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Family Dynamics in Sleep Health and Hypertension

Heather E. Gunn, PhD[1,2] and **Kenda R. Eberhardt, B.S**[2]

Department of Psychology, University of Alabama

Abstract

Purpose of Review—Present a conceptual model and review the recent literature on family dynamics, sleep, and hypertension.

Recent Findings—Family dynamics predict hypertension and hypertension risk, in part, due to shared health behaviors. Sleep health behaviors (sleep duration, quality, and efficiency) predict hypertension risk in children and youth, and are emerging as a family-level health behavior. Importantly, both family dynamics and sleep are modifiable. Family members influence one another's sleep through their physical presence and through psychological and emotional mechanisms. Family members' sleep patterns may also be coregulated. Negative family dynamics are associated with poor sleep health and predict greater cardiovascular risk. Sleep health behaviors in the family context may also interact with family dynamics to dampen or exacerbate hypertension risk factors in children and youth.

Summary—This review proposes that promoting sleep health in a family context could be one way to reduce long-term hypertension risk.

Keywords

Family relationships; family dynamics; sleep behaviors; sleep health; children; youth; hypertension; hypertension risk

Introduction

Negative family dynamics are strong contributors to the development of hypertension [1]. Hypertension risks are similar among family members, partly due to genetic dispositions inherited by children [2]. However, close family relationships also contribute to individual hypertension risk through daily interactions and the transmission of health behaviors. Cohabitating family members are one of the primary and consistent sources of relationship security or stress, which, in turn, has downstream consequences for health. For example, frequent and negative exchanges within family units are associated with increased risk for

[1]Corresponding Author, hegunn@ua.edu.
[2]Box 870348, The University of Alabama Tuscaloosa, AL 35487-0348

Conflict of Interest

The authors declare no conflicts of interest relevant to this manuscript.

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chronic illnesses (e.g., cardiovascular disease), while positive family relationships are associated with better physical health outcomes [3]. Children also learn about health behaviors from the family unit [4]. and individuals within a family tend to have concordant health and health behaviors [5]. For example, childhood obesity is strongly linked to parent's obesity [6]. Therefore, a family- based approach to the study of hypertension can inform important psychosocial targets for intervention [1]. Indeed, family-based interventions can positively influence health behaviors (e.g.,[7]).

One such family-level behavior that influences hypertension risk is sleep health. Sleep health is a multidimensional concept that refers to sleep duration, sleep continuity, timing, sleepiness versus alertness, and quality or satisfaction [8]. Associations between sleep health and cardiovascular outcomes, including hypertension have been reviewed extensively (e.g., see [9] [10]). Briefly, sleep quality, duration, timing, and fragmentation (wake after sleep onset), predict hypertension risk across the age spectrum. For example, short sleep duration (< 6 hours) is associated with increased hypertension via risk factors such as obesity in children and adolescents [11] [12]. Moreover, short sleep duration appears to have a direct association with hypertension. In one of the first longitudinal studies of…, adults with short sleep duration, and *without* other sleep disorders, were about two times more likely to develop hypertension even after controlling for other prominent risk factors (e.g., obesity, diabetes; [13]). Importantly, sleep behaviors are modifiable, and improving sleep may improve other health behaviors [14]. Thus, examining sleep in a family context allows for a greater understanding of two interrelated, and *modifiable*, predictors of hypertension: family dynamics and sleep.

In the following pages, we present and review a conceptual model of sleep, hypertension, and family relationships whereby sleep health and family relationships have a synergistic effect on the development of hypertension (Figure 1). We summarize data published in the last five years. Our summary includes studies that measured constructs of family dynamics (e.g., conflict, cohesion, structure, parenting) and how they relate to sleep behaviors and hypertension outcomes in children and youth of all ages. We focus on children because early life experiences and learned health behaviors lay the foundation for lifelong health outcomes [15-18]. Most of our review focuses on dynamics within the parent-child relationship, which also reflects the majority of the literature on family dynamics and sleep and hypertension. Furthermore, while the correlation between breathing related sleep disorders and hypertension risk is strong, we limited our studies to those focused on sleep behaviors because they are amenable to behavioral intervention and are also independently associated with hypertension [19] and hypertension risk in youth [11].

The organization of this review corresponds to the conceptual model (Figure 1). We present a framework and review of family dynamics and sleep health (Part A, Figure 1); family dynamics and hypertension risk (Part B, Figure 1); and a review of the few studies measuring family dynamics, sleep health, *and* hypertension risk (Part C, Figure 1). Finally, we discuss gaps in the literature and future directions for research and necessary steps to develop family-based approaches to improve sleep health, and ultimately, cardiovascular health.

Family relationships and sleep.

We argue that the family environment is an important social construct to consider with respect to sleep and hypertension risk for several reasons. First, there is the opportunity for an individual's sleep to be influenced by the *physical* presence of family members. For example, about 25% of children share a bed with their parents [20], and many sleep complaints are due to noise and habits of cohabitating family members [21, 22]. In addition, family members may serve as social zeitgebers (any external or environmental cue that sets biological rhythms) and regulate one another's sleep timing, and the timing of daily activities that influence circadian rhythms. Second, the presence of family members can influence sleep through psychological or emotional mechanisms. Families are often the first and primary potential source of interpersonal and emotional security, which in turn, is important for sleep [23]. Sleep requires decreased arousal and responsiveness to the external environment, which increases vulnerability. Our ancestors relied on their social network to keep them safe from prey while they slept [23, 24]. Though modern-day and westernized humans are not as susceptible to these threats, relationship insecurity increases arousal [25], which is counterproductive for sleep [26]. This suggests that sleep is vulnerable to social threat. Family relationships, therefore, are especially poised to enhance or dampen the interpersonal security that is necessary for sleep.

Third, emerging evidence suggests that coregulation or synchrony of sleep and circadian processes likely occur within family units. This is because one feature of close relationships is *coregulation* of psychological and physiological processes [27]. For example, cortisol is co-regulated in mother-child dyads [28] and mothers and infant dyads show parasympathetic synchrony [29]. Coregulation of sleep can be measured by the degree of concordance in sleep and circadian parameters. For example, family members could be concordant in their bed and wake times [30]or in sleep duration [31]. The degree to which families exhibit coregulatory sleep patterns is important for understanding the degree to which family members' sleep and circadian health are connected.

Thus, sleep appears to be a socially influenced process. Of course, the ways in which family members influence one another's sleep are interconnected and interdependent. For example, appropriate levels of parental monitoring contributes to increased feelings of security in children [32]. Monitoring also predicts consistent bedtimes and wake times, which is associated with a longer sleep duration [33]. While there are other pathways by which families influence sleep (e.g., genetic predispositions), the purpose of this framework is to highlight primary (and modifiable) targets for intervening to improve sleep and hypertension risk in a family context.

Recent findings on the family dynamics and sleep (Table 1).

In cross-sectional and longitudinal studies, family-level variables indicative of cohesion and warmth frequently predict better sleep outcomes in children of all ages [34-37]. For example, in a large, population-based study of Georgian and Swiss adolescents, more parental warmth was associated with better sleep quality and longer sleep duration [34]. Similarly, better parent-child relationship quality predicted better sleep quality over time in young adolescents [35]. In contrast, chaotic households and families with more conflict are

associated with difficulties with sleep maintenance [38] and poor sleep quality [39] in children. Family transitions also appear to have negative consequences. Changes in mother's partner status from age 11 are associated with a shorter sleep duration at age 15 [40].

It is possible that among households with more chaos and transitions, there is less parental structure, which also predicts worse sleep outcomes for children. For example, young adolescents with parent-set bedtimes have longer sleep durations than children without parent-set bedtimes [33] and parent-led bedtime routines are associated with a longer sleep duration in toddlers and preschoolers [41]. Parenting rules and routines about other activities, not just sleep behaviors, appear to be beneficial for sleep. Parenting rules and consistency in routines were associated with longer sleep durations in a large sample of 10-11 year old Finnish children [42]. Moreover, youth-reported parental monitoring was associated with more sleep on school days [43].

Lastly, of the 15 recent studies on family dynamics and sleep, two studies include assessment of concordance among families on sleep behavior. Fuligni and colleagues found that parents and adolescents had concordant sleep durations, and a supportive parent-adolescent relationship was associated with higher concordance [31]. Sleep concordance can vary within family members. Mothers' sleep fluctuations both predict, and are predicted by their partner's and their child's sleep; however father's sleep was only predicted by mother's sleep [30]. Importantly, when family members' sleep patterns were included in models of individual sleep, other health and psychological covariates were no longer significant (e.g., BMI, depression;[30]). Together these findings suggest that the sleep habits and routines of other family members (especially parents), contribute to youth sleep at the individual level, and this may be particularly pronounced in mother-child relationships.

Family dynamics and hypertension risk (Table 2)

As illustrated in the conceptual model (Figure 1), the family environment is independently linked to hypertension risk (e.g., BMI, blood pressure). This link has been well documented over the last 10-15 years. A seminal paper by Repetti and colleagues in 2002 highlighted how risky family environments increased risk for poor health, including hypertension [44]. This and other important works advanced the field by emphasizing the family environment and adverse child experiences as prominent factors in the development of cardiovascular disease[45]. Recent findings have added to this work using longitudinal and advanced methodologies. The data, which are summarized in Tables 1-3, overwhelmingly support links between the family dynamics and hypertension risk. However, significant associations between family dynamics and hypertension risk vary across studies.

Family dynamics and inflammation and sympathetic activity.

Associations between a family-level variable (neglect) and hypertension were assessed in one prospective study; more childhood neglect was associated with higher inflammation, but not hypertension, 30 years later [46]. Other studies have assessed family level variables and sympathetic and inflammatory predictors of hypertension such as baseline blood pressure (BP) and heart rate (HR). For example, adverse experiences (childhood abuse and household dysfunction), are positively associated with high resting heart rate in a cross-sectional

analysis [47], and predict elevated BP in a 23-year longitudinal study [48]. Other family-level predictors in early childhood were also associated with BP at later time points. Lower systolic BP (SBP) in middle-aged men was associated with being raised in a two-parent home [49]. Moreover, parental sensitivity (i.e., observer ratings of emotional support during a challenging task) in early years predicted lower BP when children were 15 years old [50].

A more direct measure of family-level associations with hypertension is concordance of risk factors. Few studies examine degree of concordance in hypertension risk among family members; however, results from one cross-sectional study indicate concordance in BP, hemoglobin A1c (HbA1c), and systemic inflammation [51]. In this study of Chinese children aged 7-17 and their parents, Dong found that BP was positively correlated in mothers and daughters only and CRP was correlated in fathers and sons only. HbA1c was concordant in all dyadic pairs [51]. This study suggests the importance of within family analyses (i.e., mother- child; *father*-child).

Family dynamics and Body Mass Index (BMI).

BMI is one of the strongest and earliest predictors of hypertension [52, 53]. Similar to findings on family structure and BP, 10-year old children from single-parent homes had higher BMI after two years [54]. Moreover, young adult girls who reported family transitions when they were younger (divorce, mother remarrying, etc.) had higher BMI as adults [55]. Changes in family structure could contribute to more negative events, which are also predictive of BMI. Children with more acute and chronic negative events (e.g., problems with family health and financial stability) from 4–11 years old had higher BMI at age 15 [56]. Maternal depression in early childhood, which is considered a negative family event, strongly predicted BMI in children years later [57]. Negative parenting styles are also linked to BMI. In one cross-sectional study, parents who were more authoritarian (controlling, rule-bound) and who had more rules about eating had children who were more overweight [58]. In contrast, sensitive parenting (involving support and low hostility) in early childhood predicted lower BMI during adolescence [50, 56]. Finally, in a large, longitudinal study on family cohesion and conflict, Heredia and colleagues found that less family cohesion *and* more conflict predict steeper weight loss declines in Mexican-American girls from age 12 to 17 [59].

Collectively, recent findings yield significant prospective associations between family- level variables and sympathetic activity and BMI, both putative markers for hypertension. In particular, children with two parents home, sensitive parents, and fewer adverse events also had better sympathetic outcomes and lower BMI. However, associations were not always immediately apparent. This highlights the continued importance of longitudinal and prospective study designs, particularly with respect to early markers of hypertension.

Family dynamics, sleep & hypertension risk (Table 3).

To our knowledge, only one study in the last five years measured family dynamics, sleep, *and* a proximal hypertension risk. Jones and colleagues [60] examined parental rules, short sleep, and body composition in over 100 toddlers and found that parental rules about sleep and television were associated with more total sleep time. BMI was not associated with short

sleep in this sample; however, the authors noted that the study may have been underpowered [60]. The remaining studies assessed family relationship characteristics, sleep, and psychophysiological correlates of stress (e.g., cortisol, autonomic imbalance), which have distal, downstream implications for the development of hypertension [61, 62]. For example, toddlers with blunted cortisol and critically controlling mothers had more sleep problems one year later [63]. Blunted cortisol may be a reflection of chronic stress [64]. In a different paradigm of chronic psychosocial stress (children of parents affected by HIV), children who endorsed loneliness also had blunted cortisol and poor sleep quality [65]. These findings suggest that psychosocial stress in childhood is reflected in both sleep and cortisol activity.

Measures of autonomic imbalance also reflect psychophysiological stress [62] and can be assessed through heart rate variability and respiratory sinus arrhythmia (RSA; [66]). El-Sheikh and colleagues [67] demonstrated that good sleep (more total sleep time, better sleep quality) and greater RSA were protective against psychosocial stress (marital conflict) in school-aged children. Another recent study on autonomic imbalance sought to determine whether and how family experiences are transmitted to the next generation. Results yielded an indirect effect in which maternal emotional abuse history was associated with sleep disturbances during pregnancy, which then predicted lower fetal heart rate variability [68].

In sum, examinations of family dynamics, sleep, and proximal hypertension risk are limited. Results from existing studies suggest interactive effects among sleep, cardiovascular markers of psychosocial stress, and family dynamics. In one study, sleep quality moderated a cardiovascular marker of autonomic imbalance [68] and in another, good sleep health appeared to buffer the effects of psychosocial stress in the home [67]. The remaining studies showed that negative or positive family dynamics have similar adverse effects on sleep and psychosocial markers of stress. Thus, it is not yet clear whether and how sleep interacts with family dynamics to influence hypertension risk.

Summary and considerations for future directions.

Summary

The purpose of this review was to highlight recent findings on family dynamics in sleep and hypertension risk. We presented a conceptual framework (Figure 1) for hypertension risk in which family dynamics and sleep behaviors interact to mitigate or amplify hypertension risk. Overall, separate examinations of family dynamics and sleep and family dynamics and hypertension risk suggest similar effects of family dynamics on cardiovascular and sleep health. That is, negative family dynamics, single-parent homes, and multiple transitions within the family tend to predict poor sleep outcomes and are associated with elevations in other hypertension risk factors (e.g., HR, BP, BMI).

It is not yet clear whether and how sleep behaviors interact with family dynamics on hypertension risk; however, based on recent findings, there are a few possibilities. Several studies reported that poor family dynamics were associated with the presence of poor sleep behaviors either concurrently, or within a short time frame (E.g., [34, 38]). On the other hand, the association between family dynamics and hypertension risk was not always immediately apparent (e.g. [49, 50]). In young, healthy children, it is possible that the sleep

behaviors are more immediately sensitive to fluctuations in family dynamics than other hypertension risk markers. Limited findings from studies that include measurement of family dynamics, hypertension, and sleep provide some evidence that sleep behaviors moderate the association between family dynamics and distal hypertension risk; however, only two studies [67, 68]tested for interactive associations. Sleep behaviors are now an essential component of cardiovascular health [69], thus, it will be important to continue to measure both sleep and hypertension markers in a family context to understand how these constructs interact.

Considerations for future research

The long-term goal of this research is to improve hypertension. In light of the most recent findings on family dynamics and sleep, there are several considerations for future research that will facilitate this goal. First, advanced methodological and analytic approaches to family-level variables will provide information on gaps in the literature. For example, much of the recent findings are based on aggregated family dynamic characteristics. Measurement of day-to-day family interactions allows for greater specificity on how family relationships contribute to nightly sleep patterns. This design would also allow for rigorous tests of bidirectional and lagged associations between sleep and family dynamics. For example, it is likely that family dynamics are also influenced by sleep (Figure 1) especially in families with infants and toddlers [70]. It is also necessary to consider analytic approaches that take into account the inherent interdependence of family relationships and health behaviors. Family members' behaviors are likely more similar to one another than to non-family members, and family members' behaviors are dynamically linked. Thus, behaviors of individuals within family units are *nonindependent* [71]. Statistical tests with *individuals* as the unit of analysis, as opposed to dyads or families, do not take into account the shared properties of nested, or interdependent data. Interdependent analytic techniques may result in more accurate estimates of the associations among family dynamics, sleep, and hypertension.

Second, sleep duration and sleep quality were among the most frequently studied components of sleep health. Sleep timing and circadian rhythms in a family context likely warrant further attention. Daily activities such as bedtime, wake time, and meal times (i.e., daily social rhythms) are time cues, *zeitgeibers*, for the endogenous circadian system [72]. The timing of family-based activities (such as meal time) have the propensity to influence an individual's daily social rhythmicity, and in turn, their endogenous circadian rhythms. The timing and frequency of engagement with family members can also influence endogenous biological rhythms [72]. Importantly, disruptions to circadian rhythmicity can alter cardiovascular functioning [73]. Moreover, meal timing and eating patterns, which are often family activities, are associated with cardiovascular health [74]. Thus, the timing of family activities (such as mealtime) may have a direct influence on cardiovascular health, and family social rhythms may have an indirect influence on cardiovascular health through circadian rhythms. Family-level assessment of social rhythmicity and circadian rhythms *and* sleep behaviors, will reveal novel mechanisms for hypertension risk and targets for intervention.

Third, it is also important to consider larger systemic processes that influence family- level sleep and hypertension. We focused our review on within-family dynamics and did not address systemic influences such as socioeconomic status (SES) and race and ethnicity. While several of the studies reviewed here included SES, race, and ethnicity as covariates, few studies reported on significant interactive effects of these social determinants. Nikulina and colleagues [46] found that childhood neglect was associated with CRP in white children only. Chan and colleagues [75] found that more negative feelings about the early family environment was associated with higher resting blood pressure among adolescents with higher SES. Importantly, disparities in sleep health behaviors may contribute to cardiovascular health disparities [76]. To address disparities in hypertension [77] and sleep [76], it is critical to clarify the associations among sleep, family dynamics, and hypertension risk in at-risk populations. Eliminating disparities in hypertension and sleep requires more research with diverse and underrepresented populations, and measurement of factors unique to these groups that could amplify sleep problems and hypertension risk at the family level [76]. For example, shift work and nonstandard work schedules are over represented by ethnic minorities and individuals/families who are economically challenged [78]. Shift work is associated with sleep disruption and hypertension in individuals; however, we know little about how an organizational determinant such as shift work influences sleep and circadian rhythms at the family level. There are multiple interactive systems that have direct and indirect influences on families and their sleep and cardiovascular outcomes [79]. Associations within the family system and between other organizational and community systems will be important to clarify in order to move in the direction of intervention and health promotion.

Finally, to that end, a logical next step in this area of research is to consider family-based approaches to sleep health promotion. We propose that focusing on sleep in a family context may be one way to encourage structure, collaboration, and warmth in the family unit, and that improved connection and sleep, in turn, could improve hypertension risk. One aspect of family dynamics that may be especially critical to target is parenting practices surrounding waking and nighttime behaviors. Parental factors such as monitoring, structure, and sensitivity were associated with sleep outcomes in 11 of the 15 studies on family dynamics and sleep. A focus on modifiable parenting practices has been successful in other health related outcomes. For example, increasing parental warmth, structure, and emotional support in childhood obesity interventions leads to weight reduction in children *and* parents [80]. Thus, a focus on parenting factors surrounding sleep could ostensibly have other downstream effects on family dynamics, and overall family health.

Conclusion

Components of sleep health such as poor sleep quality and insufficient sleep increase hypertension risk. Family relationship dynamics also contribute to hypertension risk. Understanding sleep health behaviors in a family context provides greater understanding of two potentially modifiable targets for reducing hypertension risk in families. Families can influence one another’s sleep (and potentially circadian rhythms) through shared activities, relationship characteristics, and concordant sleep behaviors. Parenting styles characterized by structure and warmth/sensitivity appear to be associated with better sleep health and

hypertension outcomes. However, future research will benefit from clarifying interactions among family dynamics, sleep behaviors, and hypertension risk. Clarification of these processes, in turn, will help to inform family-level approaches to improving sleep, and ultimately, hypertension outcomes.

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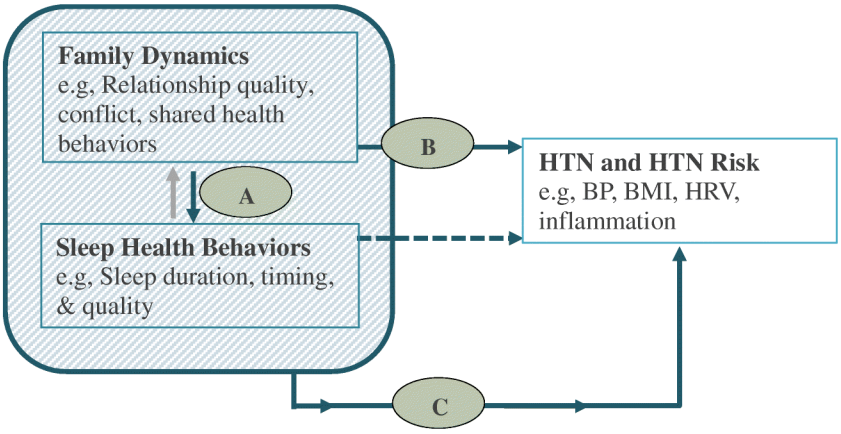

Figure 1.

A conceptual model of influence of family dynamics and sleep health behaviors on hypertension risk. Bold arrows indicate proposed directions; dashed arrow represents well-documented association between sleep behaviors and hypertension; gray arrow represents bidirectional association to consider in future research. HTN = hypertension; BP = blood pressure; BMI = body mass index; HRV = heart rate variability

Table 1.

Family Dynamics and Sleep

Author, Year	Sample (n, age, age group, race, population, gender)	Study Design	Family variable	Sleep Variable	Covariates	Results
Short, 2013	US sample: N = 302; Mage = 16.03; ages 13.8-19.9;35.15% male; Australian sample: N = 385; Mage =15.57; ages = 13.3-18.9;59.2% male	Cross sectional	Parent-set bedtime	Adolescent sleep diary: TST	gender, age, school start time, extracurricular load	**Parent-set bedtime** ↑ TST
Kelly, 2013 [81]	N = 176 US children; 3rd grade at baseline; 32% male; 31% AA, 69% White	Longitudinal	Marital aggression and emotional insecurity in 3rd and 5th	Child reported: Sleep/wake Problems in 3rd and 5th grade; Actigraphy assessed:SE and TST in 3rd and 5th grade	ethnicity, SES, gender	**Emotional insecurity** and **marital aggression** and TST in 5th grade ns. **Emotional insecurity** and **marital aggression** = ↓ SE at 5th grade **Emotional insecurity** and **marital aggression** =sleep problems in 5th grade
Ray, 2013 [42]	N = 805 Finnish child; ages 10–11 years; 50% male	Cross sectional	Parent practices (e.g. rules and routine); parental warmth/responsiveness	Adolescent reported: TST	gender, gender of parent, grade school, parent's educational level, employment status	↑ **Parenting practices** ↑ TST **warmth/responsiveness** did not moderate the association between **parenting practices** and TST
Maume, 2013 [40]	N = 974 US adolescents; 6th grade at baseline; follow up at age 15; 50% male; 80% white, 20% nonwhite	Longitudinal	Mother's partner leaves; mother has new partner; parental-adolescent bond; change in parent-adolescent bond; parental monitoring; change in parental monitoring index; consistent set bedtime	Adolescent reported: TST, sleep disruption	pubertal development, delayed phase preference, race, gender	**Mother's partner leaves** ↓ TST; ↑ sleep disruption **Mother has new partner** TST ns.; sleep dismption ns. **Parental bond** TST ns.; sleep dismption ns. **Increase in parent-adolescent bond** ↑ TST; ↓ sleep disruption ↑ **Parental monitoring** ↑ TST; sleep disruption ns. ↑ **Change parental monitoring** ↑ TST; ↓ sleep disruption ↑ **Consistent set bedtime on school nights** ↑ TST; ↓ sleep disruption
Vazsonyi,2015[34]	Georgian population: N =6,992; 40% male; Mage =15.83; ages 15–18; Swiss population: N = 5,575; 49.9% male; Mage = 17.17; ages 15– 18	Cross sectional	Family structure; parental warmth	Adolescent reported: Sleep quality, TST	age, sex, family structure, parental education	↑ **Parental warmth** ↑ Sleep quality ↑ **Parental warmth** ↑ TST

Author, Year	Sample (n, age, age group, race, population, gender)	Study Design	Family variable	Sleep Variable	Covariates	Results
Roblyer, 2015 [38]	N = 91 US adolescents; ages 11 -19; 54.9% male; 12.1% AA, 81.3% White, 6.6% Other	Cross sectional	Parental involvement; conflict; parental control	Adolescent reported: Sleep problems, TST	ethnicity, age, depressive symptoms	**Parental involvement** Difficulties initiating sleep ns.; difficulties maintaining sleep ns.; TST ns. ↑**Parent-child conflict** ↑ Difficulties maintaining sleep; difficulties initiating sleep ns.; TST ns. ↑ **Parental control** ↑ difficulties initiating sleep; ↑ difficulties maintaining sleep; TST ns.
Peltz, 2018 [39]	N = 193 US adolescents; Mage = 15.7; 45.6% male;14% Black, 71% White, 2% multiracial, 2% Latino, 2% Asian	Cross sectional	Family chaos; inconsistent discipline	Adolescent diary:TST, sleep quality	age, gender, SES, daily daytime naps, school start time	**Family chaos** ↓ Sleep quality; TST ns. **Inconsistent discipline** TST ns.; sleep quality ns.
Staples, 2015 [41]	N = 87 young US children; 30 months at baseline; 42.53% male; 4% AA, 7% Latino,82% White, 7% mixed/other	Longitudinal	Bedtime routine; inconsistent parenting at all three waves	Actigraph assessed:TST		**At 30 months, routine and inconsistency** TST ns. **At 36 months,↓ inconsistency, ↑ routine** ↑ TST **At 42 months, ↑ routine** ↑ TST **At 42 months, inconsistency** TST ns.
Millikovsky-Ayalon, 2015 [82]	N = 51 father, mother, and young child Israeli triads; (26 children with sleep disturbance and 25 children without sleep disturbance); ages 1-3; 53% male	Cross sectional	Parental stress due to acceptance of the child and restrictions imposed by the parental role; parental bedtime interaction (i.e. measure of parent assistance); parent sensitivity	Parent reported: Child sleep disturbances	matched based on age, gender, birth order, maternal education	↑ **Maternal stress (child acceptance and role restriction)** ↑ Sleep disturbance ↑ **Paternal stress (child acceptance)** ↑ Sleep disturbance **Paternal stress (role restriction)** Sleep disturbance ns. ↑ **Parental bedtime interaction** ↑ Sleep disturbance **Maternal sensitivity** Sleep disturbance ns. ↑ **Paternal sensitivity** ↓ Sleep disturbance ↑ **Parental involvement** ↓ Sleep disturbance
Fuligni, 2016 [31]	N = 421 adolescents with Mexican heritage; T1age = 15.03; 50%male; follow up =1 year later	Longitudinal	Parental support; conflict; # of people in household	Parent and Adolescent reported: Parent- adolescent sleep concordance at T1 and T2	parent education	↑ **Household size** ↑ Concordance ↑ **Parental support** ↑ Concordance **Conflict**

Author, Year	Sample (n, age, age group, race, population, gender)	Study Design	Family variable	Sleep Variable	Covariates	Results
						Concordance ns.
Meijer [35]	N = 650 German adolescents; T1age = 13.36, follow up 1 and 3 years later; 50.46% male	Longitudinal	Quality of parent adolescent relationship, autonomy granting, parental monitoring at all 3 waves	Adolescent reported: bedtime, TIB, sleep quality	sex, age, SES, ethnicity	T1: ↑ **Relationship quality** Earlier bedtime; ↑ sleep quality; TIB ns. **Autonomy** Bedtime ns.; TIB ns.; sleep quality ns. ↑ **Monitoring** Earlier bedtime; ↑ TIB; monitoring ns.; sleep quality ns. T2: ↑ **Relationship quality** Earlier bedtime;↑ (TIB; ↑ sleep quality ↑ **Autonomy** ↑ Sleep quality; bedtime ns.; TIB ns. ↑ **Monitoring** Earlier bedtime; ↑ TIB; ↑ sleep quality T3: ↑ **Relationship quality** Earlier bedtime; ↑TIB;↑ sleep quality ↑ Autonomy ↓TIB; ↑ sleep quality; bedtime ns. ↑ **Monitoring** Earlier bedtime; ↑ TIB; ↑ sleep quality
Tetreault,2017 [36]	N = 200 Canadian mother- child dyads; 5 waves from ages 1-4; 49.5% male	Longitudinal	Maternal sensitivity (e.g. cooperation/attunement, positivity, accessibility/ availability)	Parent diary: SE, TST		↑ **Cooperation/attunement** ↑ SE at age 2, age 3, age 4, TST ns. ↑ **Positivity** ↑ SE at age 3; TST ns. ↑ **Accessibillty/availability** ↑ SE at age 3; TST ns.
Kouros, 2017 [30]	N = 163 US children; Mage = 10.45; 55% male; 25% AA,75% White	Cross sectional	Parent-child concordance	Actigraph assessed:TST, SE, # of wake episodes, WASO, bedtime, morning wake time	age, race, BMI, parental marital status, family income, pubertal status, maternal depressive symptoms	**Mother-child were concordant** SE; # of wake episodes; WASO; bedtime; morning wake time **Mother-child were not concordant** TST **Father-child were concordant** TST; total WASO; wake time **Father-child were not concordant** SE; # of wake episode; bedtime

Author, Year	Sample (n, age, age group, race, population, gender)	Study Design	Family variable	Sleep Variable	Covariates	Results
Conway, 2018 [37]	N = 820 US children; T1age = age 3, T2 = 6th grade; 10.4% AA, 80.73% White, 5.1% Asian or Native American	Longitudinal	Maternal sensitivity; negative emotionality at age 3	Parent reported: Sleep problems at 6th grade	child sex, race/ethnicity, family income, maternal age, maternal education, child sleep behaviors at 3rd grade, maternal sensitivity and child negative emotionality at 5th grade, pubertal development status at 6th grade, maternal depressive symptoms at 6th	In adolescents with ↑ negative emotionality at age 3, ↑ **maternal sensitivity** predicted ↓ sleep problems in 6th In adolescents with ↓ negative emotionality at age 3, ↓ **maternal sensitivity** predicted ↑ sleep problems in 6th grade
Gunn, 2019 [43]	N = 165 US adolescents; Mage= 11.8; ages 10-14; 48% male; 78.6% AA, 15.7% White, 5.7%biracial	Cross sectional	Parental monitoring (parent and adolescent reported); parental knowledge; parental expectations of bedtime	Actigraph assessed:TST, sleep variability	gender, sex	↑ **Parental monitoring (parent reported)** ↑ Weekday TST; 7-day TST ns.; weekend TST ns.; sleep variability ns. ↑ **Parental monitoring (adolescent reported)** ↑ 7-day TST; ↑ Weekday TST; ↑ weekend TST; sleep variability ns. **Parental knowledge about bedtime** TST ns.; sleep variability ns. **Parental expectations of bedtime** TST ns; sleep variability ns.

Note. All sleep parameter results refer to child or adolescent's sleep parameters; unless otherwise stated, all sleep parameters were either parent-reported or self-reported; ns = not significant; AA = African American; SE = sleep efficiency; TIB = time in bed; BMI = body mass index; TST = total sleep time/sleep duration; WASO = wake after sleep onset.

Table 2.

Family Dynamics and Hypertension

Author, Year	Sample (n, age, age group, race, population, gender)	Study Design	Family variable	HT marker	Covariates	Results
Wang,Anderson [57]	N = 1,090 US children; 50.7% male; 12.1% AA 81.2% White,6.7% other	Longitudinal	Maternal depression when child was 1 month, age 2, and age 3	BMI in grades one, three, and six	birth weight, gender, race, maternal education, SES, breastfed, parent smoker, maternal social support, maternal sensitivity	↑ **Maternal depressive symptoms at 1 month** ↑BMI in 6th grade; BMI in 1st and 3rd grade ns. ↑ **Maternal depression age 2** ↑ BMI in 3rd and 6th grade ↑ **Maternal depression at age 3** ↑ BMI in 3rd grade **Maternal depression at more time points** ↑ BMI
Pretty, 2013 [47]	N = 1,234 Canadians adolescents;Mage =11.8;45% male	Cross sectional	Childhood adversity	Resting BP, HR, BMI, WC	family education, family income, parental history of hypertension, age, sex, physical activity	≥**4 adverse experiences** ↑ HR; ↑ BMI; ↑ WC; BP *ns.*
Lumeng,2013 [56]	N = 848 US children; age 4 at baseline, 49.5% male; 82% White; 18% other	Longitudinal	Chronicity of negative events (i.e., negative life events at multiple time points); # of negative life events; impact of negative life events; timing of negative life events; parenting sensitivity	BMI at age 15	gender, race/ethnicity, maternal education, maternal obesity	↑ **# of negative events ↑ BMI** ↑ **Chronicity of negative events =** ↑ BMI **Timing of negative life events** BMI ns. **Impact of negative life event** BMI ns. ↑ **Maternal parenting sensitivity** ↓ BMI
Nikulina,2014 [46]	N = 806 US adults with documented childhood neglect and their matched control, Mage =41; 49% male;59%	Longitudinal	Documented childhood neglect	Resting BP,CRP, pulmonary functioning	gender, smoking, hypertension medication, asthma, BMI or other pulmonary disease diagnoses, neighborhood poverty,	**Childhood neglect** hypertension ns. **In White participants, childhood neglect**

Author, Year	Sample (n, age, age group, race, population, gender)	Study Design	Family variable	HT marker	Covariates	Results
	White, 35% AA, 6% other				childhood family poverty	↑ CRP; ↑ pulmonary functioning
Hernandez, 2014 [55]	N = 3,447 US adults; Mage =21.1; 54% male; 72% White; 19% AA, 9% Hispanic	Longitudinal	# of family structure changes throughout childhood (e.g. mother remarrying, mother divorcing)	BMI	Child: race, birth weight, depressive symptoms, self-esteem, # of siblings, duration since last family structure transition, age, year of BMI assessment; Mothers: age, relationship status, education status, change in education status, BMI	Main effects ns. **In girls, ↑ family transitions** ↑ BMI
Su, 2015 [48]	N = 394 US adults; age 5 at baseline; 47% male; 54.1% AA,45.9% White	Longitudinal	Childhood adversity	Resting BP via visits every 1 to 2 years over 23 years from childhood to adulthood	ethnicity, sex, BMI	Main effect = ns; **Around age 30, ↑ACE** ↑ **SBP**; ↑ **DBP**
Gupta-Malhotra.2016 [49]	N = 515 African American men; Mage = 47.8	Retrospective	Lived with both parents from ages 1-12; lived with both parents from ages 13-19	Resting BP,MAP, PP	age, material status, education, smoker, alcohol, physical activity, BMI, type 2 diabetes, obesity, family history of hypertension, current antihypertensive medication, HDL, LDL, triglycerides, sodium/ potassium ratio	**Lived with both parents at any time between 1-12** ↑SBP; DBP ns. **Lived with both parents at any time between 13-19** SBP ns.; DBP ns **Lived with parents from 1-12 and 13-19** ↓ MAP; ↓PP
Boyer. 2015 [50]	N = 1,364 US children; pre-k at baseline; 52% male; 76% White, 24% persons of color	Longitudinal	Parental sensitivity at pre-k and 1st grade	BMI and resting BP at 6th and 9th grade	gender, ethnicity, family income-to-needs ratios	↑ **Both parent's sensitivity** ↓ BMI; ↓ SBP at 9th grade; SPB at 6th grade ns.
Gibson,2016 [54]	N = 286 community and clinical Australian population; Mage= 9.43; children; 49% male	Longitudinal	1 parent vs 2 parents; parental laxness; over-reactivity; verbosity; psychological health/ pathology of the family	BMI	Gender, age, SES, mother's depression, mother's stress, mother's negative life, mother's self-esteem, maternal obesity	**One parent home** ↑ BMI **Laxness, over-reactivity, verbosity, psychological health/pathology of the family** BMI ns. **In males, higher verbosity in parent** ↑BMI

Author, Year	Sample (n, age, age group, race, population, gender)	Study Design	Family variable	HT marker	Covariates	Results
Chan. 2016[7]	N = 259 Canadian adolescents; ages 13-16; 47% male; 49.4% White,15.3% Asian	Cross sectional	Implicit affect; implicit warmth toward family; household crowding	Resting BP, total cholesterol, glycosylated hemoglobin, WC	age, gender, ethnicity	↑ **Early life crowding** ↑SBP; DBP ns.; cholesterol ns.; HbA1c ns.; WC ns.; ↑ **Implicit negative affect** ↑ Total cholesterol; ↑SBP; ↑DBP; HbA1c ns.; WC ns. **Implicit warmth** SBP ns.; DBP ns.; HbA1c ns.; total cholesterol ns.; HbA1c ns.; WC ns.
Dong. 2017[51]	N = 940 Chinese parent-child pairs; ages 7–17, Mage =12.1; 56.17% male	Cross sectional		Resting BP,HbAlc, BMI from child and parent	age, sex, parental age, household structure, income, geographical region, parental education, urban/rural, parental smoking, caloric intake, physical activity	↑ **Parent's HbA1C** ↑ child's HbA1c ↑**Mother BP** ↑ daughter's SBP; ↑daughter's DBP ↑**Father CRP** ↑child's CRP; ↑ son's obesity
Yavuz. 2018 [58]	N = 61 normal weight Turkish children and 61 overweight or obese Turkish children; Mage = 5.18 years old; 52.36% male	Cross sectional	Parenting style; food restriction; pressure to eat; food monitoring	Normal weight vs.obese/overweight	mother's education, parent's BMI, child's NA	↑ **authoritarian parenting** ↑ obese/overweight **Authoritative parenting** BMI ns. ↑ **Pressure to eat** ↑ obese/overweight **Food restriction** BMI ns. **Food monitoring** BMI ns.
Heredia,2019 [59]	N= 1175 Mexican-American adolescents; ages 11-13 at baseline; 50% male	Longitudinal	Family cohesion; family conflict	BMI	Age, acculturation, household size, parent marital status, parent BMI, parent education, parent acculturation	**In females,** ↑ **family cohesion** Decrease in BMI **In females,** ↑ **family conflict** Decrease in BMI

Note. All results refer to child or adolescent's cardiovascular outcomes. ns = not significant. AA = African American. BMI = body mass index. SES = social economic status. BP = blood pressure, WC = waist circumference. HR = heart rate. SBP = systolic blood pressure. DBP = diastolic blood pressure. HF = high-frequency bands. LF = low-frequency bands. CRP = C-Reactive protein. MAP = mean arterial pressure. PP = pulse pressure. HDL = high- density lipoprotein. FDF = low- density lipoprotein. HbAlc = hemoglobin Alc.

Table 3.

Family dynamics, Sleep and Hypertension Risk

Author, year	Sample (n, age, age group, race, population, gender)	Study Design	Family variable	Sleep variable	HT marker	Covariates	Results
Jones,2014[60]	N = English 108 children; age 3; 53% male	Cross sectional	Presents of sleep rule, television viewing rule and/or dietary rule	Parent diary: TST	BMI; WC; triceps skinfold thickness; subscapular skinfold thickness		**Sleep rule** ↑ TST, BMI ns; WC ns.; skinfold thickness ns. **TV rule** ↑ TST; ↓ BMI; ↓ WC; ↓ subscapular skinfold thickness; triceps ns. skinfold thickness ns. **Dietary rule** ↑ TST; ↓ subscapular skinfold thickness; BMI ns.; WC ns.; triceps ns.; skinfold thickness ns.
El- Sheikh,2015 [67]	N = US 160 children; age 9.43 at baseline, 1 year follow up 53.1% male; 27% AA, 73% White	Longitudinal	Marital conflict	Actigraph assessed: TST, night time awakenings, sleep activity,SE	RSA-R	BMI, the presence of an illness and the sleep parameter at W1	↑ **Marital conflict,** ↑ **RSA-R,** ↓ **TST** ↑TST ↑ **Marital conflict,** ↑ **RSA-R, and** ↓ **SE** ↑ SE ↑**Marital conflict,** ↓ **RSA-R, and** ↓ **SE** ↓ SE ↑**Marital conflict,** ↓ **RSA-R and** ↓ **SE** ↓ SE ↑**Marital conflict,** ↑**RSA-R, and** ↓ **sleep activity** Sleep activity ↑**Marital conflict,** ↑ **RSA-R;** ↓ **long wake episodes** ↑ Wake episode
Kiel,2015 [63]	N = 51 mother-toddler dyads; T1: 18.96 months old at baseline, follow up at ages 2 and 3; 49.02% male; 82.4% White, 2.0% Latino Americans, 3.9% Asian Americans, 11.8% biracial	Longitudinal	Parental overprotection; parental critical control via questionnaires	Parent reported:Sleep problems at ages 2 and 3	Time 1:Cortisol secretion	sleep problems at age 2, parental overprotection	**Over protection** Sleep problems ns. **critical control** Sleep problems ns. **Critical control and cortisol secretion** = sleep problems at age 3
Gustafsson,2017[68]	N = 292 pregnant US adolescents; Mage =17.81; 88.28% Latina;3rd trimester	Cross sectional	Emotional abuse	Sleep disturbance	Fetal heart rate variability	age, ethnicity, income, gestational age	↑ **Emotional abuse and** ↑ **sleep disturbances**↓ Heart rate variability

Author, year	Sample (n, age, age group, race, population, gender)	Study Design	Family variable	Sleep variable	HT marker	Covariates	Results
	fetuses; fetuses = 43% female						
Zilioli,2017[65]	N = 645 Chinese participants; Mage=10.67 years; ages 8-15;51.9% male	Cross sectional	Loneliness via daily diaries and trait loneliness	Adolescent diary:Subjective sleep quality, SOL, # of awakenings,TIB	CAR	gender, parent death, health status, stressful life events, age, negative affect, depression	↑ **Trait loneliness** ↓ Morning cortisol; ↑ TIB; ↓ sle0ep quality ↑ **Daily loneliness** Flat cortisol slope; ↑ night awakenings; ↓ TIB

Note. All results refer to child or adolescent's cardiovascular or sleep outcomes; unless otherwise stated, all sleep parameters were either parent-reported or self-reported; AA = African American; T1 = initial wave of data collection; BMI = body mass index; WC = waist circumference; TST = total sleep time/sleep duration; RSA-R = respiratory sinus arrhythmia- reactivity; SE = sleep efficiency; SOL = sleep onset latency; TIB = time in bed; CAR = cortisol awakening response.