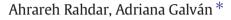
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The cognitive and neurobiological effects of daily stress in adolescents



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ABSTRACT

Increased stress reactivity during adolescence coincides with maturation of cognitive abilities and development of the prefrontal cortex. Although the effects of early-life, chronic, and pervasive stress on cognition have been extensively explored across development, very little is known about the effects of naturalistic, daily stress on adolescent cognition. In this study, our goal was to use a naturalistic approach to determine whether participants' own stressful experiences from daily life impacted cognitive performance and associated neural correlates. Adolescent and adult participants provided daily ratings of stress and underwent functional magnetic resonance imaging (fMRI) twice: once under a self-reported "high-stress" state and once under a self-reported "low-stress" state. While in the scanner, participants performed a response inhibition task. Behaviorally, all participants exhibited worse response inhibition under high, versus low, stress states, an effect that was significantly stronger in adolescents. At the neural level, there was a significant age by stress interaction, such that adolescents exhibited less recruitment of the dorsolateral prefrontal cortex (DLPFC) during inhibition under high-stress versus lowstress; adults evinced the opposite activation pattern in DLPFC. These data suggest that the developing brain may be a more vulnerable target to the cognitive and neurobiological effects of daily stress.

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Introduction

Relative to other developmental periods, adolescence is marked by stressful experiences and increased stress reactivity (Stroud et al., 2009), as well as psychosocial, physical and neurobiological changes (Persike and Seiffge-Krenke, 2012; Schlegel, 2001). Across different cultures, adolescents report having increased daily stress in the form of pressures from family (Bynner, 2000), school (Arnett, 2002), peers (Eccles et al., 1993; Hand and Furman, 2009) and romantic relationships (Kuttler and LaGreca, 2004). Daily stress arises from pressures of the recent past or pressures of the near future, and is the most common form of stress (Miller et al., 1994). The literature is rich with developmental studies examining the effects of chronic stress (a prolonged stressful period, often leading to serious physical or psychiatric illness, Baum and Polsusnzy, 1999) on cognition (e.g., Duckworth et al., 2012; Lupien et al., 2009; Pollak, 2005). Little is known, however, about the effects of daily stress on adolescent cognition. In contrast to chronic stress, daily stress in this study was defined as discrete (not prolonged) emotional strain resulting from demanding daily circumstances. This lack of empirical consideration is surprising, taking into account that both cognition and stress reactivity are in significant flux during this developmental time period. Our goal was to fill this gap in knowledge by examining the effects of naturally occurring (as opposed to

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http://dx.doi.org/10.1016/j.neuroimage.2014.02.007 1053-8119/© 2014 Elsevier Inc. All rights reserved. laboratory-induced) daily stress on cognitive control and associated neural correlates in adolescents. Understanding the specific effects of daily stress is important as stress exacerbates arousal-based decisions and behavior in adolescents (Figner et al., 2009).

During this developmental window, there are marked changes in cognitive abilities (increased reasoning and impulse control) and the neurobiological systems that support them (Casey et al., 2005). The brain undergoes remarkable development across adolescence (Eiland and Romeo, 2013; Galván et al., 2006; Gogtay et al., 2004; Shaw et al., 2008; Sowell et al., 1999), and the dorsolateral prefrontal cortex (PFC) is the last brain region to mature structurally and functionally (Casev et al., 2008; Chiron et al., 1992; Chugani et al., 1987; Fuster, 2001; Lewis, 1970). This region is critically involved in cognitive control, the regulation of emotional behaviors, and decision-making (Miller and Cohen, 2001). Furthermore, it is vulnerable to the effects of stress. In rats (Radley et al., 2006) and monkeys (Spinelli et al., 2009), stress reduces dendritic branching in the medial prefrontal cortex and neuronal reorganization in frontostriatal circuitry (Dias-Ferreira et al., 2009). In humans, laboratory-induced stressors alter prefrontal activation in response to performance anxiety (Dedovic et al., 2009), physiological (Porcelli and Delgado, 2009; Porcelli et al., 2012) and social stress (Eisenberger et al., 2007). Using a more naturalistic approach, Liston et al. (2009) found that a real-life, acute and discrete stressor (individuals preparing for a major academic exam vs. individuals undergoing no major psychosocial stress) selectively impaired attentional control and disrupted functional connectivity within the frontoparietal network (Liston et al., 2009).





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A long line of adult research shows that acute stress negatively affects cognition (Galván and McGlennen, 2012; Janis, 1993; Keinan, 1987; Mather and Lighthall, 2012; Porcelli and Delgado, 2009; Preston et al., 2007), in learning, memory, decision and inhibition domains (Mather and Lighthall, 2012; Roozendaal, 2002; Sandi, 2013; Wolf, 2006). For instance, response inhibition performance in adult males is significantly impaired following acute stress (Scholz et al., 2009). Animal research has also found that rodents (Bondi et al., 2008; Hennessy et al., 1973; Lapiz-Bluhm et al., 2009; Micco et al., 1979) and monkeys exposed to stress-level cortisol treatments have impaired response inhibition (Lyons et al., 2000), which is mediated via stressinduced atrophy of prefrontal neurons (Liston et al., 2006; Radley et al., 2004). These findings have been instrumental in establishing the mechanism by which acute stress can dysregulate cognition. However, this work has been limited to adult and animal populations so the ontogenetic effects remain unknown.

Current study

The current study examines how daily stress impacts cognitive control and its neural correlates in adolescents. We employed an Ecological Momentary Assessment (EMA) approach to monitor participants' naturalistic stress for two weeks through text messaging. This approach is a key divergence from previous work as participants were not subjected to the most commonly used tools to induce stress, such as a laboratory manipulation of stress (e.g., public speaking, cold pressor task) or recollection of previous stressors, which both incur potential limitations; the former can suffer from a lack of ecological validity, while the latter may be limited by recall bias and/or higher-order regulation of the stressful experience. EMA is an optimal way to assess daily stress, as it minimizes recall bias and maximizes ecological validity (Bolger et al., 2003). This method has been successful in the stress literature in determining how individual differences in various domains predict daily stress reactivity (e.g., Almeida and Kessler, 1998). Further, the within-subject design allowed us to use subjects as their own controls, thus allowing us to ask novel questions. For example: How does an individual's engagement of frontal circuitry change based on daily stress?

Each participant visited the laboratory twice, once on a day when they endorsed a high level of daily stress and once on a day when they endorsed a low level of daily stress. This novel approach allowed us to examine *within-person*, as well as developmental effects, thereby precluding potential confounds related to individual differences in laboratory-stress reactivity. At the lab, participants performed a Go/ No-go task while undergoing fMRI to assess cognitive control and as a probe for prefrontal function. Behaviorally, we predicted that daily stress would negatively affect cognitive control performance in both adolescents and adults, albeit with a stronger effect in adolescents. Furthermore, based on previous work in human adults (Ossewaarde et al., 2011; Porcelli and Delgado, 2009; Treadway et al., 2013) and in rodents (for review, see McEwen and Morrison, 2013) we predicted that compromised cognitive performance would be paralleled by reduced cortical engagement. Specifically, we hypothesized that the largest neural effect would be observed in the DLPFC, which undergoes significant development during adolescence.

Materials and methods

Participants

Participants included 45 right-handed English-speakers (n = 22 adolescents, ages 15–17, M = 16.5, SD = .76, 13 males; and n = 23 adults ages 25–30, M = 27.5, SD = 1.67, 10 males). Participants were recruited via advertisements on the UCLA campus, surrounding neighborhoods, and through Craigslist. Exclusion criteria included metal in the body (e.g., braces, permanent retainers), a diagnosis or a psychiatric or developmental disorder, claustrophobia, or pregnancy. Informed

consent was obtained from all adult participants, and assent was obtained from all participants under the age of 18 in accordance with procedures approved by the UCLA Institutional Review Board. The Wechsler Abbreviated Scale of Intelligence (WASI) was administered to estimate IQ; adolescents (M = 114.78) and adults (M = 117.52) did not significantly differ in IQ. Ethnic composition did not differ between age groups [adolescents: 48% Caucasian, 17% African-America, 26% Hispanic/Latino, 4% Asian-America, and 4% other; adults: 48% Caucasian, 13% African-America, 8% Hispanic/Latino, 26% Asian-America, and 4% other] nor did socioeconomic status [$\chi^2(1, 43) = .69$, p = .24], which was categorized based on maternal education.

Procedure

Participants were asked to come to the lab for an initial intake, during which a short battery of questionnaires was completed, and study procedures were explained. They then completed a baseline period to assess their normative stress assessment, followed by the experimental phase when they completed two scans (see Fig. 1 for an overview of the study design).

Ecological Momentary Assessment (EMA)

Daily stress was assessed using the EMA method, a procedure in which participants are contacted daily via smart phone in order to capture naturally-occurring stressors as they unfold (Bolger et al., 2003). EMA has previously been found to be a successful method of capturing naturally occurring stress (e.g., Almeida et al., 2009; Galván and McGlennen, 2012). In the current study, participants were contacted three times per day for two weeks and asked to indicate overall stress level using a Likert scale (1 = no stress; 7 = very stressed). Participants were not required to have a phone with a texting plan to participate, though all participants in the current study used their own personal cellular phone. The first three days of texting were used to establish a "baseline" composite stress rating for each participant, by averaging the three overall stress ratings obtained throughout the day. We then used these baseline ratings to categorize the two laboratory visits into high-stress state and low-stress state. In order to qualify as a highstress state, participants had to endorse at least one point higher than baseline. In order to qualify as a low-stress state, participants had to endorse at least one point lower than baseline. Each participant was asked to visit UCLA when experiencing a high-stress state and when experiencing a low-stress state (stress state visit order was counterbalanced across participants). During these visits participants completed an fMRI scan in which they performed a cognitive control task (i.e., Go/No-go). The average duration between stress reporting (stressor) and brain scan was 2 h and 5 min (SD = 67 min). There were no significant differences in duration based on stress state (t(44) = 1.38, p = .17) and did not differ by age group [(adolescents: t(21) = 1.40, p = .18; high-stress scan: 2 h 18 min; low-stress scan: 1 h 51 min; $M_{\text{difference}} = 27 \text{ min}$; (adults: t(22) = .42, p = .69; high-stress scan: 2 h 12 min; low-stress scan: 2 h 7 min; $M_{\text{difference}} = 5 \text{ min}$]. To determine whether the stress rating following this duration (i.e. the stress level at the scan) was most likely



Fig. 1. Study design.

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