	0.0%	97.5%	2.23	.400
Day ABPM[§]	19	5895	0.020	.02	.308	412.58	<.001	0.5%	96.9%	2.15	.209
Night ABPM[§]	18	5524	-0.019	.02	.318	688.10	<.001	0.0%	97.9%	2.05	.547
Noctural Dipping (Individual)[‖]	n/a	-	-	-	-	-	-	-	-	-	-
Diurnal Variation (Group)	19	5558	-0.008	.01	.143	450.22	<.001	7.4%	96.8%	0.61	.437
Height (cm)	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ**	**Egger**
Systolic Blood Pressure											

24h ABPM	12	3745	**0.275**	**.08**	**.001**	182.43	<.001	**50.2%**	97.3%	3.17	.993
Day ABPM	19	4757	**0.195**	**.07**	**.008**	595.06	<.001	**26.6%**	97.2%	3.38	.878
Night ABPM	19	4757	**0.214**	**.06**	**<.001**	324.31	<.001	**45.2%**	95.3%	2.59	.816
Noctural Dipping (Individual)‖	n/a	-	-	-	-	-	-	-	-	-	-
Diurnal Variation (Group)	19	4757	0.008	.01	.441	387.233	<.001	0.0%	94.2%	0.44	.619
Diastolic Blood Pressure											
24h ABPM§	12	3745	0.030	.07	.666	605.98	<.001	0.0%	98.5%	2.67	.692
Day ABPM§	19	4757	-0.048	.05	.344	526.66	<.001	0.0%	97.0%	2.32	.268
Night ABPM§	19	4757	0.023	.06	.707	335.53	<.001	0.0%	98.1%	2.85	.533
Noctural Dipping (Individual)‖	n/a	-	-	-	-	-	-	-	-	-	-
Diurnal Variation (Group)	19	4757	0.025	.01	.083	491.69	<.001	11.1%	96.7%	0.65	.362
Weight (kg)	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ**	**Egger**
Systolic Blood Pressure											
24h ABPM	12	3905	0.102	.07	.165	289.89	<.001	9.2%	98.7%	4.56	.704
Day ABPM	17	4157	**0.301**	**.11**	**.005**	596.29	<.001	**32.8%**	97.5%	3.80	.806
Night ABPM	17	4157	**0.273**	**.08**	**.001**	180.87	<.001	**45.6%**	95.4%	2.73	.279
Noctural Dipping (Individual)‖	n/a	-	-	-	-	-	-	-	-	-	-
Diurnal Variation (Group)	17	4157	-0.002	.01	.899	402.77	<.001	0.0%	94.9%	0.48	.643
Diastolic Blood Pressure											
24h ABPM§	12	3905	0.019	.05	.725	695.87	<.001	0.0%	99.1%	3.36	.801
Day ABPM§	17	4157	-0.013	.07	.852	509.85	<.001	0.0%	97.2%	2.42	.118
Night ABPM§	17	4157	0.031	.08	.691	281.73	<.001	0.0%	97.9%	2.75	.726
Noctural Dipping (Individual)‖	n/a	-	-	-	-	-	-	-	-	-	-

Diurnal Variation (Group)	17	4157	0.020	.02	.286	455.50	<.001	1.1%	96.7%	0.66	.344
BMI (kg/m²)	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ Egger**	
Systolic Blood Pressure											
24h ABPM	14	3901	0.431	.25	.089	582.71	<.001	13.9%	98.9%	5.06	0.275
Day ABPM	18	4338	**1.111**	**.34**	**.001**	757.62	<.001	**38.5%**	98.1%	4.20	.424
Night ABPM	18	4338	**1.120**	**.27**	**<.001**	248.29	<.001	**52.2%**	96.9%	3.28	.013
Noctural Dipping (Individual)‖	n/a	-	-	-	-	-	-	-	-	-	-
Diurnal Variation (Group)	18	4338	0.026	.04	.464	2556.75	<.001	0.0%	93.3%	.43	.190
Diastolic Blood Pressure											
24h ABPM§	14	3901	0.150	.15	.309	721.50	<.001	1.2%	98.6%	2.93	.383
Day ABPM§	18	4338	**0.366**	**.14**	**.008**	245.35	<.001	**28.1%**	**94.3%**	1.66	.080
Night ABPM§	18	4338	**0.391**	**.20**	**.046**	253.69	<.001	18.2%	97.3%	2.40	.080
Noctural Dipping (Individual)‖	n/a	-	-	-	-	-	-	-	-	-	-
Diurnal Variation (Group)	18	4338	0.049	.04	.254	191.02	<.001	2.5%	94.5%	0.52	.733
Family History Hypertension	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ Egger**	
Systolic Blood Pressure											
24h ABPM	5	1733	-0.030	.02	.107	6.71	.082	**52.5%**	57.9%	.95	.427
Day ABPM	9	2809	-0.032	.05	.545	328.56	<.001	0.0%	96.1%	3.50	.148
Night ABPM	9	2809	0.024	.04	.559	190.74	<.001	0.0%	94.0%	2.71	.507
Noctural Dipping (Individual)‖	n/a	-	-	-	-	-	-	-	-	-	-
Diurnal Variation (Group)	9	2809	0.006	.01	.222	57.67	<.001	11.6%	85.5%	0.31	.848
Diastolic Blood Pressure											
24h ABPM§	5	1733	-0.027	.03	.433	38.79	<.001	0.0%	93.4%	2.02	.003

Day ABPM[§]	9	2809	-0.017	.04	.664	292.29	<.001	0.0%	96.6%	2.64	.000
Night ABPM[§]	9	2809	0.001	.03	.974	95.94	<.001	0.0%	94.4%	1.97	.829
Noctural Dipping (Individual)[‖]	n/a	-	-	-	-	-	-	-	-	-	-
Diurnal Variation (Group)	9	2809	0.002	.01	.852	197.01	<.001	0.0%	94.8%	0.58	.972
Casual SBP Values	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ Egger**	
Systolic Blood Pressure											
24h ABPM	10	2168	**0.433**	**.13**	**.001**	42.96	<.001	**62.0%**	87.9%	2.01	.078
Day ABPM	14	3185	0.119	.22	.579	303.54	<.001	0.0%	95.4%	3.06	.278
Night ABPM	13	2814	-0.004	.20	.983	352.74	<.001	0.0%	94.2%	2.82	.473
Noctural Dipping (Individual)[‖]	n/a	-	-	-	-	-	-	-	-	-	-
Diurnal Variation (Group)	13	2814	-0.008	.02	.697	71.40	<.001	0.0%	80.2%	0.25	.511
Diastolic Blood Pressure											
24h ABPM[§]	10	2168	0.027	.13	.832	87.56	<.001	0.0%	95.3%	2.18	.456
Day ABPM[§]	14	3185	0.013	.15	.932	207.64	<.001	0.0%	95.1%	2.12	.633
Night ABPM[§]	13	2814	-0.026	.20	.895	228.78	<.001	0.0%	97.2%	2.88	.298
Noctural Dipping (Individual)[‖]	n/a	-	-	-	-	-	-	-	-	-	-
Diurnal Variation (Group)	13	2814	-0.014	.05	.769	318.68	<.001	0.0%	96.0%	0.65	.066
Casual DBP Values	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ Egger**	
Systolic Blood Pressure											
24h ABPM	10	2168	0.101	.26	.701	135.66	<.001	0.0%	95.8%	3.46	.900
Day ABPM	14	3185	-0.235	.14	.097	213.67	<.001	13.9%	94.9%	2.76	.755

Night ABPM	13	2814	**-0.321**	**.11**	**.003**	129.41	<.001	**42.8%**	90.3%	2.04	.910
Noctural Dipping (Individual)‖	n/a	-	-	-	-	-	-	-	-	-	-
Diurnal Variation (Group)	13	2814	-0.008	.01	.565	72.81	<.001	0.0%	81.5%	0.25	.560
Diastolic Blood Pressure											
24h ABPM§	10	2168	-0.029	.17	.862	85.33	<.001	0.0%	95.4%	2.18	0.594
Day ABPM§	14	3185	-0.048	.11	.654	203.22	<.001	0.0%	95.4%	2.10	.731
Night ABPM§	13	2814	-0.092	.14	.523	198.32	<.001	0.0%	97.2%	2.82	.354
Noctural Dipping (Individual)‖	n/a	-	-	-	-	-	-	-	-	-	-
Diurnal Variation (Group)	13	2814	-0.027	.03	.421	320.23	<.001	0.0%	96.2%	0.64	.094
Day Interval Start	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ**	**Egger**
Systolic Blood Pressure											
24h ABPM	15	4284	-2.249	1.67	.178	1336.34	<.001	4.8%	99.1%	5.09	.347
Day ABPM	22	5155	-1.298	1.50	.386	1357.81	<.001	0.0%	98.9%	5.34	.352
Night ABPM	22	5155	-1.077	1.28	.401	1194.85	<.001	0.0%	98.5%	4.57	.243
Noctural Dipping (Individual)‖	5	1577	-0.403	.95	.671	38.316	<.001	0.0%	91.8%	0.99	.006
Diurnal Variation (Group)	22	5155	-0.102	.11	.330	208.58	<.001	0.0%	91.6%	0.35	.540
Diastolic Blood Pressure											
24h ABPM§	14	4250	-0.702	.88	.425	775.20	<.001	0.0%	98.3%	2.47	.596
Day ABPM§	21	5131	0.631	.54	.241	277.43	<.001	4.2%	95.1%	1.76	.580

Night ABPM§	21	5121	-0.860	.74	.247	643.55	<.001	0.0%	98.2%	2.46	.458
Noctural Dipping (Individual)‖	4	605	1.296	.91	.153	7.50	.024	36.7%	70.8%	0.63	.138
Diurnal Variation (Group)	22	5155	-0.296	.16	.067	391.07	<.001	10.9%	95.9%	0.56	.350
Day Interval End	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ**	**Egger**
Systolic Blood Pressure											
24h ABPM	15	4284	-0.049	1.24	.969	1256.05	<.001	0.0%	99.2%	5.40	.081
Day ABPM	22	5155	-0.428	1.11	.699	1360.42	<.001	0.0%	99.0%	5.40	.235
Night ABPM	22	5155	0.176	.93	.850	1125.77	<.001	0.0%	98.6%	4.63	.116
Noctural Dipping (Individual)‖	5	1577	0.554	1.15	.631	45.14	<.001	0.0%	91.1%	0.99	.000
Diurnal Variation (Group)	22	5155	0.000	.08	>.999	202.33	<.001	0.0%	92.1%	0.36	.758
Diastolic Blood Pressure											
24h ABPM§	14	4250	-0.359	.59	.542	793.92	<.001	0.0%	98.5%	2.49	.588
Day ABPM§	21	5121	-0.701	.36	.054	222.19	<.001	15.3%	94.7%	1.65	.091
Night ABPM§	21	5121	-0.586	.54	.276	681.91	<.001	0.0%	98.4%	2.48	.529
Noctural Dipping (Individual)‖	4	605	**1.831**	**.45**	**<.001**	0.67	0.72	**100%**	0.0%	0.00	.504
Diurnal Variation (Group)	22	5155	-0.045	.12	.714	411.00	<.001	0.0%	96.6%	0.61	.631
Night Interval Start	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ**	**Egger**
Systolic Blood Pressure											
24h ABPM	15	4284	0.305	.95	.747	1016.79	<.001	0.0%	99.2%	5.37	.016
Day ABPM	22	5155	0.651	.81	.419	1079.60	<.001	0.0%	98.9%	5.29	.015

Night ABPM	22	5155	1.002	.65	.124	675.45	<.001	8.3%	98.3%	4.33	.006
Noctural Dipping (Individual)‖	5	1577	-0.382	.40	.335	30.178	<.001	0.0%	88.3%	0.89	.000
Diurnal Variation (Group)	22	5155	0.009	.06	.868	208.36	<.001	0.0%	91.8%	0.36	.792
Diastolic Blood Pressure											
24h ABPM§	14	4250	0.059	.45	.894	649.84	<.001	0.0%	98.4%	2.52	.301
Day ABPM§	21	5121	0.013	.29	.965	318.80	<.001	0.0%	95.6%	1.85	.407
Night ABPM§	21	5121	-0.197	.39	.616	688.29	<.001	0.0%	98.3%	2.53	.339
Noctural Dipping (Individual)‖	4	605	**1.831**	**.45**	**<.001**	0.66	0.720	**100%**	0.0%	0.00	.504
Diurnal Variation (Group)	22	5155	-0.060	.09	.512	453.62	<.001	0.0%	96.4%	0.61	.490

Night Interval End	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ Egger**	
Systolic Blood Pressure											
24h ABPM	15	2284	**-4.275**	**1.60**	**.008**	733.53	<.001	**31.1%**	98.7%	4.33	.110
Day ABPM	22	5155	**-4.151**	**1.17**	**<.001**	839.65	<.001	**36.4%**	98.3%	4.22	.122
Night ABPM	22	5155	**-4.163**	**.89**	**<.001**	505.59	<.001	**52.3%**	96.7%	3.13	.038
Noctural Dipping (Individual)‖	5	1577	**1.038**	**.39**	**.007**	11.30	.010	**70.13%**	75.6%	0.48	.001
Diurnal Variation (Group)	22	5155	-0.132	.10	.192	199.36	<.001	4.0%	91.4%	0.34	.933
Diastolic Blood Pressure											
24h ABPM§	14	4250	**-2.007**	**.77**	**.010**	501.212	<.001	**31.4%**	97.5%	2.01	.243
Day ABPM§	21	5121	-0.717	.52	.164	320.14	<.001	2.7%	95.2%	1.77	.558
Night ABPM§	21	5121	-1.104	.67	.099	622.33	<.001	7.0%	97.8%	2.37	.137

Noctural Dipping (Individual)‖	4	605	1.296	.91	.153	7.50	.024	**36.7%**	70.8%	0.63	.138
Diurnal Variation (Group)	22	5155	-0.170	.17	.317	419.76	<.001	0.3%	96.4%	.60	.806
Day Sampling Frequency	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ Egger**	
Systolic Blood Pressure											
24h ABPM35	22	6417	0.160	.19	.409	1488.42	<.001	0.0%	99.1%	5.15	.122
Day ABPM	35	8218	0.205	.14	.133	1789.89	<.001	3.4%	98.7%	4.96	.649
Night ABPM	34	7847	0.059	.12	.636	1374.54	<.001	0.0%	98.3%	4.40	.102
Noctural Dipping (Individual)‖	5	1577	-0.027	.10	.783	42.94	<.001	0.0%	92.2%	1.02	.001
Diurnal Variation (Group)	34	7847	-0.017	.01	.140	372.90	<.001	5.9%	92.7%	0.39	.510
Diastolic Blood Pressure											
24h ABPM§	21	6383	0.019	.10	.847	1018.75	<.001	0.0%	98.4%	2.55	.683
Day ABPM§	34	8184	0.116	.06	.059	582.73	<.001	7.8%	96.5%	2.16	.151
Night ABPM§	33	7813	-0.052	.08	.504	701.13	<.001	0.0%	98.3%	2.67	.345
Noctural Dipping (Individual)‖	5	1577	0.434	.70	.535	734.75	<.001	0.0%	99.7%	7.64	.391
Diurnal Variation (Group)	34	7847	**-0.039**	**.02**	**.013**	516.67	<.001	15.7%	95.5%	0.55	.694
Night Sampling Frequency	**k**	**N**	**B**	**SE**	**p***	**Q_E**	**p†**	**$R^{2‡}$**	**I^2**	**τ Egger**	
Systolic Blood Pressure											
24h ABPM	22	6417	0.006	.07	.928	1448.42	<.001	0.0%	99.1%	5.23	.143
Day ABPM	35	8218	0.037	.05	.491	1826.28	<.001	0.0%	98.8%	5.09	.663
Night ABPM	34	7847	0.035	.05	.451	1520.82	<.001	0.0%	98.3%	4.37	.136
Noctural Dipping (Individual)‖	5	1577	-0.001	.04	.979	39.361	<.001	0.0%	93.9%	1.02	.002
Diurnal Variation (Group)	34	7847	0.000	.005	.933	546.33	<.001	0.0%	93.5%	0.41	.555

Diastolic Blood Pressure									
24h ABPM[§]	21	6383	-0.052	.03	.126	671.20	<.001	6.9%	98.2%	2.40	.837
Day ABPM[§]	34	8184	0.006	.03	.822	724.96	<.001	0.0%	96.9%	2.28	.091
Night ABPM[§]	33	7813	-0.031	.03	.281	731.28	<.001	0.8%	98.1%	2.64	.277
Noctural Dipping (Individual)[‖]	5	1577	0.174	.29	.543	778.14	<.001	0.0%	99.7%	7.66	.587
Diurnal Variation (Group)	34	7847	-0.009	.006	.140	786.80	<.001	3.9%	96.2%	0.59	.761

Note. *Significance value of B (slope); $p < .05$ indicates slope is significantly different than 0.

†Significance value of Q_E (Residual Heterogeneity); $p < .05$ indicates heterogeneity not explained by predictor is significant.

‡R^2 indicates percentage of between-study heterogeneity explained by predictor.

§Pooled estimate excludes extreme outlier (Davis et al., 1996).

‖Pooled estimate excludes extreme outlier (Zhu et al., 2008).

Appendix C: *Categorical effect modifiers in Study 1.*

Table 7. *Meta-Analytic Models: Categorical Effect Modifiers.*

REGION – SYSTOLIC BLOOD PRESSURE							
Systolic – 24hr ABPM	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Geographical Region	Q_M=11.60, p=.009, I^2=98.5%, τ=4.58, Egger=.020						
North America	9	1981	118.77	1.50	(115.84, 121.71)	293.79	<.001
South America	-	-	-	-	-	-	-
Europe	10	2746	111.91	1.33	(109.30, 114.515)	326.22	<.001
Asia	5	1878	115.05	2.64	(109.88, 120.22)	228.45	<.001
Oceania	-	-	-	-	-	-	-
Systolic – Day ABPM	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Geographical Region	Q_M=4.97, p=.290, I^2=98.6%, τ=5.01, Egger=.261						
North America	17	3928	119.19	1.22	(116.80, 121.58)	605.91	<.001
South America	2	129	112.63	.99	(110.70, 114.57)	1.63	.201
Europe	9	1917	116.20	1.19	(113.88, 118.53)	295.25	<.001
Asia	8	2392	116.75	2.38	(112.08, 121.41)	685.00	<.001
Oceania	-	-	-	-	-	-	-
Systolic – Night ABPM	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**P**
Geographical Region	Q_M=13.29, p=.010, I^2=97.60, τ=3.94, Egger=.029						
North America	16	3557	107.68	.94	(105.85, 109.52)	242.70	<.001
South America	2	129	100.74	2.04	(96.74, 104.73)	9.65	.002
Europe	9	1917	103.20	1.04	(101.17, 105.23)	164.49	<.001
Asia	8	2392	103.13	1.95	(99.31, 106.94)	219.95	<.001
Oceania	-	-	-	-	-	-	-
Systolic – Dipping	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**P**
Geographical Region	Q_M=3.95, p=.138, I^2=77.4%, τ=.68, Egger=.007						
North America	-	-	-	-	-	-	-
South America	2	129	10.13	.75	(8.66, 11.60)	6.89	.009

Europe	-	-	-	-	-	-	-
Asia	2	476	10.56	.27	(10.02, 11.09)	1.30	.254
Oceania	-	-	-	-	-	-	-
Systolic – Diurnal	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Geographical Region					Q_M=5.48, p=.245, I^2=92.6%, τ=0.39, Egger=.413		
North America	16	3557	-1.20	.11	(-1.41, 1.00)	179.27	<.001
South America	2	129	-1.47	.14	(-1.74, -1.19)	0.33	.565
Europe	9	1917	-1.54	.14	(-1.81, -1.27)	82.40	<.001
Asia	8	2392	-1.54	.16	(-1.85, -1.23)	129.80	<.001
Oceania	-	-	-	-	-	-	-
REGION – DIASTOLIC BLOOD PRESSURE							
Diastolic – 24hr ABPM	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Geographical Region					Q_M=3.44, p=.329, I^2=99.6%, τ=5.61, Egger=.277		
North America	9	1981	69.97	2.82	(64.43, 75.51)	749.68	<.001
South America	-	-	-	-	-	-	-
Europe	10	2746	65.33	.78	(63.80, 66.86)	438.02	<.001
Asia	5	1878	66.13	1.59	(63.02, 69.23)	115.93	<.001
Oceania	-	-	-	-	-	-	-
Diastolic – Day ABPM	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Geographical Region					Q_M=1.20, p=.878, I^2=99.2%, τ=4.64, Egger=.635		
North America	17	3928	71.41	1.51	(68.44, 74.38)	878.86	<.001
South America	2	129	68.87	.71	(67.48, 70.26)	1.98	.160
Europe	9	1917	70.01	.36	(69.31, 70.71)	30.63	<.001
Asia	8	2392	69.92	1.13	(67.70, 72.14)	257.63	<.001
Oceania	-	-	-	-	-	-	-
Diastolic – Night ABPM	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Geographical Region					Q_M=1.50, p=.827, I^2=99.4%, τ=4.96, Egger=.240		
North America	16	3557	59.61	1.58	(56.50, 62.71)	1,257.04	<.001
South America	2	1917	57.05	1.29	(54.52, 59.58)	6.74	.009

Europe	9	2392	57.71	1.10	(55.56, 59.86)	127.17	<.001
Asia	8	129	57.49	1.24	(55.07, 59.91)	183.98	<.001
Oceania	-	-	-	-	-	-	-
Diastolic – Dipping	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Geographical Region	Q_M=390.19, p<.001, I^2=54.8%, τ=0.55, Egger=.077						
North America	-	-	-	-	-	-	-
South America	2	129	16.24	.62	(15.4, 17.45)	2.52	.11
Europe	-	-	-	-	-	-	-
Asia	2	476	17.28	.42	(16.45, 18.11)	1.88	.17
Oceania	-	-	-	-	-	-	-
Diastolic – Diurnal	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Geographical Region	Q_M=1.50, p=.827, I^2=96.2%, τ=0.61, Egger=.676						
North America	16	3557	-1.83	.17	(-2.15, -1.50)	431.78	<.001
South America	2	129	-2.07	.18	(-2.47, -1.66)	1.61	.204
Europe	9	1917	-2.06	.23	(-2.52, -1.60)	136.12	<.001
Asia	8	2392	-2.11	.18	(-2.46, -1.77)	89.45	<.001
Oceania	-	-	-	-	-	-	-
DEVICE TYPE – SYSTOLIC BLOOD PRESSURE							
Systolic – 24hr ABPM	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Device Type	Q_M=236.4, p<.001, I^2=98.5%, τ=<.001, Egger=.000						
Oscillometric	21	6307	112.63	.10	(112.44, 112.83)	1421.53	<.001
Auscultatory	3	346	123.48	.70	(122.11, 124.84)	5.98	.05
Systolic – Day ABPM	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Device Type	Q_M=69.5, p<.001, I^2=98.2%, τ=<.001, Egger=.000						
Oscillometric	29	7570	117.47	.10	(117.28, 117.66)	1529.81	<.001
Auscultatory	7	844	120.60	.36	(119.89, 121.31)	309.01	<.001
Systolic – Night ABPM	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Device Type	Q_M=59.8, p<.001, I^2=97.9%, τ=<.001, Egger=.000						

Oscillometric	28	7199	103.78	.10	(103.59, 103.97)	1369.08	<.001
Auscultatory	7	844	107.21	.43	(106.35, 108.05)	167.95	<.001
Systolic – Dipping	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Device Type	Q_M=9292.1, p<.001, I^2=91.4%, τ=<.001, Egger=.000						
Oscillometric	5	1577	9.44	.10	(9.25, 9.63)	46.37	<.001
Auscultatory	-	-	-	-	-	-	-
Systolic – Diurnal	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Device Type	Q_M=76.79, p<.001, I^2=93.7%, τ=<.001, Egger=.006						
Oscillometric	28	7199	-1.52	.02	(-1.55, -1.48)	492.93	<.001
Auscultatory	7	844	-1.03	.05	(-1.13, -.93)	26.95	<.001
DEVICE TYPE – DIASTOLIC BLOOD PRESSURE							
Diastolic – 24hr ABPM	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Device Type	Q_M=162.2, p<.001, I^2=98.7%, τ=<.001, Egger=.000						
Oscillometric	21	6307	65.55	.07	(65.42, 65.68)	1646.50	<.001
Auscultatory	3	346	71.29	.45	(70.42, 72.17)	15.29	<.001
Diastolic – Day ABPM	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Device Type	Q_M=8.90, p=.003, I^2=97.2%, τ=<.001, Egger=.000						
Oscillometric	29	7570	70.61	.07	(70.47, 70.75)	1080.02	<.001
Auscultatory	7	844	71.32	.23	(70.87, 71.77)	142.33	<.001
Diastolic – Night ABPM	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Device Type	Q_M=188.4, p<.001, I^2=97.5%, τ=<.001, Egger=.000						
Oscillometric	28	7199	57.72	.07	(57.59, 57.86)	1022.38	<.001
Auscultatory	7	844	56.20	.09	(56.03, 56.37)	321.62	<.001
Diastolic – Dipping	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**
Device Type	Q_M=7892.4, p<.001, I^2=99.6%, τ=<.001, Egger=.000						
Oscillometric	5	1577	14.35	.16	(14.04, 14.67)	1015.58	<.001
Auscultatory	-	-	-	-	-	-	-
Diastolic – Diurnal	**k**	**N**	**Estimate**	**SE**	**CI**	**Q_W**	**p**

Device Type					Q_M=94.13, p<.001, I^2=95.3%, τ=<.001, Egger=.113		
Oscillometric	28	7199	-2.06	.02	(-2.10, -2.02)	531.08	<.001
Auscultatory	7	844	-1.48	.06	(-1.59, -1.37)	176.47	<.001

Appendix D: *Group comparison results from Study 3.*

Table 4. *Group comparisons of blood pressure Z-score and sleep dimensions by sex.*

	Boys	Girls		
	M (*SD*)	*M* (*SD*)	*t*	*p*
Blood pressure				
SBP Z-score	-.75 (1.05)	-.86 (1.11)	.828	.204
DBP Z-score	-.49 (.88)	-.25 (.78)	-2.485	.007
Actigraphy				
Bedtime	23:26 (1:13)	23:25 (1:18)	.103	.918
Wake time	07:41 (1:00)	07:44 (0:54)	-.394	.694
Midpoint	03:34 (1:03)	03:35 (1:03)	-.120	.905
Duration	495.03 (47.15)	499.50 (46.72)	-.652	.258
Sleep onset latency	11.41 (13.06)	10.25 (10.21)	.664	.254
Number of awakenings	4.73 (4.73)	4.23 (4.53)	.728	.234
WASO	53.09 (31.88)	48.65 (37.71)	.882	.190
PSG				
Sleep onset latency	25.50 (32.17)	29..23 (39.72)	-.825	.205
Number of awakenings				
WASO	30.99 (41.36)	20.70 (26.41)	2.310	.011*
Efficiency	86.30 (11.93)	88.32 (10.63)	-1.399	.082
Arousal Index				
% time in N1	2.81 (3.76)	2.25 (2.57)	1.297	.098
% time in N2	49.19 (10.38)	53.21 (9.78)	-3.017	.001*
% time in N3	27.88 (10.37)	25.63 (8.92)	1.751	.038*
% time in REM	13.02 (7.34)	14.36 (6.54)	-1.462	.073
Self-report sleep quality	7.23 (.98)	7.15 (1.24)	.458	.324

Note. *$p<.05$

Table 5. *Group comparisons of blood pressure Z-score and sleep dimensions by race.*

	White	Black	Asian	Other/Mixed race		
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*F*	*p*
Blood pressure						
SBP Z-score	-.73 (1.08)	-.70 (.82)	-1.01 (1.13)	-1.05 (1.31)	.908	.438
DBP Z-score	-.46 (.81)	.027 (.75)	-.45 (.84)	-.19 (.61)	2.401	.068
Actigraphy						
Bedtime	23:20 (1:06)	23:36 (0:52)	23:19 (1:06)	23:53 (1:09)	1.204	.310
Wake time	07:37 (0:54)	07:46 (0:40)	07:38 (1:00)	08:26 (0:51)	3.402	.019*
Midpoint	03:29 (0:56)	03:41 (0:41)	03:29 (0:59)	04:09 (0:59)	2.327	.077
Duration	497.38 (47.73)	489.76 (42.15)	499.31 (45.68)	512.99 (31.23)	.644	.588
Sleep onset latency	11.22 (13.32)	8.99 (8.15)	10.15 (10.08)	13.06 (11.45)	.262	.853
Number of awakenings	4.16 (3.93)	4.02 (5.32)	7.23 (7.64)	6.95 (6.94)	2.967	.034*
WASO	51.73 (31.54)	42.27 (21.09)	53.22 (24.05)	54.89 (49.29)	.424	.736
PSG						
Sleep onset latency	26.01 (31.08)	18.50 (20.14)	44.05 (68.94)	31.13 (18.34)	2.003	.115
Number of awakenings	17.39 (8.12)	17.82 (7.59)	17.10 (8.64)	19.69 (6.63)	.026	.994
WASO	23.83 (27.35)	29.91 (64.78)	35.43 (62.89)	21.13 (45.23)	2.194	.090
Efficiency	88.10 (9.48)	86.27 (12.07)	81.56 (18.12)	85.90 (8.91)	2.396	.069
Arousal Index	12.24 (5.16)	9.47 (2.76)	13.42 (4.57)	11.74 (5.82)	1.915	.128
% time in N1	2.61 (3.36)	3.49 (3.83)	2.41 (2.83)	1.97 (2.32)	.492	.688
% time in N2	50.36 (10.24)	54.17 (10.81)	51.17 (13.68)	50.11 (11.64)	.454	.715
% time in N3	27.69 (9.67)	21.82 (9.37)	24.88 (9.34)	29.12 (10.39)	1.871	.139
% time in REM	13.88 (7.16)	9.49 (7.52)	14.66 (6.21)	11.74 (5.82)	1.853	.136
Self-report sleep quality	7.32 (.92)	6.16 (1.33)	6.87 (1.09)	7.43 (1.34)	4.106	.008*

Note. *$p<.05$

Table 6. *Group comparisons of blood pressure Z-score and sleep dimensions by family history of hypertension*.

	Family history	No family history		
	M (*SD*)	*M* (*SD*)	*t*	*p*
Blood pressure				
SBP Z-score	-.80 (.98)	-.68 (1.28)	.727	.234
DBP Z-score	-.33 (.88)	-.42 (.84)	-.608	.272
Actigraphy				
Bedtime	23:33 (1:22)	23:04 (0:59)	-2.046	.021*
Wake time	07:50 (0:59)	7:23 (0:47)	-2.650	.005*
Midpoint	03:41 (1:08)	03:13 (0:47)	-2.780	.008*
Duration	497.61 (47.75)	498.89 (48.73)	.144	.443
Sleep onset latency	11.05 (12.92)	11.10 (13.14)	.023	.491
Number of awakenings	4.29 (4.35)	4.47 (5.65)	.208	.418
WASO	48.23 (61.37)	61.37 (43.43)	1.967	.026*
PSG				
Sleep onset latency	25.16 (38.56)	26.16 (38.56)	.209	.417
Number of awakenings	16.75 (8.00)	16.94 (8.22)	.149	.441
WASO	26.65 (34.69)	24.27 (39.76)	-.412	.340
Efficiency	87.87 (9.38)	87.02 (13.90)	-.442	.330
Arousal Index	11.67 (4.55)	12.22 (4.95)	.762	.224
% time in N1	2.68 (3.57)	2.14 (2.41)	-1.065	.144
% time in N2	50.59 (10.29)	50.94 (10.05)	.215	.415
% time in N3	26.65 (10.53)	28.32 (9.35)	1.120	.132
% time in REM	14.19 (6.76)	12.96 (7.37)	1.122	.132
Self-report sleep quality	7.21 (1.05)	7.18 (1.12)	-.127	.450

Note. *p<.05