AERA Open
July-September 2017, Vol. 3, No. 3, pp. 1–17
DOI: 10.1177/2332858417713488

Stress, Sleep, and Performance on Standardized Tests: Understudied Pathways to the Achievement Gap

Jennifer A. Heissel
Northwestern University
Dorainne J. Levy
Indiana University
Emma K. Adam
Northwestern University

Stress exposure, and subsequent biological responses to stress exposure, can negatively affect cognitive functioning and test performance. Stress exposure affects multiple biological systems, including sleep and the activity of the hypothalamic-pituitary-adrenal (HPA) axis and its primary hormonal product, cortisol. Sleep and cortisol interact with each other, and both affect cognitive performance. Stress, sleep, and cortisol also vary by race-ethnicity and socioeconomic status, and as a result, they may serve as potential pathways contributing to racial-ethnic and socioeconomic achievement gaps. Effects of stress on academic performance could occur either during the learning process or in response to the acute stress of testing. Interventions to limit stress exposure, reduce perceived stress, and promote optimal stress hormone levels and sleep may help students arrive in the classroom prepared to learn and to perform well on tests. These measures may particularly benefit low-income and minority students and may play a role in reducing achievement gaps.

Keywords: *testing, sleep, cortisol, HPA axis, stress/coping, achievement gap, high-stakes testing, educational policy*

Children from low–socioeconomic status (SES) and racial-/ethnic-minority groups score lower on average on standardized academic tests relative to high-SES and White families, respectively (Bradbury, Corak, Waldfogel, & Washbrook, 2015; Reardon, 2011). Many factors contribute to the achievement gap, including class size, teacher characteristics, parent participation, and hunger and nutrition (Barton & Coley, 2009). Although acknowledging these factors, the present paper focuses on an understudied component of the achievement gap: how biological stress responses associated with stress exposure affect performance on standardized tests. Stress exposures come from school, home, and neighborhood factors and can affect the functioning of multiple stress-sensitive biological systems. We focus here on two such systems: the hypothalamic-pituitary-adrenal (HPA) axis and sleep. As we show, stress-related alterations in these systems can affect students' learning and test-taking experience. We argue that achievement gaps are at least partly driven by differential stress exposure and biological stress responses between groups.

The Stress Disparity Model

Here we provide a brief overview of a theoretical model of how stress exposure and resulting biological stress responses might affect academic achievement as measured by standardized tests. We call this model the *stress disparity model*. Figure 1 displays the pathways of this model; Figure 2 focuses on the outcomes measured as an achievement gap. We present evidence for this model throughout the paper.

We begin by noting that low-SES and racial-/ethnic-minority children are more likely to be exposed to stressful life events relative to higher-income or White students (Pathway A; Hatch & Dohrenwend, 2007; Repetti, Taylor, & Seeman, 2002). Exposure to stressors affects the HPA axis, one of the body's key biological stress systems (Pathway B; Adam, 2012; Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004). HPA axis activation can be advantageous by assisting individuals in meeting the demands of daily life (Del Giudice, Ellis, & Shirtcliff, 2011), but long-term stress exposure can lead to changes in

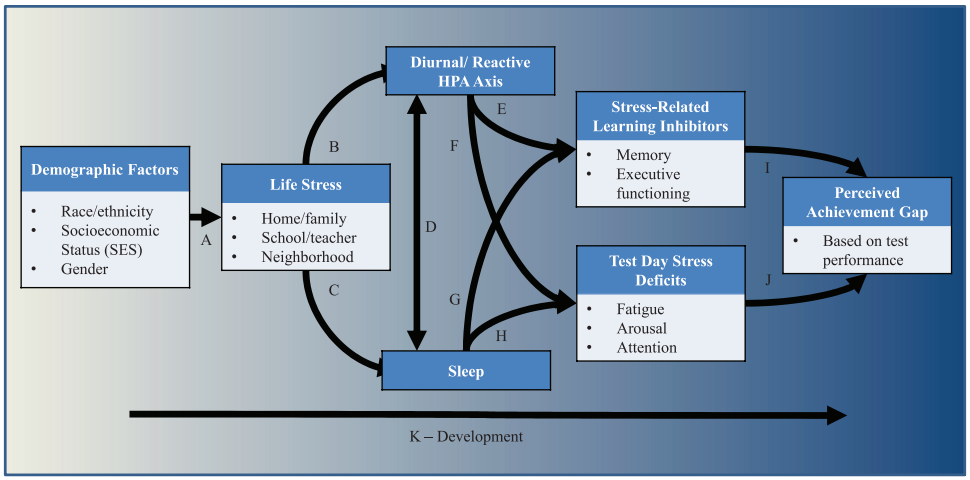

FIGURE 1. *Stress disparity pathways. Pathways of the stress disparity model lead from demographic factors to perceived achievement gaps. The outcomes component of the stress disparity model (Pathways I and J in this figure) are further elaborated in Figure 2.*

the HPA axis that can be maladaptive in some contexts (McEwen & Gianaros, 2010). HPA axis changes can affect students' ability to learn new material and their ability to respond to an acute cognitive challenge, such as a standardized test (Pathways E and F; Schwabe, Joëls, Roozendaal, Wolf, & Oitzl, 2012). Similarly, exposure to stressors can affect sleep (Pathway C; Sadeh, Raviv, & Gruber, 2000), which subsequently affects students' ongoing learning (Pathway G; Walker & Stickgold, 2006) and ability to function in the face of a cognitive challenge, such as an academic test (Pathway H; Gillen-O'Neel, Huynh, & Fuligni, 2013). The HPA axis and sleep systems can also affect one another (Pathway D; Zeiders, Doane, & Adam, 2011).

Bringing this literature together, we theorize that the effects of stress biology on students' learning before a test and their ability to bring optimal levels of arousal and cognitive readiness to the testing environment affect their performance on standardized tests (Pathways I and J, respectively). In Pathway K, we note that the transition to adolescence brings increases in stress exposure (Romeo, 2010), as well as changes to the HPA axis and sleep systems, which may make the impact of stress exposure on academic performance particularly important during adolescence (Gunnar, Wewerka, Frenn, Long, & Griggs, 2010; Jenni & Carskadon, 2012).

We provide a second figure to argue that the achievement gap, as measured by standardized tests, reflects a gap in actual knowledge and a gap in cognitive performance during test taking, both of which are affected by home, neighborhood, and other stressors. We explore these pathways in the review that follows.

Demographic Factors and Life Stress (Pathway A)

Low-SES and racial-/ethnic-minority children (particularly, Black children) in the United States are more likely than higher-SES or White children to experience negative life events, such as a very violent incident; harsh or inconsistent parenting; or parental divorce, death, or alcohol abuse (Glasscock, Andersen, Labriola, Rasmussen, & Hansen, 2013; Hatch & Dohrenwend, 2007; Repetti et al., 2002). Rising inequality (Duncan & Murnane, 2011; Heathcote, Perri, & Violante, 2010) may lead to increasingly divergent childhoods in low- and high-income families. Stressful events may also affect the way children interpret the world, with low-SES children more likely to interpret ambiguous events as hostile (Chen & Matthews, 2001).

Likewise, some racial-ethnic minorities face more stressors than White youth, on average. For instance, Black children are more likely to witness domestic violence than White children (Roberts, Gilman, Breslau, Breslau, & Koenen, 2011). Some of these stressors are income-related (Myers, 2008). Other stressors are specific to the experience of race-ethnicity, such as exposure to stereotype threat (i.e., the fear of confirming negative stereotypes concerning one's social group) and perceived discrimination (Adam et al., 2015; Clark, Anderson, Clark, & Williams, 1999; Levy, Heissel, Richeson, & Adam, 2016; Steele & Aronson, 1995). Past experiences with discrimination can increase vigilance for potential future racism (Bennett, Merritt, Edwards, & Sollers, 2004; Chen & Matthews, 2001; Myers, 2008).

Life Stress and the HPA Axis (Pathway B)

The biological stress response includes multiple systems, but we focus here on the HPA axis and its primary hormonal product, cortisol. Cortisol levels show a strong circadian rhythm across the day, known as the diurnal cortisol rhythm, with highest cortisol levels occurring shortly after waking and lowest levels occurring about 30 minutes after sleep begins (Adam, Hawkley, Kudielka, & Cacioppo, 2006). Researchers often measure the waking cortisol level and the

daily cortisol slope, which is the rate at which cortisol levels drop from wake to bedtime. They also measure the area under the diurnal cortisol curve (AUC), which is the estimated total cortisol exposure across the day from wake to bedtime. A sharp increase in cortisol in the 30 to 40 minutes after waking is called the cortisol awakening response, or CAR.

Real or perceived stressors can increase cortisol above typical diurnal levels (called acute cortisol reactivity; Adam, 2012; Miller, Chen, & Zhou, 2007; Sapolsky, Romero, & Munck, 2000). For routine stressors, cortisol levels return to the typical diurnal pattern approximately an hour after the stressor has passed (Adam, 2012; Miller et al., 2007). The cortisol stress response is typically adaptive rather than problematic: The HPA axis mobilizes psychological and physiological responses when presented with a stressor (Del Giudice et al., 2011; Shirtcliff, Peres, Dismukes, Lee, & Phan, 2014).

Normally, the CAR is helpful in promoting the transition from sleep to waking, and it is thought to provide an energetic boost to help individuals meet the expected demands of the upcoming day (Adam et al., 2006; Clow, Hucklebridge, Stalder, Evans, & Thorn, 2010; Doane & Adam, 2010; Fries, Dettenborn, & Kirschbaum, 2009; Vrshek-Schallhorn et al., 2012). Frequently experiencing large CARs may indicate the individual faces excessive perceived demands—or insufficient perceived resources to meet those demands (Adam et al., 2010; Vrshek-Schallhorn et al., 2012).

Chronic stress system activation can have substantial, long-lasting repercussions on HPA axis functioning. The concept of *allostatic load* suggests that the body undergoes "wear and tear" as it experiences repeated biological stress responses during stressful events (Juster, McEwen, & Lupien, 2010; McEwen & Stellar, 1993). Prolonged periods of hyperactivity of the HPA axis may be followed by *hypocortisolism*, wherein the AUC is lower, the cortisol slope is flattened, the CAR is muted, and the system no longer sufficiently activates in response to perceived stressors (Fries et al., 2009; Fries, Hesse, Hellhammer, & Hellhammer, 2005; Gunnar & Vazquez, 2001). This pattern is often accompanied by fatigue (Fries et al., 2005, 2009). In particular, low waking cortisol and nonexistent or very small CARs may not provide the individual with sufficient energetic mobilization (Adam et al., 2006). Exposure to international orphanages, serious financial strain, and physical abuse have been associated with hypocortisolism in children (Badanes, Watamura, & Hankin, 2011; Cicchetti & Rogosch, 2001; Koss, Mliner, Donzella, & Gunnar, 2016).

Changes to the stress response system are not necessarily maladaptive. The adaptive calibration model argues that humans take in environmental inputs and calibrate their stress response system to match their surroundings (Del Giudice et al., 2011; Shirtcliff et al., 2014). High or rising cortisol is expected in cases when individuals are engaged with their environment, are in situations that are personally relevant, or are facing a difficult (but manageable) challenge; low or diminishing cortisol is expected in cases when an individual is disengaged from the environment or a challenge is impossible or no longer novel (Del Giudice et al., 2011; Dickerson & Kemeny, 2004; Shirtcliff et al., 2012, 2014). The particular diurnal pattern likely depends on what is adaptive in the primary socialization environment. For instance, whereas physically abused children display symptoms of shutting out the world through hypocortisolism, children who experience sexual abuse tend to have a hypervigilant pattern that maintain high levels of cortisol throughout the day (Cicchetti & Rogosch, 2001). As we argue in more detail below, changes that are adaptive for the child's primary context may not serve children well in school settings.

SES and Racial-Ethnic Differences in Cortisol Patterns (Pathways A and B)

On average, low-SES and Black individuals have flatter diurnal cortisol slopes, with both lower wake-up and higher bedtime cortisol levels, relative to higher-SES and White individuals (Adam, 2012; S. Cohen et al., 2006; DeSantis et al., 2007; DeSantis, Adam, Hawkley, Kudielka, & Cacioppo, 2015; Hajat et al., 2010). Researchers occasionally, but not consistently, find HPA axis patterns differ between Hispanic and non-Hispanic White populations (DeSantis et al., 2007; Hajat et al., 2010). These differences by group are hypothesized to occur due to differences in stress exposure and resulting changes to the HPA axis (DeSantis et al., 2007).

Not all low-SES or racial-/ethnic-minority individuals exhibit the flattened rhythms associated with higher stress exposure, particularly among those with lower levels of life stress or those who have positive coping resources, such as positive parenting, high family or community support, or strong racial-ethnic identities (Ai, Aisenberg, Weiss, & Salazar, 2014; Levy et al., 2016; Taylor & Stanton, 2007; Thoits, 2011). There are also likely differences in susceptibility to environmental influences (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Boyce & Ellis, 2005).

We acknowledge that resources such as coping and buffering processes may affect which individuals experience the optimal levels of stress reactivity (Gilbert, Mineka, Zinbarg, Craske, & Adam, 2017; Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996). These differences in resources may also play a role in both learning and test-taking performance. On average, however, racial-ethnic minorities and low-SES students experience more life stress and show differences in HPA axis functioning, which may be the result of chronic stress.

HPA Axis, Learning, and Cognition (Pathways E and F)

The level of cortisol that students bring into the classroom is a combination of their diurnal pattern and their acute

stress responses, which may have implications for learning new material and mentally retrieving material during tests.

HPA Axis and Learning (Pathway E)

We review evidence here suggesting that HPA axis functioning has implications for the learning that occurs in the weeks and months leading up to a high-stakes test. Learning is a complex phenomenon (e.g., Bransford, Brown, Cocking, Donovan, & Pellegrino, 2000), and we do not review all aspects of it here. Instead we focus on memory formation and student attention, both of which are important for learning and are associated with HPA axis functioning.

Successful learning requires both forming new memories and making connections to old ones. Memory formation is composed of multiple systems, only some of which are affected by the HPA axis (Schwabe et al., 2012; Squire, 2004). Memories are initially formed after engagement with a novel input. Memory consolidation occurs as the memory is stabilized, maintained, reorganized, and integrated with past experience and knowledge (McKenzie & Eichenbaum, 2011). Cognitive performance involves both memory consolidation and retrieval, which occur continuously and simultaneously while awake (Roozendaal, 2002).

The relationship between stress and learning is complex, and whether stress helps or hampers cognition depends on the situation. Sandi and Pinelo-Nava (2007) identify several important factors in their model of how stress affects learning: learning type (e.g., spatial learning), whether the stress is intrinsic (related to the cognitive task) or extrinsic (originating from the outside world and unrelated to the cognitive task), duration (acute or chronic), stressor intensity, and timing relative to the memory phase (e.g., during memory acquisition, storage, or retrieval).

Although the complexities noted above provide exceptions, worse cognitive performance typically occurs at zero or high levels of stress response and better performance occurs at moderate levels of stress response, resulting in an inverse-*U*-shaped association between cortisol levels and cognitive performance (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; Salehi, Cordero, & Sandi, 2010). At low levels of stress and cortisol, arousal and energy may not be sufficient to successfully engage the task at hand; at higher levels, performance begins to be impaired. This may be partially explained by the different types of receptors to which cortisol binds. Specifically, cortisol attaches to two types of receptors: mineralocorticoid receptors (MRs) and glucocorticoid receptors (GRs; Gunnar & Quevedo, 2007). Cortisol binds more readily to MRs, and most MRs are occupied before cortisol begins to bind to GRs. The effects of GRs and MRs tend to counteract each other, in part as a mechanism to shut off the stress response (Gunnar & Quevedo, 2007). Cortisol attachment to MRs can improve memory and focus by enhancing neural plasticity and increasing glucose availability in the brain, whereas GR attachment hampers plasticity and glucose availability (de Kloet, Oitzl, & Joëls, 1999; Gunnar & Quevedo, 2007; Lupien et al., 2007).

Below, we discuss the relationship between both acute and chronic stress and learning. Although we acknowledge that some students are substantially stressed by learning itself, our main concern involves the impact of acute and chronic extrinsic stress on student learning.

Acute stress and learning. Acute extrinsic stress unrelated to the task can affect learning. Learning in stressful situations may lead to more rigid, habit-based memory formation rather than more flexible memories that connect concepts together (Schwabe & Wolf, 2010; Schwabe, Wolf, & Oitzl, 2010). As humans evolved, it may have been beneficial to have improved attention to and memory of stress-related material in particular. We argue that this form of memory consolidation may not be adaptive in a school learning environment, where students need to focus on and connect a broad range of material largely unrelated to the stressors in their lives.

In rats, the relationship between external acute stressors and cognition appears only in more complex cognitive tasks (e.g., when the reward is moved, thus requiring more flexible thinking; Sandi & Pinelo-Nava, 2007). Time of day may matter as well, perhaps because GRs are less likely to be occupied in the afternoon. Administration of exogenous cortisol in the morning, when levels are typically high, negatively affects memory formation, whereas the same in the afternoon, when levels are typically lower, has a small positive effect (Het, Ramlow, & Wolf, 2005). Although the effects are complex and may involve other biological processes besides the HPA axis (Sauro, Jorgensen, & Pedlow, 2003; Shields, Sazma, & Yonelinas, 2016), increases in cortisol due to acute stressors unrelated to the learning context are thought to hamper memory formation (Joëls, Pu, Wiegert, Oitzl, & Krugers, 2006).

Chronic stress and learning. Chronically stressful situations are also associated with a reduced ability to learn, perhaps due to the associated damage to the hippocampus (Lupien et al., 1998; Sandi & Pinelo-Nava, 2007). Children and adolescents who report high daily stress have lower levels of cortisol at waking and during the CAR relative to those who report lower stress; these same children also have slower memory speed and reduced attention continuity relative to their less-stressed peers (Maldonado et al., 2008). In research on adults, people with more dynamic HPA activity (namely, a higher CAR and a steeper slope of decline in cortisol levels across the day) have better executive functioning and cognitive performance than those with smaller CARs and flatter slopes (Clow et al., 2014; Evans et al., 2011; Evans, Hucklebridge, Loveday, & Clow, 2012). Perhaps the flattened cortisol rhythms displayed by those with chronic stress exposure mean that the HPA axis does not properly deploy to respond optimally to challenges or focus attention on important tasks (Adam et al., 2006).

In summary, we contend in Pathway E that the difficulty focusing and connecting concepts negatively affects students' ability to learn under acute and chronic stress.

HPA Axis and the Acute Stress of Test Taking (Pathway F)

HPA axis activity may affect cognitive performance during test taking through effects on arousal, attentional focus, and memory recall (Abercrombie, Kalin, & Davidson, 2005; Chapotot, Buguet, Gronfier, & Brandenberger, 2001; Joëls et al., 2006). However, the effects of stress on these systems may differ depending on whether the stress is intrinsic (related to the test) or extrinsic (not related to the test) and, for extrinsic stress, whether it is acute or chronic (Sandi & Pinelo-Nava, 2007). We argue that students may perform best on tests if they feel moderately challenged and their cortisol levels are responsive to that challenge such that they feel energetic and alert. We further argue that students will perform worse when facing overwhelming psychological and biological stress levels, particularly when the stress is from sources extrinsic to the test itself. Below, we further elaborate and provide evidence for Pathway F.

Arousal and attention. Higher levels of cortisol correspond to greater arousal, which can at times even offset drowsiness due to short-term sleep deprivation (Abercrombie et al., 2005; Chapotot et al., 2001). Children from lower-income backgrounds exhibit a lower ability to filter irrelevant information relative to higher-income children (Stevens, Lauinger, & Neville, 2009), but among a group of low-income Head Start students, moderate cortisol reactivity was associated with better self-regulation, including better attention, relative to low-income Head Start students who displayed limited cortisol reactivity (Blair, Granger, & Razza, 2005). In our context, increased arousal and attention may allow students to focus on the test and improve measured performance.

Recall. Intrinsic stress is more likely to help recall, whereas extrinsic stress is more likely to hamper it (Joëls et al., 2006; Sandi & Pinelo-Nava, 2007). As with learning, the relation of cortisol levels to memory recall likely has an inverse-*U* shape, with the best outcomes with a moderate amount of perceived stress and moderate activation of the HPA axis (Lupien et al., 2002).

Individual differences and coping with intrinsic stress. Experience of the same test may not necessarily produce the same biological stress response in all students. Differences in anxiety and ability can change the relationship between cortisol and task performance, with stronger relationships in more difficult tasks and for those who perceive the test as stressful (Malarkey, Pearl, Demers, Kiecolt-Glaser, & Glaser, 1995; Mattarella-Micke, Mateo, Kozak, Foster, & Beilock, 2011; Schlotz, Schulz, Hellhammer, Stone, & Hellhammer, 2006). Positive coping strategies may also reduce the effects of stress on test performance, although this is in need of further study, particularly with inclusion of biological stress measures and in school-age children (M. Cohen, Ben-Zur, & Rosenfeld, 2008; Struthers, Perry, & Menec, 2000). One study that did focus on school-age children showed that greater perceived stress during an academic test was associated with lower test performance and a larger increase in cortisol during the test (Lindahl, Theorell, & Lindblad, 2005). Moreover, students who reported reacting to demanding academic tasks with "I say to myself: I can solve this task" had a lower morning CAR; students who said they responded to demanding academic tasks with "I get worried and will have problems solving other tasks too" had a larger increase in cortisol during the testing period.

Impact of acute extrinsic stress. Acute extrinsic stress unrelated to the test can also affect test outcomes. Researchers can study this relationship in multiple ways: by synthetically administering cortisol, creating stressful situations in labs, or finding naturally occurring stressors. When cortisol is administered synthetically, humans generally have worse memory recall (Het et al., 2005), with larger effects for more emotional information (Kuhlmann, Kirschbaum, & Wolf, 2005). There is again an inverse-*U* shape to this recall pattern, with the best performance when small doses of cortisol are synthetically supplied and the worst outcomes with zero or very large doses (Schilling et al., 2013).

Studies in laboratory settings often employ a protocol called the Trier Social Stress Test (TSST), which involves giving a speech in front of others or via videotape. Following the speech and subsequent increase in cortisol, studies generally find decreases in recall ability (Kuhlmann, Piel, & Wolf, 2005; Smeets, Jelicic, & Merckelbach, 2006). Some studies also find a decrease in working memory, but others find no effect (Hoffman & al'Absi, 2004; Kuhlmann, Piel, et al., 2005; Schoofs, Preuß, & Wolf, 2008; Smeets et al., 2006). In rats, more flexible forms of memory recall are disrupted by the presence of a cat, but less complicated cognition is not (Sandi & Pinelo-Nava, 2007), indicating that more difficult tasks may be more susceptible to the detrimental effects of acute extrinsic stress.

There is also some evidence that acute external stressors can affect test performance in the real world. Students exposed to a homicide within a block of their homes 1 to 7 days before an academic assessment scored over half a standard deviation lower on vocabulary and reading tests, relative to students from the same neighborhood who happened to be tested on days without recent homicides (Sharkey, 2010). Despite similar experiences in the months leading up to the assessment, an acute external stressor

affected student outcomes during the test-taking period. Similarly, being near the Beltway Sniper attacks was associated with drops in academic performance, particularly in schools with high proportions of racial-minority and low-SES students (Gershenson & Tekin, 2015). Although the authors did not examine neurobiological mediators of these effects, changes in the HPA axis are one plausible candidate. In our own research, acute local violent crimes are associated with increases in adolescents' CAR on the day immediately following the crime, relative to days without crime for the same student (Heissel, Sharkey, Torrats-Espinosa, Grant, & Adam, in press).

Impact of chronic extrinsic stress. We also argue that chronic extrinsic stress can change how students respond to the acute intrinsic stress of a test. Individuals in long-term stressful situations have flattened diurnal cortisol rhythms and dampened cortisol reactions to acutely stressful situations (Juster et al., 2010; McEwen & Stellar, 1993). Reduced cortisol reactivity may limit students' ability to respond to challenges such as tests. In research on low-income children in Head Start, those with higher cortisol reactivity had better cognition and behavior outcomes than children without a cortisol response (Blair et al., 2005). When the CAR is synthetically suppressed in adults, recall of text and pictures learned 3 days earlier is reduced (Rimmele, Meier, Lange, & Born, 2010).

Overall, the research supporting Pathway F suggests that a moderate increase in cortisol in response to the acute stress of a test may improve students' concentration in the testing environment. However, too much cortisol, whether due to overabundant test anxiety or acute external stressors, may cause impairments in cognitive performance. We argue that students with chronic out-of-school stress may have dampened biological responses to the testing situation, which could inhibit their ability to respond optimally to the test. These patterns have implications for estimated achievement gaps if more-advantaged populations are more likely to have optimal reactions to the test.

Stress, Sleep, and Academic Performance (Pathways C, G, and H)

Sleep is an important influence on cognitive functioning. Sleep can be measured in everyday settings both for quantity (e.g., asking participants, "How many hours did you sleep last night?") and quality (e.g., "How rested do you feel?). It can also be objectively measured using actigraphy/accelerometry tools like sleep watches for quantity (e.g., how many hours are spent sleeping) and quality (e.g., sleep efficiency, a measure of the percentage of time from the first start of sleep at night to the final end of sleep in the morning that is spent in waking bouts). Children and teenagers in the United States sleep less than recommended (Adam, Snell, & Pendry, 2007; National Sleep Foundation, 2004, 2006).

Stress Exposure and Sleep (Pathway C)

Although sleep is affected by individual biological rhythms, daily schedules, and activity choices (Adam et al., 2007), sleep is also a stress-sensitive system. Perceived stressors contribute to changes in both sleep quantity and quality (Hanson & Chen, 2010; Hicken, Lee, Ailshire, Burgard, & Williams, 2013; Sadeh, 1996; Sadeh et al., 2000). In children, separation from loved ones, child abuse, natural disasters, and acute violence are associated with poor sleep (Sadeh, 1996). Abused children take longer to fall asleep and have more nocturnal activity than a comparison group of children (Glod, Teicher, Hartman, & Harakal, 1997). Family stress (based on loss, illness, hospitalization, moving, and emotional turmoil) is associated with more night wakings and lower sleep efficiency (Sadeh et al., 2000).

Acute and chronic stress may have different effects on different aspects of sleep, although to our knowledge this has not been studied comprehensively. Still, the effects of stress exposure on sleep likely have a dose-dependent relationship, with more traumatic stress having larger effects. In our own research, we show that children exposed to a violent crime have later bedtimes and less sleep, relative to that same child's nights without nearby violent crimes (Heissel et al., in press). The effects are larger for a nearby homicide or sexual assault than a nearby robbery, and they are also larger the closer the crime is to the home. The effects disappear after the night immediately following the violent crime (Heissel et al., in press).

More traumatic events, with more direct exposure, might have larger and longer-lasting effects. One study examined children exposed to a sniper attack on an elementary school playground, in which one child and a passerby were killed and 13 children were injured (Pynoos et al., 1987). One month after the event, 77.1% of students who had been on the playground during the attack reported sleep disturbances. In contrast, some of the students in this year-round school happened to be on vacation that week and were not exposed. Among those students, only 23.8% reported sleep disturbances. For children who were at school but not on the playground that day, rates fell between those extremes (Pynoos et al., 1987). This study demonstrates that very traumatic acute events can affect sleep for weeks. Childhood psychosocial stressors, such as family conflict, can also exert long-term effects on the relationship between stress and sleep outcomes through early adulthood (Hanson & Chen, 2010).

SES and Racial-Ethnic Differences in Sleep (Pathways A and C)

Black individuals, particularly those who experience greater perceived racial discrimination, and low-SES adults have lower average sleep quantity and quality, relative to White and high-SES groups (Buckhalt, El-Sheikh, & Keller, 2007; Mezick et al., 2008; Petrov & Lichstein, 2015; Thomas,

Bardwell, Ancoli-Israel, & Dimsdale, 2006). Buckhalt (2011) posits that sleep differences contribute to the SES achievement gap, as sleepy children cannot focus as well as more alert children in the classroom. We further argue that poor sleep may contribute to the Black/White achievement gap. Black Americans have shorter sleep duration, lower sleep efficiency, different sleep structures, and a greater prevalence of sleep disorders than their White counterparts (Mezick et al., 2008; Petrov & Lichstein, 2015; Ruiter, DeCoster, Jacobs, & Lichstein, 2010, 2011; Tomfohr, Ancoli-Israel, & Dimsdale, 2010). In turn, sleepiness in Black students is associated with a bigger drop in performance than it is for non-Black students (Bub, Buckhalt, & El-Sheikh, 2011).

There is some evidence that differences in sleep by SES and race are at least partially mediated by differences in stress, at least among adults (Hall et al., 2009; Sekine, Chandola, Martikainen, Marmot, & Kagamimori, 2006). Race-based stress in particular is associated with differences in sleep architecture, sleep timing, and subjective sleep quality between White and racial-/ethnic-minority populations (Thomas et al., 2006; Tomfohr, Pung, Edwards, & Dimsdale, 2012). Although differences in stress exposure have been hypothesized to be a part of the sleep gap among children (Buckhalt, 2011), this is an area of our model in need of additional study among children.

Sleeping, Learning, and Testing (Pathways G and H)

Students with less sleep and higher reported levels of sleepiness generally have lower grades and alertness (Bub et al., 2011; Dewald, Meijer, Oort, Kerkhof, & Bögels, 2010; Sadeh, Gruber, & Raviv, 2003; Wolfson & Carskadon, 1998, 2003). Sleep is important both in the learning process and during testing for several reasons, including its contributions to memory formation and recall, emotional functioning and behavior, and executive functioning. First, sleep is essential to the memory formation and consolidation processes that affect learning in adults (Fogel & Smith, 2011; Maquet et al., 2000; Sasaki et al., 2007; Walker & Stickgold, 2006). Patterns of brain activity exhibited during learning tasks are replayed during sleep, as the brain continues to learn and connect concepts (Maquet et al., 2000; Sasaki et al., 2007). This suggests that adequate sleep is important in the consolidation of daily learning. We note, however, that sleep duration and efficiency are not consistently related to memory in children (ages 5 to 12), perhaps due to the immaturity of their still-developing brains (Astill, Van der Heijden, Van IJzendoorn, & Van Someren, 2012). The relationship may also be task dependent; increased sleep in children has been shown to improve declarative memory but not procedural memory (Wilhelm, Diekelmann, & Born, 2008).

Second, sleep matters for behavioral and emotional functioning during the day. The consequence of insufficient sleep—more fatigue, sleepiness, and difficulty concentrating (Alapin et al., 2000; Baum et al., 2014)—can affect students' ability to learn and their ability to perform during a test. Fatigued students may take fewer notes during learning or skip more questions on the test, although we know of no studies that have studied this. A recent meta-analysis confirms that sleep duration is consistently associated with both internalizing and externalizing behavior problems in children (Astill et al., 2012), which could also disrupt learning and test-taking performance in students.

Third, sleep matters for the regulation of cognitive processes. In cross-sectional studies, more sleepiness and lower sleep quality are associated with worse executive functioning (e.g., Anderson, Storfer-Isser, Taylor, Rosen, & Redline, 2009; Sadeh, Gruber, & Raviv, 2002). Experimental studies in children are rare, but what evidence exists does support a strong relationship between sleep duration and executive functioning. Of eight experimental studies, longer sleep duration is consistently associated with improved executive functioning, whereas sleep efficiency has no relationship with executive functioning (Astill et al., 2012).

In adults, 24 hours of sleep deprivation is associated with worse executive functioning and more errors but only on more difficult tasks (Joo, Yoon, Koo, Kim, & Hong, 2012). Total sleep deprivation studies may not be ethically possible in children and adolescents, but partial sleep deprivation/extension studies do exist. In one experiment that extended or reduced children's sleep by an hour, students (ages 10 to 13) with extended sleep demonstrated significantly better performance on sustained visual attention, response inhibition, and motor control (Sadeh et al., 2003). These measures, which the authors link to school performance, could matter for learning or test taking. In similar studies, sleep-deprived adolescents (ages 14 to 17) also had reduced executive functioning according to parent and student self-reports (Baum et al., 2014; Beebe et al., 2008). Moreover, the weeklong sleep restriction led to reduced attention and worse test scores in a simulated classroom with educational videos and quizzes (Beebe, Rose, & Amin, 2010).

To this point, we have reviewed sources of acute and chronic stress that are external to the learning and testing environment. However, the stress of examinations themselves can affect sleep. In one study, high school students slept about an hour less per night during the regular school week than they did during a week of vacation, and they compensated for this decrease in sleep during the school week by increasing sleep efficiency (Astill, Verhoeven, Vijzelaar, & Van Someren, 2013). Students slept about 20 minutes less per night during a week of testing than during a regular school week, and their sleep efficiency decreased (Astill et al., 2013). Future studies should examine to what extent stress hormones, such as cortisol, mediate the different sleep patterns observed during stressful and nonstressful periods for students. Notably, not all students will respond to school-related stress in the same way. A high emotion–focused coping strategy predicted a reduction in sleep time among students being

evaluated for acceptance to graduate programs in clinical psychology, with no relationship to sleep for those who demonstrated problem-focused coping or disengagement (Sadeh, Keinan, & Daon, 2004).

Sleep may be more important on a regular school day than an exam day, as anxiety or motivation can temporarily displace sleepiness in affecting test performance (Chapotot et al., 2001; Chmiel, Totterdell, & Folkard, 1995; Horn & Dollinger, 1989; Wolfson & Carskadon, 2003). Thus, there may be mechanisms that can reduce the importance of sleep on the test day that do not come to students' aid during learning. Notably, students who sacrifice sleep for additional studying have more trouble with tests and understanding material taught in class the following day (Gillen-O'Neel et al., 2013), indicating that lack of sleep on test days does matter.

HPA Axis Activity and Sleep (Pathway D)

The effects of perceived stressors on the HPA axis and on sleep are not independent but, rather, interact with one another in ways that may accentuate effects on academic outcomes. The HPA axis helps regulate the sleep-wake cycle, and HPA axis alterations associated with acute or chronic stress exposure can change sleep timing and architecture (Clow et al., 2010; Van Reeth et al., 2000). Higher afternoon cortisol levels in children have been associated with worse subjectively and objectively measured sleep quality as well as shorter objectively measured sleep duration (El-Sheikh, Buckhalt, Keller, & Granger, 2008). Among adolescents, more hours of objectively measured sleep and a later waking time predicted a higher waking cortisol level and steeper cortisol slope over the day, and higher waking cortisol and steeper cortisol slope subsequently predicted more sleep the next evening (Zeiders et al., 2011). A 24-hour period of sleep deprivation increased stress hormones, including cortisol, in adult men (Joo et al., 2012). In children, shorter sleep duration was associated with a higher and longer-lasting CAR and elevated cortisol levels throughout the day (Räikkönen et al., 2010). Relative to those with average or high sleep efficiency, those with lower sleep efficiency demonstrated a larger cortisol increase after exposure to the TSST for Children (Räikkönen et al., 2010). In other words, those with poor sleep quality had a larger cortisol reaction to a stressful situation.

Cortisol also plays a role in the memory consolidation process that occurs during sleep. Artificially elevating cortisol during sleep impaired consolidation of newly learned words, and inactivation of the GRs may be an essential part of the memory consolidation process during sleep (Plihal & Born, 1999).

Overall, we argue that stress-related changes in the HPA axis and sleep can interact with one another to influence and exacerbate each other's impacts on cognitive performance during learning and test taking.

Gender Differences and Developmental Changes in Stress and Sleep (Pathway K)

Although we do not focus extensively on developmental changes in this paper, we note that the transition from childhood to adolescence is associated with changes in many of the variables in the model. As children move into adolescence, they are exposed to multiple novel stressors, including stress associated with puberty and additional social and academic demands (Arnett, 1999; Compas, Orosan, & Grant, 1993; Romeo, 2010). Adolescents have higher cortisol levels and flatter slopes relative to younger children, and HPA axis reactivity also increases with the pubertal transition (Gunnar et al., 2010; Romeo, 2010; Shirtcliff et al., 2012; Stroud et al., 2009). At the same time, adolescents experience delays in their circadian rhythm that mean they are biologically inclined to stay up later than younger children and adults (Carskadon, Acebo, & Jenni, 2004; Crowley, Acebo, & Carskadon, 2007; Jenni & Carskadon, 2012; Laberge et al., 2001; Sadeh, Dahl, Shahar, & Rosenblat-Stein, 2009). Changes in social and activity demands and in how strictly parents enforce bedtimes may also be a factor (Adam et al., 2007). Academic schedules are rarely synchronized with adolescents' naturally "owl-like" sleep patterns (American Academy of Pediatrics, 2014; Kirby, Maggi, & D'Angiulli, 2011), leading to decreases in sleep quantity (Crowley et al., 2007; Jenni & Carskadon, 2012). Increases in stress exposure, increases in biological stress reactivity, and changes in sleep resulting in increased sleep deprivation imply that the impact of stress exposure on academic performance may be particularly important during adolescence.

There are also differences in stress responses by gender that we do not thoroughly explore in this paper (Del Giudice et al., 2011; Sauro et al., 2003; Stroud, Salovey, & Epel, 2002; Weekes et al., 2006). In particular, males may be more responsive to achievement-related stressors, whereas females may be more responsive to social rejection (Stroud et al., 2002). This may mean that our model is particularly salient for adolescent males.

Stress Disparity Outcomes (Pathways I and J)

Throughout this paper, we have argued that differences in life stress affect HPA axis functioning and sleep patterns, which in turn lead to changes to learning and test-taking performance. This section introduces the outcomes component of our model, which explains how differences in knowledge and testing performance can create achievement gaps. The primary contribution of the outcomes component of the overall model is to make clear that the observed achievement gap reflects the cumulative effects of stress exposure on both learning and cognitive readiness for the test-taking environment as well as the differences in input and ability that tests are meant to measure.

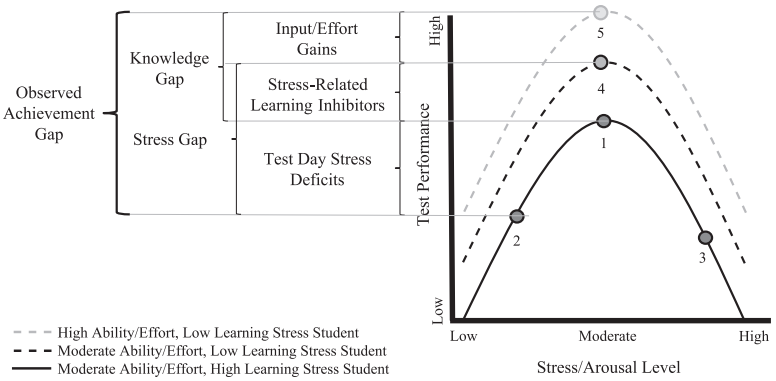

FIGURE 2. *Stress disparity outcomes. The observed achievement gap is composed of input/effort gains, stress-related learning inhibitors, and test-day stress deficits. Figure displays three hypothetical students with the following characteristics: (a) moderate inputs/effort (from students, families, teachers, and schools) and high stress during the learning period (solid black line), (b) moderate inputs/effort and low stress during the learning period (dashed black line), and (c) high inputs/effort and low stress during the learning period (dashed gray line). Each inverse U-shaped curve represents potential performance on the standardized test on the testing day, with optimal performance occurring with moderate stress. Learning determines students' potential curve on testing day, and their response to the test determines where they fall on the curve on the day of the test. Together, stress-related learning inhibitors and the test-day stress deficits form the stress gap due to differences in stress exposure during learning and the stress response during the test, and stress-related learning inhibitors and input/effort gains form the knowledge gap due to differences in learning.*

We call differences in what groups of students know in the days leading up to the test a *knowledge gap*. The knowledge gap accumulates across days and years of schooling and is broken into two parts: (a) *input and effort gains* attributed to the work of students, families, and schools and (b) *stress-related learning inhibitors* attributed to attention and memory problems induced by stress-related HPA axis and sleep dysregulation. The combination of input/ability gains and stress-related learning inhibitors can lead to a gap in the knowledge base that groups of students bring to the testing situation, depicted as different curves in Figure 2.

We argue that in addition to differences in knowledge, student performance on a standardized test is also affected by differences in stress/arousal during testing, or what we call a *test-day stress deficit*. This response, depicted as specific points on the curve in Figure 2, represents how a student performs on the test day, although the availability of an appropriate acute biological stress response may be influenced by longer-term stress exposure. The deficit is the difference between a student's peak potential performance that day (the top of the inverse-*U* curve) and where they actually perform. Moderate intrinsic stress, to the extent that it leads to students being alert and focused on the test, improves performance in the model, as it results in performing at the peak of the curve. We argue that it is the students with either no reaction or a very large reaction to the test who experience this deficit. The test-day stress deficit can create a divergence between what students know and how much they are judged to know by a given standardized test. The *stress gap* is the combination of the test-day stress deficit and stress-related learning inhibitors.

Researchers currently do not have data that can distinguish between input and effort gains, stress-related learning inhibitors, and test-day stress deficits, but it seems unlikely that stress gaps and demonstrated ability are independently distributed. Low-achieving students may be more likely to be burdened both by true input/effort gaps and stress-induced achievement gaps, for an overall higher *observed achievement gap*. For instance, the gap between Point 2 and Point 5 in Model 2 includes differences in input/effort gains, stress-related learning inhibitors, and test-day stress deficits.

The model presented in this section is a simplification of the complicated, simultaneous processes that occur in humans. Individuals' stress response to a given event (such as a test) depends on their interpretation of the situation, their past experiences, their biological systems, their mood states, and their trait personalities (Adam et al., 2006; Doane & Adam, 2010). Some students are more susceptible to perceiving events as stressful, relative to others, and some students may have greater biological responses to the same perceived stressor (Belsky et al., 2007; Del Giudice et al., 2011; Malarkey et al., 1995; Shirtcliff et al., 2014). A more nuanced model could show some students with flatter stress-cognition curves or shift curves laterally to have peak performance at different points of stress. The key takeaway, however, is that in standardized testing, what appears as a difference in achievement may actually be a combination of a true knowledge gap and a stress gap.

Suggested Future Research

Although evidence supports components of the proposed pathways, the overall model remains theoretical. No research has comprehensively examined all of these processes together. The magnitudes of stress-induced achievement gaps

remain unclear, and future research should attempt to quantify the existence and degree of stress-related impairments to learning and test-taking as well as racial-ethnic and SES differences in these processes. Any research on this model will be complicated by the fact that input/effort gains, stress-related learning inhibitors, and test-day stress deficits may be correlated with each other, so any component of the model will need to carefully consider other potential factors.

We suggest that researchers begin by addressing small components of the model. For instance, Sharkey (2010) demonstrates that homicides affect test-day performance in a way that cannot be associated with learning, but it is unclear how this stressor "gets under the skin." Recent data from our research team shows that local violent crime increased bedtime the following night and increased the CAR the following morning (Heissel et al., in press). Future research could combine these analyses, examining an acute stressful event, potential mechanisms (i.e., cortisol and sleep), and cognitive outcomes in the same study. Given the higher prevalence of violent crime in low-income and high-minority areas, this is a pathway by which demographic factors can lead to the perceived achievement gap (that is, Pathway A→Pathway C→Pathway H→Pathway J).

We note that much of the present research is difficult to fully compare and integrate, given the many different measures in sleep (subjective vs. objective, quantity vs. quality) and cortisol (waking levels, CAR, slope, AUC, or specific time points throughout the day). Future research should utilize multiple methods of measurement for both cortisol and sleep over a multiday period; such a study could identify when and why different measures of sleep and cortisol are associated with acute stressors, chronic stressors, and performance on academic tests.

Further complicating such studies, different measures of the diurnal pattern may change on different time scales (Adam, 2012; Doane, Chen, Sladek, Van Lenten, & Granger, 2015; Ross, Murphy, Adam, Chen, & Miller, 2014; Wang et al., 2014). These differences by measure are important for researchers considering using cortisol as a measure of acute or chronic stress to test components of our proposed model. For instance, the CAR may be a better measure of acute versus chronic stress.[1] Researchers should carefully consider the timing relative to the stressor and the time of day as they design studies.

Future research should also explore ways to reduce student stress and modify how students respond to stress. Improvements in neighborhood and family climates, such as reduced exposure to violence and financial stress, could improve student performance. Preliminary research indicates "mindfulness" approaches in K–12 education may provide psychological, social, and cognitive benefits (Meiklejohn et al., 2012). Programs could try to promote positive coping resources, such as positive parenting, high family or community support, or strong racial-ethnic identities (Ai et al., 2014; Taylor & Stanton, 2007; Thoits, 2011; Umaña-Taylor, Wong, Gonzales, & Dumka, 2012). Such research should examine whether perceived stress is reduced, how biological stress responses change, and whether and how intervention effects cascade through the pathways of the model.

Success in sleep intervention studies indicates that parents and school-based programs can change children's sleep behavior, at least in the short term (Cassoff, Knäuper, Michaelsen, & Gruber, 2013; Hiscock et al., 2015; Sadeh et al., 2003). Morning exercise regimens can also improve sleep, mood, and concentration in healthy adolescents (Kalak et al., 2012). Changes to school start times could also lead to widespread increases in sleep quantity and improved academic outcomes (Carrell, Maghakian, & West, 2011; Edwards, 2012; Heissel & Norris, 2017).

One important note is that the expansion of research into the importance of stress exposure and stress responses on student performance could exacerbate the achievement gap if already-advantaged families are more likely to act on such research. To reduce the achievement gap, information and policies must be particularly targeted to those from disadvantaged backgrounds.

Finally, we reiterate that not every low-SES and racial-/ethnic-minority individual will face the types of stressors that affect their HPA axis and sleep, and given the same event, some may perceive a situation as more stressful than others. Policymakers and practitioners should consider within-group variability as they attempt to address disparities in stress exposure and stress responses, in terms of both the policies they pursue and how they interact with and interpret the test scores of individual students. Between-group variability may matter as well. Future studies should examine outcomes separately by sex, age, and pubertal status; for instance, given differences in stress responsivity and sleep by pubertal status, the processes may work differently in different stages of the pubertal transition.

Conclusion

Differences in stress exposure are not the only causes of the academic achievement gap, but they are a potentially important and understudied part of the story. Human biological systems, such as the HPA axis and sleep, both respond to external stressors and affect one another—and in turn affect outcomes like test performance. Stress exposure can change how students gain knowledge during the learning process and how students respond to the acute stress of high-stakes testing. Differences in stress exposure and stress response may explain why two students with the same level of knowledge may have different results on the same standardized tests—or why one student does not accumulate the same level of knowledge in the first place. Developmental increases in HPA axis reactivity and changes to the circadian

rhythm, combined with increased stress exposure, may make the stress gap proposed in the present paper even more relevant for adolescents than for younger children.

High-stakes tests, such as end-of-course tests, course placement exams, high school competency tests, and college entrance exams, are used to make important decisions. The current focus on test-based accountability means that some students, teachers, and schools are penalized and may receive sanctions for standardized test scores that are influenced by stress-related factors outside of their control. The passage of the Every Student Succeeds Act (ESSA) means that states have more authority over their accountability systems and how testing results are used. As states design their new policies under ESSA or its eventual replacement, they should pay particular attention to ways that stress exposure might affect student learning and test-taking performance. More holistic measures of student achievement, such as projects or interim unit tests, may reduce acute stress, although this requires additional study. Still, students' learning may be affected by stress-related learning inhibitors, and using alternative measures would not solve this problem. Thus, the overall goal of preventing excess stress exposure for children and adolescents remains important.

Given that stress exposure is not randomly distributed across the population, the effects of stress have important implications for interpreting racial-ethnic and socioeconomic achievement gaps measured by standardized tests. The current paper is as much a call for additional research as it is a call to action. Nonetheless, we believe that an accumulating body of research supports the claims that factors outside the classroom—including students' stress exposure and their biological responses to stress—may play an important and understudied role in America's achievement gaps.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: We acknowledge funding from the Institute for Policy Research Faculty Fellowship to Emma K. Adam, as well as the Institute for Policy Research and the Sybil N. Heide Fellowship for their graduate support for Jennifer A. Heissel.

Note

1. Morning cortisol levels are more stable within individuals across several months than the cortisol awakening response or cortisol slope, which have high day-to-day variability (Doane, Chen, Sladek, Van Lenten, & Granger, 2015; Wang et al., 2014).

References

Abercrombie, H. C., Kalin, N. H., & Davidson, R. J. (2005). Acute cortisol elevations cause heightened arousal ratings of objectively nonarousing stimuli. *Emotion*, *5*(3), 354–359. https://doi.org/10.1037/1528-3542.5.3.354

Adam, E. K. (2012). Emotion-cortisol transactions occur over multiple time scales in development: Implications for research on emotion and the development of emotional disorders. *Monographs of the Society for Research in Child Development*, *77*(2), 17–27. https://doi.org/10.1111/j.1540-5834.2012.00657.x

Adam, E. K., Doane, L. D., Zinbarg, R. E., Mineka, S., Craske, M. G., & Griffith, J. W. (2010). Prospective prediction of major depressive disorder from cortisol awakening responses in adolescence. *Psychoneuroendocrinology*, *35*(6), 921–931. https://doi.org/10.1016/j.psyneuen.2009.12.007

Adam, E. K., Hawkley, L. C., Kudielka, B. M., & Cacioppo, J. T. (2006). Day-to-day dynamics of experience–cortisol associations in a population-based sample of older adults. *Proceedings of the National Academy of Sciences*, *103*(45), 17058–17063. https://doi.org/10.1073/pnas.0605053103

Adam, E. K., Heissel, J. A., Zeiders, K. H., Richeson, J. A., Ross, E. C., Ehrlich, K. B., . . . Eccles, J. S. (2015). Developmental histories of perceived racial discrimination and diurnal cortisol profiles in adulthood: A 20-year prospective study. *Psychoneuroendocrinology*, *62*, 279–291. https://doi.org/10.1016/j.psyneuen.2015.08.018

Adam, E. K., Snell, E. K., & Pendry, P. (2007). Sleep timing and quantity in ecological and family context: A nationally representative time-diary study. *Journal of Family Psychology*, *21*(1), 4–19. https://doi.org/10.1037/0893-3200.21.1.4

Ai, A. L., Aisenberg, E., Weiss, S. I., & Salazar, D. (2014). Racial/ethnic identity and subjective physical and mental health of Latino Americans: An asset within? *American Journal of Community Psychology*, *53*(1/2), 173–184. https://doi.org/10.1007/s10464-014-9635-5

Alapin, I., Fichten, C. S., Libman, E., Creti, L., Bailes, S., & Wright, J. (2000). How is good and poor sleep in older adults and college students related to daytime sleepiness, fatigue, and ability to concentrate? *Journal of Psychosomatic Research*, *49*(5), 381–390. https://doi.org/10.1016/S0022-3999(00)00194-X

American Academy of Pediatrics. (2014). School start times for adolescents. *Pediatrics*, *134*(3), 642–649. https://doi.org/10.1542/peds.2014-1697

Anderson, B., Storfer-Isser, A., Taylor, H. G., Rosen, C. L., & Redline, S. (2009). Associations of executive function with sleepiness and sleep duration in adolescents. *Pediatrics*, *123*(4), e701–e707. https://doi.org/10.1542/peds.2008-1182

Arnett, J. J. (1999). Adolescent storm and stress, reconsidered. *American Psychologist*, *54*(5), 317–326. https://doi.org/10.1037/0003-066X.54.5.317

Astill, R. G., Van der Heijden, K. B., Van IJzendoorn, M. H., & Van Someren, E. J. W. (2012). Sleep, cognition, and behavioral problems in school-age children: A century of research meta-analyzed. *Psychological Bulletin*, *138*(6), 1109–1138. https://doi.org/10.1037/a0028204

Astill, R. G., Verhoeven, D., Vijzelaar, R. L., & Van Someren, E. J. W. (2013). Chronic stress undermines the compensatory sleep efficiency increase in response to sleep restriction in adolescents. *Journal of Sleep Research*, *22*(4), 373–379. https://doi.org/10.1111/jsr.12032

Badanes, L. S., Watamura, S. E., & Hankin, B. L. (2011). Hypocortisolism as a potential marker of allostatic load in children: Associations with family risk and internalizing disorders.

Development and Psychopathology, *23*(3), 881–896. https://doi.org/10.1017/S095457941100037X

Barton, P. E., & Coley, R. J. (2009). *Parsing the achievement gap II*. Princeton, NJ: Policy Information Center, Educational Testing Service. Retrieved from http://www.ets.org/Media/Research/pdf/PICPARSINGII.pdf

Baum, K. T., Desai, A., Field, J., Miller, L. E., Rausch, J., & Beebe, D. W. (2014). Sleep restriction worsens mood and emotion regulation in adolescents. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *55*(2), 180–190. https://doi.org/10.1111/jcpp.12125

Beebe, D. W., Fallone, G., Godiwala, N., Flanigan, M., Martin, D., Schaffner, L., & Amin, R. (2008). Feasibility and behavioral effects of an at-home multi-night sleep restriction protocol for adolescents. *Journal of Child Psychology and Psychiatry*, *49*(9), 915–923. https://doi.org/10.1111/j.1469-7610.2008.01885.x

Beebe, D. W., Rose, D., & Amin, R. (2010). Attention, learning, and arousal of experimentally sleep-restricted adolescents in a simulated classroom. *Journal of Adolescent Health*, *47*(5), 523–525. https://doi.org/10.1016/j.jadohealth.2010.03.005

Belsky, J., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). For better and for worse: Differential susceptibility to environmental influences. *Current Directions in Psychological Science*, *16*(6), 300–304. https://doi.org/10.1111/j.1467-8721.2007.00525.x

Bennett, G. G., Merritt, M. M., Edwards, C. L., & Sollers, J. J. (2004). Perceived racism and affective responses to ambiguous interpersonal interactions among African American men. *American Behavioral Scientist*, *47*(7), 963–976. https://doi.org/10.1177/0002764203261070

Blair, C., Granger, D., & Razza, R. P. (2005). Cortisol reactivity is positively related to executive function in preschool children attending Head Start. *Child Development*, *76*(3), 554–567. https://doi.org/10.1111/j.1467-8624.2005.00863.x

Boyce, W. T., & Ellis, B. J. (2005). Biological sensitivity to context: I. An evolutionary-developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology*, *17*(2), 271–301. https://doi.org/10.1017/S0954579405050145

Bradbury, B., Corak, M., Waldfogel, J., & Washbrook, E. (2015). *Too many children left behind: The U.S. achievement gap in comparative perspective*. New York, NY: Russell Sage Foundation.

Bransford, J. D., Brown, A. L., Cocking, R. R., Donovan, M. S., & Pellegrino, J. W. (Eds.). (2000). *How people learn: Brain, mind, experience, and school. Expanded edition*. Washington, DC: National Academies Press. Retrieved from http://www.nap.edu/catalog/9853

Bub, K. L., Buckhalt, J. A., & El-Sheikh, M. (2011). Children's sleep and cognitive performance: A cross-domain analysis of change over time. *Developmental Psychology*, *47*(6), 1504–1514. https://doi.org/10.1037/a0025535

Buckhalt, J. A. (2011). Insufficient sleep and the socioeconomic status achievement gap. *Child Development Perspectives*, *5*(1), 59–65. https://doi.org/10.1111/j.1750-8606.2010.00151.x

Buckhalt, J. A., El-Sheikh, M., & Keller, P. (2007). Children's sleep and cognitive functioning: Race and socioeconomic status as moderators of effects. *Child Development*, *78*(1), 213–231. https://doi.org/10.1111/j.1467-8624.2007.00993.x

Carrell, S. E., Maghakian, T., & West, J. E. (2011). A's from Zzzz's? The causal effect of school start time on the academic achievement of adolescents. *American Economic Journal: Economic Policy*, *3*(3), 62–81. https://doi.org/10.1257/pol.3.3.62

Carskadon, M. A., Acebo, C., & Jenni, O. G. (2004). Regulation of adolescent sleep: Implications for behavior. *Annals of the New York Academy of Sciences*, *1021*, 276–291. https://doi.org/10.1196/annals.1308.032

Cassoff, J., Knäuper, B., Michaelsen, S., & Gruber, R. (2013). School-based sleep promotion programs: effectiveness, feasibility and insights for future research. *Sleep Medicine Reviews*, *17*(3), 207–214. https://doi.org/10.1016/j.smrv.2012.07.001

Chapotot, F., Buguet, A., Gronfier, C., & Brandenberger, G. (2001). Hypothalamo-pituitary-adrenal axis activity is related to the level of central arousal: Effect of sleep deprivation on the association of high-frequency waking electroencephalogram with cortisol release. *Neuroendocrinology*, *73*(5), 312–321. https://doi.org/54648

Chen, E., & Matthews, K. A. (2001). Cognitive appraisal biases: An approach to understanding the relation between socioeconomic status and cardiovascular reactivity in children. *Annals of Behavioral Medicine*, *23*(2), 101–111. https://doi.org/10.1207/S15324796ABM2302_4

Chmiel, N., Totterdell, P., & Folkard, S. (1995). On adaptive control, sleep loss, and fatigue. *Applied Cognitive Psychology*, *9*(7), S39–S53. https://doi.org/10.1002/acp.2350090705

Cicchetti, D., & Rogosch, F. A. (2001). Diverse patterns of neuroendocrine activity in maltreated children. *Development and Psychopathology*, *13*(3), 677–693.

Clark, R., Anderson, N. B., Clark, V. R., & Williams, D. R. (1999). Racism as a stressor for African Americans: A biopsychosocial model. *American Psychologist*, *54*(10), 805–816. https://doi.org/10.1037/0003-066X.54.10.805

Clow, A., Hucklebridge, F., Stalder, T., Evans, P., & Thorn, L. (2010). The cortisol awakening response: More than a measure of HPA axis function. *Neuroscience & Biobehavioral Reviews*, *35*(1), 97–103. https://doi.org/10.1016/j.neubiorev.2009.12.011

Clow, A., Law, R., Evans, P., Vallence, A.-M., Hodyl, N. A., Goldsworthy, M. R., . . . Ridding, M. C. (2014). Day differences in the cortisol awakening response predict day differences in synaptic plasticity in the brain. *Stress*, *17*(3), 219–223. https://doi.org/10.3109/10253890.2014.905533

Cohen, M., Ben-Zur, H., & Rosenfeld, M. J. (2008). Sense of coherence, coping strategies, and test anxiety as predictors of test performance among college students. *International Journal of Stress Management*, *15*(3), 289–303. https://doi.org/10.1037/1072-5245.15.3.289

Cohen, S., Schwartz, J. E., Epel, E., Kirschbaum, C., Sidney, S., & Seeman, T. (2006). Socioeconomic status, race, and diurnal cortisol decline in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Psychosomatic Medicine*, *68*(1), 41–50. https://doi.org/10.1097/01.psy.0000195967.51768.ea

Compas, B. E., Orosan, P. G., & Grant, K. E. (1993). Adolescent stress and coping: Implications for psychopathology during adolescence. *Journal of Adolescence*, *16*(3), 331–349. https://doi.org/10.1006/jado.1993.1028

Crowley, S. J., Acebo, C., & Carskadon, M. A. (2007). Sleep, circadian rhythms, and delayed phase in adolescence. *Sleep Medicine*, *8*(6), 602–612. https://doi.org/10.1016/j.sleep.2006.12.002

de Kloet, E. R., Oitzl, M. S., & Joëls, M. (1999). Stress and cognition: Are corticosteroids good or bad guys? *Trends in Neurosciences*, *22*(10), 422–426. https://doi.org/10.1016/S0166-2236(99)01438-1

Del Giudice, M., Ellis, B. J., & Shirtcliff, E. A. (2011). The adaptive calibration model of stress responsivity. *Neuroscience & Biobehavioral Reviews*, *35*(7), 1562–1592. https://doi.org/10.1016/j.neubiorev.2010.11.007

DeSantis, A. S., Adam, E. K., Doane, L. D., Mineka, S., Zinbarg, R. E., & Craske, M. G. (2007). Racial/ethnic differences in cortisol diurnal rhythms in a community sample of adolescents. *Journal of Adolescent Health*, *41*(1), 3–13. https://doi.org/10.1016/j.jadohealth.2007.03.006

DeSantis, A. S., Adam, E. K., Hawkley, L. C., Kudielka, B. M., & Cacioppo, J. T. (2015). Racial/ethnic differences in diurnal cortisol rhythms: Are they consistent over time? *Psychosomatic Medicine*, *77*(1), 6–15. https://doi.org/10.1097/PSY.0000000000000131

Dewald, J. F., Meijer, A. M., Oort, F. J., Kerkhof, G. A., & Bögels, S. M. (2010). The influence of sleep quality, sleep duration and sleepiness on school performance in children and adolescents: A meta-analytic review. *Sleep Medicine Reviews*, *14*(3), 179–189. https://doi.org/10.1016/j.smrv.2009.10.004

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*(3), 355–391. https://doi.org/10.1037/0033-2909.130.3.355

Doane, L. D., & Adam, E. K. (2010). Loneliness and cortisol: Momentary, day-to-day, and trait associations. *Psychoneuroendocrinology*, *35*(3), 430–441. https://doi.org/10.1016/j.psyneuen.2009.08.005

Doane, L. D., Chen, F. R., Sladek, M. R., Van Lenten, S. A., & Granger, D. A. (2015). Latent trait cortisol (LTC) levels: Reliability, validity, and stability. *Psychoneuroendocrinology*, *55*, 21–35. https://doi.org/10.1016/j.psyneuen.2015.01.017

Duncan, G. J., & Murnane, R. J. (2011). *Whither opportunity? Rising inequality, schools, and children's life chances*. New York, NY: Russell Sage Foundation.

Edwards, F. (2012). Early to rise? The effect of daily start times on academic performance. *Economics of Education Review*, *31*(6), 970–983. https://doi.org/10.1016/j.econedurev.2012.07.006

El-Sheikh, M., Buckhalt, J. A., Keller, P. S., & Granger, D. A. (2008). Children's objective and subjective sleep disruptions: Links with afternoon cortisol levels. *Health Psychology*, *27*(1), 26–33. https://doi.org/10.1037/0278-6133.27.1.26

Evans, P., Fredhoi, C., Loveday, C., Hucklebridge, F., Aitchison, E., Forte, D., & Clow, A. (2011). The diurnal cortisol cycle and cognitive performance in the healthy old. *International Journal of Psychophysiology*, *79*(3), 371–377. https://doi.org/10.1016/j.ijpsycho.2010.12.006

Evans, P., Hucklebridge, F., Loveday, C., & Clow, A. (2012). The cortisol awakening response is related to executive function in older age. *International Journal of Psychophysiology*, *84*(2), 201–204. https://doi.org/10.1016/j.ijpsycho.2012.02.008

Fogel, S. M., & Smith, C. T. (2011). The function of the sleep spindle: A physiological index of intelligence and a mechanism for sleep-dependent memory consolidation. *Neuroscience & Biobehavioral Reviews*, *35*(5), 1154–1165. https://doi.org/10.1016/j.neubiorev.2010.12.003

Fries, E., Dettenborn, L., & Kirschbaum, C. (2009). The cortisol awakening response (CAR): Facts and future directions. *International Journal of Psychophysiology*, *72*(1), 67–73. https://doi.org/10.1016/j.ijpsycho.2008.03.014

Fries, E., Hesse, J., Hellhammer, J., & Hellhammer, D. H. (2005). A new view on hypocortisolism. *Psychoneuroendocrinology*, *30*(10), 1010–1016. https://doi.org/10.1016/j.psyneuen.2005.04.006

Gershenson, S., & Tekin, E. (2015). *The effect of community traumatic events on student achievement: Evidence from the Beltway Sniper attacks* (Working Paper No. 21055). Cambridge, MA: National Bureau of Economic Research. Retrieved from http://www.nber.org/papers/w21055

Gilbert, K., Mineka, S., Zinbarg, R. E., Craske, M. G., & Adam, E. K. (2017). Emotion regulation regulates more than emotion: Associations of momentary emotion regulation with diurnal cortisol in current and past depression and anxiety. *Clinical Psychological Science*, *5*(1), 37–51. https://doi.org/10.1177/2167702616654437

Gillen-O'Neel, C., Huynh, V. W., & Fuligni, A. J. (2013). To study or to sleep? The academic costs of extra studying at the expense of sleep. *Child Development*, *84*(1), 133–142. https://doi.org/10.1111/j.1467-8624.2012.01834.x

Glasscock, D. J., Andersen, J. H., Labriola, M., Rasmussen, K., & Hansen, C. D. (2013). Can negative life events and coping style help explain socioeconomic differences in perceived stress among adolescents? A cross-sectional study based on the West Jutland cohort study. *BMC Public Health*, *13*(1), 532. https://doi.org/10.1186/1471-2458-13-532

Glod, C. A., Teicher, M. H., Hartman, C. R., & Harakal, T. (1997). Increased nocturnal activity and impaired sleep maintenance in abused children. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*(9), 1236–1243. https://doi.org/10.1097/00004583-199709000-00016

Gunnar, M. R., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology*, *58*(1), 145–173. https://doi.org/10.1146/annurev.psych.58.110405.085605

Gunnar, M. R., & Vazquez, D. M. (2001). Low cortisol and a flattening of expected daytime rhythm: Potential indices of risk in human development. *Development and Psychopathology*, *13*(3), 515–538.

Gunnar, M. R., Wewerka, S., Frenn, K., Long, J. D., & Griggs, C. (2010). Developmental changes in hypothalamus–pituitary–adrenal activity over the transition to adolescence: Normative changes and associations with puberty. *Development and Psychopathology*, *21*(1), 69–85. https://doi.org/10.1017/S0954579409000054

Hajat, A., Diez-Roux, A., Franklin, T. G., Seeman, T., Shrager, S., Ranjit, N., . . . Kirschbaum, C. (2010). Socioeconomic and race/ethnic differences in daily salivary cortisol profiles: The Multi-Ethnic Study of Atherosclerosis. *Psychoneuroendocrinology*, *35*(6), 932–943. https://doi.org/10.1016/j.psyneuen.2009.12.009

Hall, M. H., Matthews, K. A., Kravitz, H. M., Gold, E. B., Buysse, D. J., Bromberger, J. T., . . . Sowers, M. (2009). Race and financial strain are independent correlates of sleep in midlife women: The SWAN Sleep Study. *Sleep*, *32*(1), 73–82.

Hanson, M. D., & Chen, E. (2010). Daily stress, cortisol, and sleep: The moderating role of childhood psychosocial environments. *Health Psychology*, *29*(4), 394–402. https://doi.org/10.1037/a0019879

Hatch, S. L., & Dohrenwend, B. P. (2007). Distribution of traumatic and other stressful life events by race/ethnicity, gender, SES and

age: A review of the research. *American Journal of Community Psychology*, *40*(3/4), 313–332. https://doi.org/10.1007/s10464-007-9134-z

Heathcote, J., Perri, F., & Violante, G. L. (2010). Unequal we stand: An empirical analysis of economic inequality in the United States, 1967–2006. *Review of Economic Dynamics*, *13*(1), 15–51. https://doi.org/10.1016/j.red.2009.10.010

Heissel, J. A., & Norris, S. (2017). Rise and shine: The effect of school start times on academic performance from childhood through puberty. *Journal of Human Resources*. Advance online publication. https://doi.org/10.3368/jhr.53.4.0815-7346R1

Heissel, J. A., Sharkey, P. T., Torrats-Espinosa, G., Grant, K. E., & Adam, E. K. (in press). Violence and vigilance: The acute effects of community violent crime on sleep and cortisol. *Child Development*.

Het, S., Ramlow, G., & Wolf, O. T. (2005). A meta-analytic review of the effects of acute cortisol administration on human memory. *Psychoneuroendocrinology*, *30*(8), 771–784. https://doi.org/10.1016/j.psyneuen.2005.03.005

Hicken, M. T., Lee, H., Ailshire, J., Burgard, S. A., & Williams, D. R. (2013). "Every shut eye, ain't sleep": The role of racism-related vigilance in racial/ethnic disparities in sleep difficulty. *Race and Social Problems*, *5*(2), 100–112. https://doi.org/10.1007/s12552-013-9095-9

Hiscock, H., Sciberras, E., Mensah, F., Gerner, B., Efron, D., Khano, S., & Oberklaid, F. (2015). Impact of a behavioural sleep intervention on symptoms and sleep in children with attention deficit hyperactivity disorder, and parental mental health: Randomised controlled trial. *BMJ*, *350*, h68. https://doi.org/10.1136/bmj.h68

Hoffman, R., & al'Absi, M. (2004). The effect of acute stress on subsequent neuropsychological test performance (2003). *Archives of Clinical Neuropsychology*, *19*(4), 497–506. https://doi.org/10.1016/j.acn.2003.07.005

Horn, J. L., & Dollinger, S. J. (1989). Effects of test anxiety, tests, and sleep on children's performance. *Journal of School Psychology*, *27*(4), 373–382. https://doi.org/10.1016/0022-4405(89)90014-9

Jenni, O. G., & Carskadon, M. A. (2012). Sleep behavior and sleep regulation from infancy through adolescence: Normative aspects. *Sleep Medicine Clinics*, *7*(3), 529–538. https://doi.org/10.1016/j.jsmc.2012.06.002

Joëls, M., Pu, Z., Wiegert, O., Oitzl, M. S., & Krugers, H. J. (2006). Learning under stress: How does it work? *Trends in Cognitive Sciences*, *10*(4), 152–158. https://doi.org/10.1016/j.tics.2006.02.002

Joo, E. Y., Yoon, C. W., Koo, D. L., Kim, D., & Hong, S. B. (2012). Adverse effects of 24 hours of sleep deprivation on cognition and stress hormones. *Journal of Clinical Neurology*, *8*(2), 146–150. https://doi.org/10.3988/jcn.2012.8.2.146

Juster, R.-P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience & Biobehavioral Reviews*, *35*(1), 2–16. https://doi.org/10.1016/j.neubiorev.2009.10.002

Kalak, N., Gerber, M., Kirov, R., Mikoteit, T., Yordanova, J., Pühse, U., . . . Brand, S. (2012). Daily morning running for 3 weeks improved sleep and psychological functioning in healthy adolescents compared with controls. *Journal of Adolescent Health*, *51*(6), 615–622. https://doi.org/10.1016/j.jadohealth.2012.02.020

Kirby, M., Maggi, S., & D'Angiulli, A. (2011). School start times and the sleep–wake cycle of adolescents: A review and critical evaluation of available evidence. *Educational Researcher*, *40*(2), 56–61. https://doi.org/10.3102/0013189X11402323

Koss, K. J., Mliner, S. B., Donzella, B., & Gunnar, M. R. (2016). Early adversity, hypocortisolism, and behavior problems at school entry: A study of internationally adopted children. *Psychoneuroendocrinology*, *66*, 31–38. https://doi.org/10.1016/j.psyneuen.2015.12.018

Kudielka, B. M., Buske-Kirschbaum, A., Hellhammer, D. H., & Kirschbaum, C. (2004). HPA axis responses to laboratory psychosocial stress in healthy elderly adults, younger adults, and children: impact of age and gender. *Psychoneuroendocrinology*, *29*(1), 83–98. https://doi.org/10.1016/S0306-4530(02)00146-4

Kuhlmann, S., Kirschbaum, C., & Wolf, O. T. (2005). Effects of oral cortisol treatment in healthy young women on memory retrieval of negative and neutral words. *Neurobiology of Learning and Memory*, *83*(2), 158–162. https://doi.org/10.1016/j.nlm.2004.09.001

Kuhlmann, S., Piel, M., & Wolf, O. T. (2005). Impaired memory retrieval after psychosocial stress in healthy young men. *Journal of Neuroscience*, *25*(11), 2977–2982. https://doi.org/10.1523/JNEUROSCI.5139-04.2005

Laberge, L., Petit, D., Simard, C., Vitaro, F., Tremblay, R. E., & Montplaisir, J. (2001). Development of sleep patterns in early adolescence. *Journal of Sleep Research*, *10*(1), 59–67. https://doi.org/10.1046/j.1365-2869.2001.00242.x

Levy, D. J., Heissel, J. A., Richeson, J. A., & Adam, E. K. (2016). Psychological and biological responses to race-based social stress as pathways to disparities in educational outcomes. *American Psychologist*, *71*(6), 455–473. https://doi.org/10.1037/a0040322

Lindahl, M., Theorell, T., & Lindblad, F. (2005). Test performance and self-esteem in relation to experienced stress in Swedish sixth and ninth graders: Saliva cortisol levels and psychological reactions to demands. *Acta Pædiatrica*, *94*(4), 489–495. https://doi.org/10.1111/j.1651-2227.2005.tb01922.x

Lupien, S. J., de Leon, M., de Santi, S., Convit, A., Tarshish, C., Nair, N. P. V., . . . Meaney, M. J. (1998). Cortisol levels during human aging predict hippocampal atrophy and memory deficits. *Nature Neuroscience*, *1*(1), 69–73. https://doi.org/10.1038/271

Lupien, S. J., Maheu, F., Tu, M., Fiocco, A., & Schramek, T. E. (2007). The effects of stress and stress hormones on human cognition: Implications for the field of brain and cognition. *Brain and Cognition*, *65*(3), 209–237. https://doi.org/10.1016/j.bandc.2007.02.007

Lupien, S. J., Wilkinson, C. W., Brière, S., Ménard, C., Ng Ying Kin, N. M. K., & Nair, N. P. V. (2002). The modulatory effects of corticosteroids on cognition: Studies in young human populations. *Psychoneuroendocrinology*, *27*(3), 401–416. https://doi.org/10.1016/S0306-4530(01)00061-0

Malarkey, W. B., Pearl, D. K., Demers, L. M., Kiecolt-Glaser, J. K., & Glaser, R. (1995). Influence of academic stress and season on 24-hour mean concentrations of ACTH, cortisol, and β-endorphin. *Psychoneuroendocrinology*, *20*(5), 499–508. https://doi.org/10.1016/0306-4530(94)00077-N

Maldonado, E. F., Fernandez, F. J., Trianes, M. V., Wesnes, K., Petrini, O., Zangara, A., . . . Ambrosetti, L. (2008). Cognitive performance and morning levels of salivary cortisol and alpha-

amylase in children reporting high vs. low daily stress perception. *Spanish Journal of Psychology*, *11*(1), 3–15. https://doi.org/10.1017/S1138741600004066

Maquet, P., Laureys, S., Peigneux, P., Fuchs, S., Petiau, C., Phillips, C., . . . Cleeremans, A. (2000). Experience-dependent changes in cerebral activation during human REM sleep. *Nature Neuroscience*, *3*(8), 831. https://doi.org/10.1038/77744

Mattarella-Micke, A., Mateo, J., Kozak, M. N., Foster, K., & Beilock, S. L. (2011). Choke or thrive? The relation between salivary cortisol and math performance depends on individual differences in working memory and math-anxiety. *Emotion*, *11*(4), 1000–1005. https://doi.org/10.1037/a0023224

McEwen, B. S., & Gianaros, P. J. (2010). Central role of the brain in stress and adaptation: Links to socioeconomic status, health, and disease. *Annals of the New York Academy of Sciences*, *1186*, 190–222. https://doi.org/10.1111/j.1749-6632.2009.05331.x

McEwen, B. S., & Stellar, E. (1993). Stress and the individual: Mechanisms leading to disease. *Archives of Internal Medicine*, *153*(18), 2093–2101. https://doi.org/10.1001/archinte.1993.00410180039004

McKenzie, S., & Eichenbaum, H. (2011). Consolidation and reconsolidation: Two lives of memories? *Neuron*, *71*(2), 224–233. https://doi.org/10.1016/j.neuron.2011.06.037

Meiklejohn, J., Phillips, C., Freedman, M. L., Griffin, M. L., Biegel, G., Roach, A., . . . Saltzman, A. (2012). Integrating mindfulness training into K–12 education: Fostering the resilience of teachers and students. *Mindfulness*, *3*(4), 291–307. https://doi.org/10.1007/s12671-012-0094-5

Mezick, E. J., Matthews, K. A., Hall, M., Strollo, P. J., Buysse, D. J., Kamarck, T. W., . . . Reis, S. E. (2008). Influence of race and socioeconomic status on sleep: Pittsburgh Sleep SCORE Project. *Psychosomatic Medicine*, *70*(4), 410–416. https://doi.org/10.1097/PSY.0b013e31816fdf21

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*, *133*(1), 25–45. https://doi.org/10.1037/0033-2909.133.1.25

Myers, H. F. (2008). Ethnicity- and socio-economic status-related stresses in context: An integrative review and conceptual model. *Journal of Behavioral Medicine*, *32*(1), 9–19. https://doi.org/10.1007/s10865-008-9181-4

Nachmias, M., Gunnar, M., Mangelsdorf, S., Parritz, R. H., & Buss, K. (1996). Behavioral inhibition and stress reactivity: The moderating role of attachment security. *Child Development*, *67*(2), 508–522. https://doi.org/10.2307/1131829

National Sleep Foundation. (2004). *Children and sleep (Sleep in America Poll)*. Arlington, VA: Author. Retrieved from http://www.sleepfoundation.org/

National Sleep Foundation. (2006). *Teens and sleep (Sleep in America Poll)*. Arlington, VA: Author. Retrieved from http://www.sleepfoundation.org/

Petrov, M. E., & Lichstein, K. L. (2015). Differences in sleep between black and white adults: An update and future directions. *Sleep Medicine*. Advance online publication. https://doi.org/10.1016/j.sleep.2015.01.011

Plihal, W., & Born, J. (1999). Memory consolidation in human sleep depends on inhibition of glucocorticoid release. *Neuroreport*, *10*(13), 2741–2747.

Pynoos, R. S., Frederick, C., Nader, K., Arroyo, W., Steinberg, A., Eth, S., . . . Fairbanks, L. (1987). Life threat and posttraumatic stress in school-age children. *Archives of General Psychiatry*, *44*(12), 1057–1063. https://doi.org/10.1001/archpsyc.1987.01800240031005

Räikkönen, K., Matthews, K. A., Pesonen, A.-K., Pyhälä, R., Paavonen, E. J., Feldt, K., . . . Kajantie, E. (2010). Poor sleep and altered hypothalamic-pituitary-adrenocortical and sympatho-adrenal-medullary system activity in children. *Journal of Clinical Endocrinology & Metabolism*, *95*(5), 2254–2261. https://doi.org/10.1210/jc.2009-0943

Reardon, S. F. (2011). The widening academic achievement gap between the rich and the poor: New evidence and possible explanations. In G. J. Duncan, & R. J. Murnane (Eds.), *Whither opportunity? Rising inequality, schools, and children's life chances* (pp. 91–116). New York, NY: Russell Sage Foundation.

Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin*, *128*(2), 330–366. https://doi.org/10.1037/0033-2909.128.2.330

Rimmele, U., Meier, F., Lange, T., & Born, J. (2010). Suppressing the morning rise in cortisol impairs free recall. *Learning & Memory*, *17*(4), 186–190. https://doi.org/10.1101/lm.1728510

Roberts, A. L., Gilman, S. E., Breslau, J., Breslau, N., & Koenen, K. C. (2011). Race/ethnic differences in exposure to traumatic events, development of post-traumatic stress disorder, and treatment-seeking for post-traumatic stress disorder in the United States. *Psychological Medicine*, *41*(1), 71–83. https://doi.org/10.1017/S0033291710000401

Romeo, R. D. (2010). Pubertal maturation and programming of hypothalamic–pituitary–adrenal reactivity. *Frontiers in Neuroendocrinology*, *31*(2), 232–240. https://doi.org/10.1016/j.yfrne.2010.02.004

Roozendaal, B. (2002). Stress and memory: Opposing effects of glucocorticoids on memory consolidation and memory retrieval. *Neurobiology of Learning and Memory*, *78*(3), 578–595. https://doi.org/10.1006/nlme.2002.4080

Ross, K. M., Murphy, M. L. M., Adam, E. K., Chen, E., & Miller, G. E. (2014). How stable are diurnal cortisol activity indices in healthy individuals? Evidence from three multi-wave studies. *Psychoneuroendocrinology*, *39*, 184–193. https://doi.org/10.1016/j.psyneuen.2013.09.016

Ruiter, M. E., DeCoster, J., Jacobs, L., & Lichstein, K. L. (2010). Sleep disorders in African Americans and Caucasian Americans: A meta-analysis. *Behavioral Sleep Medicine*, *8*(4), 246–259. https://doi.org/10.1080/15402002.2010.509251

Ruiter, M. E., Decoster, J., Jacobs, L., & Lichstein, K. L. (2011). Normal sleep in African-Americans and Caucasian-Americans: A meta-analysis. *Sleep Medicine*, *12*(3), 209–214. https://doi.org/10.1016/j.sleep.2010.12.010

Sadeh, A. (1996). Stress, trauma, and sleep in children. *Child and Adolescent Psychiatric Clinics of North America*, *5*(3), 685–700.

Sadeh, A., Dahl, R. E., Shahar, G., & Rosenblat-Stein, S. (2009). Sleep and the transition to adolescence: A longitudinal study. *Sleep*, *32*(12), 1602–1609.

Sadeh, A., Gruber, R., & Raviv, A. (2002). Sleep, neurobehavioral functioning, and behavior problems in school-age children. *Child Development*, *73*(2), 405–417.

Sadeh, A., Gruber, R., & Raviv, A. (2003). The effects of sleep restriction and extension on school-age children: What a difference an hour makes. *Child Development*, *74*(2), 444–455. https://doi.org/10.1111/1467-8624.7402008

Sadeh, A., Keinan, G., & Daon, K. (2004). Effects of stress on sleep: The moderating role of coping style. *Health Psychology*, *23*(5), 542–545. https://doi.org/10.1037/0278-6133.23.5.542

Sadeh, A., Raviv, A., & Gruber, R. (2000). Sleep patterns and sleep disruptions in school-age children. *Developmental Psychology*, *36*(3), 291–301. https://doi.org/10.1037/0012-1649.36.3.291

Salehi, B., Cordero, M. I., & Sandi, C. (2010). Learning under stress: The inverted-U-shape function revisited. *Learning & Memory*, *17*(10), 522–530. https://doi.org/10.1101/lm.1914110

Sandi, C., & Pinelo-Nava, M. T. (2007). Stress and memory: Behavioral effects and neurobiological mechanisms. *Neural Plasticity*, *2007*, e78970. https://doi.org/10.1155/2007/78970

Sapolsky, R. M., Romero, L. M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Reviews*, *21*(1), 55–89. https://doi.org/10.1210/er.21.1.55

Sasaki, Y., Yotsumoto, Y., Chan, P., Vasios, C., Nanez, J., Shimojo, S., . . . Bonmassar, G. (2007). Brain activity related to consolidation of perceptual learning during sleep. *Journal of Vision*, *7*(15), 103. https://doi.org/10.1167/7.15.103

Sauro, M. D., Jorgensen, R. S., & Pedlow, T. (2003). Stress, glucocorticoids, and memory: A meta-analytic review. *Stress: The International Journal on the Biology of Stress*, *6*(4), 235–245. https://doi.org/10.1080/1025389031000161648 2

Schilling, T. M., Kölsch, M., Larra, M. F., Zech, C. M., Blumenthal, T. D., Frings, C., & Schächinger, H. (2013). For whom the bell (curve) tolls: Cortisol rapidly affects memory retrieval by an inverted U-shaped dose–response relationship. *Psychoneuroendocrinology*, *38*(9), 1565–1572. https://doi.org/10.1016/j.psyneuen.2013.01.001

Schlotz, W., Schulz, P., Hellhammer, J., Stone, A. A., & Hellhammer, D. H. (2006). Trait anxiety moderates the impact of performance pressure on salivary cortisol in everyday life. *Psychoneuroendocrinology*, *31*(4), 459–472. https://doi.org/10.1016/j.psyneuen.2005.11.003

Schoofs, D., Preuß, D., & Wolf, O. T. (2008). Psychosocial stress induces working memory impairments in an n-back paradigm. *Psychoneuroendocrinology*, *33*(5), 643–653. https://doi.org/10.1016/j.psyneuen.2008.02.004

Schwabe, L., Joëls, M., Roozendaal, B., Wolf, O. T., & Oitzl, M. S. (2012). Stress effects on memory: An update and integration. *Neuroscience & Biobehavioral Reviews*, *36*(7), 1740–1749. https://doi.org/10.1016/j.neubiorev.2011.07.002

Schwabe, L., & Wolf, O. T. (2010). Learning under stress impairs memory formation. *Neurobiology of Learning and Memory*, *93*(2), 183–188. https://doi.org/10.1016/j.nlm.2009.09.009

Schwabe, L., Wolf, O. T., & Oitzl, M. S. (2010). Memory formation under stress: Quantity and quality. *Neuroscience & Biobehavioral Reviews*, *34*(4), 584–591. https://doi.org/10.1016/j.neubiorev.2009.11.015

Sekine, M., Chandola, T., Martikainen, P., Marmot, M., & Kagamimori, S. (2006). Work and family characteristics as determinants of socioeconomic and sex inequalities in sleep: The Japanese Civil Servants Study. *Sleep*, *29*(2), 206–216.

Sharkey, P. T. (2010). The acute effect of local homicides on children's cognitive performance. *Proceedings of the National Academy of Sciences*, *107*(26), 11733–11738. https://doi.org/10.1073/pnas.1000690107

Shields, G. S., Sazma, M. A., & Yonelinas, A. P. (2016). The effects of acute stress on core executive functions: A meta-analysis and comparison with cortisol. *Neuroscience & Biobehavioral Reviews*, *68*, 651–668. https://doi.org/10.1016/j.neubiorev.2016.06.038

Shirtcliff, E. A., Allison, A. L., Armstrong, J. M., Slattery, M. J., Kalin, N. H., & Essex, M. J. (2012). Longitudinal stability and developmental properties of salivary cortisol levels and circadian rhythms from childhood to adolescence. *Developmental Psychobiology*, *54*(5), 493–502. https://doi.org/10.1002/dev.20607

Shirtcliff, E. A., Peres, J. C., Dismukes, A. R., Lee, Y., & Phan, J. M. (2014). Hormones: Commentary. Riding the physiological roller coaster: Adaptive significance of cortisol stress reactivity to social contexts. *Journal of Personality Disorders*, *28*(1), 40–51. https://doi.org/10.1521/pedi.2014.28.1.40

Smeets, T., Jelicic, M., & Merckelbach, H. (2006). The effect of acute stress on memory depends on word valence. *International Journal of Psychophysiology*, *62*(1), 30–37. https://doi.org/10.1016/j.ijpsycho.2005.11.007

Squire, L. R. (2004). Memory systems of the brain: A brief history and current perspective. *Neurobiology of Learning and Memory*, *82*(3), 171–177. https://doi.org/10.1016/j.nlm.2004.06.005

Steele, C. M., & Aronson, J. (1995). Stereotype threat and the intellectual test performance of African Americans. *Journal of Personality and Social Psychology*, *69*(5), 797–811. https://doi.org/10.1037/0022-3514.69.5.797

Stevens, C., Lauinger, B., & Neville, H. (2009). Differences in the neural mechanisms of selective attention in children from different socioeconomic backgrounds: An event-related brain potential study. *Developmental Science*, *12*(4), 634–646. https://doi.org/10.1111/j.1467-7687.2009.00807.x

Stroud, L. R., Foster, E., Papandonatos, G. D., Handwerger, K., Granger, D. A., Kivlighan, K. T., & Niaura, R. (2009). Stress response and the adolescent transition: Performance versus peer rejection stressors. *Development and Psychopathology*, *21*(1), 47–68. https://doi.org/10.1017/S0954579409000042

Stroud, L. R., Salovey, P., & Epel, E. S. (2002). Sex differences in stress responses: Social rejection versus achievement stress. *Biological Psychiatry*, *52*(4), 318–327. https://doi.org/10.1016/S0006-3223(02)01333-1

Struthers, C. W., Perry, R. P., & Menec, V. H. (2000). An examination of the relationship among academic stress, coping, motivation, and performance in college. *Research in Higher Education*, *41*(5), 581–592. https://doi.org/10.1023/A:1007094931292

Taylor, S. E., & Stanton, A. L. (2007). Coping resources, coping processes, and mental health. *Annual Review of Clinical Psychology*, *3*(1), 377–401. https://doi.org/10.1146/annurev.clinpsy.3.022806.091520

Thoits, P. A. (2011). Mechanisms linking social ties and support to physical and mental health. *Journal of Health and Social Behavior*, *52*(2), 145–161. https://doi.org/10.1177/0022146510395592

Thomas, K. S., Bardwell, W. A., Ancoli-Israel, S., & Dimsdale, J. E. (2006). The toll of ethnic discrimination on sleep architecture and fatigue. *Health Psychology*, *25*(5), 635–642. https://doi.org/10.1037/0278-6133.25.5.635

Tomfohr, L. M., Ancoli-Israel, S., & Dimsdale, J. E. (2010). Childhood socioeconomic status and race are associated with adult sleep. *Behavioral Sleep Medicine*, *8*(4), 219–230. https://doi.org/10.1080/15402002.2010.509236

Tomfohr, L. M., Pung, M. A., Edwards, K. M., & Dimsdale, J. E. (2012). Racial differences in sleep architecture: The role of ethnic discrimination. *Biological Psychology*, *89*(1), 34–38. https://doi.org/10.1016/j.biopsycho.2011.09.002

Umaña-Taylor, A. J., Wong, J. J., Gonzales, N. A., & Dumka, L. E. (2012). Ethnic identity and gender as moderators of the association between discrimination and academic adjustment among Mexican-origin adolescents. *Journal of Adolescence*, *35*(4), 773–786. https://doi.org/10.1016/j.adolescence.2011.11.003

Van Reeth, O., Weibel, L., Spiegel, K., Leproult, R., Dugovic, C., & Maccari, S. (2000). Interactions between stress and sleep: From basic research to clinical situations. *Sleep Medicine Reviews*, *4*(2), 201–219. https://doi.org/10.1053/smrv.1999.0097

Vrshek-Schallhorn, S., Doane, L. D., Mineka, S., Zinbarg, R. E., Craske, M. G., & Adam, E. K. (2012). The cortisol awakening response predicts major depression: Predictive stability over a 4-year follow-up and effect of depression history. *Psychological Medicine*, *43*(3), 483–493. https://doi.org/10.1017/S0033291712001213

Walker, M. P., & Stickgold, R. (2006). Sleep, memory, and plasticity. *Annual Review of Psychology*, *57*(1), 139–166. https://doi.org/10.1146/annurev.psych.56.091103.070307

Wang, X., Sánchez, B. N., Golden, S. H., Shrager, S., Kirschbaum, C., Karlamangla, A. S., . . . Diez Roux, A. V. (2014). Stability and predictors of change in salivary cortisol measures over six years: MESA. *Psychoneuroendocrinology*, *49*, 310–320. https://doi.org/10.1016/j.psyneuen.2014.07.024

Weekes, N., Lewis, R., Patel, F., Garrison-Jakel, J., Berger, D. E., & Lupien, S. J. (2006). Examination stress as an ecological inducer of cortisol and psychological responses to stress in undergraduate students. *Stress*, *9*(4), 199–206. https://doi.org/10.1080/10253890601029751

Wilhelm, I., Diekelmann, S., & Born, J. (2008). Sleep in children improves memory performance on declarative but not procedural tasks. *Learning & Memory*, *15*(5), 373–377. https://doi.org/10.1101/lm.803708

Wolfson, A. R., & Carskadon, M. A. (1998). Sleep schedules and daytime functioning in adolescents. *Child Development*, *69*(4), 875–887. https://doi.org/10.2307/113235

Wolfson, A. R., & Carskadon, M. A. (2003). Understanding adolescents' sleep patterns and school performance: A critical appraisal. *Sleep Medicine Reviews*, *7*(6), 491–506. https://doi.org/10.1016/S1087-0792(03)90003-7

Zeiders, K. H., Doane, L. D., & Adam, E. K. (2011). Reciprocal relations between objectively measured sleep patterns and diurnal cortisol rhythms in late adolescence. *Journal of Adolescent Health*, *48*(6), 566–571. https://doi.org/10.1016/j.jadohealth.2010.08.012

Authors

JENNIFER A. HEISSEL is a doctoral student in human development and social policy at Northwestern University. Working with the Institute for Policy Research, she studies K–12 education policy, and she is particularly interested in how stress and sleep affect cognitive performance in children and adolescents.

DORAINNE J. LEVY is a postdoctoral researcher at Indiana University. She is primarily interested in research that examines the effects of discrimination on affective, cognitive, behavioral, and physiological outcomes.

EMMA K. ADAM, a developmental psychologist, has been with Northwestern's School of Education and Social Policy since 2000. She is interested in how everyday life factors, such as work, school, family, and peer relationships, influence levels of stress, health, and well-being in parents and their children.