



Review article

Sleep disturbance and the long-term impact of early adversity

Andrew J. Fuligni^{a,*}, Jessica J. Chiang^b, Nim Tottenham^c

^a University of California, Los Angeles, United States

^b Georgetown University, United States

^c Columbia University, United States



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ABSTRACT

Sleep disturbance may be a central, yet underappreciated mechanism by which early adversity has a long-term impact upon mental and physical health. The fundamental regulatory processes shaped by early adversity – neural, neuroendocrine, and immune – are also central to sleep. Sleep problems, in turn, lead to a similar constellation of chronic health problems that have been linked to early adversity. We bring together work from the fields of early adversity and sleep in order to suggest a model by which sleep disturbance plays a critical role in the far-reaching impacts of early adversity on health. Future research should employ more longitudinal designs and pay particular attention to the impact of developmental periods such as adolescence and midlife when maturational and environmental factors conspire to create a unique time of sleep disturbance. We also suggesting that intervening to minimize sleep disturbance may be a promising means by which to test the model, as well as potentially blunt the long-term impact of early adversity on health.

1. Introduction

The long-term impact of early adversity on mental and physical health across the lifespan has generated much empirical and clinical interest (Danese and McEwen, 2012; Miller et al., 2011; Nelson, 2017). At the same time, the study of sleep expanded significantly, showing the impact of chronic sleep disturbance on many aspects of health (Carskadon, 2011; Dahl and Harvey, 2007; Kripke et al., 2002; Vgontzas et al., 2013). Curiously, the two fields have had little cross-talk with one another despite a striking overlap in health outcomes.

Children and adolescents exposed to adversities, particularly those chronic or severe in nature, evidence numerous mental and physical health challenges throughout their lives. For instance, parental death, parental divorce, caregiver psychopathology, separation from parents, socioeconomic disadvantage, maltreatment, and violence, have been linked to 1.3–2.2 odds for developing a range of psychiatric disorders, including mood, substance use, and fear disorders among adolescents (Lansford et al., 2002; McLaughlin et al., 2012) and adults (Green et al., 2010; Kessler et al., 2010; Lewis et al., 2019). Growing up in a household marked by adversities such as maltreatment, chaos, conflict, and parent psychopathology, as well as specific adversities (namely maltreatment and socioeconomic disadvantage), have been linked to greater risk for cardiometabolic and respiratory health problems in youth (Cesaroni

et al., 2003; Gong et al., 2014; Goodman et al., 2007; Lynch et al., 2016) and cardiovascular disease, diabetes, certain cancers, and respiratory and autoimmune disorders in adults (Brown et al., 2013; Cohen et al., 2004; Felitti et al., 1998; Galobardes et al., 2006; Wegman and Stetler, 2009). Furthermore, increasing evidence suggests that early adversity has implications for adult mortality. One study, for instance, showed that midlife women who reported childhood abuse were at greater risk for premature mortality (Chen et al., 2016). Other studies have shown this same pattern of elevated mortality risk for composite measures aggregating various early adversities, including maltreatment, parental separation, witnessing domestic violence, and living with family members with mental illness or who were substance abusers (Brown et al., 2009; Kelly-Irving et al., 2013).

A parallel, but distinct line of research has shown a comparable constellation of health effects from sleep disturbance (e.g., duration, continuity, quality, variability; Buysse et al., 1989; Lockley et al., 1999; van de Water et al., 2011; see Box 1) throughout the lifecourse. For instance, experimental sleep restriction studies show that shorter sleep duration decreases positive affect and increases depressed mood, anger, confusion, anxiety, threat perception in youth and adults alike (Minkel et al., 2014; Short and Louca, 2015; Talbot et al., 2010). Observational studies of youth and adults show the same pattern of findings and extend them to greater risk for substance abuse and psychiatric conditions

* Corresponding author.

E-mail addresses: afuligni@ucla.edu (A.J. Fuligni), jessica.chiang@georgetown.edu (J.J. Chiang), nlt7@columbia.edu (N. Tottenham).

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Box 1

Indicators of Sleep Disturbance

In addition to laboratory studies experimentally inducing sleep disturbance, studies non-invasively assess sleep disturbance with larger samples in naturalistic settings using wrist-based actigraphy devices that derive sleep parameters from movement (Van De Water et al., 2011), daily sleep logs in which individuals report their sleep time and quality (Lockley et al., 1999), and established self-report scales that assess multiple dimensions of sleep and related functioning (Buysse et al., 1989).

Common indicators of sleep disturbance include:

Very Low or Very High Average Sleep Duration
average total time actually asleep from initial sleep onset to final waking

Poor Sleep Continuity
greater sleep onset latency (time it takes to fall asleep), greater time spent awake after sleep onset, and lower sleep efficiency (proportion of time actually spent sleeping from onset to final waking)

Poor Sleep Quality
subjective sense of not having restful and restorative sleep

High Sleep Variability
nightly variability in aspects of sleep such as duration, continuity, quality

(James and Hale, 2017; Roberts et al., 2009). Sleep disturbances also are associated with greater cardiometabolic risk. In youth, shorter sleep duration, lower sleep continuity, and greater sleep variability have been associated with greater odds of being overweight or obese (Fatima et al., 2015; He et al., 2015; Magee and Hale, 2012; Matthews and Pantescio, 2016), lower insulin sensitivity, and higher cholesterol, triglycerides, and blood pressure (Feliciano et al., 2018; Gangwisch et al., 2010; He et al., 2015; Javaheri et al., 2008; Kuula et al., 2016; Mezick et al., 2012). In adults, shorter sleep duration, sleep disturbances, and poorer subjective quality of sleep are linked to greater risk for cardiovascular disease (Hall et al., 2018) and metabolic disorders such as diabetes (Cappuccio et al., 2010). Shorter sleep duration in youth and adults has also been associated with lower self-rated physical health (Hale et al., 2010; James and Hale, 2017; Moore et al., 2002) and to respiratory-related problems (Cohen et al., 2009; Han et al., 2019; Meltzer et al., 2020, 2015; Prather and Leung, 2016). There also is abundant evidence indicating that both short and long sleep duration are linked to premature mortality in adults (Gallicchio and Kalesan, 2009).

Despite the similar health effects of both early adversity and sleep disturbance, these areas of research have tended to remain disparate. Integrating these lines of research, we suggest a heuristic model positing that sleep disturbances may function as a mechanism by which early adversity engenders greater risk for poor mental and physical health throughout the lifespan (Fig. 1). Early adversity can disrupt sleep concurrently (e.g., greater neighborhood deprivation is cross-sectionally linked with poorer infant sleep, Grimes et al., 2019; foster care

arrangements are associated with poorer sleep in young children, Tinienko et al., 2010) and most likely does so through many of the same mechanisms that we will be discussing in the model. The primary focus of our discussion, however, is on the potential longer-term impact of adversity upon sleep and health.

We focus on the most commonly-studied early adversities (e.g., caregiving and material deprivation, abuse, and socioeconomic disadvantage) and behavioral indicators of sleep disruption suggested to be reflective of poor sleep health (e.g., duration, continuity, quality, variability; Buysse, 2014). We evaluate evidence for the model by first highlighting the shared underlying neurobiological regulatory mechanisms linking both early adversity and sleep disturbance to disease risk, with a specific focus on the brain, hypothalamic-pituitary-adrenal (HPA) axis, and immune system. This approach may simplify a highly complex set of biological interactions between sleep and responses to early adversity, but it provides a reasonable and relatively tractable starting point for scientific inquiry into the overlap between sleep and adversity.

Next, we review emerging work that points to sleep as a plausible mechanism linking early adversity to mental and physical health. We then offer two potential directions for future research. First, given that sleep disturbance often occurs during certain developmental transitions, such as adolescence and midlife, we propose that these normative sleep disturbances may act as potential “second-hits” for individuals with a history of early adversity. Second, studies can further test the model by examining whether sleep is a promising target of intervention for reducing the long-term health impacts of early adversity given its bidirectional relations with stress, biology, and health.

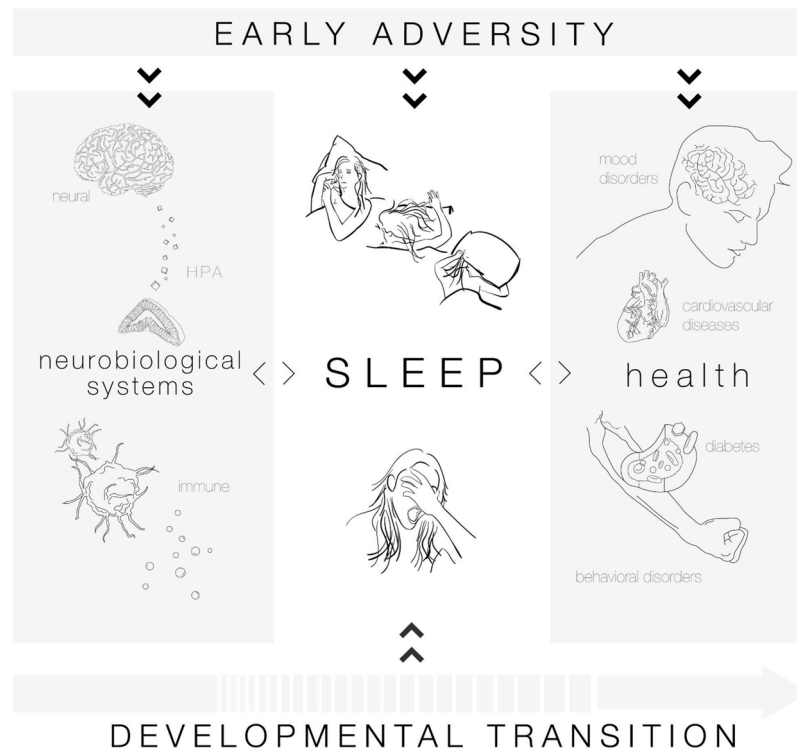


Fig. 1. Heuristic Model of Sleep Disturbance as a Mechanism of the Long-Term Impact of Early Adversity.

2. Shared neurobiological mechanisms of early adversity & sleep disturbance

Predominant models of the regulatory mechanisms by which early adversity impacts long-term health highlight brain development and functioning of the HPA axis and immune system as key pathways (e.g., Chiang et al., 2015; Danese and McEwen, 2012; Kim et al., 2018; Nusslock and Miller, 2016; Repetti et al., 2011; Tottenham, 2012; Tottenham and Sheridan, 2010). The brain, HPA axis, and immune systems dynamically interact with one another over the course of development to both influence growth and help orchestrate sleep, in part through the actions of key hormonal systems involved in growth, eating, and other homeostatic functions (Johnson et al., 2011; Kotronoulas et al., 2009).

Various early adversities involving threats of harm or unpredictability of environments, such as caregiving and maternal deprivation, abuse, and socioeconomic disadvantage, can alter structural and functional development of cortical and subcortical networks involving the amygdala, hippocampus, and prefrontal cortex (Chiang et al., 2015; Kim et al., 2018; McLaughlin et al., 2014; Nusslock and Miller, 2016; Tottenham, 2014, 2012; Tottenham and Sheridan, 2010). Together, these brain regions subserve cognitive, affective, social, executive, and self-regulatory processes, all of which are implicated in mental and physical health. The regions additionally interact as systems that help to regulate physiological and emotional vigilance to threats in the environment. As vigilance is diminished during sleep and greater vigilance can be more adaptive in adverse environments, it can be more difficult for those who experience early adversity to achieve healthy sleep (Dahl, 1996; Matthews et al., 2017; Silvers et al., 2017). Consequently, early adversity-related alterations in these areas can increase risk for poor health outcomes. Indeed, they have been linked to depression, post-traumatic stress disorder, borderline personality disorder, conduct disorder, and substance use disorders (Dom et al., 2005; Holz et al., 2015; Merz et al., 2018; Sala et al., 2004; Yang et al., 2016). These early-adversity alterations in the brain can also manifest as enhanced responses to threat and difficulties in regulating those enhanced

responses, in turn promoting engagement in poor health behaviors that give rise to disease (e.g., smoking, lack of exercise, poor diet) as a means of coping and greater or sustained biological responses to threat, which can eventually damage tissues and organs (Chiang et al., 2015; Miller et al., 2011; Nusslock and Miller, 2016). Supporting this notion, amygdala, hippocampus, and prefrontal cortical alterations have been linked to downstream markers of risk for cardiometabolic health problems, such as inflammation, heart rate variability, carotid intima-media thickness, and insulin resistance (Kraynak et al., 2018b, 2018a).

Two key regulatory systems highlighted in mechanistic models of early adversity and health include the HPA axis and the immune system. Substantial bodies of work provide evidence that early adversity can disrupt these systems. With respect to the HPA axis, various forms for early adversities have been associated with lower cortisol levels in response to acute stressors (Bunea et al., 2017) and with alterations in the cortisol awakening response, diurnal slope, and total cortisol output (Chiang et al., 2015; King et al., 2017; Kumari et al., 2013; Pendry & Adam, 2007). Alterations in HPA axis functioning, in turn, has been shown to predict worse mental and physical health outcomes including depression, externalizing disorders, obesity, and mortality (Adam et al., 2017; Turner et al., 2020). For the immune system, adversities such as maltreatment, socioeconomic disadvantage, peer victimization, household dysfunction, and targeted rejection, have been linked to chronic low-grade inflammation (Baumeister et al., 2016; Milaniak and Jaffee, 2019; Miller et al., 2011), which may be due to their links with heightened inflammatory responses to biological and psychosocial threat (Miller et al., 2011; Prather et al., 2013), and lower sensitivity to anti-inflammatory signals (Miller et al., 2011). Chronic low-grade inflammation, in turn, has been implicated in poor health outcomes, including depression, cardiovascular disease, and certain cancers (Pearson et al., 2003; Shrotriya et al., 2018; Valkanova et al., 2013). Beyond influencing HPA and immune processes in the periphery, early adversity may influence HPA and immune processes in the brain to elevate risk for poor health. For instance, prolonged exposure to glucocorticoids from the HPA axis can shape both neurogenesis and neural

functioning in ways that promote psychopathology (Lupien et al., 2009), and proinflammatory cytokines can affect reward, threat, and cognitive systems in ways that promote greater risk for depression, substance misuse, cardiometabolic problems, certain cancers, and autoimmune conditions (Nusslock and Miller, 2016).

Notably, virtually all of these neural and biological mechanisms have been implicated in sleep disturbances and are thought to at least partly mediate sleep's connection to poor health. Many studies have been done among adults, but an emerging body of research has documented similar patterns during childhood and adolescence. Neuroimaging studies among adults indicate that sleep deprivation results in greater amygdala responses to negative stimuli (Prather et al., 2013; Yoo et al., 2007), lower hippocampal engagement (Yoo et al., 2007), altered PFC function (Feng et al., 2018b; Ma et al., 2015), and decreased functional connectivity between the amygdala and PFC (Feng et al., 2018a) and between the hippocampus and PFC (Chee and Chuah, 2007). More normative sleep difficulties also have been linked to alterations in the structure and function of the corticolimbic regions with naturalistic studies showing that lower sleep duration and slow wave sleep are linked to reduced volumes in the hippocampus during childhood and adolescence, (Taki et al., 2012) and reduced volumes of the PFC and to decreased connectivity between the hippocampus and PFC among adults (Cheng et al., 2018). Sleep problems similarly have been linked cross-sectionally to altered PFC function and weaker corticolimbic connectivity during adolescence (Galván, 2019).

Sleep difficulties also have been tied to alterations of the HPA axis and immune system. Sleep deprivation, poorer self-reported sleep quality, lower actigraphy-assessed sleep efficiency, and more actigraphy-assessed sleep awakenings have been associated with increased HPA reactivity to stress and alterations in the cortisol awakening response among both children and adults (Fekedulegn et al., 2018; Kuhlman et al., undefined/ed; Leggett et al., 2016; Minkel et al., 2014; van Dalsen and Markus, 2018). Likewise, lab-induced sleep deprivation or restriction over several days results in enhanced inflammatory molecular signaling and elevated levels of circulating pro-inflammatory cytokines among adults, though not all studies show the same pattern for circulating cytokines (Irwin and Opp, 2017). Natural variations in sleep disturbances and shorter sleep duration are also linked to elevated levels of CRP and IL-6 (Irwin et al., 2016) and to poorer adaptive immune responses to vaccinations (Irwin, 2015) among adults. Linkages with inflammation also have been observed among adolescents, with poorer self-reported sleep quality and greater actigraphy-assessed sleep variability being associated with higher levels of CRP (Hall et al., 2015; Park et al., 2020) and shorter actigraphy-assessed sleep duration being related to an upregulation of inflammation-related genes (Chiang et al., 2019).

3. Sleep disturbances as a pathway in the early adversity-health link

Overlapping health effects and biological regulatory systems between early adversity and sleep suggest that sleep disturbance could function as a critical pathway linking early adversity to health. Indeed, a growing number of studies have begun to demonstrate a link early adversity to poor sleep across the lifespan.

Several epidemiological studies have documented cross-sectional associations between retrospective self-reports of early adversities such as maltreatment, accidents, disasters, and household dysfunction to greater self-reported sleep disturbances in adults (Greenfield et al., 2011; Koskenvuo et al., 2010; McWhorter et al., 2019). This association has been observed prospectively in relation to documented reports of sexual abuse among female adults (Noll et al., 2006) and extended to actigraphy-based measures of sleep in midlife adults (Brindle et al., 2018). The early adversity-sleep association also has been observed in earlier life stages. For instance, one study found that socioeconomic disadvantage at 12 months age predicted shorter sleep duration, longer

sleep latency, and greater sleep variability as assessed via actigraphy during middle childhood (Doane et al., 2018). Similarly, other studies have found that conflict at age 7–15 years predicted higher rates of insomnia at age 18 (Gregory et al., 2006), and that a lack of routine from ages 2–4 predicted more parent-reported sleep problems at 3, 6, and 10 years old and shorter sleep duration and longer sleep onset latency assessed using accelerometers at 11–12 years of age (Koopman-Verhoeff et al., 2019).

Additional evidence comes from several recent studies statistically testing mediation. One of these studies found that self-reported childhood abuse in midlife prospectively predicted functional limitations in physical activities (e.g., walking, carrying groceries, climbing stairs) nearly a decade later and that poor sleep quality explained this association (Mishra et al., 2019). A study of midlife adults found that greater sleep disturbances mediated an association between retrospective reports of early abuse and low-grade chronic inflammation, which in turn predicted elevated hypertension (Petrov et al., 2016). In other studies, poor sleep quality and greater sleep disturbances were shown to explain the links between childhood abuse and greater smoking behavior, and depressive symptoms in adults (Jones et al., 2018; Oshri et al., 2017) and smaller volumes of corticolimbic structures in late adolescents (Teicher et al., 2018).

Collectively, these findings provide initial evidence for sleep disturbance as a pathway linking early adversity to health across the lifespan. Sleep and health are intimately connected throughout development, and as described in the earlier sections, the insults experienced earlier in life can set up trajectories of growth and development through multiple systems that make it difficult to maintain optimal mental and physical functioning. However, it is important to bear in mind that these findings are largely based on retrospective self-reports of early adversity and subjective sleep. Further work based on more rigorous longitudinal designs of early adversity that incorporate repeated assessments of both subjective and objective assessments of sleep can help address these limitations. Additionally, research should explore the possibility that the trajectories of growth, development, and health established by early adversity may be exacerbated or perhaps ameliorated later in life through changes in sleep. As described below, research should: 1) focus on the particular role of heightened sleep disturbance that occurs during times of developmental transition, and 2) employ intervention paradigms to manipulate sleep and potentially mitigate the long-term effects of early adversity on health.

4. Sleep disturbance during developmental transitions as a “second hit”

Research efforts examining the role of sleep disturbance as a pathway linking early adversity to health should pay particular attention to periods of developmental transition. Maturation and contextual changes that occur at times of transition in the lifespan can interact to create periods at which sleep difficulties become more normative for the population. The endogenous circadian rhythm associated with the sleep-wake cycle demonstrates significant shifts from infancy, through adolescence and young adulthood, and into midlife and aging (Fischer et al., 2017; Roenneberg et al., 2004; Tonetti et al., 2008). Sleep pressure, the homeostatic mechanism by which the need for sleep increases during waking hours, changes in rate at different stages of life and interacts with the circadian rhythm to influence sleep (Cajochen et al., 2006; Jenni et al., 2005; Jenni and LeBourgeois, 2006). The sensitivity of the suprachiasmatic nucleus (SCN) to the effects of light in shifting the circadian rhythm may also change across development (Hagenauer and Lee, 2012). At the same time, developmental transitions typically involve changes in the social and structural constraints on sleep – school, work, parenting – that, in combination with simultaneous shifts in biology, can create self-perpetuating cycles of sleep disturbance that reach clinical significance and compromise health.

Adolescence is one such period of normative sleep disturbance. Sleep

pressure continues to slow during the teen years (Jenni et al., 2005; Jenni and LeBourgeois, 2006). The circadian sleep-wake cycle undergoes a phase-delay shift such that endogenous sleep and wake times move forward (Carskadon, 2011; Roenneberg et al., 2004). As a result, adolescents find it increasingly difficult to fall asleep and wake up as earlier as they did when they were younger. The collision of these maturational shifts with age-graded social and structural changes creates what appears to be a period of normative sleep disturbance for many youth in industrialized and post-industrial societies. School start-times move earlier during adolescence in many countries (Wahlstrom and Owens, 2017). The salience of peers, magnified by age-graded universal schooling and the ability to electronically interact with friends late into the evening, can push sleep times later. The same is true for easy consumption of electronic media. As a result, sizeable proportions of adolescents around the globe obtain fewer than the recommended range of 8–10 hours of sleep per night (Hirshkowitz et al., 2015; Keyes et al., 2015; Lehto et al., 2016; Paiva et al., 2016; Seo et al., 2017). Other features of healthy sleep – consistent durations, bed and wake times, and high quality, restful sleep – decline as children move into and through secondary school (Crabtree and Williams, 2009; Galland et al., 2018; Park et al., 2019; Pesonen et al., 2014; Sadeh et al., 2009).

Similar problems with sleep have been observed among adults transitioning from mid to later life. The endogenous circadian phase-delay shift observed during adolescence reverses direction around the age of 20 and continues to move earlier throughout the lifespan (Fischer et al., 2017; Tonetti et al., 2008). This may make bedtime easier, but it produces early wake times that coupled with evening activities can result in a reduction in total sleep time. Homeostatic and circadian changes relevant for sleep (Dijk et al., 2001; Kim and Duffy, 2018), in combination with physical difficulties, pain, and discomfort that accumulate with age, can make it challenging to obtain sleep of sufficient quantity (7–9 hours per night; Watson et al., 2015) and quality. Nightly sleep duration declines due to extended nighttime awakenings, an increasingly common complaint as individuals move through mid and later life (Ancoli-Israel and Ayalon, 2006; Foley et al., 1995). Older adults also have less restorative deep, slow-wave sleep during the night (Cauter et al., 2000). Adults during this period report more sleep problems than at any other time of life. Rates of insomnia nearly double among those older than 55 years as compared to adults between the ages of 30–50 years (Ohayon, 2002).

The normative increases in sleep disturbance at periods such as adolescence and mid to older adulthood may create a “second hit” for those with a history of early adversity who already experience altered biological regulation and compromised physical and mental health. The alteration in the HPA axis, proinflammatory profile, and greater anxiety and depression discussed earlier may set up those with early adversity for even worse sleep problems during these periods of normative sleep disturbance than their peers. More sleep problems, in turn, might amplify the HPA and inflammatory alterations of those with early adversity, creating an inflection point at which their risk for chronic adult health conditions such as obesity, Type II diabetes, and some cancers becomes elevated. Sleep disturbances also may interact with the dysregulation of these systems to potentiate risk for mental health disorders, such as depression, that can emerge during these developmental periods.

Such a “second hit” hypothesis requires longitudinal examinations of the model portrayed in Fig. 1 before, during, and after periods of normative sleep disturbance. To our knowledge, no such studies have yet been published (though it may be that some such datasets exist). Analyses could test two potential scenarios shown in Fig. 2. In one scenario, early adversity differences in sleep disturbance appear during a period of developmental transition such as adolescence (Panel A), altering neurobiological systems and bringing those with a history of early adversity past a threshold that increases risk for mental and physical health. A second possibility is that any early adversity differences in sleep disturbance already existed prior to the period of

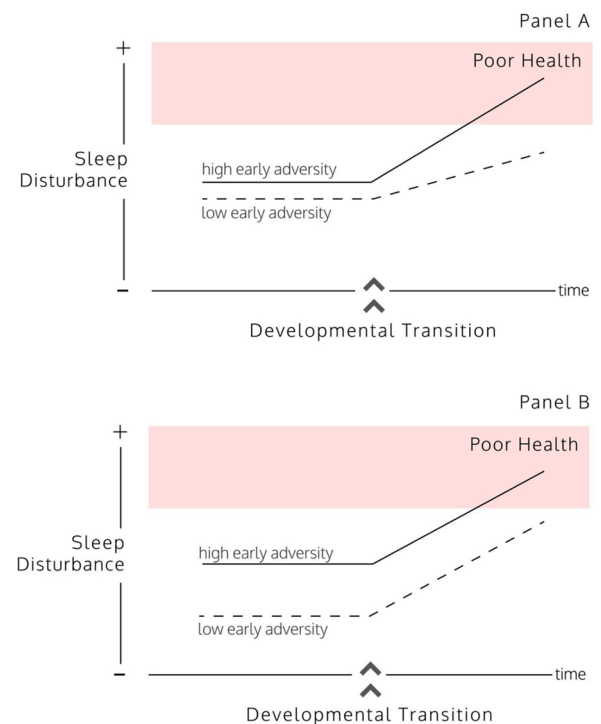


Fig. 2. Sleep Disturbances at Developmental Transitions as a “Second Hit” for Those with High vs. Low Levels of Early Adversity.

developmental transition, but normative increases in sleep disturbance bring those with a history of early adversity past the health risk threshold (Panel B). Such thresholds are becoming more apparent now that investigators have begun to examine non-linear associations between sleep and health (e.g., Eide and Showalter, 2012; El-Sheikh et al., 2019; Fuligni et al., 2018; James and Hale, 2017). Thresholds also are increasingly recognized by professional organizations issuing sleep recommendations that acknowledge that while sleep variations within a certain range may be fine (e.g., 7–10 hrs. for adolescents), going outside the range likely creates greater risk for health.

5. Improving sleep to mitigate the health impact of early adversity

The potential additional risk for sleep disturbance for those with early adversity highlights the need and value of intervening during the transition into and during developmental periods such as adolescence and midlife. The shared neurobiological pathways linking early adversity and sleep to health offer the intriguing possibility that improving sleep could reduce the impact of early adversity on long-term mental and physical health. Given that biological and psychological dysregulation are key mechanisms of the long-term impact of early adversity, targeting and improving sleep may provide a direct way to improve regulation across several systems simultaneously. Manipulating sleep through such interventions also would provide additional tests of the proposed role of sleep disturbance in the links between early adversity and health. To our knowledge, however, there have yet to be published empirical investigations of the use of sleep interventions to mitigate the impact of early adversity on physical or mental health.

Numerous observational studies highlight how better sleep can blunt the impact of stress on health in everyday life. In our own work, for instance, everyday family demands were associated with smaller cortisol awakening responses only among adolescents with longer sleep latency and lower sleep efficiency, but not among those with shorter sleep latency and higher efficiency (Chiang et al., 2016). Using the same data, we also found that family-related major life events were more strongly

associated with greater depressive symptoms among adolescents with lower sleep efficiency compared to those with higher sleep efficiency; the same pattern was observed for everyday family demands and everyday negative affect (Chiang et al., 2019, 2017; Kuhlman et al., 2020). Other labs have similarly documented this pattern of findings for other youth and other stressors. For example, perceived discrimination, peer victimization, and greater interparental conflict have also been more strongly associated with adjustment outcomes in adolescents with poorer sleep quality, lower sleep efficiency, and shorter sleep duration compared to their peers with better sleep (Lemola et al., 2012; Tu et al., 2015; Yip, 2015).

Sleep is a modifiable health behavior, with downstream effects on some of the same biological mechanisms and health outcomes as early adversity. Education in appropriate sleep hygiene, such as minimizing stimulation and maintaining a regular sleep schedule, is necessary but not sufficient (Cassoff et al., 2013; Friedrich and Schlarb, 2018; Morin et al., 1994). More targeted interventions are necessary. Medication demonstrates short-term effectiveness for insomnia, but brings risks of potential dependence, tolerance, and daytime dysfunction (Qaseem et al., 2016; Silber, 2005). Cognitive behavioral therapy for insomnia (CBT-I) is an empirically-proved method of improving sleep in both adolescents and adults, with effect sizes comparable to that of medication (Okajima et al., 2011; van Straten et al., 2018). CBT-I combines education about healthy sleep habits such as maintaining a regular schedule, controlling stimuli such as light and sound that can disrupt sleep, and managing dysfunctional thoughts and beliefs about sleep. Mindfulness-based practices that provide strategies for reducing reactivity and rumination have also evidenced sleep improvements similar in magnitude as CBT-I (Black et al., 2015; Blake et al., 2016; Rusch et al., 2018). Importantly, both of these approaches have been shown to be effective during both adolescence and later life, key times of normative sleep disturbance (Black et al., 2015; Blake et al., 2019, 2017). And they impact some of the impairments in psychological well-being and biological regulation associated with history of early adversity. These include improvements in affect, mood, fatigue, and a reduction in inflammatory processes (Black et al., 2015; Blake et al., 2019; Rash et al., 2019).

Non-therapeutic approaches that focus on the structural constraints on sleep offer the possibility of improving sleep for larger segments of the population. Changing second school start-times for adolescents and improving work schedules among adult workers improve the duration and regularity of sleep. Several experimental and observational studies of varying the times at which secondary schools begin in the morning have shown improvements in sleep duration and associated psychological functioning during the day (Dunster et al., 2018; Minges and Redeker, 2016). During mid and later adulthood, interventions that focus on work-life balance work schedules have shown improvements upon sleep (Robbins et al., 2019). For example, an intervention designed to promote employee control and flexibility in work schedules, as well as employer support for employees' work-family issues, significantly increased the sleep duration for all employees (Lee et al., 2016). There were additional positive effects for employees at the midlife transition (56–70 yrs. of age): reduced nap time during the day and decreased variability in waking during the night after the onset of sleep. One study suggested that a work-place intervention to promote employee control over work schedules actually improved the latency, variability, and quality of the sleep of the employee's adolescent children (McHale et al., 2015). Little research, however, has examined how school and work-place sleep interventions may influence key biological regulatory mechanisms such as HPA and inflammatory activity.

Although not targeted to those with a history of early adversity, community and structural-based approaches such as these could

nevertheless be beneficial for this population by lessening the extent to which they would encounter a "second hit" of normative sleep disturbance at key periods of development.

6. Conclusion

The time is ripe to bring together the sciences of early adversity and sleep – two flourishing fields with increasingly deep penetration into the study and treatment of physical and mental health across the lifespan. Shared health effects and underlying neurobiological mechanisms suggest potential payoff in our understanding of the legacy of stress, deprivation, and maltreatment in childhood. Methods and measures have been well-validated in each line of study and can be shared easily. Longitudinal designs, particularly those that cover developmental periods of normative sleep disturbance, are necessary to examine the experiential, neurobiological, and health pathways outlined in our model. Such designs could help to untangle the inherent bidirectionality in which neurobiological mechanisms and poor health influence each across the lifespan. Evidence-based sleep interventions exist and to our knowledge, have yet to be applied or adapted to populations with a history of early adversity. Collectively, these efforts could yield promising ways to help break the feed-forward cycles of dysregulation and poor health across the lifecourse, thereby mitigating the long-term health effects of early adversity.

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