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Development of infant physiological self-regulatory capacities across the first year of life: the role of parenting

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DEVELOPMENT OF INFANT PHYSIOLOGICAL SELF-REGULATORY CAPACITIES ACROSS THE FIRST YEAR OF LIFE: THE ROLE OF PARENTING

by

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DEDICATION

To my father, who was my source of motivation and strength, and to my mother, siblings, and husband for their constant love and support.

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ABSTRACT

Sleep and cortisol function are two physiological self-regulatory processes that codevelop during infancy. Dysregulation of each system is linked to enduring health problems, so it is critical to understand factors contributing to the development of physiological self-regulation. However, it is not clear how infant sleep and cortisol interact with each other or with the parenting context.

This project examined (1) the interplay of infant sleep and cortisol; (2) how cortisol interacts with parent characteristics in relation to infant sleep; and (3) whether consistent parenting buffers infant cortisol dysregulation. Study 1 (86 parent-infant dyads) investigated whether average nighttime sleep onset and duration predicted cumulative cortisol exposure, indexed by hair cortisol concentration (HCC). As hypothesized, infants who fell asleep earlier at night had lower HCC regardless of their family income and household chaos. Additionally, I expected that sleep characteristics on one night would predict total salivary cortisol exposure (AUC_g) the next day, and that salivary cortisol at bedtime would predict sleep the same night. Partially supporting expectations, time-based analyses revealed that infants with lower cortisol on a particular evening fell asleep earlier the same night. In Study 2 (84 parent-infant dyads), I hypothesized that the link between parent characteristics (i.e., bedtime parental involvement and parental sensitivity) and infant sleep would differ by AUC_g. Falling asleep independently predicted earlier sleep onset only for infants with dysregulated cortisol, whereas bedtime parental involvement did not predict sleep for infants with well-regulated cortisol. Infants with emotionally warm and appropriately responsive parents fell asleep earlier at night only if their cortisol was well-regulated. Utilizing archival data of 82 mother-infant dyads, Study 3 assessed consistency in parenting behaviors (i.e., smiles and laughter, and positive vocalizations), cortisol, and socioeconomic status (SES). As hypothesized, higher-SES infants experienced consistency, whereas lower-SES infants experienced inconsistency, in maternal smiles and laughter across 6 to 12 months of infancy. Contrary to expectations, inconsistent parenting did not predict cortisol. Findings highlight the intricate relation between two vital physiological processes codeveloping in the first year of life – sleep and cortisol regulation – and the role cortisol plays in moderating how parenting characteristics contribute to infant sleep.

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LIST OF ABBREVIATIONS

ANCOVA ....Analysis of covariance

AUC_g..Area under the curve with respect to ground

CHAOS .... Confusion, Hubbub, and Order Scale

EAS... Emotional Availability Scale

GED...General Education Development

HCC .... Hair cortisol concentration

HPA.. Hypothalamic-pituitary-adrenocortical

ICC.. Intraclass correlation coefficients

IDS...Infant-directed speech

ITN .... Income-to-needs

OLS...Ordinary least-squares

PCA..Principal component analysis

SCC...Salivary cortisol concentration

SD .... Standard deviation

SES .... Socioeconomic status

GENERAL BACKGROUND

Sleep and the physiological stress system are two fundamental biological self-regulatory processes. Sleep is vital for the restoration of the body and the brain (Assefa, Diaz-Abad, Wickwire, & Scharf, 2015) and children spend more time sleeping than adults (Iglowstein, Jenni, Molinari, & Largo, 2003). When individuals get inadequate sleep or have difficulties falling and staying asleep, they experience a host of physiological and neuropsychological malfunctions (see Assefa et al., 2015; McEwen, 2006a). For example, sleep deprivation leads to increased appetite, obesity, physiological stress dysregulation, cardiometabolic health problems, and dampened immune response, memory formation, and motor skill learning. Further, sleep disturbances are associated with mood disorders such as depression and anxiety (Franzen & Buysse, 2008; Staner, 2003) Thus, it is absolutely important for the body to rest and recuperate at night. During the day, however, the body is alert, active, and ready to perform daily tasks. This is because the hypothalamic-pituitary-adrenal (HPA) axis, a physiological stress system, prepares the body to combat daily life challenges by mediating the production of the stress hormone cortisol (Cone, Low, Elmquist, & Cameron, 2002). Cortisol production directs bodily resources to performing actions by mobilizing energy to muscles, enhanced cardiovascular output, and heightened cognition (Sapolsky, Romero, & Munck, 2000). In adults, the workings of the HPA axis is closely connected to sleep. Cortisol, produced by the HPA axis, reaches its minimum level during the early phase of nighttime sleep and peaks during the late phase of sleep through waking (Born & Fehm, 1998). As with sleep deprivation, failure of the HPA axis to optimally regulate cortisol is also known to be detrimental for a range of mental and physiological health outcomes. In fact, there is a substantial overlap between the health outcomes associated with cortisol dysregulation and sleep difficulties. For example, cortisol dysregulation is also associated with vulnerability to depression, anxiety, cardiovascular diseases and immune-mediated diseases (Bauer, 2005; Laurent, Gilliam, Wright, Fisher, 2015; McEwen, 2006b).

Sleep is immature at birth and undergoes drastic changes over the first year of life. Infants progress from having irregular and fragmented sleep distributed across 24 hours to the emergence of a circadian rhythm that leads to more consolidated nighttime sleep by 12 months of age (Anders, Sadeh, & Appareddy, 1995; Davis, Parker & Montgomery, 2004; Sheldon, 2002). Early life adversities can obstruct proper development of sleep during infancy, leading to irregular sleep schedules and sleep deprivation (McEwen, 2006a, 2006b). There are indications that such early sleep difficulties are enduring as they predict sleep problems in later childhood (Scher, Epstein, & Tirosh, 2004; Tikotzky & Shaashua, 2012; Zuckerman, Stevenson, & Bailey, 1987). Moreover, we know from adult studies that such dysregulation in sleep is a stressor (McEwen & Karatsoreos, 2015), contributing to the wear and tear on the body and leading to impairment of multiple regulatory systems. This phenomenon is referred to as the allostatic load (McEwen, 2006a, 2006b).

Cortisol regulation is also immature at birth and develops throughout infancy (Antonini, Jorge & Moreira 2000, De Weerth, Zijl & Buitelaar 2003, Spangler, 1991). Like sleep, the HPA axis develops a circadian rhythm as it matures. There is a daily pattern of cortisol peaking in the morning and dropping at night. This cyclic change in cortisol levels is referred to as the diurnal cortisol rhythm. The upsurge in cortisol in the morning prepares the body to perform activities throughout the day, and the decrease in cortisol at night helps the body wind down and fall asleep. As with sleep, early life insults maladaptively influence the developing HPA axis (Boyce & Ellis, 2005; Gunnar 1994). The HPA axis adapts to become vigilant to challenges, i.e., hyperactive, in order to survive in a threatening environment. Following prolonged periods of hyperactivity due to early life stressors, there is downregulation of the HPA axis represented by a pattern of hypoactivity (Friese, Hesse, Hellhammer, & Hellhammer, 2005). An indication of hypoactivity is the flattening of the diurnal cortisol activity due to a slower decline in cortisol through the day with lower morning cortisol levels and higher evening cortisol levels (Gunnar & Vazquez, 2001). This pattern of cortisol dysregulation is seen in children who have suffered severe early deprivation, neglect, and abuse (Carlson & Earls, 1997; De Bellis et al., 1999; Meinlschmidt & Heim, 2005; Shea, Walsh, MacMillan, & Steiner 2004). Thus, dysregulation of the HPA axis in early life gives rise to maladaptive HPA axis functioning later in life. In turn, HPA axis dysregulation adds to the allostatic load, ensuing a range of lasting health problems (McEwen, 2006b). Thus, infancy is a time of vulnerability when early life dysfunction of both sleep and cortisol systems plays a role in later dysregulation of each of the systems, increasing the allostatic load.

Along with the establishment of a circadian rhythm during infancy, sleep and cortisol systems develop in parallel as they become characterized by consolidated nighttime sleep and the onset of adult-like diurnal cortisol pattern (Antonini et al., 2000, De Weerth et al., 2003). The development of these systems during infancy is foundational for how they regulate later in life. It is suggested that early insults on the development of one system not only leads to dysregulation in that system, but can also maladaptively influence the maturation of the other system (Palagini, Drake, Gehrman, Meerlo, & Riemann, 2015; Van Reeth et al., 2000). This sets the stage for lasting sleep and cortisol dysregulation adding to the allostatic load, which ultimately increases an individual's risk for enduring health problems (McEwen, 2006a, 2006b).

Infancy is a time when humans depend the most on their caregivers to regulate their physiological systems including sleep and cortisol function (Hostinar, Sullivan, & Gunnar, 2014; Sadeh, Tikotzky, & Scher, 2010). In the case of sleep, parents play a role in infants' bedtime routines, environment, feeding habits, sleeping arrangement, and instilling a sense of safety. Parental sensitivity, in general helps, with biobehavioral regulation (Spangler, Schieche, Ilg, & Maier, 1994), and sleep regulation could also be one of the bioregulatory process shaped by sensitive parenting. All these factors contribute to infant sleep development, and early sleep characteristics have been linked to later sleep-wake organization (Scher et al., 2004; Tikotzky & Shaashua, 2012; Zuckerman et al., 1987). Therefore, parent characteristics have implications for concurrent and lasting sleep health. In the case of cortisol regulation, prior research has garnered substantial evidence suggesting that parental sensitivity has a social buffering effect on cortisol dysregulation (Hostinar & Gunnar, 2015). The social buffering effect refers to the protective role that social factors play by dampening cortisol responses. Given that the HPA axis is immature during infancy and vulnerable to early life adversities, it is well established that infants rely on sensitive caregivers for proper regulation of cortisol (Hostinar & Gunnar, 2015; Hostinar, Sullivan, & Gunnar, 2014; Tarullo & Gunnar, 2006). This has even led to a developmental model, which postulates that early sensitive caregiving shapes the HPA axis by influencing how future social relationships impact later HPA axis functioning (Hostinar & Gunnar, 2015). Thus, parenting is a common factor that is known to play a role in both sleep and cortisol function. This makes it a popular target for interventions directed towards promoting sleep health (e.g., Morgenthaler et al., 2006; Mindell, Kuhn, Meltzer, & Sadeh, 2006). Parenting has also been targeted for interventions to improve cortisol regulation (see Slopen, Mclaughlin, & Shonkoff, 2014).

Motivation for the Current Research

Sleep literature and cortisol literature both establish that early maladaptive development of these two vital self-regulatory processes have implications for lasting mental and physical health problems. To address these health concerns, the sleep literature and the cortisol literature have individually investigated risk and protective factors of early regulation of each of these physiological processes. These two systems develop in parallel during infancy, are vulnerable to early perturbations, and have implications for common long-term health outcomes. Despite these remarkable similarities between the two systems, the literatures on sleep and cortisol have largely remained separate. Given the parallel between the two systems in their early development, vulnerabilities, and enduring health implications, maladaptive development of one system could be a risk factor for dysfunction in the other system. Thus, there is a need to bridge the disconnection between the sleep literature and the cortisol literature, and investigate the early interplay between sleep and cortisol systems. Moreover, because both sleep and cortisol are immature during infancy and humans heavily depend on their caregivers for physiological regulation, it is important to understand the role of parents in the development of both these systems. While research has begun investigating this matter, the role of parenting in both these systems is not fully understood. The pressing need to understand the factors that influence the early development of these two crucial physiological regulatory systems motivates the studies presented in this dissertation research.

First, while the development of sleep or cortisol function is believed to be intertwined during infancy, the directionality of their relation is not known. For instance, it is possible that sleep difficulties contribute to cortisol dysregulation or vice versa. A third factor such as parenting could also interact with both the systems. A deeper understanding of the directionality of relation between sleep and cortisol, as well as the overall relation between sleep and chronic cortisol function in the first year of life is needed to further our knowledge on early co-development of these systems. This will have implications for promoting healthy sleep and cortisol function early on to ultimately reduce risks for later health adversities. One way to understand the directionality is by examining how one system relates to the other on a daily basis. There are daily variations in sleep characteristics and cortisol levels, and research attempting to disentangle temporal dependencies between these regulatory processes is lacking. There is evidence from a couple of time-based studies on children and adults, suggesting that the link between sleep and cortisol is likely bidirectional (Gribbin, Watamura, Cairns, Harsh, &

LeBourgeois, 2012; Van Cauter & Spiegel, 1997). With the exception of these studies, temporal relations between daily sleep and diurnal cortisol has not been thoroughly examined, especially in the first year of life. Apart from the daily interplay between sleep and cortisol, infant sleep difficulties also predict chronic cortisol levels (Flom, St. John, Meyer, & Tarullo, 2017). However, comprehensive research on this matter using objective assessment of multiple sleep characteristics still remains to be conducted.

Second, in addition to understanding the interplay between sleep and cortisol in depth, it is important to know what their association means for the protective or risk value of other factors (i.e., parent characteristics) contributing to infant sleep. Such knowledge can inform sleep interventions. Infancy is a vulnerable time when humans are most reliant on their caregivers. Naturally, parenting has been a popular candidate examined as a risk or protective factor for infant sleep (see Camerota, Propper, & Teti, 2019; Tikotzky, 2017; Sadeh et al., 2010). Though parent characteristics such as bedtime parental involvement and cosleeping have been linked to poor sleep outcomes, whether parental sensitivity buffers or exacerbates infant sleep difficulties is still not clear due to contradictory findings from prior literature. Furthermore, given that sleep and cortisol are primarily physiological processes that codevelop, cortisol regulation could play a role in how sensitive infant sleep is to parent characteristics. Thus, there is a need to assess the nuances of how parent characteristics interact with infant sleep in the context of cortisol regulation. Examining this will elucidate the likelihood of infant sleep being responsive to parent interventions when infants have well-regulated or dysregulated cortisol function.

Third, the role of parenting quality on infant cortisol function has been widely investigated, but whether consistency in the level of parental sensitivity across the first year of life matters for proper regulation of infant cortisol is not known. Unpredictability is a core characteristic of a stressor (Lupien, Maheu, Tu, Fiocco & Schramek, 2007). Variability in maternal response to infants at a crucial time when infants are forming attachments and expectations about their caregivers' behaviors, may ensue a sense of unpredictability, potentially adding to cortisol dysregulation (Manczak, Leigh, Chin & Chen, 2017). Young children exposed to consistent levels of parental sensitivity have better social and cognitive outcomes (Landry et al., 2001). A mechanism for this may be proper cortisol regulation. Thus, investigating how consistency in maternal sensitivity during infancy is related cortisol regulation can help us determine whether cortisol regulation is should be examined as an underlying mechanism in the relation between consistent parental sensitivity and optimal social and cognitive outcomes. Moreover, because parenting infants, especially in the first year of life, can be a turbulent time for some mothers who are vulnerable to financial strain (Bornstein, Hahn, Suwalsky, & Haynes, 2014), it is important to assess whether family income is a contributor of consistency in parental sensitivity.

Overview of the Current Research

The early development of two interconnected physiological self-regulatory processes, sleep and cortisol function, plays a crucial role in lasting health outcomes. Understanding the early interplay between these systems and the role of parents in their development has implications for promoting better sleep health and cortisol function.

Given this, the aim of this dissertation was to better understand how the two systems are intertwined and shaped by parenting in the first year of life. In Study 1, I examined the overall relation between sleep and chronic cortisol function in 12-month-old infants. I measured actigraphy-derived nighttime sleep duration and onset, and used hair cortisol concentration (HCC) as an index of cumulative exposure to cortisol. Additionally, I performed time-based analyses investigating whether sleep characteristics on one night related to salivary cortisol the next day, and whether salivary cortisol at bedtime related to sleep the same night. In Study 2, I used the same dataset as Study 1 to assess how parent characteristics (i.e. bedtime parental involvement, cosleeping, and parental sensitivity) related to nighttime sleep onset and duration, and whether these associations varied as a function of infant cortisol regulation indexed by total cortisol exposure across the day. In Study 3, I used archival data from a different dataset and assessed consistency in maternal parenting behaviors (i.e., smiles and laughter, and positive vocalizations) from 6 to 12 months of age, and the role of parenting consistency in HCC and total cortisol exposure across the day after accounting for socioeconomic status (SES). Jointly, these studies attempt to uncover the intricate association between two vital physiological processes co-developing in the first year of life – sleep and cortisol regulation – and the role of parents in their development.

INFANT EVENING CORTISOL LEVELS PREDICT SLEEP ONSET

Sleep patterns change dramatically over the first year of life. Infants progress from having irregular and fragmented sleep distributed throughout 24 hours to the emergence of a circadian rhythm that leads to more consolidated nighttime sleep by 12 months of age (Anders, et al., 1995; Davis, et al., 2004; Sheldon, 2002). In parallel, the hypothalamic-pituitary-adrenal (HPA) axis, a biological stress system also develops throughout infancy along with the establishment of a circadian rhythm (Antonini et al., 2000, De Weerth et al., 2003, Spangler, 1991). At night, sleep plays an important role in the restoration of the body and the brain (Assefa et al., 2015). During the day, the HPA axis prepares the body to combat daily life stressors by mediating the production of the stress hormone, cortisol (Cone et al., 2002). It is well established that the regulation of both these systems is closely intertwined in adults (Van Reeth et al., 2000), and their proper functioning is essential for concurrent and lasting positive health outcomes (McEwen, 2006b; Palagini et al., 2015). However, the early interplay between sleep and the HPA axis, when both these systems are maturing simultaneously, is not fully understood. The development of these systems during infancy is foundational for how they regulate later in life. Early perturbations on one system during such a vulnerable time can maladaptively influence the maturation of the other system. This sets the stage for lasting sleep and HPA axis dysregulation, which ultimately increases an individual's risk for enduring health problems (McEwen, 2006b; Palagini et al., 2015; Van Reeth et al., 2000). Thus, there may be an interplay between sleep and the HPA axis during infancy through which early adversities such as poverty get "under the skin," leading to lasting adverse health outcomes.

Both sleep and the HPA axis follow a circadian rhythm (Antonini et al., 2000; Borbely, 1982) and get dysregulated due to early life adversities (Flannery et al., 2017; Fisher, Stoolmiller, Gunnar, & Burraston, 2007; Fisher, Van Ryzin, Gunnar & Gunnar 2011; Gunnar & Vazquez, 2001; Hofer, 1976, 1994; Mrdalj et al., 2013; Palagini et al., 2015; Saridjan et al., 2010; Tiba, Tufik, Suchecki, 2004, 2008). On a daily basis, the HPA axis produces peak levels of cortisol during the final phases of sleep as the body prepares itself to wake for the day, and minimum levels of cortisol at night during early phase of nighttime sleep as the body winds down (Born & Fehm, 1998). This cyclic change in cortisol levels that is dependent on the circadian rhythm is referred to as the diurnal cortisol rhythm. While decline in cortisol from waking to bedtime is normative, severe early adversity has been associated with slower decline in cortisol, indexing a dysregulated diurnal cortisol rhythm during early and later childhood (Flannery et al., 2017; Fisher et al., 2007; Fisher et al., 2011; Gunnar & Vazquez, 2001). Early adversity is also related to increased cortisol levels throughout the day - another marker of dysregulated diurnal cortisol rhythm (Saridjan et al., 2010). In the context of sleep, early stress impacts sleep architecture and consolidation in rodents (Hofer, 1976; Hofer 1994; Mrdalj et al., 2013; Tiba, Tufik, Suchecki, 2004, 2008). In humans, it has been theorized that early adversity disrupts the circadian sleep-wake cycle, which ultimately leads to hyperactivated wake-promoting physiological systems (Palagini et al., 2015). A proposed mechanism is that early adversities lead to higher activation of the HPA axis which then distorts the sleep-wake cycle.

When dysregulation of the circadian sleep-wake cycle and diurnal cortisol rhythm is prolonged, it has adverse consequences for long-term health. Sleep dysregulation includes sleep deprivation disrupting the circadian rhythm, which is a stressor in itself leading to elevated cortisol levels in young adults (McEwen & Karatsoreos, 2015). This contributes to the wear and tear on the body and the impairment of multiple regulatory systems, a phenomenon referred to as allostatic load (McEwen, 2006). Likewise, diurnal cortisol dysregulation over a long period leads to higher cumulative exposure to cortisol over several months (e.g., Flom et al., 2017). Cumulative exposure to cortisol is assessed from cortisol deposited in the hair shafts (Meyer & Novak, 2012). Increased cumulative exposure to cortisol also contributes to the allostatic load, consequently increasing an individual's risk for poor health outcomes. Given this, examining the relation between sleep and cumulative exposure to cortisol during infancy is imperative to understand the emerging pattern of their association.

In infants, sleep deprivation, or poor sleep consolidation, is indexed by shorter nighttime sleep duration. Shorter nighttime sleep during infancy could also disrupt circadian rhythm and contribute to elevated cumulative exposure to cortisol, making infants vulnerable to a myriad of health problems (McEwen, 2006a). In line with this view, a recent study found that parent-reported, shorter nighttime sleep duration in the first year of life was correlated with increased cumulative exposure to cortisol (Flom et al., 2017). However, parent reports of sleep durations are prone to inaccuracy (Goodwin et al., 2007), and confirming this finding using objective measures of nighttime sleep duration is crucial. Another sleep characteristic that has been associated with shorter nighttime sleep duration during infancy is the later onset of nighttime sleep (Cheung, Bedford, Saez De Urabain, Karmiloff-Smith & Smith, 2017). Later sleep onset disrupts circadian rhythms, including diurnal cortisol rhythm, and early childhood studies reveal that delay in sleep onset in infants and toddlers is related to measures of diurnal cortisol such as higher cortisol awakening response and lower waking cortisol levels (Gribbin et al., 2012; Stalder et al., 2013). Thus, it is possible that later sleep onset also relates to cumulative exposure to cortisol during infancy. To our knowledge, this has not been explored. Findings from research on the association of poor sleep characteristics with cumulative exposure to cortisol, from as early as infancy, could have implications for improving infant and later health outcomes through early sleep interventions.

In addition to investigating the relations of sleep consolidation and onset with cumulative exposure to cortisol, understanding the interplay between sleep characteristics and diurnal cortisol function can provide insight on how short-term interplay between sleep and cortisol function could eventually lead to long-term associations. More research has examined the relation of sleep consolidation and onset with measures of diurnal cortisol by averaging the measures across multiple days (e.g. Flom et al., 2017; Gribbin et. al, 2012; Stalder et al., 2013). However, both sleep and cortisol levels vary on a daily basis, and it is necessary to disentangle temporal dependencies between daily sleep and diurnal cortisol regulation. There are indications that the direction of their association is likely bidirectional. Induced sleep onset delay in toddlers and preschoolers result in dysregulated cortisol levels the next morning, suggesting that sleep onset delay could influence diurnal cortisol rhythm (Gribbin et al., 2012). However, cortisol dysregulation may be impacting sleep as well. For example, temporal analyses show that adults with higher cortisol levels at night had delayed sleep onset (Van Cauter & Spiegel, 1997). With the exception of these few studies, research examining the relation between sleep and cortisol has not assessed time-based relations between daily sleep and diurnal cortisol, especially in the first year of life. Gaining further insights into the potential bidirectional nature of their association early in development will be important for obtaining a more comprehensive understanding of how sleep and cortisol systems begin to influence each other early in their development.

Given that both sleep and cortisol systems develop in parallel, and early perturbations have implications for lasting health outcomes, the goal of the current study was to understand how relatively objective measures of sleep consolidation and onset related to cumulative as well as diurnal cortisol regulation in 12-month-old infants. By this age, both sleep and diurnal cortisol rhythm are relatively established with consolidated nighttime sleep and adult-like diurnal cortisol patterns, providing an ideal window to explore the early interplay between these two systems. In this study, we compared actigraphy-derived measures of nighttime sleep onset and consolidation to cumulative exposure to cortisol, as measured by hair cortisol, and daily cortisol regulation, as measured by salivary cortisol. First, to understand the overall relation between sleep and chronic cortisol function, we assessed whether average sleep onset and nighttime sleep duration related to cumulative exposure to cortisol. Based on previous research (Flom et al., 2017), we hypothesized that later nighttime sleep onset and shorter nighttime sleep duration would relate to higher hair cortisol. Given that both poor sleep characteristics and dysregulated cortisol function are linked to lower household income (Flom et al., 2017; Mezick et al., 2008; Patel, Grandner, Xie, Branas, Gooneratne, 2010, Vaghri et al., 2013; Vliegenthart et al., 2016) and greater exposure to chaos at home (Boles et al., 2017; Doom et al., 2018; Lumeng et al., 2014), these environmental factors were considered as covariates. Additionally, given that sleep and cortisol vary daily, to disentangle the temporal associations between daily sleep and diurnal cortisol during infancy, we examined whether sleep onset and nighttime sleep duration have time-based associations with indices of diurnal cortisol rhythm. Specifically, we assessed how nighttime sleep onset and duration on a given night related to total cortisol exposure over the next day, and how bedtime cortisol on a particular night was associated with nighttime sleep onset and duration the same night. Based on literature showing that later nighttime sleep onset and shorter duration related to dysregulated diurnal cortisol indices (Flom et al., 2017; Gribbin et. al, 2012; Stalder et al., 2013), we hypothesized that later nighttime sleep onset and shorter nighttime sleep duration on a given night would relate to greater exposure to cortisol the next day, and greater bedtime cortisol on a given day would relate to later nighttime sleep onset and shorter nighttime sleep duration the same night.

METHODS

Participants

We recruited ninety parent-infant dyads from the greater Boston metropolitan area through a departmental database, online advertising, publicly available state birth records, and community recruitment events. Four of the infants did not have usable hair nor salivary cortisol data and were excluded from the analyses. This resulted in a final sample of 86 healthy, singleton one-year-olds (43 female, M_{age}=12.24 months, SD=0.82) with no known hearing, visual, neurological or developmental disorders and their parents (N=86, 80 female). Infants were accompanied by the parent with whom they spent most of their time. All infants were full-term and born at 37 weeks of gestation or later. Infants taking oral steroid medications or using topical steroid creams on the scalp were ineligible due to potential effects on cortisol levels. Demographic information is provided in Table 1. The number of participants with data for each variable is presented in Table 2.

Table1

Demographic Characteristics of Participating Families

Variables	
Infant race	
Caucasian	62.2%
Black or African American	13.3%
Asian	7.8%
Native American	4.4%
Multiracial	12.2%
Maternal Education (with at least 4 year college degree)	79.9%
Paternal Education (with at least 4 year college degree)	67.4%

Maternal occupation prestige (1-5 scale; 1 requiring less to no preparation))	
M(SD)	3.52(1.07)
Paternal Occupational prestige (1-5 scale; 1 requiring less to no preparation)	
M(SD)	3.41(1.16)
Economically Strained Participants (ITN =<3)	42.4%

Table 2

Descriptive Statistics of All the Variables

Variables	*n*	*M (SD)*
Raw Cortisol		
HCC (pg/mg)	86	301.16(1111.25)
Average AUCg (µg/dl/hr)	70	157.81 (189.33)
Average bedtime SCC (µg/dl)	76	0.22 (0.47)
Sleep		
Average Nighttime Sleep Onset (hours:minutes)	81	21:12 (01:29)
Average Nighttime Sleep Duration (hours)	81	9.83 (1.16)
ITN	85	4.09 (2.89)
Household Chaos	85	2.04 (0.57)

Procedure

This study was approved by Boston University's Institutional Review Board. Parent-infant dyads visited the laboratory for 1.5 hours. Following informed parental consent, parents were trained on home salivary sample collection procedures and how to put the actigraphs on their infants before bedtime. Hair samples were collected from the infants in the laboratory. Parents completed questionnaires on infant sleep environment, home environment, infant hair habits and family demographics. Additional behavioral measures were also collected as a part of a larger study, but were not included in this study. Upon completion of three days of diurnal salivary cortisol collection and actigraph recording at home by the parents from their infants, cortisol samples and actigraphs were picked up.

Measures

Hair cortisol. Hair cortisol measurement procedures followed our validated methods (Davenport, Tiefenbacher, Lutz, Novak, & Meyer, 2006; Meyer, Novak, Hamel, & Rosenberg, 2014). Hair samples were collected from the infants and were then assayed to determine hair cortisol concentrations (HCC) indexing cumulative exposure to cortisol. Samples of 3 cm length from the scalp and weighing 15–30 mg were cut from the posterior vertex of the head. Because washing may affect HCC (Hoffman, Karban, Benitez, Goodteacher, & Laudenslager, 2014), parents were asked about how frequently infants got their hair got wet. Human scalp hair grows at approximately 1 cm per month (LeBeau, Montgomery, & Brewer, 2011), so the 3 cm sample indexed cortisol output over the past several months prior to the lab visit. Hair samples were stored in plastic tubes labeled with subject ID, and were frozen at −20°C until cortisol analysis. Hair samples were weighted, washed twice with isopropanol to remove contaminants, dried, and ground into a fine powder. Cortisol was extracted into methanol, which was then evaporated, and the residue was reconstituted in assay buffer. Reconstituted extracts were analyzed for cortisol using a sensitive and selective commercially available enzyme immunoassay (Salimetrics, LLC; Carlsbad, CA). Assay readout was converted to pg cortisol per mg of dry hair weight. The average intra-assay and inter-essay coefficients of variation were 3.3% and 10.8% respectively. Raw HCC levels (*Mdn* = 41.10, *M* = 301.16, *SD* = 1111.25) were natural log-transformed because the data were not normally distributed. Most participants provided useable HCC values (92%). Of those without useable HCC, four infants had biologically implausible HCC values (greater than 1500 pg/mg), two used topical or oral steroid medication within the three months prior to the lab visit, and one did not provide a hair sample. HCC was unrelated to frequency of washing, $rs(83) = 0.085$, $p=0.451$. Therefore, it was not necessary to correct for variation frequency of hair washing.

Diurnal salivary cortisol. Parents were instructed to place a synthetic stick in the infant's mouth for a total of 60 seconds (Salimetrics, State College, PA). They were asked to collect saliva samples at waking, early afternoon, and bedtime across three days when the parents spent most of their day with the infants, and saliva samplings days were scheduled during the lab visit. We provided parents with a home kit including all collection materials, instructions, and a home diary to record information about time of sampling, bed and wake times, feeding and napping, and other factors that can affect cortisol levels. The experimenter was available via text to respond to parents' questions at the time of saliva sample collection. Parents were instructed not to collect saliva samples if infants were sick on the prescheduled saliva collection days. In such cases, we rescheduled saliva collection to a week after the onset of the sickness. Parents collected saliva samples within 3 to 87 days (*M* = 13, *SD* = 13.69) after the lab visit. Collected samples were frozen at -20° C until they were sent to Trier Laboratories in Germany for cortisol assay. Biologically plausible values for waking, afternoon and bedtime cortisol values were used to calculate area under the curve with respect to ground (AUC_g) from waking to bedtime as an estimate of diurnal cortisol exposure over the day (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003). We computed summary scores of AUC_g across three days of sampling as well as individual scores for each day of sampling. Bedtime salivary cortisol concentration (SCC) for each day of sampling was also recorded.

Actigraphy. Sleep onset and offset times were computed based on the absence of movement, as measured by actigraphy. Motion data was recorded to quantify onset and offset of sleep on the three nights preceding the collection of saliva sample. Parents were trained to attach the actigraph (Mini Mitter Actical) to the infants' right ankle before the infants went to sleep. Sleep onset times and durations were calculated from actigraphy data in MATLAB 9.2.0.556344 (MathWorks Inc., MA, USA) using the algorithms and thresholds put forth by Galland and colleagues to measure sleep-wake states in infants with actigraphy (Galland, Kennedy, Mitchell, & Taylor, 2012). The algorithms put forth by Galland and colleagues (2012) are reliable in extracting sleep onset and offset times using actigraphy, and are compatible with the actigraph used in this study. The actigraphy-based data for sleep onset and offset times were validated by parent report home diaries to ensure that the actigraphy-derived sleep offset occurred before the parent report of infant wake and that the actigraphy-derived sleep offset occurred after the parent report of infant bedtime. When participants were missing actigraphy data on all the three days or just for specific times or days, information about the onset and offset of sleep times were obtained from home diary parent reports. Statistical analyses were considered for both sleep onset and duration across individual days and as average values calculated across all three days.

Income-to-needs ratio (ITN) was calculated using household income and family composition based on the federal poverty guidelines. For example, an income-to-needs ratio of 2 indicates that the household income is twice the federal poverty line for that household size.

Household chaos was measured using the short version of the Confusion, Hubbub, and Order Scale (CHAOS). It is a widely-used six-item scale (α = .50) that assesses the level of chaos in the home environment (Matheny, Wachs, Ludwig, & Phillips, 1995). Mothers rated the extent to which they agreed with the six statements reflecting chaotic or calm and organized household environment (1= *definitely true* and 5 = *definitely untrue*). Examples of items from this scale are, "it's a real zoo in our home" and "there is usually a television turned on somewhere in our home."

Analysis Plan

Pearson's correlation analyses between average sleep characteristics and HCC were performed to assess whether sleep characteristics and cumulative exposure to cortisol were associated. It is well established from prior research that economically strained population has poor sleep quality (Mezick et al., 2008; Patel et al., 2010) and higher HCC (Vliegenthart et al., 2016). Additionally, chaotic home environment is related to delayed sleep onset (Boles et al., 2017). Thus, when ITN and household chaos correlated with either HCC or average sleep characteristics, they were included as covariates in the subsequent main analysis examining the association between average sleep characteristics and cumulative exposure to cortisol.

In order to examine whether average sleep characteristics predicted cumulative exposure to cortisol, we conducted linear regression analyses. First we regressed HCC on average nighttime sleep onset and duration to determine whether both sleep characteristics uniquely predicted HCC. In the case that household chaos and ITN correlated with either of these variables, we added them as covariates in the next model, determining whether both average nighttime sleep onset and duration uniquely predicted HCC after controlling for household chaos and ITN.

To understand whether sleep characteristics on a given day contributed to the diurnal cortisol exposure the next day, correlation analyses between sleep characteristics on a given night and AUC_g on the following day were run including data from all the three saliva collection days. Each subject had three measures of AUC_g as well as nighttime sleep onset and duration. To examine whether sleep characteristics on a given day predicted the diurnal cortisol exposure the next day beyond the influences of shared variance due to repeated measures, multilevel regression models clustered by participants were then performed using the software MPlus 7 (Los Angeles, CA) (Muthen & Muthen, 1998–2012). Missing cortisol and sleep values were accounted for by the this software using maximum likelihood estimations.

To understand whether cortisol level on a given night predicted sleep characteristics the same night, correlation analyses between bedtime SCC on a given night and sleep characteristics on the same night were run by including data from the first two saliva collection days. Each subject had two measures of bedtime SCC as well as measures of nighttime sleep onset and duration. Thus, to examine whether evening cortisol level on a given night predicted sleep characteristics the same night beyond the influences of the shared variance due to repeated measures, multilevel regression models clustered by participants were again performed.

RESULTS

Preliminary Analyses

Descriptive statistics of hair and salivary cortisol measures, nighttime sleep onset and duration, ITN and household chaos are provided in Table 2. Correlations between all the variables are presented in Table 3. As ITN and household chaos related to HCC as well as nighttime sleep onset and duration, both variables were considered as covariates in the multiple linear regression analysis examining whether average nighttime sleep onset and duration both uniquely predicted HCC.

Table 3

Correlations Among all the Variables

Measures	1	2	3	4	5	6
1. HCC	-					
2. AUC_g across 3 days	0.362^{**}	-				
3. Bedtime cortisol across 2 days	0.541^{**}	0.735^{**}	-			
4. Average nighttime sleep onset	0.484^{**}	0.226	0.246	-		
5. Average nighttime sleep duration	-0.334^{**}	-0.216	-0.091	-0.629^{**}	-	
6. ITN	-0.282^{*}	-0.169	-0.272^{*}	-0.369^{**}	0.238^{*}	-
7. Household chaos	0.236^{*}	0.363^{**}	0.277^{*}	-0.382^{**}	-0.347^{**}	-0.367^{**}

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

Average Sleep and Infant Hair Cortisol

As shown in Table 3, later average nighttime sleep onset and shorter average nighttime sleep duration were associated with greater infant HCC. To assess whether average nighttime sleep onset and nighttime sleep duration uniquely predicted HCC, we regressed HCC on both the sleep variables. A linear regression model revealed that average nighttime sleep onset uniquely predicted HCC above and beyond the effects of average nighttime sleep duration (Table 4).

In the next model, we added ITN and household chaos as covariates. The linear regression analysis with HCC regressed on average nighttime sleep onset, average nighttime sleep duration, ITN and household chaos revealed that average nighttime sleep onset predicted HCC above and beyond average nighttime sleep duration even after controlling for ITN and household chaos (Table 4). Thus, infants who went to bed later at night had higher exposure to chronic physiological stress irrespective of how long they slept, their family ITN, and the level of chaos at home.

Table 4

Linear Regression Models Predicting HCC

	HCC		
		Model 2	
Variable	Model 1 *B*	*B*	95% CI
Constant	-3.380	-1.748	[-9.228,5.733]
Average nighttime sleep onset	0.364*	0.318*	[0.072,0.562]
Average nighttime sleep duration	-0.001	-0.002	[-0.007,0.003]
ITN		-0.043	[-0.144,0.57]
Household Chaos		-0.03	[-0.524,0.464]
*R*2	0.235	0.228	
F	11.551***	4.959**	
*Changed R*2		0.007	
Changed F		6.592	

Note. In model 1, HCC was regressed on average nighttime sleep onset and average nighttime sleep duration. In model 2, HCC was regressed on average nighttime sleep onset, average nighttime sleep duration, ITN and household chaos. **p*<.01; ***p*<.001

Day-to-day Sleep and Salivary Cortisol

To examine time-based relations between sleep and cortisol, only measures collected on a daily basis were included in the model such as daily actigraphy and cortisol measures (i.e., AUC_g and bedtime SCC).

Sleep predicting cortisol. Initial correlation analyses without accounting for repeated measures of cortisol and sleep revealed that later sleep onset, $r(190) = 0.18$, p =.016, and shorter sleep duration, $r(190)$=-.19, p=.011, on a given night were associated with higher AUC_g the next day. To account for repeated measures of sleep and cortisol, further multilevel regression analyses clustered by participants were conducted. First AUC_g on a given day was regressed onto sleep onset the night before, clustered by subjects. Then AUC_g on a given day was regressed onto sleep duration the night before, clustered by subjects. Both these models yielded non-significant associations indicating that sleep onset and sleep duration on a given night did not significantly predict the level of AUC_g in infants the next day. Thus, although initial analyses revealed that later sleep onset and shorter sleep duration on a given night were associated with higher AUC_g, once we corrected for repeated measures of sleep and cortisol data, the associations was no longer statistically significant.

Cortisol predicting sleep. Initial correlation analyses without accounting for repeated measures of cortisol and sleep revealed that higher bedtime SCC on a given night was associated with later sleep onset, $r(139) = 0.22$, $p = .010$, but not shorter sleep duration on the same night. Further multilevel regression analysis, regressing sleep onset on a given night onto bedtime SCC the same night clustered by participants, was performed. The model revealed that higher bedtime cortisol on a given night significantly predicted later sleep onset the same night after accounting for repeated measures of bedtime SCC and sleep onset ($b = 0.259$, $SE = 0.107$, $p = .015$). Thus, there was a temporal relation between sleep and cortisol levels such that infants who had higher cortisol levels at bedtime tended to fall asleep later in the night.

DISCUSSION

We investigated the relation of nighttime sleep onset and duration to HCC, an index of cumulative exposure to cortisol, in 12-month-old infants. Further, we explored time-based associations between nighttime sleep and salivary cortisol. Specifically, we assessed whether nighttime sleep onset and duration on a particular evening predicted diurnal cortisol exposure the next day and whether salivary cortisol on a particular evening predicted nighttime sleep onset and duration the same night. Overall, nighttime sleep onset uniquely predicted HCC, suggesting that infants who generally fall asleep earlier at night have lower cumulative exposure to cortisol, regardless of how long they sleep through the night, their economic status, and the level of chaos they experience in their homes. Temporally, salivary cortisol levels in the evening predicted how late infants fell asleep that night, such that infants who had higher evening cortisol levels on a given night fell asleep later the same night. Evening salivary cortisol level was not related to nighttime sleep duration. What time infants fell asleep and how long they slept on a given night were not associated with the diurnal cortisol exposure the following day. Therefore, our results suggest that the directionality of the relation between daily sleep and cortisol is that evening cortisol levels predict how early or late the infants fall asleep that night, but sleep characteristics over a night do not predict the diurnal cortisol exposure in infants the next day.

Prior literature has reported an association between parent-reported infant nighttime sleep duration and HCC in 12-month-olds (Flom et al., 2017). Our study bolsters this finding by replicating the results using a relatively objective measure of nighttime sleep duration. Our findings expand on previous literature by underscoring the importance of nighttime sleep onset for cumulative exposure to cortisol. It is striking that sleep onset uniquely predicted cumulative exposure to cortisol above and beyond sleep duration, ITN and chaos at home. To our knowledge, ours is the first study to demonstrate this by using actigraphy-derived sleep measures. Based on this finding, future studies should explore whether parenting interventions targeted to help infants fall sleep earlier in the night would buffer infants from having chronically heightened levels of cortisol. This could be especially effective for infants who are at risk for higher cumulative exposure to cortisol due to a variety of adversities such as chaotic home environments and belonging to economically strained families.

Another novel finding was that there was a temporal association between evening cortisol level and nighttime sleep onset. As mentioned earlier, we know from the adult literature that diurnal cortisol reaches its minimum level in the earliest phase of sleep as the body winds down (Born & Fehm, 1998; Van Cauter & Spiegel, 1997). In line with this research, we found that lower evening cortisol level was predictive of earlier sleep onset in infants as young as 12 months old. By 12 months, infants have consolidated nighttime sleep and diurnal circadian rhythm similar to that of adults. We speculate that the functioning of the sleep-wake system and the HPA axis begin to synchronize at this age. Limited literature on time-based relations between sleep and cortisol in toddlers (Gribbin et al., 2012) and adults (Van Cauter & Spiegel, 1997) suggests that dysregulation of one system leads to dysregulation of the other system, creating a vicious cycle. Our findings, however, suggest that evening cortisol level is likely driving this cyclical relation early in development. Thus, factors that elevate evening cortisol in infants are unfavorable for their HPA axis functioning. Moreover, it can also adversely impact their sleep-wake system by contributing to delayed sleep onset. For infants who are at risk for late sleep onset, interventions could include parent training on how to eliminate potential stressors from the infants' environment – especially in the evening. Future studies should also explore whether interventions helping parents develop bedtime routines would calm the infants and lower their cortisol levels.

Although evening cortisol predicted sleep onset on the same night, it was not associated with sleep duration that night. While lower cortisol level on a given evening may facilitate early sleep onset, there could be a multitude of other factors contributing to how long the infant sleeps during the night. For example, duration and number of naps during the prior day, as well as morning environment such as onset of daylight in the room, parental or family interference and noise could all factor into how long the infant sleeps during the night. Thus, cortisol level in the evening seems to be more of an antecedent to the sleep process that ensues immediately, i.e., sleep onset.

In our study, nighttime sleep onset and duration on a particular evening did not predict diurnal cortisol exposure the following day. Previous literature on toddlers and adults examining temporal associations between sleep and cortisol suggests that sleep onset and duration may lead to dysregulated diurnal cortisol the next day (Bostock & Steptoe, 2013; Gribbin et al., 2012). However, sleep onset delay and short sleep duration were externally manipulated in these studies. While forced sleep onset delay and shortened sleep duration due to early morning work-shift may influence cortisol levels the next day, this may not be the case for our infants who did not have induced sleep disruptions. Moreover, it is possible that the sleep-wake system and the HPA axis have not completely synchronized at 12 months, and hence sleep on a given night may not predict cortisol levels the next day at this age. Additionally, salivary AUC_g is a measure of diurnal cortisol exposure throughout the day. Apart from how well the infants slept the night before, there may be a number of stressors throughout the day that added to the diurnal cortisol exposure that day.

A few limitations must be noted. Infants' experiences throughout the day, such as increased evening activity level and routine disruptions, could impact evening cortisol levels (Doom et al., 2018; Kertes & Gunnar, 2004). Hence, it is not clear whether evening cortisol mediates the impact of daily experiences on sleep onset or whether daily experiences influence both evening cortisol and sleep onset. To answer this question, it is necessary to collect data on these infant experiences that are also variable day-to-day along with actigraphy and salivary cortisol data. The present study has shown a specific, directional temporal association between evening cortisol and sleep onset the same night. However, we cannot conclude causality from our findings. Additionally, our goal of obtaining a representative infant sample and maintaining a high compliance rate presented a few limitations. First, when investigating temporal associations, it is ideal to collect data for seven to ten days to better capture the variability in cortisol levels and sleep characteristics. However, acquiring usable salivary cortisol and actigraphy data for so many days would require parents to be extremely dedicated and compliant, and could select for a certain group of parents who can meet such demands. To obtain a more representative sample and maintain a high compliance rate, we minimized the data collection burden on families by having them collect data for only three nights and three days. Second, although actigraphy is an objective assessment of sleep, polysomnography is considered the gold standard of sleep assessment. Polysomnography records multiple physiological changes that occur during sleep whereas, actigraphy only records changes in motion during sleep. However, polysomnography requires complex equipment set-up in a laboratory, creating a barrier to collect infant sleep data in naturalistic settings (Sadeh, 2015). Given that our study assessed individual differences in the relation between infant sleep and cortisol, it was important to collect sleep data from a representative sample. To increase participant compliance, and due to the feasibly of training parents on accurately placing the actigraphs on their infants at home, we used actigraphy. Third, we used actigraphs to recorded only nighttime sleep data. As our participants were 12-month-old infants, placing actigraphs on them throughout the day when they were awake would have yielded low compliance rates. Hence, we did not have actigraphy data on daytime sleep.

In conclusion, this study demonstrates that on a daily basis, infants' evening cortisol levels predict sleep onset, and average sleep onset predicts cumulative exposure to cortisol beyond the effects of sleep duration, ITN and chaos at home. Taking measures to maintain low evening cortisol levels in infants and ensuring earlier sleep onset may, therefore, be important to reduce their chronic exposure to cortisol. This, in turn, will decrease their allostatic load, ultimately diminishing their risks of long-term health problems.

CORTISOL MODERATES THE ASSOCIATIONS BETWEEN PARENT CHARACTERISTICS AND INFANT SLEEP

Sleep is a bioregulatory process that is vital for the restoration of the body and the brain (see Assefa et al., 2015). Early sleep characteristics have been linked to later sleep-wake organization (Scher et al., 2004; Tikotzky & Shaashua, 2012; Zuckerman et al., 1987), as well as to socioemotional, cognitive, behavioral and physiological developmental outcomes during childhood (Kelly, Kelly & Sacker, 2013, Mindell, Leichman, Dumod, & Sadeh 2017, Palagini et al., 2015; Sadeh 2007; Sadeh et al., 2015; Sivertsen et al., 2015; Vaughn, Elmore-Staton, Shin, & El-Sheik, 2015). This suggests that early development of sleep defines present and later sleep characteristics, subsequently contributing to a myriad of childhood developmental outcomes. Thus, promoting optimal sleep development in early childhood is crucial for later health outcomes. To do so, there is a need to first understand factors contributing to infant sleep development.

Infant sleep maturation is primarily a physiological process; however, sociocontextual factors play an integral role in its developmental trajectory (Sadeh, Tikotzky, & Scher, 2010; Tikotzky 2017). Experiences during the first year of life structurally and functionally impact the developing brain (Kolb, Gibb, & Robinson, 2003) and hence, sleep physiology (Hofer, 1976; Hofer 1994; Mrdalj et al., 2013; Palagini et al., 2015, Tiba, et al., 2004, 2008). According to a transactional model, proximal environmental influences on infant sleep include intrinsic infant factors like temperament, maturation, and physiology, and extrinsic factors such as parenting (Sadeh et al., 2010). Distal environmental factors influencing infant sleep include culture, environment and broader family characteristics. Proximal factors that are more directly linked to infant sleep and parenting characteristics seem to be of paramount importance, especially for developing healthy sleep characteristics during infancy (Sadeh et al., 2010). Infant sleep is immature at birth and progresses from irregular and fragmented sleep distributed throughout 24 hours of the day to consolidated nighttime sleep with only one to two daytime naps by 12 months of age (Anders et al., 1995; Davis et al., 2004; Sheldon, 2002). Infants lack self-regulation of sleep. Their bedtime routines and environment, and feeding habits interact with parental caregiving. To a large extent, infants also depend on their parents to cope with emotional and physiological distress. All of these factors contribute to infant sleep development (Sadeh et al., 2010). Thus, how parents help or hinder infants from forming and maintaining adult-like consolidated nighttime sleep during this foundational time could play a vital role in shaping early sleep development.

Parent Characteristics and Infant sleep

Parent characteristics that may play a role in infant sleep include, but are not limited to, bedtime parental involvement with the infant, cosleeping, and parental sensitivity. The most widely studied parent characteristic in its relation to infant sleep is bedtime parental involvement. A large body of research has shown that infants who fall asleep with greater parental involvement (i.e., being soothed actively by holding, feeding, rocking or passively through physical proximity) experience more sleep difficulties such as frequent and longer nighttime awakenings, shorter nighttime sleep duration and difficulty falling asleep at night (Anuntaseree et al. 2008; Burnham, Goodlin-Jones, Gaylor, & Anders, 2002; Morell & Cortina-Borja, 2002; Philbrook & Teti, 2016; Touchette et al., 2005). Greater parental involvement during infancy has also been linked to later child sleep problems (Sheridan et al., 2013). Furthermore, parent interventions promoting infant independence and self-soothing while falling asleep and through the night have been linked with longer and more consolidated nighttime sleep (Paul et al., 2016; Sadeh, Mindell, Luedtke, & Wiegand, 2009; Mindell et al., 2006; Kempler, Sharpe, Miller, Bartlett, 2015). Together, these studies suggest that infants with greater parental involvement during bedtime lack independence and self-soothing behaviors during sleep, which ultimately contributes to sleep difficulties. However, given that sleep is primarily a physiological process, it is also possible that infants who are physiologically dysregulated to begin with and have sleep difficulties that require greater parental involvement. Therefore, the role of parental involvement on infant sleep may vary by intrinsic infant physiological factors.

Cosleeping is another parenting characteristic that has been associated with sleep difficulties, including increased sleep disruptions and decreased total sleep in infancy and early childhood (Lozoff, Askew & Wolf, 1996; Mao, Burnham, Goodlin-Jones, Gaylor & Anders, 2004, Mindell et al., 2010). However, only a limited number of studies have examined how this relation varies by SES, race and culture. For example, Lozoff et al. (1996) found that cosleeping was linked to sleep difficulties only in White infants with low socioeconomic background and African American infants with higher socioeconomic background. Mindell et al. (2010) found that the link between cosleeping and sleep difficulties was more profound in countries with a predominantly White population, compared to countries with predominantly Asian populations. Furthermore, this link was mediated by bedtime parental involvement, as most of the infants from predominantly White countries who shared a bed with their parents fell asleep with more parental assistance. This, however, was not the case in infants from predominantly Asian countries, as almost all of them engaged in cosleeping, but, not everybody fell asleep with parental assistance. Thus, for infants from cultures in which cosleeping is not normative, cosleeping may be tapping into another underlying factor that is associated with sleep difficulties, i.e., bedtime parental involvement, rather than cosleeping alone being responsible for sleep difficulties. These nuanced findings highlight the need for more research examining the interplay between cosleeping and infant sleep, and the role of other contextual as well as intrinsic infant factors.

A vital parental characteristic that has not been examined extensively as a predictor of infant sleep is parental sensitivity. Parental sensitivity indexes how warm, appropriately responsive, emotionally available and well-attuned the mother is to her infant's needs (Ainsworth, 1978). It has been argued that parental sensitivity helps infants regulate their emotional and physiological states, including their sleep-wake organization (Sadeh et. al, 2010). However, limited research in this area has yielded mixed results. In a small sample, Scher (2001) found that maternal sensitivity evaluated during mother-infant free play interaction was not related to sleep onset and duration. Yet, other studies assessing parental sensitivity during bedtime have found that it is associated with fewer sleep disruptions (Teti, Kim, Mayer & Countermine, 2010) and extended nighttime sleep

(Philbrook & Teti, 2016), especially in younger infants. Specifically, Philbrook and Teti (2016) showed that when mothers were more emotionally available, infants were less distressed and slept for a longer duration throughout the night. Higher maternal emotional availability at bedtime, combined with less physical proximity to the mother during bedtime, was associated with infants sleeping longer through the night. Infant sleep rapidly increased with age when they experienced higher maternal emotional availability combined with fewer arousing bedtime activity. Following these findings, it has been argued that parental sensitivity (i.e., attending to emotional aspects of infant needs), specifically during bedtime rather than during free play, could play an integral role in instilling a sense of safety and helping infants regulate sleep (Philbrook & Teti, 2016; Teti et al., 2010). However, the parenting sensitivity assessment of emotional availability used in all these studies is designed to represent parents' global and general sensitivity levels (Biringen & Easterbrooks, 2012). Theoretically, parental emotional availability during bedtime should relate to emotional availability at other times. Thus, it is possible that there are other factors contributing to Scher's (2001) findings regarding null associations between parental sensitivity and infant sleep. For example, parental sensitivity was measured in the context of a pleasurable free play interaction designed to avoid any infant frustrations in this study. Putting infants to sleep, on the other hand, may create situations that frustrate infants, subsequently presenting the parents with challenges. Thus, free play may not have elicited a full range of maternal behaviors, as evident in the lack of variability present in some of Scher's (2001) parenting variables. If general parental sensitivity is measured in the context of parent-infant interactions with mild challenges presented to the parents, it could relate to infant sleep similarly to bedtime parental sensitivity.

Hypothalamic-pituitary-adrenal (HPA) Axis and Infant sleep

Sleep and the hypothalamic-pituitary-adrenal (HPA) axis are both bioregulatory systems that are immature at birth and develop in parallel during infancy (Antonini et al., 2000; Spangler, 1991). The HPA axis is a biological stress system that prepares the body to combat daily life stressors by mediating the production of the stress hormone, cortisol (Cone et al., 2002). On a daily basis, the HPA axis produces its maximum level of cortisol during the late phase of sleep through waking as the body prepares itself for the day, and its minimum level of cortisol at night during the early phase of nighttime sleep as the body winds down (Born & Fehm, 1998). This is referred to as the diurnal cortisol rhythm. Infancy is characterized by dramatic changes in both systems, including consolidation of nighttime sleep and the onset of adult-like diurnal cortisol pattern. When both sleep and HPA axis regulation are immature, they are susceptible to early environmental insults. Perturbation of one system during such a vulnerable time can maladaptively influence the maturation of the other system (McEwen, 2006a, 2006b; Van Reeth et al., 2000). Indeed, there is evidence that infant sleep difficulties characterized by shorter sleep duration and later sleep onset are associated with diurnal cortisol dysregulation (Bright Frick, Out, & Granger, 2014; Flom et al., 2017; Gribbin et al., 2012; Stalder et al., 2013).

Sleep and cortisol function are two bioregulatory processes codeveloping during infancy, and there is evidence demonstrating a link between the two systems. Given this, it is likely that proper regulation of one system could contribute to healthy functioning of the other system. Thus, the role that other factors, such as parent characteristics play in infant sleep may vary as a function of infant cortisol regulation. For instance, well-regulated cortisol in infants could reflect healthy functioning of multiple physiological processes, including sleep. Moreover, proper regulation of cortisol itself could contribute to better sleep outcomes. In such cases, infants could be less dependent on bedtime parenting, and factors such as increased bedtime parental involvement and cosleeping may not contribute to infant sleep outcomes. Dysregulation of cortisol on the other hand, may heighten infants' vulnerability to sleep difficulties. Hence, bedtime parenting may be more salient in shaping sleep in infants who are already vulnerable to sleep difficulties due to cortisol dysregulation.

Cortisol regulation could also have a role in how responsive infant sleep outcomes are to general parental sensitivity. Both sleep and cortisol are primarily physiological processes. Parental sensitivity could be a protective factor for infant sleep only when infants have healthy cortisol regulation. To gain a better understanding of the intricate relations between parent characteristics and infant sleep, it is crucial to investigate the extent to which parent characteristics facilitate or hinder infant sleep in the context of infant cortisol regulation. To our knowledge, this remains to be explored.

Current Study

Infant sleep difficulties have been linked to problematic psychological and physical health outcomes in later childhood. Given that parents play a role in infant sleep, modification of parent characteristics has been a part of sleep intervention programs.

However, the relation between parent characteristics and infant sleep is not straight forward. Sleep is a bioregulatory process closely related to other intrinsic physiological states such as cortisol function. This makes it is critical to understand how parent characteristics help or hinder infant sleep in the context of infants' cortisol regulation. Thus, the goal of this study was to understand (1) how parent characteristics (i.e. bedtime parental involvement, cosleeping, and parental sensitivity) related to relatively objective measures of sleep onset and consolidation, and (2) whether these associations varied as a function of infant cortisol regulation in 12-month-old infants. By this age, both sleep and diurnal cortisol rhythm are relatively established, and dysregulation of one system may have implications for the development of the other system. We used actigraphy-derived measures of nighttime sleep onset and consolidation, and measured daily cortisol regulation through salivary cortisol. Parent reports of bedtime parental involvement and cosleeping were also assessed. We obtained observational measures of parental sensitivity from a parent-infant interaction consisting of free play, structured play and clean-up sessions, using global ratings as well as frequency-based, fine-grained analysis of specific parenting behaviors. Prior studies have assessed parental sensitivity in one or both ways. If both assessments do not tap into the same construct of parental sensitivity, the association between parental sensitivity and infant sleep could differ depending on the sensitivity measures used. Thus, we used both measures of parental sensitivity.

To understand how various parent characteristics associated with infant sleep, we examined whether bedtime parental involvement, cosleeping, and parental sensitivity related to nighttime infant sleep onset and duration. Based on previous literature discussed, we hypothesized that limited parental involvement would relate to earlier nighttime sleep onset and longer nighttime sleep duration. Our participants were from the U. S. where cosleeping is not a predominant practice. It is possible that parents engage in cosleeping because they have infants who have sleep difficulties. Thus, we hypothesized that solitary sleeping arrangements would relate to earlier nighttime sleep onset and longer nighttime sleep duration. Based on previous findings about bedtime parental sensitivity (Philbrook & Teti, 2016; Teti et al., 2010), and our argument for why Scher (2001) might not have found a relation between parental sensitivity and infant sleep, we hypothesized that greater parental sensitivity would relate to earlier nighttime sleep onset and longer nighttime sleep duration.

Additionally, to investigate whether the relations between parent characteristics and infant sleep are strengthened or weakened by how infants regulate physiological stress, we examined whether diurnal cortisol exposure moderated the associations between parent characteristics and infant sleep. As sleep characteristics are linked to lower household income (Mezick et al., 2008; Patel et al., 2010) and higher level of chaotic home environment (Boles et al., 2017), these environmental factors were considered as covariates. Sleep is primarily a bioregulatory process closely related to cortisol function. Cortisol dysregulation could reflect problematic sleep. Moreover, cortisol dysregulation itself could add to sleep difficulties. Given this, extrinsic factors such as parenting may not contribute to sleep outcomes when infants have dysregulated cortisol. Thus, we hypothesized that associations of parent characteristics with better sleep outcomes would be strengthened by less diurnal cortisol exposure (i.e., better physiological stress regulation). If infants have more diurnal cortisol exposure, parent characteristics would not be salient enough to promote better sleep characteristics.

METHODS

Study 1 and 2 utilized the sample from the same research project.

Participants

We recruited 90 parent-infant dyads from the greater Boston metropolitan area through a departmental database, online advertising, publicly available state birth records and community recruitment events. Six of the infants did not provide usable data on sleep measures and were excluded from the analyses. This resulted in a final sample of 84 healthy, singleton one-year-olds (43 female, M_{age}=12.24 months, *SD*=0.82) with no known hearing, visual, neurological, or developmental disorders and their parents (*N*=84, 79 female). Infants were accompanied by the parent with whom they spent most of their time. All infants were full-term and born at 37 weeks of gestation or later. Infants taking oral steroid medications or using topical steroid creams on the scalp were also excluded due to potential effects on cortisol levels. Demographic information is provided in Table 5. The number of participants with data for each variable is presented in Table 7.

Table 5

Demographic Characteristics of Participating Families

Variables	
Infant race	
Caucasian	64.3%
Black or African American	10.7%
Asian	7.1%
Native American	4.8%
Multiracial	13.1%

Maternal Education (with at least 4 year college degree)	79.5%
Paternal Education (with at least 4 year college degree)	67.4%
Economically Strained Participants (ITN =< 3)	43%

Procedure

This study was approved by the university Institutional Review Board. Parent-infant dyads visited the laboratory for 1.5 hours. Following informed parental consent, parents were trained on home salivary sample collection procedures and putting the actigraphs on their infants before bedtime. Parents completed questionnaires on infant sleep environment, home environment, infant hair habits, and family demographics. Then the dyads engaged in a 12-minute interaction session. Additional behavioral measures were also collected as a part of a larger study, but were not included in this study. Upon completion of three days of diurnal salivary cortisol collection and actigraph recording by the parents from their infants, cortisol samples and actigraphs were picked up from their homes.

Measures

Actigraphy. Sleep onset and offset times were computed based on the absence of movement, as measured by actigraphy. Motion data was recorded to quantify onset and offset of sleep on the three nights preceding the collection of saliva sample. Parents were trained to attach the actigraph (Mini Mitter Actical) to the infants' right ankle before the infants went to sleep. Sleep onset times and durations were calculated from actigraphy data in MATLAB R2017a (9.2.0.556344). Sleep-wake onsets were determined based on the algorithms and thresholds put forth by Galland and colleagues (Galland et al., 2012). The algorithms were compatible with the actigraph used in this study The script output for sleep onset and offset times was validated by the home diary to ensure that the acticgraphy derived sleep offset occurred before the parent report of infant wake and that the actigraph sleep offset occurred after the parent report of infant bedtime. For participants who were partially and fully missing actigraphy data, information about the onset and offset of sleep times were obtained from home diary parent reports. Statistical analyses were performed for both sleep onset and duration across individual days and as average values calculated across all three days.

Parenting. To assess parenting, parent-infant dyads engaged in a 12-minute interaction session. The session was divided into a 5-minute free play, a 5-minute structured play, and a 2-minute clean-up task. The parents were instructed to play as they normally would throughout the interaction session. The dyads sat on the floor facing the camera. Parents were provided with a floor bumbo seat. Parents had the option to let their infants sit on the bumbo chair or on the floor. The dyads were left in the room with five age-appropriate toys for the infants for the free play. After 5 minutes of free play, the experimenter entered the room with a shape-sorter toy designed for toddlers. The experimenter instructed the parents to play with just the shape-sorter toy. All the other toys from the free play were also left in the room. This structured play was designed to challenge the parents to draw and sustain their infants' attention only to the shape-sorter toy. It was a toy that infants would easily lose their interests in because of its difficulty level and due to the presence of other age appropriate toys. Parents were also informed that the experimenter would knock on the door at the end of the 5 minutes of the structured play, and that it would signal them to clean up for the following 2 minutes. Parents were instructed to try their best to get their infants to put away the toys into a bin during clean-up. This was designed to present the parents with another set of challenges. The structured play and the clean-up task were designed to obtain variability of parenting in our sample by eliciting parental behaviors that occur naturally during challenging parent-infant interactions.

Global parenting in a dyadic context was coded from the videotaped interaction session using the sensitivity, structuring and non-intrusiveness subscales of the Emotional Availability Scale (EAS). The EAS captures the capacity of a dyad to share an emotionally healthy relationship and assess the affect and behavior of both the child and caregiver during an interaction (Biringen & Easterbrooks, 2012; Biringen, 2000; Pipp-Siegel & Biringen, 2000). EAS is well validated and has been widely used in various cultural contexts with children of ages 0 - 14 years old (Bornstein et al., 2008). The scales designed for early childhood, ages 0 - 5 years old were used in this study. The sensitivity scores are based on the parent's ability to correctly read the infant's cues and appropriately respond to them in a timely manner that is well accepted by the infant. This includes clear, accurate perceptions of emotions, responsiveness, ability to handle conflictual situations, and awareness of timing (Biringen & Easterbrooks, 2012). The structuring scores are based on the parent's ability to effectively provide guidance to their infants, leading them to succeed in tasks. The structuring score takes into consideration whether the parent uses multiple strategies to teach the infant, breaks down steps, teaches at the level of the infant's understanding, uses vocal and gestural instructions, sets appropriate limits, and provides just the right amount of , ultimately leading the infant to sustain attention and succeed in tasks (Biringen & Easterbrooks, 2012). The non-intrusiveness scores are based on the parent's ability to allow the child to lead during play, enter the interaction smoothly without interrupting or upsetting the infant, and not engaging in physical intrusion by taking toys away, moving the infant or rough handling of the infant (Biringen & Easterbrooks, 2012). Although these three subscales are assessments of parenting in a dyadic context, they do not yield dyadic scores. However, the subscales reflect parenting in a dyadic context because the ratings of how sensitive, structuring, or non-intrusive the parents are take into consideration how well-received the parent behaviors are by the infants. Higher scores on all the subscales indicate optimal sensitivity while lower scores reflect emotional detachment. Two trained and certified coders who completed the EA training program coded the videos, and 20% of the sample was double coded for reliability. Intraclass correlation coefficients (ICC) were calculated to assess inter-rater reliability. The ICC for sensitivity was 0.97, for structuring was 0.93, and for non-intrusiveness was 0.84.

Frequency-based parenting behaviors including positive affect, positive tone, affectionate touch, soothing touch, and prolonged body contact, were coded using the software Observer XT – Noldus 11.0 (Wageningen, the Netherlands). The coding scheme was adapted from Moore and colleagues' (2009) work. Positive affect was coded when parents laughed and smiled in a positive, warm, and loving manner. Positive tone was coded when parents directed speech at the infant with a "cooing" pattern of intonation, high in pitch, and exaggerated vowel sounds. Affectionate touch was coded when the parent hugged, kissed or caressed the baby to express her affection with no intention to soothe the infant. Soothing touch was coded when the parent hugged, kissed or caressed the infant to soothe the infant when they cried or were distressed or frustrated. Passive body contact was coded when the infant's body was in contact with the parent due to their body positioning. For instance, when the infant sat on the parent's lap, leaned on the parent, or rested an arm on the parent, it was coded as passive body contact.

All these parental behaviors were coded for presence or absence every 10 seconds (i.e., a score of 0 or 1 every 10 seconds). If the behaviors occurred for a part of the 10-second interval, they were coded as present. If there were multiple occurrences of a particular behavior in a single interval, the behavior was counted as 1 for the interval. The total number of 10-second intervals in which each parenting behavior occurred was calculated. Scores were computed as percentages of total valid interaction time. Trained coders coded 20% of the same sample: Inter-rater reliability ranged from 0.81 to 0.89 for positive affect, 0.81 to 0.83 for positive tone, 0.82 to 0.96 for affectionate touch, 0.87 to 1 for soothing touch and 0.82 to 0.90 for passive body contact.

Diurnal salivary cortisol. Parents were instructed to place a synthetic stick in the infant's mouth for a total of 60 seconds (Salimetrics, State College, PA). They were asked to collect saliva samples at waking, early afternoon, and bedtime across three days when the parents spent most of their day with the infants, and saliva samplings days were scheduled during the lab visit. We provided parents with a home kit including all collection materials, instructions, and a home diary to record information about time of sampling, bed and wake times, feeding and napping, and other factors that can affect cortisol levels. The experimenter was available via text to respond to parents' questions at the time of saliva sample collection. Parents were instructed not to collect saliva samples if infants were sick on the prescheduled saliva collection days. In such cases, we rescheduled saliva collection to a week after the onset of the sickness. On average, parents collected saliva samples within approximately 13 days (*SD* = 13.69 days, range = 3 to 87 days) after the lab visit. Collected samples were frozen at -20° C until they were sent to Trier Laboratories in Germany for cortisol assay. Biologically plausible values for waking, afternoon and bedtime cortisol values were used to calculate area under the curve (AUC_g) with respect to ground from waking to bedtime as an estimate of diurnal cortisol exposure over the day (Pruessner et al., 2003).

Bedtime parental involvement. Information about whether infants fell asleep in the presence of a parent was obtained from one of the items of the Brief Infant Sleep Questionnaire (BISQ; Sadeh, 2004). Bedtime parental involvement included falling asleep in bed near a parent and/or while being fed, rocked and/or held by a parent.

Cosleeping. An adapted version of the Sleep Practice Questionnaire (SPQ; Goldberg & Keller, 2007) was used to examine cosleeping which was defined by infants sharing a bed with their parents for at least a part of the night.

Income-to-needs ratio (ITN) was calculated using annual household income and family composition based on the federal poverty guidelines. For example, an ITN of 2 indicates that the household income is twice the federal poverty line for that household size.

Household chaos was measured using the short version of the Confusion, Hubbub, and Order Scale (CHAOS). It is a widely-used six-item scale (α = .50) that assesses the level of chaos in the home environment (Matheny et al., 1995). Mothers rated the extent to which they agreed with the six statements reflecting chaotic or calm and organized household environment (1= *definitely true* and 5 = *definitely untrue*). Examples of items from this scale are, “it’s a real zoo in our home” and “there is usually a television turned on somewhere in our home.”

Analysis Plan

All the variables were checked for normality before proceeding with the preliminary analyses.

Preliminary analyses. A Pearson’s chi-squared test of independence between bedtime parental involvement and cosleeping was run to determine whether they were associated. Previous literature showed that cosleeping was confounded by bedtime parental involvement when predicting sleep in a predominantly White sample (Mindell et al., 2010). Thus, when cosleeping and bedtime parental involvement were significantly associated, each variable was included as covariate in the subsequent analyses examining each of their associations with sleep characteristics.

To examine the relations among global parenting variables from EAS and among frequency-based parenting behaviors, correlation analyses were run. For data reduction and to group parental sensitivity variables within each scale into statistically meaningful components, principle component analyses (PCA) were run separately for EAS variables and frequency-based parenting variables if the variables within each scale were correlated and met the assumptions for PCA. PCA was run separately for each parenting scale because we wanted to assess whether parental sensitivity measured by global rating and frequency-based coding of specific behaviors related similarly to infant sleep.

ITN and household chaos were tested as possible covariates because it is well established from previous research that economically strained populations have poor sleep quality (Mezick et al., 2008; Patel et al., 2010) and chaotic home environment is related to delayed sleep onset (Boles et al., 2017). When these two variables correlated with average sleep characteristics, they were included as covariates in the subsequent analyses examining the associations of parenting characteristics with sleep characteristics.

Main Analyses. Parent characteristics included bedtime parental involvement, cosleeping, a global parental sensitivity component and frequency-based parental sensitivity components. Infant sleep characteristics included nighttime sleep onset and nighttime sleep duration. First, correlation analyses were performed to assess whether parent characteristics were associated with sleep characteristics. When parent characteristics were significantly related to sleep characteristics, each association was considered for subsequent analyses.

To examine whether physiological stress regulation indexed by AUC_g played a role in the relations of parent characteristics and sleep characteristics, separate moderation models for each parent characteristic as a predictor were run. In all the models, AUC_g was entered as the moderator, and ITN and household chaos were entered as covariates.

RESULTS

Preliminary Analyses

All the variables were normally distributed except salivary cortisol. Raw diurnal cortisol exposure values for each saliva sampling day were winsorized to within 3 standard deviations of the mean and then log-transformed after which the variable was normally distributed. Diurnal cortisol exposure across the three sampling days was then averaged to obtain an aggregate score. A Pearson's chi-squared test of independence revealed that most infants who engaged in cosleeping experienced more bedtime parental involvement, $\chi^2(1, n=84) = 24.5$, $p < .001$.

Parental sensitivity, structuring, and non-intrusiveness were significantly correlated with each other, as shown in Table 6. These variables were appropriate for PCA as all of them correlated with at least one other variable with *r* ranging from 0.45 to 0.71. The Kaiser-Meyer-Olkin measure of sampling adequacy was 0.64, which is above the commonly accepted value of 0.50 (Kaiser, 1970), and Bartlett's test of sphericity was significant ($\chi^2(3, n=84) = 108.49$, $p < .01$). Thus, PCA was used to extract a single component as the eigenvalue of 2.22 indicated that the first component accounted for 73.8% of the total variance. The other two components had eigen values that were less than 0.55. The first component was interpreted as a 'global parental sensitivity' component. Parental sensitivity, structuring, and non-intrusion loaded positively to this component with factor loadings of 0.92, 0.84, and 0.81 respectively. Frequency-based parenting behaviors were also determined as appropriate for PCA. Although the correlations between each variable with another variable ranged from an *r* of -0.04 to

0.26, the Kaiser-Meyer-Olkin measure of sampling adequacy was 0.51, just above the commonly accepted value of 0.50, and Bartlett's test of sphericity was significant ($\chi^2(10, n=84) = 19.10, p = .039$). In a PCA on frequency-based parenting behaviors, the eigenvalues of 1.45 and 1.19 indicated that first two components explained 29% and 23.87% of the variance. The other three components had eigen values below 0.97. The first component (4 items) included use of more positive affect and tone, and less soothing touch and passive body contact. This component accounted for 29% of the total variance. Positive affect and tone loaded positively to this component with factor loadings of 0.78 and 0.46, respectively. Soothing touch and passive body contact loaded negatively to this component with factor loadings of -0.46 and -0.62, respectively. The component was interpreted as positivity shown by the parent and will be referred to as 'parental positivity' from here onwards. The other component (4 items) included greater use of affectionate touch, soothing touch, passive body contact, and positive tone, and it accounted for 23.87% of the total variance. Affectionate touch, soothing touch, passive body contact, and positive tone all loaded positively to this component with factor loadings of 0.48, 0.58, 0.36 and 0.69, respectively. The component was interpreted as sensory stimulations provided by the parents and will be referred to as 'sensory stimulatory behaviors' from here onwards.

Table 6

Correlations between Global and Frequency-based Parenting Variables

Measures	1	2	3	4	5	6	7
1. Sensitivity (EAS)	-						
2. Structuring (EAS)	0.708^{**}	-					

3. Non-intrusiveness (EAS)	0.669**	0.447*	-				
4. Positive affect	0.351*	0.257*	0.222*	-			
5. Positive tone	0.345*	0.330**	0.227*	0.244*	-		
6. Affectionate touch	0.110	0.111	0.072	0.068	0.137	-	
7. Soothing touch	-0.076	-0.103	-0.110	-0.179	0.095	-0.001	-
8. Passive body contact	-0.339**	-0.228*	-0.406**	-0.194	-0.015	0.011	0.201

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

Descriptive statistics of the parent characteristics, sleep characteristics, ITN, household chaos are provided in Table 7.

Table 7

Descriptive Statistics of All the Variables

Variables	*n*	*M (SD)*
Sensitivity score (EAS)	84	5.01 (1.19)
Structuring score (EAS)	84	4.81 (1.32)
Non-intrusiveness score (EAS)	84	4.95 (1.47)
Positive affect (%)	84	41.27(22.53)
Positive tone (%)	84	56.26(27.06)
Affectionate touch (%)	84	2.40(2.90)
Soothing touch (%)	84	1.79(3.17)
Passive body contact (%)	84	21.78(19.37)
Sleep		
Average Nighttime Sleep Onset (hours:minutes)	84	21:12 (01:29)
Average Nighttime Sleep Duration (hours)	84	9.82 (1.12)
Raw Cortisol		
AUC_g (μg/dl/hr)	70	219.07 (205.63)
ITN	79	4.01 (2.84)
Household Chaos	83	2.06 (0.58)
Sleep Environment		Percentage (%)
Cosleepers	81	48.1

Bedtime parental presence	82	59.8

Note. Descriptive statistics for all the parental sensitivity variables are provided in this table. However, principal components of EAS and frequency-based parenting variables were used in the main analyses. EAS scores ranged from 1 to 7. Frequency-based parenting behaviors reflect percentage of 10-second intervals with the parenting behavior present.

Main Analyses

Independent samples *t*-tests indicated that infants with bedtime parental involvement had later sleep onset (M = 21:41 hh:mm, SD = 1:31 hh:mm) compared to those who did not have bedtime parental involvement (M = 20:28 hh:mm, SD = 1:08 hh:mm), $t(82) = -3.884$, Cohen's $d = -0.90$, $p < .001$. Infants with bedtime parental involvement had shorter nighttime sleep duration (M = 9.59 hours, SD = 1.23) compared to those who did not have bedtime parental involvement (M = 10.13 hours SD = 0.87), $t(82) = 2.188$, Cohen's $d = -0.61$, $p = .032$. Independent samples *t*-tests also indicated that infants who engaged in cosleeping had later sleep onset (M = 21:45 hh:mm, SD = 1:35 hh:mm) compared to those who were solitary sleepers (M = 20:44 hh:mm, SD = 1:14 hh:mm), $t(81) = -3.235$, Cohen's $d = -0.72$, $p = .002$. Cosleeping did not significantly associate with nighttime sleep duration.

Pearson's correlations between all the variables are presented in Table 8. Higher score on global parental sensitivity was significantly correlated with earlier nighttime sleep onset, but not nighttime sleep duration. Higher parental positivity was significantly associated with earlier nighttime sleep onset and longer nighttime sleep duration. Sensory stimulatory behavior, however, was unrelated to both sleep characteristics. All the significant associations were followed up with six individual moderation models examining whether diurnal cortisol exposure across the day moderated the relations between parent characteristics and sleep characteristics. As ITN and household chaos related to nighttime sleep onset and duration (see Table 8), both variables were considered as covariates in all the six moderation analyses.

Table 8

Correlations between All the Variables

Measures	1	2	3	4	5	6	7
1. Nighttime sleep onset	-						
2. Nighttime sleep duration	-0.624**	-					
3. Global parental sensitivity	-0.258*	0.115	-				
4. Parental positivity	-0.232*	0.286**	0.500**	-			
5. Sensory stimulatory behaviors	-0.131	0.130	0.113	-0.005	-		
6. Diurnal cortisol AUC_g across 3 days	0.224	-0.230	-0.006	-0.040	-0.104	-	
7. ITN.	-0.369**	0.240*	0.273*	0.229*	0.290**	-0.169	-
8. Household chaos	0.394**	-0.377**	-0.087	0.009	-0.160	-0.363**	0.372**

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

Predicting nighttime sleep onset. First, we examined whether diurnal cortisol exposure moderated the relation between bedtime parental involvement and nighttime sleep onset after controlling for cosleeping, ITN, and household chaos. In an OLS regression analysis, nighttime sleep onset was regressed on bedtime parental involvement, diurnal cortisol exposure and their interaction term. Cosleeping, ITN and household chaos were entered as covariates. The model was statistically significant (R^2 = 0.27, F (6, 54) = 3.37, p = .007), and revealed no significant main effects. However, there was a significant interaction between bedtime parental involvement and diurnal cortisol exposure in predicting nighttime sleep onset, with bias corrected bootstrapped confidence intervals at 95% level based on 5000 samples, CI: 0.1050–1.6772. Inclusion of this interaction effect significantly improved the model (R^2 change = 0.07 F(1, 54) = 5.16, p = .0271), indicating that diurnal cortisol exposure moderated the association between bedtime parental involvement and nighttime sleep onset. Follow-up analyses revealed that infants who were exposed to bedtime parental involvement fell asleep later at night only if they had dysregulated cortisol levels, indexed by higher diurnal cortisol exposure. Parental involvement during bedtime did not significantly predict later sleep onset for infants who had well-regulated cortisol levels as indexed by lower diurnal cortisol exposure.

Next, we examined whether diurnal cortisol exposure moderated the relation between cosleeping and nighttime sleep onset after controlling for bedtime parental involvement, ITN, and household chaos. In another OLS regression analysis, nighttime sleep onset was regressed on cosleeping, diurnal cortisol exposure and their interaction term. Bedtime parental involvement, ITN and household chaos were entered as covariates. Although this model was statistically significant (R^2 = 0.25, F (6, 54) = 3.01, p = .013), there were no significant main effects nor interaction effects. Thus, cosleeping did not predict sleep onset after controlling for bedtime parental involvement, ITN and household chaos, and diurnal cortisol exposure did not interact with cosleeping to predict sleep onset.

Then, we examined whether diurnal cortisol exposure moderated the relation between global parenting sensitivity and nighttime sleep onset after controlling for ITN and household chaos. In an ordinary least squares (OLS) regression analysis conducted using the PROCESS macro in SPSS (Hayes, 2013), nighttime sleep onset was regressed on global parental sensitivity, diurnal cortisol exposure, and their interaction term. ITN and household chaos were entered as covariates. The overall model was statistically significant ($R^2 = 0.26$, F (5, 58) = 5.84, $p = .003$), but revealed no significant main effects. There was a significant interaction between global parental sensitivity and diurnal cortisol exposure in predicting nighttime sleep onset, with bias corrected bootstrapped confidence intervals at 95% level based on 5000 samples, CI: 0.1503–0.8407. Inclusion of this interaction effect significantly improved the model (R^2 change = 0.11 $F(1, 58)$ = 8.26, $p = .0057$), indicating that diurnal cortisol exposure moderated the association between global parental sensitivity and nighttime sleep onset. Follow-up analyses revealed that infants who experienced greater global parental sensitivity fell asleep earlier at night only if the infants had well-regulated cortisol levels indexed by lower diurnal cortisol exposure. Having globally sensitive parents did not predict earlier sleep onset for infants who had dysregulated cortisol levels indexed by higher diurnal cortisol exposure.

We also examined whether diurnal cortisol exposure moderated the relation between parental positivity and nighttime sleep onset after controlling for ITN and household chaos. In another OLS regression analysis, nighttime sleep onset was regressed on parental positivity, diurnal cortisol exposure, and their interaction term. ITN and household chaos were entered as covariates. This model was not statistically significant.

Predicting nighttime sleep duration. We examined whether diurnal cortisol exposure moderated the relation between bedtime parental involvement and nighttime sleep duration after controlling for ITN and household chaos. In an OLS regression analysis, nighttime sleep duration was regressed on bedtime parental involvement, diurnal cortisol exposure, and their interaction term. ITN and household chaos were entered as covariates. The model was statistically significant ($R^2 = 0.44$, $F\ (5, 57) = 2.66$, $p = .032$) and revealed only one significant main effect of household chaos (B = -0.69, $p = .009$), indicating that infants living in homes with greater levels of chaos sleep for a shorter duration at night. There was no significant interaction between bedtime parental involvement and diurnal cortisol exposure in predicting nighttime sleep duration. Thus, while more bedtime parental involvement initially related to shorter nighttime sleep duration, it was not a significant predictor after controlling for household chaos.

Finally, we examined whether diurnal cortisol exposure moderated the relation between parental positivity and nighttime sleep duration after controlling for ITN and household chaos. In an OLS regression analysis, nighttime sleep duration was regressed on parental positivity, diurnal cortisol exposure, and their interaction term. ITN and household chaos were entered as covariates. The model was statistically significant ($R^2 = 0.46$, $F\ (5, 58) = 3.09$, $p = .015$) and revealed only one significant main effect of household chaos (B = -0.61, $p = .014$). There was no significant interaction between parental positivity and diurnal cortisol exposure in predicting nighttime sleep duration.

Thus, although less parental positivity initially related to shorter nighttime sleep duration, it was not a significant predictor after controlling for household chaos.

DISCUSSION

We explored the relations between parent characteristics and infant sleep, and whether cortisol function modulated these associations in 12-month-old infants. Parent characteristics included bedtime parental involvement, cosleeping, global parental sensitivity, parental positivity, and parental sensory stimulatory behaviors. Infant sleep characteristics assessed were nighttime sleep onset and duration. Cortisol function was indexed by diurnal cortisol exposure across the day (i.e., AUC_g). First, we assessed predictors of infant sleep onset. Infants with minimal bedtime parental assistance fell asleep earlier at night only if they had dysregulated cortisol levels. On the other hand, infants with globally sensitive parents fell asleep earlier at night only if they had well-regulated cortisol levels. Next, we examined predictors of infant sleep duration. Infants with minimal bedtime parental assistance, and greater exposure to parental positivity slept longer through the night. Yet, these associations did not differ by infant cortisol function. Our study adds to the existing literature by revealing the moderation effect that cortisol regulation has on how parent characteristics are linked to infant sleep outcomes. It illuminates how parent characteristics interact with one measure of infant physiological reactivity to predict infant sleep outcomes. These findings have implications for identifying infants whose sleep outcomes may be the most responsive to parenting interventions targeted at improving infant sleep outcomes.

In agreement with previous studies, we found that limited bedtime parental involvement was optimal for healthy infant sleep characteristics. We extend the prior literature by making the important distinction that this association between bedtime parental involvement and infant sleep does not necessarily hold true for all infants when it comes to falling asleep at night. Minimal parental assistance during bedtime was linked to earlier nighttime sleep onset only for infants who had increased cortisol exposure across the day. This suggests that limited bedtime parental involvement could buffer sleep initiation difficulties only for infants with dysregulated cortisol. For infants who had well-regulated cortisol, the amount of parental assistance they received during bedtime did not predict how early they fell asleep. Prior literature has suggested that parents should refrain from soothing their infants during bedtime in order to foster independence and self-regulated sleep in their infants. Based on our findings, this recommendation would not apply to parents who have infants with optimal physiological stress regulation.

Bedtime parental involvement and cosleeping were significantly associated within our diverse U.S. sample. We wanted to investigate whether each of these parent characteristics made independent contributions to infant sleep onset. It is noteworthy that among infants with dysregulated cortisol, minimal bedtime parental involvement was related to falling asleep earlier at night even after controlling for family income, household chaos, and especially cosleeping. On the other hand, the converse was not true. Cortisol dysregulation did not strengthen the association between solitary sleeping arrangements and falling asleep earlier at night after accounting for bedtime parental involvement, family income, and household chaos. Together, these findings illuminate the fact that it is limited bedtime parental involvement, and not solitary sleeping, that contributes to earlier nighttime sleep onset in this sample. Moreover, our initial analyses had revealed that solitary sleepings related to earlier sleep onset before we investigated the moderating role of cortisol regulation, accounting for bedtime parental presence. The initial association between solitary sleeping and earlier sleep onset can be attributed to the possibility that cosleeping in our sample could have been "reactive". Reactive cosleeping refers to parents sharing a bed with their infants in response to infant sleep, whereas intentional cosleeping is a sleeping arrangement voluntarily chosen by the parents (Sadeh et al., 2010; Tikotzky, 2017). Cosleeping might have been reactive in our sample for a few reasons. First, cosleeping is not the predominant sleeping culture in the U.S. When cosleeping did occur, it might have been a response to soothe an infant with sleep difficulties. Second, greater bedtime parental involvement is seen particularly in infants with sleep difficulties. The fact that bedtime parental involvement frequently occurred with cosleeping in our sample suggests that parents might have allowed their infants to share a bed with them in response to infant sleep difficulties. Given that cosleeping might have been mostly reactive in our sample, and that cosleeping and infant sleep onset were not associated after accounting for bedtime parental involvement, our findings lend support to the speculation that non-reactive cosleeping is not problematic for infant sleep outcomes.

Although prior findings regarding the role of parental sensitivity on infant sleep outcomes have been inconsistent, our data revealed that infants exposed to global parental sensitivity and parental positivity fell asleep earlier at night. A reason for this finding could be that our parent-infant interaction session challenged the parents, eliciting variability in their behaviors and sensitivity levels. The level of challenge presented to the parents in this context could have been similar to that faced by the parents when putting their infants to sleep. Parental sensitivity during bedtime has indeed been linked to better sleep outcomes (Philbrook & Teti, 2016; Teti et al., 2010). Furthermore, our sample was also relatively larger and more diverse than samples in studies that previously found no relation between parental sensitivity and infant sleep (e.g. Scher, 2001). This could have contributed to the variability in parental behaviors, sensitivity levels, and infant sleep onset. Another explanation for this finding could be that we used PCAs to come up with aggregate scores for (1) the global ratings of parental sensitivity, and (2) fine-grained analyses of specific parenting behaviors. This was not always the approach taken in prior studies.

To our knowledge, the interaction between parental sensitivity and cortisol regulation in predicting infant sleep has not been previously examined. It is quite striking that global parental sensitivity was associated with earlier nighttime sleep onset only for infants with well-regulated cortisol. For infants with dysregulated cortisol, global parental sensitivity did not relate to nighttime sleep onset. The moderation of cortisol regulation was significant even after controlling for family income and household chaos, so our result was not confounded by these factors. The novel finding emphasizes that exposure to increased global parental sensitivity is not related to earlier nighttime sleep onset for all infants. This could be one of the reasons for the inconsistencies found in the prior literature about whether parental sensitivity related to infant sleep. Prior studies have not assessed cortisol regulation as a moderator between parental sensitivity and infant sleep, and it is a possibility that infants with dysregulated cortisol in their samples contributed to the null associations found between parental sensitivity and infant sleep (e.g. Scher, 2001). Parental sensitivity is integral for the development of self-regulation in general (Bernier, Carlson, & Whipple, 2010), and hence essential for self-regulation of sleep (Sadeh et al., 2010). It has been thought to promote a sense of safety in infants making them less vigilant during bedtime and helping them with sleep initiation (Dahl, 1996). Theoretically then, parental sensitivity would buffer infant sleep dysregulation. Our finding supports this notion, and expands on this theory by suggesting that infant sleep may be responsive to the buffering effect of parental sensitivity only when infants have well-regulated physiological stress systems. Given that cortisol dysregulation is intertwined with maladaptive sleep, for infants with increased cortisol levels throughout the day, cortisol dysregulation might dampen the buffering role of parental sensitivity on sleep difficulties. Thus, parental sensitivity may have little effect on infant sleep when infants have dysregulated cortisol.

Our findings illustrate the complexities of how distinct infant sleep characteristics are differentially receptive to specific measures of parental sensitivity. While global parental sensitivity only predicted nighttime sleep onset, parental positivity related to both nighttime sleep onset and duration. Moreover, parental sensory stimulations did not relate to any sleep characteristic. The nature of each of these parenting measures and the core construct that they represent could help explain these differential associations. First, we assessed global parental sensitivity using the EAS. EAS takes into account parents' levels of emotional availability tailored to the needs of their infants in an interaction session. Emotional availability has been thought to instill a sense of security in infants helping them to feel safe and initiate sleep during bedtime (Dahl, 1996; Teti et al., 2010). This might explain why global parental sensitivity measured by EAS predicted nighttime sleep onset, but not nighttime sleep duration. General emotional availability of the parent could be more salient in helping infants feel secure at night and fall asleep in a timely manner, but it may not be as relevant for how long the infants stay asleep at night. Second, the parental positivity factor included parents using more positive affect and vocalization, and less soothing touch and passive body contact. This measure was aggregated from fine-grained assessment of specific parental behaviors and related to both the sleep characteristics. More parental soothing during bedtime has been linked to sleep difficulties in infants (Morrell & Cortina-Borja, 2002; Sadeh et al., 2010; Tikotzky, 2017). It is believed that such parental engagement could promote infants' reliance on the parents, preventing infants from self-regulating sleep. We speculate that parents who generally engaged in more soothing touch and passive body contact with the infant in our sample could have discouraged infants from independently regulating sleep at night. On the other hand, positive affect and vocalization are emotional elements of maternal sensitivity that could foster a sense of security in the infants, facilitating sleep. Thus, parents who displayed more positive affect and vocalization, and engaged in less soothing touch and passive body contact could have facilitated both infant sleep outcomes. Third, parental sensory stimulations it did not relate to any sleep outcome suggesting that sensory stimulatory behaviors may not be important for infant sleep.

Together, these findings indicate that global parental sensitivity, parental positivity and parental sensory stimulations are tapping into separate constructs and contribute differentially to infant sleep characteristics.

We found that exposure to household chaos uniquely predicted the duration of nighttime sleep in infants. Although minimal bedtime parental assistance and greater parental positivity initially related to longer nighttime sleep duration in our study, the two associations were not significant in subsequent models including cortisol regulation as a moderator, and family income, and household chaos as covariates. Only minimal exposure to household chaos significantly predicted longer sleep duration in both the models. It is striking that household chaos predicted nighttime sleep duration overpowering the effects of bedtime parental involvement, parental positivity and family income. This elucidates the robust predictive value of household chaos for nighttime sleep duration, and adds to the findings of previous literature on household chaos and sleep (e.g., Boles et al., 2017). Although we assessed general exposure to household chaos, a chaotic environment during bedtime and waking may especially play a role in predicting nighttime sleep duration. It is possible that infants in chaotic home environments are subjected to disruptions in bedtime and morning routines, more noise and distractions. Thus, they may have difficulty falling asleep at night due to bedtime chaos and could wake up earlier in the morning due to noise or disruptions, cumulatively contributing to reduced nighttime sleep duration. Another possibility is that infants who are generally exposed to chaotic home environments throughout the day are generally more aroused. This could contribute to infants' difficulty falling asleep and/or staying asleep through the night. Future studies investigating the antecedents of infant sleep difficulties should assess exposure to chaos specifically in the morning and during bedtime as a predictor or a covariate of infant sleep. Studies should also explore bedtime and morning routine disruptions, noise levels and distractions, and overall infant arousal as mechanisms underlying the link between household chaos and infant sleep duration.

Infant physiological regulation interacted with parental characteristics when predicting nighttime sleep onset, but not nighttime sleep duration. Although limited bedtime parental assistance and greater parental positivity initially associated with longer nighttime sleep duration, these relations did not vary as a function of infant cortisol regulation. Our measure of cortisol regulation was diurnal cortisol exposure across the day. This measure of cortisol interacted with parent characteristics to predict when infants fell asleep at night, yet it might not have been the best measure of cortisol regulation interacting with parent characteristics when predicting infant nighttime sleep duration. For example, nocturnal cortisol exposure also contributes to nighttime sleep duration in adults. Adults with higher nocturnal cortisol exposure sleep for a shorter duration at night (Späth-Schwalbe, Schöller, Kern, Fehm, & Born, 1992). Nocturnal exposure to cortisol could be a better candidate than total cortisol exposure across the day as a moderator for the association between parent characteristics and infant nighttime sleep duration. Unfortunately, we did not collect data on nocturnal cortisol exposure. When examining cortisol regulation as a potential moderator in the association between parental characteristics and infant sleep, future research should carefully select and discern indices of cortisol dysregulation that are relevant for the specific measure of infant sleep in question.

Although the present study revealed novel findings and extend our knowledge about the complex relations between parent characteristics and infant sleep, the study is not without its limitations. First, our sample consisted of a diverse U.S. population, but the number of participants in each racial group was not large. As a result, we were unable to examine whether cosleeping related differentially to infant sleep onset in a variety of American households. Second, we did not collect data on bedtime parental sensitivity. Future studies should compare parental sensitivity in an interaction session and during bedtime in the same sample to determine whether both measures tap into the same construct, and whether they relate similarly to infant sleep outcomes. Third, the findings of this study are correlational. The directionality of whether parent characteristics shape infant sleep or whether infant sleep outcomes elicit particular parent characteristics, is not clear. Transactional models suggest that the relation is likely bidirectional. Prior intervention studies examining the role of bedtime parental involvement and infant sleep suggest a causal relationship (see Camerota. et al., 2019). However, more work is needed to determine the directionality of the relation between parental sensitivity and infant sleep. Temporal assessments between parental sensitivity and infant sleep could shed light on the directionality of their relation. Fourth, our study used actigraphy to assess infant sleep. When actigraphy data was missing, we used data from sleep diaries. Although actigraphy is an objective assessment of sleep, polysomnography is considered the gold standard of sleep assessment. Polysomnography records multiple physiological changes that occur during sleep, whereas actigraphy only records changes in motion during sleep. However, polysomnography requires complex equipment set-up in a laboratory, creating a barrier to collect infant sleep data in naturalistic settings (Sadeh, 2015). Given that our study assessed individual differences in the relation between infant sleep and its predictors, it was important to collect sleep data from a representative sample. To increase participant compliance with the sleep data collection protocol, we used actigraphy. It was feasible to train parents on accurately placing the actigraphs on their infants before the infants went to bed at home. Finally, our study uncovered how cortisol function interacts with parent characteristics to predict infant sleep onset. However, cortisol function is only one of many other bioregulatory systems intertwined with sleep. Future studies should examine the moderating role of other physiological systems in the relation between parent characteristics and infant sleep.

Sleep and physiological stress systems are intertwined and relatively mature by 12 months of age. The present study demonstrates how regulation of one system begins to play a part in the regulation of the other system by interacting with parent characteristics towards the end of the first year of life. Our findings substantiate the idea that whether or not a specific sleep characteristic is sensitive to extrinsic factors, such as parent characteristics may depend on other intrinsic infant physiological regulatory systems connected to sleep. Furthermore, this study implies that when infants have dysregulated physiological stress systems, a parent characteristic thought to help infant sleep regulation (i.e., parental sensitivity) may be of little importance for infant sleep. In contrast, infants with dysregulated cortisol are particularly vulnerable to sleep difficulties when they are exposed to a parenting factor known to impede sleep regulation (i.e., bedtime parental involvement). This has implications for identifying infants who are most likely to benefit from specific types of parenting interventions targeted to foster better infant sleep regulation.

Apart from intrinsic physiological regulation in infants, it is also important to take cultural contexts into account when interpreting the role of parent characteristics on infant sleep outcomes. For example, cosleeping is often associated with problematic sleep in infants. In our American sample though, it is clear that cosleeping is often accompanied by increased bedtime parental assistance. It is actually bedtime parental presence that is associated with sleep difficulty, and not cosleeping. Thus, our results underscore the need for comprehensive research on how parent characteristics relate to infant sleep in culturally diverse samples. Examining how parent characteristics differentially predict infant sleep in the presence of intrinsic physiological as well as extrinsic cultural contexts will bring us a step closer to fully understanding the role of parenting in infant sleep. Ultimately, it will help promote optimal development of infant sleep and cortisol regulation, both of which have implications for better mental and physiological health outcomes

CONSISTENCY IN MATERNAL AFFECT AND POSITIVE VOCALIZATION OVER THE FIRST YEAR OF LIFE

Early quality parenting is vital for positive infant development across multiple domains including physiological, cognitive, language, and socioemotional development (e.g. Jahromi, Putnum & Stifter, 2004, Landry, Smith, & Swank, 2006, Propper & Moore, 2006, Saint-Georges et al., 2013). Maternal sensitivity, a key characteristic of quality parenting, indexes how warm, appropriately responsive and well-attuned the mother is to her infant (Ainsworth, 1978). Mothers are relatively consistent with maternal sensitivity throughout early development (Belsky, Gilstrap, & Rovine, 1984; Bornstein & Tamis-Le-Monda, 1990; Dallaire & Weinraub, 2005, Else-Quest, Clark, & Owen, 2011, Madigan, Plamondon, Browne, & Jenkins, 2016). Consistency, also called relative or individual-order stability, in parenting refers to the degree to which parents maintain their positions relative to other parents over time on measures of parenting (Forehand & Jones, 2002). Although the link between early parenting and infant development has been widely studied, minimal research has examined the role of parenting consistency on infant development.

Emerging evidence suggests that not only the quality of parenting but also consistency in parenting during infancy has concurrent and lasting positive developmental outcomes. When parents are consistent with the level of parenting sensitivity they engage in, infants are known to have better social and cognitive outcomes (Landry et al., 2001) that last through middle childhood irrespective of later parenting (Landry, Smith, & Swank, 2003). Consistency in parenting sensitivity is crucial during infancy as it instills a sense of predictability. Infancy is a period of rapid development when caregivers play a foundational role in molding many aspects of their present and later development (e.g. Ainsworth, Blehar, Waters, & Wall, 1978; Bornstein, 1985, Hostinar et al., 2014). Typically, mothers are the first primary caregivers whom infants bond with and heavily depend on (Hostinar et al., 2014). Unpredictability of maternal behaviors during such a time is an early stressor leading to adverse health outcomes (Lupien et al., 2007). Hence, inconsistent parenting during infancy could be detrimental for behavioral and physiological health. Predictable parenting can facilitate proper development of processes such as attachment formation, social referencing, joint attention and self-regulation which emerge during the first year of life. These processes are precursors of socioemotional and cognitive development. Perhaps consistency in parenting infants is essential for these areas of development. It is then important to examine contributors of variability in consistency in early parenting to help identify infants at risk for poor developmental outcomes.

Children living in poverty are at risk for insensitive caregiving (Bornstein et al., 2014) and are likely experiencing inconsistent parenting. Parenting infants, especially in the first year of life, is a turbulent time for mothers who are vulnerable to financial strain (Bornstein et al., 2014; Feldman, 2007) rendering it difficult for them to consistently display certain parenting behaviors. This has not been well explored during infancy. A study on toddlerhood through preschool age found that SES, indexed by family finances did not moderate consistency of maternal sensitivity during toddlerhood and preschool years (Madigan et al., 2016). Whether the same holds true during infancy is not clear.

SES could matter more for consistency in maternal sensitivity during infancy as mothers may still be adjusting to the added demands of caring for a new infant. Moreover, in addition to family finances, parental education and occupational prestige should also be included when assessing SES in the context of parenting (Hoff, Laursen, & Tardif, 2002). The link between higher education levels and better parenting is well documented (Richman, Miller, & Levine 1992; Suizzo, & Stapleton, 2007; Tamis-Lemonda, Briggs, McClowry, & Snow 2009). Likewise, higher occupational prestige is also linked to better parenting (Raver, 2003). Both of these factors could also influence consistency in maternal sensitivity. To better understand the role SES plays in the consistency with which mothers engage in maternal sensitivity when parenting infants, research using a multidimensional assessment of SES is warranted.

Maternal sensitivity, a broad multimodal parenting construct, is a conglomerate of various discrete maternal behaviors (Mesman & Emmen, 2013). Two such building blocks of maternal sensitivity that are known to facilitate physiological, socioemotional and language development are maternal positive affect (Gunnar & Stone, 1984; Hanley, Brain, & Oberlander, 2013) and infant-directed speech (Bigelow et al., 2010; Saint-Georges et al., 2013). Maternal positive affect typically refers to maternal smiling, positive tone of voice, and often also to physical affection like caressing (see Mesman & Emmen, 2013). It is known to facilitate better infant physiological regulation (Martinez-Torteya et al., 2014; Moore et al., 2009), social referencing in infants (Gunnar & Stone, 1984), and socioemotional development (Hanley et al., 2013). Infant-directed speech (IDS) refers to the way in which adults speak to infants involving overall higher and variable pitch, intonation contours, and exaggerated vowels conveying positive affect. Additionally, it is characterized by slower repetitive speech, long pauses, shorter utterances and use of syntactically and semantically simple language (e.g. Cristia, 2013; Fernald, Taeschner, Dunn & Papousek, 1989; Singh, Morgan & Best, 2002; Soderstrom 2007; Spinelli, Fasolo & Mesman, 2017). IDS is linked with affect, attention, physiological regulation and language learning in infants and predicts formation of secure attachment (Bigelow et al., 2010; Fillipa et al., 2017; Jahromi et. al, 2004; Saint-Georges et al., 2013). Although maternal positive affect and IDS are evidently associated with better infant developmental outcomes, the consistency in how much infants are exposed to these maternal behaviors is not fully understood.

There are indications that mothers engage in these behaviors consistently across infancy (Belsky et al., 1984; Bornstein & Tamis-LeMonda, 1990), but whether SES has a role on such consistency is not well understood. For example, early research by Belsky et. al, (1984) found that positive maternal affect is consistent across the first nine months of infancy. However, the participants were predominantly middle-income families and whether consistency of maternal affect differed by SES was not reported. Longitudinal studies on IDS have assessed whether specific characteristics of IDS, such as prosodic modifications in pitch, exaggerated vowels and vocal affect, change in development and whether consistencies in those characteristics have implications for language development (see Burnham et al., 2015; Cristia & Seidl, 2014; Kalashnikova & Burnham 2018; Kitamura, Thanavishuth, Burnham & Luksaneeyanawin, 2002). However, it is important to note that mothers do not always speak to their infants using positive vocalizations, prosodic modifications associated with IDS. The extent to which positive vocalizations are a part of the infant environment can make a difference in infant language and socioemotional development. There is limited research assessing whether there is consistency in the extent to which infants hear maternal positive vocalizations. An early study found that exposure to IDS, characterized by extreme or fluctuating pitch, is consistent across the first 5 months of infancy (Bornstein & Tamis-LeMonda, 1990). This study also included middle to higher SES families, and it is not clear if consistency in the use of IDS varies by SES. Given that minimal research has been done on consistency of maternal affect and IDS in the context of SES, there is a need to better understand the role of SES on consistency of these behaviors.

In addition to examining the modulators of consistency in maternal sensitivity, it is also crucial to understand mechanisms through which consistency in maternal sensitivity relates to its developmental outcomes. Consistency in maternal sensitivity could be associated with positive social and cognitive developmental outcomes by way of well-regulated physiological stress system. Thus, there is a need to understand how consistency in maternal sensitivity during infancy relates to cortisol regulation, a marker of physiological stress system. Consistency of maternal sensitivity in early development could contribute to the shaping of HPA axis regulation, a physiological stress system of which the end product is cortisol. Unpredictability is a core characteristic of a stressor (Lupien et al., 2007). Variability in maternal response to infants may ensue a sense of unpredictability, which in itself is a direct source of stress that may add to cortisol dysregulation (Manczak et al., 2017). When infants are forming attachments and expectations about caregivers' behaviors, engaging in joint attention and social referencing, they may especially benefit from a predictable environment. Unpredictable caregiving during such a vulnerable period of development may not only be a stressor, but may also leave infants without necessary social scaffolding for cortisol regulation. It is possible that such early inconsistent maternal sensitivity could shape an individuals' cortisol systems in maladaptive ways, ultimately making them susceptible to poor mental and physical health outcomes (McEwen, 2006b).

The present study investigated whether there is consistency in the use of maternal smiling and laughter and maternal positive vocalization in the context of SES from 6 to 12 months of infancy. Additionally, we examined whether consistency of maternal behaviors related to diurnal and cumulative measures of cortisol. Maternal smiling and laughter are components of maternal positive affect, and maternal positive vocalization refers to prosodic components of IDS such as higher and variable pitch, intonation contours and exaggerated vowels. SES was indexed by family income, parental education and parental occupational prestige. Based on previous literature, we hypothesized that maternal smiling and laughter, and positive vocalization at 6 and 12 months would be consistent. We predicted that SES would moderate consistency of maternal smiling and laughter and positive vocalization, such that mothers from higher SES would display more consistency in these behaviors. We also predicted that consistency in these maternal behaviors from 6 to 12 months of infancy would relate to better regulated chronic and acute cortisol function and cumulative exposure to cortisol at 12 months of age.

Participants were 82 mothers (*M* = 33.37 years, *SD* = 3.70) and their healthy, singleton infants with no known hearing, visual, neurological or developmental disorders. Families were recruited from the greater Boston area using public birth records and a departmental database of families who had expressed interest in participating in research. The dyads were assessed at home when infants were 6 months old (*M* = 6.69 months; *SD* = 0.43; 38 females) and then in the laboratory when their infants were 12 months old (*M* = 12.18 months; *SD* = 0.73). Table 9 summarizes demographic characteristics of all the participants.

Table 9

Demographic Characteristics of Participating Families

Variables	
Infant Ethnicity	
White	69.5%
Black or African American	2.4%
Asian	3.7%
Hispanic	1.2%
Native American	0.0%
Multiracial/Other	22.0%
Maternal Ethnicity	
White	75.6%
Black or African American	4.9%
Asian	11.0%
Hispanic	2.4%
Native American	1.2%
Multiracial/Other	2.4%
Income to Needs Ratio (ITN)	
M (SD)	5.79 (3.18)
Economically Strained Participants (ITN =< 3)	21.2%
Maternal Education percent with at least a 4 yr college degree	90.30%

Paternal Education percent with at least a 4 yr college degree	81.70%
Maternal Job Prestige (ranged from 1 to 5; 1 requiring less to no preparation)	
M (SD)	3.99 (0.94)
Paternal Job Prestige (ranged from 1 to 5; 1 required less to no preparation)	
M (SD)	3.92 (1.03)
CES-D (above the clinical cutoff score, 16)	18.30%

Home visits at six months were conducted during the day at a convenient time for the mothers and when the infants were well rested. Following informed consent, mothers engaged with their infants in a free play session after which they provided demographic information and filled out a questionnaire on depressive symptomatology. Six months after the initial visit, the dyads participated in a follow-up free play session at the laboratory and collected saliva samples as a part of a larger study.

Maternal parenting behaviors were assessed at both time points by micro-coding videotapes of 6-minute mother-infant free play sessions designed to elicit maternal behaviors that occur naturally during mother-infant interactions (Feldman & Eidelman, 2004; Feldman, Gordon, & Zagoory-Sharon, 2011). The dyads sat on the floor with five age-appropriate toys laid before them and were left by themselves in a room with the instructions to play as they normally would. A bumbo floor seat was provided and parents were free to place the infants in the seat or allow them to move around the room. Coders were trained to a kappa of .80. They assessed maternal behaviors every 33.33 milliseconds, frame by frame (Feldman & Eidelman, 2004) using the software Observer XT – Noldus 11.0 (Wageningen, the Netherlands). One minute of each video was double-coded for reliability. Interrater reliability kappa ranged from .84 to .97. At every 30th of a second, the software paused the video so that the coders could code for presence of maternal smiles or laughter and positive vocalization to obtain the exact duration of the behaviors. When the mothers smiled or laughed looking at the baby, it was coded as engaging in maternal smiles and laughter. Maternal smile and laughter was calculated as the proportion of time mothers displayed smile or laughter during free play. Positive vocalization was calculated as the proportion of time mothers used higher and variable pitch, intonation contours, and exaggerated vowels directed at the infant during the free play. These variables were corrected for the time when mothers' could not be coded either because their faces were out of the camera view or because their voices were inaudible. Proportion of each discrete maternal behaviors at 6-months and 12-months were standardized and difference scores were computed as a measure of consistency of each maternal behavior.

Mothers reported on family income, maternal and paternal occupation, and highest level of maternal and paternal education. An income-to-needs ratio (ITN) was calculated using total household size, household income and federal poverty guidelines. For example, an ITN of 2 indicates the household income is twice the federal poverty line for that household size. Highest education level was coded from 1 to 5 where a score of 1 represented elementary, middle and junior high school, 2 represented high school and general educational development (GED) certificate, 3 represented vocational school and community college, 4 represented college (4-year degree) and 5 represented graduate school. Occupational prestige of both parents was coded using the Job Zone coding scheme from the Occupational Information Network (O*NET, http://www.onetonline.org/help/online/zones), which ranks U. S. Census-based occupational categories on a 5-point scale based on the education, experience, and training required (1 = *requiring the least preparation* and 5 = *requiring the most extensive preparation*). A composite SES was calculated by averaging the standardized scores of ITN, total parental education level and total parental occupational prestige.

As some mothers are prone to depressive symptomatology when parenting infants in the first year of life (Feldman, 2007), maternal depressive status was tested as a potential covariate. Mothers' depressive symptomatology was assessed using the Center for Epidemiologic Studies - Depression Scale (CES-D). It is a 20-item (α = .84) self-report questionnaire designed to measure depressive symptoms in the general population (Radloff, 1977).

During the 12-month visit, mothers were trained on collecting saliva samples from their infants. Mothers were instructed to place a synthetic swab in the infant's mouth for a total of 60 seconds (Salimetrics, State College, PA). They were asked to collect saliva samples at waking and bedtime across 3 days. We provided them with a home kit including collection materials, instructions, and a home diary to record information about time of sampling, bed and wake times, feeding and napping, and other factors that can affect cortisol levels. Collected samples were frozen at -20° C until they were sent to Trier Laboratories in Germany for cortisol assay. Biologically plausible values for waking and bedtime across the 3 days of sampling were used to compute the area under the curve with respect to ground (AUC_g) from waking to bedtime. It was an estimate of total cortisol exposure over the day, also referred to as diurnal cortisol exposure. We computed an aggregate diurnal cortisol exposure value by averaging the AUC_g values across 3 saliva sampling days.

At 12 months of age, infant hair cortisol concentration (HCC) was measured by sampling 3 cm of hair (15-20 mg) closest to the scalp from the posterior vertex. It indexed infants' cumulative exposure to cortisol over few months prior to the 12-month lab visit. Hair samples were shipped to the University of Massachusetts, Amherst, for cortisol analysis following the methods of Meyer et al. (2014).

Preliminary analyses indicated that all the variables, except maternal positive vocalization, HCC, and AUC_g were normally distributed. To ensure normal distribution of the data, maternal positive vocalization at both time points was winsorized, AUC_g was also winsorized and HCC was log-transformed. Descriptive statistics of maternal behaviors at both time points are presented in Table 10. ITN, parental job prestige and education were significantly correlated with each other (see Table 11) and a composite SES was calculated. An independent sample *t*-test revealed that SES did not significantly differ based on White ($M = 6.05$, $SD = 2.78$) or non-White participants ($M = 5.17$, $SD = 3.97$), $t(78) = 1.14$, $p > .05$.

Table 10

Descriptive Statistics of Maternal Behaviors

Variables	*M* (*SD*)
At 6 months	
Maternal Smiling and Laughter	.153 (.145)
Maternal Positive Vocalization	.221 (.204)
At 12 months	

Maternal Smiling and Laughter	.273 (.239)
Maternal Positive Vocalization	.162 (.169)

Table 11

Correlations among Individual Components of SES

Measure	1	2	3
1. ITN	-		
2. Parental Education	-.405**	-	
3. Parental Job Prestige	.435**	.631**	-

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

To explore whether there was developmental mean-group level change in maternal smile and laughter and positive vocalization, we ran two separate repeated-measures analyses of covariance (ANCOVAs) with time as a within-subjects factor. To assess whether SES also had a main effect on developmental changes in maternal smile and laughter, and positive vocalization, it was entered as a predictor in both the models. Statistically, SES was entered as a covariate because it was a continuous measure (Hoffman et al., 2015; Sweet & Grace-Martin, 2011). There was a main effect of infant age on maternal smile and laughter ($F(1, 77) = 23.58$, $p < 0.001$, $\eta_p = .234$), such that proportion of maternal smile and laughter increased from 6 months to 12 months of infant age at the mean level. There was also a main effect of age on maternal positive vocalization ($F(1, 78) = 10.51$, $p < 0.01$, $\eta_p = .119$), such that proportion of maternal positive vocalization decreased from 6 to 12 months of age at the mean level. However, SES did not have a significant main nor interaction effect on either of the models. That is, neither smile and laughter nor maternal vocalization varied by SES, and age-related shifts in the prevalence of these maternal behaviors were observed independent of SES.

For the main regression analyses assessing individual-order continuity in maternal behaviors and the role of SES, we examined maternal depressive symptomatology at 6 months as a potential covariate. Associations of maternal depressive symptomatology at 6 months with maternal smile and laughter, and positive vocalization concurrently as well as at 12 months were examined. Maternal depressive status at 6 months was not significantly associated with maternal smile and laughter concurrently nor at 12 months, and thus was not included as a covariate in the main regression analysis assessing the role of SES on consistency of maternal smile and laughter. Depressive symptomatology was not correlated with maternal positive vocalization concurrently, but did correlate with maternal positive vocalization at 12 months ($r(77) = -.264$, $p<.021$) and therefore was included as a covariate in the regression analysis examining the role of SES on consistency of positive vocalization. In our sample, maternal depressive symptomatology and SES were not related.

For the main analysis examining individual-order consistency in maternal smile and laughter, and positive vocalization, both maternal behaviors were standardized at each age (6 and 12 months) to control for mean-level developmental changes. Pearson's correlation analyses revealed individual-order continuity in the use of maternal smile and laughter ($r(80) = 0.33$, $p = .003$) and positive vocalization ($r(81) = 0.50$, $p < .001$) from 6 months to 12 months. Next, we examined whether SES moderated consistency of maternal smile and laughter from 6 to 12 months. In an ordinary least squares (OLS) regression analysis conducted using the PROCESS macro in SPSS (Hayes, 2013), maternal smile and laughter at 12 months was regressed on maternal smile and laughter at

6 months, SES, and their interaction term. This was a single model in which all independent variables were entered simultaneously. Results presented in Table 12 show that the overall model was statistically significant and revealed a significant main effect of maternal smile and laughter at 6 months, but not of SES. However, there was a significant interaction between maternal smile and laughter at 6 months and SES in predicting 12-month maternal smile and laughter, with bias corrected bootstrapped confidence intervals at 95% level based on 5000 samples, CI: 0.006–0.7573. Inclusion of this interaction effect significantly improved the model (R^2 change = 0.045 , $F(1, 75)$ = 3.98, $p < .05$) indicating that SES moderated consistency of maternal smile and laughter from 6 months to 12 months. Follow-up analyses revealed that only mothers who had higher SES showed significant consistency in maternal smile and laughter (Table 13). In the another OLS regression model examining the role of SES on consistency of maternal positive vocalization, with maternal depressive symptomatology included as a covariate, we regressed maternal positive vocalization at 12 months on maternal positive vocalization at 6 months, SES, maternal depressive symptomatology, and the interaction term between positive vocalization at 6 months and SES. This was a single model in which we entered maternal positive vocalization at 6 months as a predictor, SES as a moderator and maternal depressive symptomatology as covariate simultaneously. As presented in Table 14, this model was statistically significant but, only maternal positive vocalization at 6 months had a significant main effect on maternal positive vocalization at 12 months. Thus, maternal positive vocalization at 6 months uniquely predicted maternal positive vocalization at 12 months above and beyond SES and maternal depressive

symptomatology. Moreover, SES did not moderate consistency of maternal positive vocalization when controlled for maternal depressive symptomatology.

Table 12

OLS Regression Model Examining the Moderation of the Effect of Maternal Smile and Laughter at 6 Months on Maternal Smile and Laughter at 12 Months by SES

						95% confidence interval for coeff.	
		Coeff.	*SE*	*t*	*p*	Lower bound	Upper bound
Constant	i^1	0.022	0.117	0.192	.8483	-0.209	0.255
Maternal Smile and Laughter (6 months)	b^1	0.322	0.127	2.533	.0134	0.069	0.574
SES	b^2	0.059	0.178	0.330	.7425	-0.295	0.412
Maternal Smile and Laughter (6 months) X SES	b^3	0.379	0.189	1.995	.0497	0.001	0.757
				$R^2 = 0.385$, $MSE = 1.004$			
				$F(3, 75) = 4.347$, $p = .007$			

Note. Coeff.= unstandardized coefficient

Table 13

Conditional Effect of Focal Predictor at Values of the Moderator Variable for the *OLS Regression Model Examining the Moderation of the Effect of Maternal Smile and Laughter at 6 Months on Maternal Smile and Laughter at 12 Months by SES*

					95% confidence interval for effect	
SES	Effect	*SE*	*t*	*p*	Lower bound	Upper bound
-0.707	0.054	0.202	0.266	.7911	-0.349	0.456
0.167	0.385	0.125	3.086	.0028	0.136	0.633
0.702	0.587	0.165	3.559	.0007	0.259	0.916

Note. Effect = estimated effect of maternal smile and laughter at 6 months on maternal smile and laughter at 12 for the given value of SES.

Table 14

OLS Regression Model Examining the Moderation of the Effect of Maternal Positive Vocalization at 6 Months on Maternal Positive Vocalization at 12 Months by SES, Controlling for Maternal Depressive Symptomatology at 6 Months

						95% confidence interval for coeff.	
		Coeff.	*SE*	*t*	*p*	Lower bound	Upper bound
Constant	i^1	0.189	0.152	1.241	.2179	-0.114	0.493
Maternal Positive Vocalization (6 mo.)	b^1	0.482	0.101	4.785	.0000	0.281	0.682
SES	b^2	0.119	0.131	0.906	.3681	-0.142	0.379
Maternal Positive Vocalization (6 mo.) X SES	b^3	0.085	0.151	0.565	.5738	-0.216	0.386
Maternal Depressive Symptomatology	b^4	-1.143	0.735	-1.555	.1242	-2.607	0.322
				$R^2 = 0.310$, $MSE = 0.691$			
				$F(4, 72) = 8.090$, $p < .001$			

Note. Coeff.= unstandardized coefficient

Pearson's correlation analyses revealed that consistency scores of maternal smiles and laughter and maternal positive vocalizations were not significantly correlated with HCC and AUC_g. Thus, consistency of maternal behaviors from 6 to 12 months of infancy did not relate to cortisol regulation at 12 months of age.

The present study investigated the associations of maternal smile and laughter and positive vocalizations from 6 to 12 months during infancy; the contribution of SES to consistency of maternal behaviors; and whether consistency in maternal behaviors from 6 to 12 months during infancy related to cortisol regulation at 12 months of age. Overall, infants were exposed to maternal smile and laughter, and positive vocalizations consistently across the first year of life. However, infants from lower SES families were vulnerable to inconsistent parenting, experiencing no stability in the level of maternal smile and laughter they received from 6 to 12 months of age. This implies that while privileged infants experience more predictability in terms of their mothers' facial affective responses, less privileged infants are more likely to be challenged with uncertainty about their mothers' facial affective responses. Unpredictable reactions from mothers when infants depend on them for facilitation of processes like attachment formation and social referencing can be detrimental to their socioemotional development. Moreover, such unpredictability is an additional stressor for infants already facing other adversities which place them at a greater risk for maladaptive physiological development.

Given that consistency in maternal smiling and laughing, but not positive vocalization, varied by SES, the results emphasize how factors contributing to parenting consistency may differ by the parenting behavior in question. SES may not have played a role in consistency of positive vocalization in our sample because most of our participants were highly educated despite some variability in their household incomes. Higher SES measured by income and education is a predictor of greater use of IDS when parenting toddlers. Rowe (2008) found that mothers who have higher levels of education are more knowledgeable about child development and those who are more knowledgeable about child development use more IDS. It is possible that maternal education is crucial for consistency of maternal positive vocalization as well. It is likely that the majority of the mothers in our sample, who are well educated, were also knowledgeable about child development and engaged in positive vocalizations consistently. Future studies examining the role of SES on consistency of parenting should also consider parental knowledge on child development.

Previous literature has not found that SES was important for consistency of maternal sensitivity when parenting toddlers (Madigan et al., 2016). However, they relied only on financial indices of SES whereas we employed a multidimensional measure of SES including parental education and occupational prestige. Our finding that SES mattered in order for mothers to consistently display similar levels of positive facial affect to their infants underscores the importance of including parental education and occupational prestige as measures of SES when exploring its role in consistency of parenting. There is a possibility that the role of SES on consistency of parenting varies by child age, and the measure of parenting. We indexed maternal sensitivity by specific parenting behaviors which likely overlap with global measures of maternal sensitivity. SES was associated with consistency of only maternal smile and laughter and not positive vocalization in this study. This finding, considered in concert with previous literature on consistency of the broader construct of maternal sensitivity (Madigan et al., 2016), raises a question regarding whether the role of SES in consistency of parenting is specific to particular parenting behaviors or if it is also applicable to broader measures of maternal sensitivity. It is yet to be fully understood to what extent these specific parenting behaviors index broader measures of maternal sensitivity and how SES influences consistency of these constructs. To address this question, future studies should assess the role of SES in consistency of both broad as well as discrete parenting measures of maternal sensitivity in various child age groups.

When interpreting the findings, it is important to consider that we assessed maternal behaviors in different settings at two time points: in the home at 6 months and in the laboratory at 12 months. We cannot tease apart whether mothers with low SES were generally inconsistent with their smiles and laughter over time, or if they appeared inconsistent because they were uncomfortable in a laboratory setting at 12 months. Mothers of low SES or from racial or ethnic minority groups may have responded differently to the laboratory setting, with differential effects on observed parenting in this context. While it is important to note that racial and ethnic minority status did not relate to SES in our sample, we did not have a large enough sample to allow for analysis of subgroups of specific racial and ethnic minorities. Future studies of maternal consistency and SES should assess these behaviors in the same environment across time, ideally the home, and should include larger numbers of individuals of specific racial and ethnic minority groups to allow for examination of the interplay of SES and racial and ethnic identity in consistency of parenting.

Contrary to expectations, consistency in maternal behaviors from 6 to 12 months of infancy did not relate to cortisol regulation at 12 months. Although evidence from prior literature suggests that quality caregiving buffers cortisol dysregulation (Hostinar et al., 2014), current findings suggest that how consistent mothers are with the specific parenting behaviors they engage in may not matter for cortisol regulation at this age. However, it is important to note that most studies that have found that quality caregiving predicts cortisol regulation have used global rating measures of maternal sensitivity (Hostinar & Gunnar 2015). It is possible that infant cortisol regulation is sensitive to consistencies in maternal sensitivity measured by global rating assessments that take into account infants' responses to parent behaviors. Future studies should investigate whether consistency in globally sensitive caregiving predicts cortisol regulation. This study was limited because it used archival data and did not have data on global ratings of maternal sensitivity.

In the present study, consistency of maternal behaviors was analyzed in the context of a brief free play session. Maternal behaviors assessed in this study might have primarily reflected consistency of maternal behaviors when the infants were calm. Consistency of maternal behaviors might have been different if maternal behaviors were assessed when the infants were distressed or overstimulated. Thus, investigating how consistency of maternal behaviors relates to infant cortisol dysregulation when the infants are distressed might yield different results than when it is assessed in a free play setting with no infant distress. Future studies investigating the role of parenting consistency in infant cortisol regulation should pay attention to the context in which parenting is examined.

The finding that SES moderated consistency of maternal smile and laughter from 6 to 12 months even in a sample facing relatively low adversity highlights how consistency of maternal smile and laughter is sensitive to SES. Thus, this study demonstrates the role that SES has in consistency of an aspect of parenting. This study establishes that while individual differences in discrete maternal behaviors are consistent across the second half year of infancy overall, lower multidimensional SES contributes to infants' risk for experiencing inconsistent parenting.

GENERAL DISCUSSION

Sleep difficulties and cortisol dysregulation have each been associated with a multitude of health problems (Assefa et al., 2015; Bauer, 2005; Franzen & Buysse, 2008; Laurent et al., 2015, McEwen, 2006a, 2006b; Staner, 2003). There is a considerable overlap between the nature of health outcomes that is associated with each system (e.g., cardiovascular diseases, immune health problems, mood disorders). Given that sleep and cortisol systems are intertwined and regulated by a circadian rhythm, it is not surprising that they have implications for common health issues. Moreover, these systems codevelop during infancy (Antonini et al., 2000; De Weerth et al., 2003). It is believed that early perturbations in one system, not only lead to dysregulation in that system, but can also maladaptively influence the maturation of the other system (Palagini et al., 2015; Van Reeth et al., 2000). This subsequently could increase an individual's risk for enduring health problems (McEwen, 2006a; 2006b). Thus, to better understand how early development of both the systems contributes to lasting health outcomes, it is first important to comprehensively investigate how the two systems interact with each other during infancy. Moreover, because humans heavily depend on their caregivers during infancy, it is important to unravel the nuances of the role of parenting in the development of these systems. This dissertation contributes to the current literature by examining how sleep and cortisol systems interact with each other; how cortisol interacts with parent characteristics to predict infant sleep; and whether consistent parenting buffers infant cortisol dysregulation.

Together, the present studies demonstrate that in 12-month-old infants, well-regulated cortisol at bedtime predicts healthy sleep characteristics (i.e., falling asleep earlier at night) on a daily basis, and infants with generally healthy sleep characteristics have well-regulated chronic cortisol levels; the sensitivity of infant sleep to particular parent characteristics depends on infants' cortisol regulations; and SES plays a role in infants' experiences of consistent parenting from 6 to 12 months of age. Study 1 highlighted that there is a time-based association between bedtime cortisol and infant sleep on the same night; however, infant sleep on a given evening does not relate to diurnal cortisol exposure the next day. Lower cortisol in the evening predicted earlier sleep onset. Given that prior literature on adults had also found the same relation between daily evening cortisol and sleep onset (Van Cauter & Spiegel, 1997), the findings of this study extend the literature by showing that the temporal relation between bedtime cortisol and sleep onset is established as early as by 12 months of age. Prior research with preschoolers and adults suggested a bidirectional influence between daily sleep and cortisol (Gribbin et al., 2012; Van Cauter & Spiegel, 1997). Importantly, by contrast, this study brought to light that at 12 months only bedtime cortisol predicts infant sleep the same night, but infant sleep does not predict cortisol levels the following day. It is possible that bedtime cortisol predicts infant sleep the same night because they occur together closely in time. Infant sleep on a given night may not have associated with diurnal cortisol exposure the following day because apart from sleep on the prior night, other factors infants are exposed to during the day could be strongly contributing to total cortisol exposure during the day. Further, the fact that bedtime cortisol predicted only sleep onset and not duration the same night implies that evening cortisol levels are closely tied with sleep regulation at night, but there may be other factors contributing to how long the infants sleep through the night. Additionally, this study found that infants who generally fell asleep earlier at night had lower chronic cortisol levels, regardless of how long they slept through the night, their economic status, and the level of chaos they experienced in their homes. This finding expands on previous literature by underscoring the powerful association of nighttime sleep onset with chronic cortisol levels, beyond the effects of other predictors of chronic cortisol that were assessed in this study.

Study 2 illustrated that the relation of both bedtime parenting and general sensitive parenting throughout the day with infant sleep onset varied as a function of cortisol regulation. Infants with minimal bedtime parental assistance fell asleep earlier at night only if they had dysregulated cortisol levels. On the other hand, infants who were exposed to general sensitive caregiving during the day fell asleep earlier at night only if they had well-regulated cortisol levels. These findings imply that infants with dysregulated cortisol are particularly vulnerable to sleep difficulties when they are exposed to a parenting factor known to impede sleep regulation (i.e., bedtime parental involvement). In contrast, when infants have dysregulated cortisol systems, a parent characteristic previously known to help infant sleep regulation (i.e., parental sensitivity) may be of little importance for infant sleep. This has implications for identifying infants who are most likely to benefit from specific types of parenting interventions targeted to foster better infant sleep regulation.

Study 2 also found that solitary sleep and sleep onset were no longer associated after accounting for bedtime parental involvement. This finding illuminated the fact that it is limited bedtime parental involvement, and not solitary sleeping, that contributes to earlier nighttime sleep onset. In the U.S. sample of this study, cosleeping often occurred with greater bedtime parental involvement. Cosleeping could have occurred in response to infant sleep difficulties, and cosleeping when non-reactive may not be associated with difficulties falling asleep. Thus, it is also important to take cultural contexts into account when interpreting the role of parent characteristics on infant sleep outcomes.

Study 3 revealed that SES played a role in consistency of certain parenting behaviors across 6 to 12 months of infancy, but consistency in parenting behaviors across the second half of the first year of life was independent of cortisol regulation at 12 months. Infants from lower SES families were vulnerable to inconsistent parenting, experiencing no stability in the level of maternal smile and laughter they received from 6 to 12 months of age. This implies that while privileged infants experience more predictability in terms of their mothers' facial affective responses, less privileged infants are more likely to be challenged with uncertainty about their mothers' facial affective responses. Contrary to expectations, maternal consistency did not predict chronic nor diurnal cortisol exposure at 12 months. Thus, although evidence from prior literature suggests that quality caregiving buffers cortisol dysregulation (Hostinar et al., 2014), findings imply that how consistent mothers are with the specific parenting behaviors they engage in may not matter for cortisol regulation at this age. However, it is important to note that this study used archival data and parenting consistency was analyzed in the context of a brief free play session. Parenting behaviors assessed in this study might have primarily reflected consistency of parenting behaviors when the infants were calm. Parenting consistency might have been different if parenting was assessed when the infants were distressed or overstimulated. Thus, investigating how consistency of parenting behaviors relates to infant cortisol dysregulation when the infants are distressed might yield different results than when it is assessed in a free play setting with no infant distress. Future studies investigating the role of parenting consistency in infant cortisol regulation should pay attention to the context in which parenting is examined.

Collectively, these studies demonstrate that sleep and cortisol systems are intertwined early in development by 12 months of age, a time when the circadian rhythm is relatively well-established. It is clear through these studies that there is an early interplay between sleep and cortisol systems. Additionally, regulation of one system begins to play a part in the regulation of the other system by interacting with contributors such as parent characteristics. The early relation of sleep and cortisol demonstrated in this dissertation lends support to the idea that the workings of both these systems together contribute to the allostatic load (McEwen, 2006b). More importantly, findings suggest that the two systems may begin jointly contributing to the allostatic load very early in development. Increased allostatic load, in turn, has implications for physiological dysregulations at a multisystemic level, giving rise to enduring health outcomes. Given that sleep and cortisol systems mature together and interact with one other, it is not surprising that dysregulation of each of these systems is associated with a common set of maladaptive health outcomes. Thus, the findings have implications for the importance of early interventions on populations at risk for early sleep and cortisol dysregulation to reduce the public health burden.

Early interventions for sleep problems have garnered a lot of attention as sleep difficulties are behaviorally observable and are highly prevalent during early childhood (Morgenthaler et al., 2006; Mindell et al., 2006). Nonpharmaceutical interventions include behavioral interventions that focus on parent education and training to help parents cultivate predictable sleep schedules for their infants, and also adapt their bedtime parenting behaviors to reduce infants' dependency on parents for sleep regulation. Given the current finding from Study 2 that sensitivity of infant sleep to parenting depends on infants' cortisol regulation, it is important for future interventions to take cortisol regulation into consideration to help identify which infants are most likely to benefit from their interventions. Apart from parenting related to sleep, interventions should also target fostering general sensitive caregiving among parents with infants who have sleep difficulties. In contrast to intervention efforts for early sleep difficulties, interventions for early cortisol dysregulation have been limited. A family based intervention has shown to buffer preschoolers in foster care from cortisol dysregulation (Fisher et al., 2007). Findings from this dissertation suggest that such interventions should begin as early as infancy, and that special attention should be given to help infants reduce cortisol levels at bedtime. Given that cortisol regulation plays an important role in infant sleep and in the sensitivity of infant sleep to parenting, there is a need for more family based interventions that jointly target improvement of both infant sleep and cortisol regulation

Sleep, cortisol, and parenting are all multidimensional constructs. For example, nighttime sleep onset indexes sleep regulation, whereas nighttime sleep duration indexes consolidated nighttime sleep. Likewise, HCC is a marker of chronic physiological stress, whereas diurnal cortisol exposure is a marker of acute physiological stress exposure. Parental sensitivity measured in an interaction session is meant to capture parents' general sensitivity levels unlike bedtime parental presence which is specific to parent behaviors directly related to infant sleep. Some researchers have attributed the inconsistent findings regarding the relation between parenting and sleep, or cortisol and sleep to the differences in how each construct is measured, and what they represent (Camerota et al., 2019; Tétreault, Bouvette-Turcot, Bernier, & Bailey, 2017; Scher, Hall, Zaidman-Zait, & Weinberg, 2010). This dissertation suggests that mixed findings from prior literature are not simply inconsistencies, but rather reflections of the nuanced meaningful differences among the associations between various dimensions of cortisol, sleep and parenting. For example, cortisol, parenting and sleep were associated when the measure of sleep was nighttime sleep onset and not nighttime sleep duration. In Study 1, daily evening cortisol predicted sleep onset the same night, but not sleep duration. It is possible that this is because evening cortisol is more relevant for sleep onset on the same night, but other factors such as morning routines or distractions could impact sleep duration. Moreover, in Study 2, diurnal cortisol exposure and parenting interacted to predict sleep onset, but not sleep duration. If the current dissertation had only assessed sleep duration and not sleep onset, the findings would have been misleading in that they would imply no link between daily cortisol and sleep, and no moderating role of cortisol regulation in the association between parenting and sleep. Likewise, our choice of cortisol variable could also have played a role in which sleep characteristic was sensitive to parent characteristics. In Study 2, sensitivity of sleep onset to parenting depended on infants' diurnal cortisol exposure throughout the day. In contrast, sensitivity of nighttime sleep duration to parenting did not depend on infants' diurnal cortisol exposure throughout the day and might have depended on nocturnal cortisol regulations instead. Indeed, nocturnal cortisol exposure contributes to nighttime sleep duration in adults (Späth-Schwalbe et al., 1992), and it could have been a better candidate than diurnal cortisol exposure across the day for the moderating cortisol variable. Similarly, in Study 2 there were differences in how parent characteristics predicted sleep characteristics and how these relations varied by cortisol regulation for daytime parental sensitivity and bedtime parental involvement. In Study 3, consistency of parenting behaviors did not predict cortisol regulation. However, quality caregiving has been associated with cortisol regulation in the prior literature (see Hostinar & Gunnar 2013, 2015). This underscores the complexities of how various dimensions of parenting differentially relate to sleep and cortisol. Thus, when analyzing the interplay between cortisol, sleep, and parenting, it is important not to treat all dimensions of each factor as the same construct. The relations between cortisol, sleep, and parenting are nuanced depending on various aspects of the factor being examined. To obtain a fuller picture of how the three factors interact, future research should carefully select and discern indices of cortisol dysregulation, parenting and sleep that are most relevant for the specific measures being assessed.

It is also important to note that broader contextual factors such as SES and household chaos cannot be discounted when investigating parenting and sleep. In Study

3, findings elucidated the vulnerability of lower-SES infants to inconsistent parenting. Results from Study 2 illustrated that infants' exposure to chaos at their homes was a powerful predictor of their nighttime sleep duration, beyond the effects of parenting. These findings underscore the need for future studies to investigate the role of parenting in sleep by taking into consideration such broader contextual factors.

Generalizability of the findings of the current dissertation is limited because of socioeconomically and culturally similar samples across the three studies. Study 1 and 2 included a socioeconomically diverse sample. The sample for study 3 was comparatively less diverse: The majority of the sample was not economically strained and had parents who were highly educated. While the findings of studies 1 and 2 may be more representative of families from poorer educational backgrounds compared to study 3, findings from all the studies are generalizable to infants in the U.S. only. Culture plays a big role in both parenting and sleep (Jenny & O'Connor, 2005; Mindell et al., 2010; Rothbaum, Weisz, Pott, Miyake, & Morelli, 2000). For example, there are cultural differences in what is normative regarding sleeping arrangements, nature of bedtime routines, parental involvement, sensitive parenting, consolidated sleep distribution, sleep duration and such. Based on these cultural differences, the interplay between parenting and sleep could be different in cultures outside the U.S. Moreover, cortisol and sleep codevelop in the presence of contextual factors, so the roles of cortisol regulation in infant sleep, as well as in the relation between parenting and sleep could be more complex in the context of cultural differences. Thus, the present work is limited in that findings cannot be extended to infants from other cultures. Though some researchers have begun investigating the association between parenting and infant sleep in culturally diverse populations, research on the role of cortisol regulation in infant sleep, and in the relation between parenting and infant sleep is yet to be examined in a culturally diverse sample. Future studies should investigate the interplay between cortisol, parenting and infant sleep in other cultures as well.

As a whole this dissertation research elucidates the complexities underlying the relation between two vital physiological processes codeveloping in the first year of life – sleep and cortisol regulation – and the role cortisol plays in modulating the associations between parent characteristics and infant sleep. The multidimensional nature of each of these factors, the settings in which they are assessed, and the broader contexts in which infant cortisol and sleep develop all contribute to the nuances in the associations among infant cortisol, sleep, and parenting. While more research is still required to uncover these nuances, the current research brings us a step closer to fully understanding the directionality of the relation between infant cortisol and sleep. Moreover, the research goes beyond studying the main effects of parenting on infant sleep, and adds to the limited body of work supporting a claim of the transactional model that infant physiological characteristics and parenting jointly influence the development of infant sleep. Lastly, this work highlights the need for holistic family interventions targeted to promote multiple physiological regulatory processes from as early as 12 months of age. Such interventions will not only have implications for proper development of the targeted physiological processes, but may also have implications for minimizing future risks for a myriad of health problems associated with dysregulation of those systems.

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