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Conceptual precision is key in acute stress research: A commentary on Shields, Sazma, & Yonelinas, 2016

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ABSTRACT

A recent meta-analytic review by Shields, Sazma, & Yonelinas (2016) brings to the fore several conceptual issues within the stress and executive function (EF) literatures. We present a critique of these issues, using the review as an exemplar of how stress and EF are often examined empirically. The review summarizes research suggesting that EF is not only trait-like, but can be also state-like, influenced by factors such as acute stress. It has numerous strengths including its scope in examining EF across domains, inclusion of moderators, and timeliness, given the rapidly expanding field of stress research. We argue that the conclusions would be less equivocal with a more precise and neurally-informed consideration of EF, stressor, and timing assessments. A detailed discussion of these issues is provided, using the inhibition EF domain as an example, in order to illustrate key limitations and potential consequences of broad inclusion criteria. We endeavor to promote precise, shared definitions in the service of delineating a more complete and consistent account of acute stress effects on EF.

1. Introduction

A growing body of work shows that variance in executive function (EF) performance is not only trait-like, but can be also state-like, influenced by contextual factors such as acute stress. This research is particularly exciting as it begins to incorporate the role of the environment into EF research, which has key implications for interventions seeking to support effective goal-directed behavior in the real-world (Arnsten, 2009; Hofmann et al., 2012). Shields et al. (2016) recently conducted a meta-analysis of studies that examined effects of acute stress on EF across domains of inhibition, working memory, and cognitive flexibility. We applaud the authors of the meta-analysis for conducting such a timely and ambitious investigation in the service of reconciling inconsistencies in the literature regarding the size and direction of the effects of acute stress on EF. In addition, this meta-analysis raises some interesting and important questions with respect to the value of its broad inclusion strategy and its approach of collapsing results across studies with highly variable designs and EF measures. In this commentary on Shields et al. (2016) meta-analysis "The effects of acute stress on core executive functions: A meta-analysis and comparison with cortisol," we emphasize the importance of employing precise criteria that are informed by biobehavioral theory on the mechanisms through which acute stress affects neural processes. We further highlight the equivocal validity of conclusions about links between stress and EF in the absence of such precision.

We provide a detailed discussion of conceptual and measurement issues, using the inhibition EF domain as an example, in order to illustrate key limitations and potential consequences of the broad inclusion criteria used in the Shields et al. (2016) meta-analysis. Topics include: (a) Distinct and heterogeneous neurocognitive processes underlie performance on 'inhibition' tasks; (b) Consistent benchmark criteria must be used to establish that 'stress' occurred; and (c) Precise timing is critical when examining the effect of acute stress on EF given what is known about the neurobiology of stress systems. We wish to emphasize that there are many valuable aspects of the meta-analysis, especially the comprehensive examination of moderators across stressor paradigms and individual participant characteristics. By identifying areas that would benefit from greater conceptual precision, informed by biological and neuroscience research, our intention is to highlight the advantage of a more mechanism-focused approach to studying the effects of acute stress on EF. In turn, a better understanding of these mechanisms will suggest a more refined approach for subsequent meta-analyses and identify important questions for future inquiry.

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2. Distinct and heterogeneous neurocognitive processes underlie performance on 'inhibition' tasks

Although prior research has established that performance on EF tasks are moderately correlated, there is strong evidence across behavioral and neuroimaging literatures for dissociation of EF into the domains of inhibition, working memory, and flexibility (Miyake et al., 2000; Collette et al., 2006; Duncan and Owen, 2000). This "unity and diversity" principle is appropriately highlighted in the Shields et al. (2016) introduction as rationale for examining the separable effects of acute stress in each EF domain. A similar consideration of the heterogeneity in neurocognitive processes assessed by tasks *within* a given domain, however, is not sufficiently addressed.

Using 'inhibition' as an exemplar, the broad inclusionary approach is apparent in the search terms, which included "*cognitive inhibition, response inhibition, selective attention, executive attention, emotional interference, and sustained attention.*" Some of these search terms (e.g., sustained attention) represent constructs that are, at most, only partially overlapping with inhibition (Garavan et al., 2006; Aron et al., 2014). The breadth of search terms is also inconsistent with longstanding recommendations for specificity within the inhibition umbrella, based on the multiplicity of distinct processes and neural systems underlying inhibition (e.g., action versus thought versus emotion; Friedman and Miyake, 2004; Dillon and Pizzagalli, 2007; Aron, 2007). We appreciate that the authors recognized this distinction in comparing 'cognitive inhibition' and 'response inhibition' tasks and also examined similarly meaningful moderators in other EF domains (e.g., high versus low working memory load). However, the wide variability of tasks included within each subdomain (e.g., response inhibition: Stop-signal, Go/No-Go, Stroop color reading; cognitive inhibition: Stroop word reading, Emotional Stroop task, Simple forward span Flanker task) limits the inferences that can be drawn by that comparison.

For example, a strong case could be made to classify individual tasks differently (e.g., Stroop color reading as cognitive inhibition *or* attentional control). Furthermore, there are commonalities in recruited neural systems, but important differences also exist across tasks (Wager et al., 2005; Cieslik et al., 2015). Although the goal of such meta-analytic procedures is to offer the benefit of identifying alterations in performance linked to shared underlying neural systems, it is also possible that (1) the presence of significant results could be due to impairment (or enhancement) of neural processes unique to a subgroup of tasks OR (2) the impacts of stress on performance in a subgroup of tasks could be overlooked due to null meta-analytic results. Consistent with such potential risks, Aron (2007) suggests that when seeking to draw conclusions across diverse 'inhibition' tasks, it is prudent to only employ tasks with similar established underlying neurocircuitry in order to draw more meaningful mechanism-informed conclusions. The distinction may be particularly relevant for the response inhibition domain conclusions because the largest effect size (and sample size) of the five included studies employed the Stroop task. Although, behaviorally, the Stroop has been found to load similarly on a latent inhibition construct to the stop-signal and anti-saccade tasks (Miyake et al., 2000), it has relatively distinct neurocircuitry compared to other inhibition tasks (i.e., more left lateralized and reduced right inferior frontal gyrus activation; Chajut and Algom, 2003; Cieslik et al., 2015).

Another important distinction to be made, even between tasks within the same domain, concerns whether the task stimuli contain affective content. There are well-established differences in the neural systems recruited during affective and non-affective versions of the same task (Joëls et al., 2011; Arnsten and Rubia, 2012; Pessoa, 2009). For example, resolving response conflict, as required by a Flanker or Stroop task, activates dorsolateral prefrontal regions when the conflicting stimuli are non-emotional, and activates rostral anterior cingulate cortex/medial prefrontal cortex when the conflicting stimuli are emotional (Egner et al., 2008; Ochsner et al., 2009). Affective versions of inhibitory control tasks also tend to recruit activity in the amygdala and insula to a greater degree than their non-affective counterparts (Berkman et al., 2009). Taken together with the fact that the effects of acute stress are particularly pronounced in mesolimbic cortical structures such as the amygdala and rostral anterior cingulate cortex (Joëls et al., 2011; Arnsten, 2009), it stands to reason that acute stress might have different effects on affective and non-affective inhibitory control tasks. To their credit, Shields and colleagues note their intention to investigate emotional stimuli as a potential moderator along with their inability to do so, due to the small number of studies including affective content. Given this challenge, it would have been useful to determine if the meta-analytic results replicate when studies employing emotional stimuli were excluded. To our knowledge, no other research has tested the differential effects of acute stress on EF based on affective content, but this could be examined in the future and used as a moderator in subsequent meta-analyses, once more studies are published in this area.

Although it may not be possible to conduct a meta-analysis on identical tasks given the limited acute stress research to date, a critical comparison of the task demands could advocate for a more nuanced interpretation of the results. Even tasks that are more closely related (e.g., Go/No-Go versus Stop-Signal; affective versus non-affective Stroop) have non-trivial variability in demand characteristics, as well as differences in functional neuroanatomy with respect to stress-responsive systems, both of which are relevant for understanding effects of acute stress on inhibition performance (Eagle et al., 2008 ; Aron et al., 2014). Additionally, it may be possible that stress has diverging influences on performance because tasks rely on different neurotransmitters. For example, Stop Signal performance is sensitive to noradrenaline (a fast-acting signal prominently implicated in the effects of stress on the brain; Joëls et al., 2011), while Go/No-Go inhibition is associated with serotonin signaling (Eagle et al., 2008), so acute stressors might have greater effects on Stop Signal (consistent with a recent finding by our research group; Roos et al., 2017), compared with Go/No-Go inhibition performance.

3. Acute stress research requires consistent benchmark criteria to establish the onset of a stress response

In over half a decade of research on acute stress, a common critique across reviews is the subjective terminology regarding (a) what is considered stressful (versus frustrating or arousing) and (b) the variability in such paradigms' ability to produce a biological measure of stress validation (e.g., cortisol, Dickerson and Kemeny, 2004; Gunnar et al., 2009). In contrast to the sympathetic adrenal medullary (SAM) axis, which is activated in response to effortful, arousing, or challenging tasks, evidence suggests that the HPA axis only responds when such challenges are linked to socially evaluative distress (reviewed in Kudielka et al., 2007). Accordingly, cortisol reactivity has become the gold standard stress response benchmark (Dickerson and Kemeny, 2004).

Critically, stress-induction paradigms do not universally elicit a cortisol response across time and across labs, so cortisol reactivity cannot be assumed when using a given paradigm, even when that paradigm has previously elicited a cortisol response. However, the meta-analytic inclusionary criteria included any paradigms "previously validated" by either a biological measure of stress (i.e., a cortisol response) or the presence of face-valid elements that should theoretically induce a cortisol response (i.e., motivated performance with socio-evaluative threat). The limitation in this approach is that it assumes a stress response occurred without verifying significant cortisol reactivity. In healthy populations, defining a 'stressor' by the documentation of HPA axis reactivity has considerable value for increasing precision in acute stress research and our understanding of the effects of *stress* on EF, as opposed to the effects of frustration, disappointment, or challenge. We note that certain individual characteristics (e.g. history of childhood maltreatment, psychological/psychiatric disorders) have been linked to blunted cortisol responses, which can make employing