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

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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

Challenging students 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...
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, Labirola, 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, Szirma, & Yonelinas, 2016), increases in cortisol due to acute stressors unrelated to the learning context are thought to hamper memory formation (Joëls, Pu, Wieghert, 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,
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; Ruitter, 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, & Börgels, 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, Vijzelraar, & 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 (Zeidler 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 (Rääikkönen 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 (Rääikkönen 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 (Plilhal & 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; Shirlcliff 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.
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. 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.

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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 Lente, & Granger, 2015; Wang et al., 2014).

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