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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 neurocircuity 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

the cortisol reactivity benchmark less relevant in studies of such subpopulations (e.g. Ginty et al., 2012). However, because the meta-analysis did not examine such subpopulations, employing the cortisol reactivity benchmark would be of substantial value to guide future work.

We acknowledge that applying stringency in such criteria limits the number of studies eligible for inclusion and power for examining the effects of acute stress on EF. Although we believe that this cost is outweighed by the benefits of employing reactivity inclusionary criteria, we appreciate the steps taken by Shield's and colleagues to investigate biological reactivity validation as a possible moderator of the effects acute stress on EF, with results indicating null moderation effects. We would encourage researchers conducting future acute stress meta-analyses who prefer to not employ biological reactivity validation criteria to include similar moderation analyses in order to examine if different methodological approaches significantly bias results.

Given that cortisol reactivity was not used as a benchmark, a careful consideration of the inclusion of paradigms that have qualities that 'should' elicit a stress response (i.e., motivated performance with socio-evaluative threat) but fall short of the set of stressor criteria provided by Dickerson and Kemeny (2004) is of particular importance. For example, Dickerson and Kemeny (2004) define 'social-evaluative stress' paradigms as those that include "*a permanent recording, presence of an evaluative audience, or presence of a negative social comparison* (*i.e. the real or potential out-performance by a confederate or other participant*". However, the authors of the Chajut and Algom (2003) study claim their task elicits social-evaluative stress because participants are told in the task instructions that the selective attention task is a "*measure of cognitive ability*" and are requested to enter their names on a computer, "*increasing the social relevance"* and that they "*would be able, should they desired to do so, to compare performance to normative data.*" We argue that these factors are likely insufficient to qualify as a true social-evaluative threat because the social comparison is only to aggregate de-identified data (i.e., not to a specific individual/confederate) and the proposed comparison is described as 'optional.' Further, only 1 of 4 possible uncontrollability factors outlined by Dickerson and Kemeny (2004) was met (i.e., manipulation of task difficulty). This is of further concern because neither the Chajut and Algom (2003) study, nor the studies from which this task was adapted, had established biological reactivity validation.[1]

There are multiple other factors related to acute stress cortisol reactivity that would be of particular value to discuss in detail and emphasize for future research. For example, numerous reviews have emphasized the substantial heterogeneity in cortisol response for the same stressor paradigm, with extensive work directed at determining and describing responders vs. non-responders (Dickerson and Kemeny, 2004; Miller et al., 2013). Accordingly, there is likely high heterogeneity in the actual cortisol responses amongst articles reviewed, both between studies and within subjects. With such individual differences in mind, indexing a given study's cortisol response effect size may not be the most appropriate analysis for the purpose of estimating links between cortisol responsivity and EF vulnerability to acute stress. Instead, a more focused examination of the links between individual cortisol responses predicting EF, within a given study, would more clearly link cortisol reactivity to any putative changes in EF.

A second consideration related to stressor definitions is that type of stressor employed varied widely across systemic, psychological, and cognitive load type. Although Shields et al. (2016) address this by examining the stressor type as a potential moderator, the paper would benefit from a discussion about the theoretical importance of such an examination. There are established reasons why different types of stressors would have disparate effects on biological and neurological processes (Smeets et al., 2012). As appropriately noted by the Shields and colleagues in the limitation section, the small number of studies included within each EF domain type, particularly in the inhibition and cognitive flexibility domains, renders the null effects regarding 'stressor type' particularly difficult to interpret; the field will benefit from focused investigations into the role of stressor type and severity once there is a larger literature base.

4. Precision in timing is important to understanding EF measurement in relation to acute stress, given what is known about the circuitry of the stress system and how it functions

Finally, precision of the timing between the stress induction and cognitive performance is critical in studies measuring the effects of acute stress on EF performance. Acknowledging this, Shields et al. (2016) examined timing as a moderator based on the delay between *stressor onset* and the assessment of executive function. This approach was based on another meta-analysis on the effects of cortisol *administration* on executive function, which examined timing parameters based on cortisol's short-term (i.e., theorized to be non-genomic; rapid, within minutes) and longer-term (i.e., theorized to be genomic; slow, greater than 1 h) effects on different neural systems (Joëls et al., 2011; Shields et al., 2015). However, the nature and timing of the stress response, which is the focus of the Shields et al. (2016) meta-analysis, is systematically different from that of exogenous cortisol administration in which participants receive a bolus dose of cortisol at one time point and no ongoing psychosocial stressor context. These considerations are particularly relevant in endogenous stress response research given that the 'stress-inducing' qualities of acute stress (e.g., uncontrollability, experiences of failure) may not be consistently present during a stressor and can emerge gradually (Del Giudice et al., 2011; Dickerson and Kemeny, 2004).[2]

One key concern about anchoring to the onset of the stressor paradigm in examining the effects of acute stress on behavior is that this approach ignores task context, which evolutionary accounts suggest would moderate the effects of stress (de Kloet et al.1999; Del Giudice et al., 2011; McEwen, 1998). Specifically, biological responses to acute stress evolved to promote a specific and coordinated 'fight or flight' response (Del Giudice and Belsky, 2011; McEwen, 1998). Accordingly, stress system responsivity is expected to facilitate certain types of cognitive function *during* stress (e.g., those that require exaggerated vigilance and additional metabolic resources) and perhaps attenuate those same responses *after* stress, in the recovery period (Arnsten, 2009; Del Giudice and Belsky, 2011; Linden et al., 1997).

In light of this distinction, the effects of acute stress on cognitive performance might be better conceptualized as two separate questions. First, does acute stress and its biological consequences support concurrent cognitive performance under stress? Second, are there residual consequences of biological reactivity on subsequent EF function after an acute stressor has ended? Time since stressor conclusion (i.e., when recovery should start) would be key for addressing questions about the effects of prolonged stress system activation and homeostatic recovery processes on EF. The potential consequences of this distinction are particularly relevant for the inhibition analyses: one of six articles examines response inhibition under stress (i.e., time pressure and psychosocial threat) and five of six articles examine response inhibition at variable intervals after acute stress. Notably, the study with the largest sample size and effect size linking acute stress to facilitating response inhibition is the sole study that examines EF performance *during* such stressful conditions and may bias the results (Chajut and Algom, 2003).

A further source of timing confounds is the variability in the time

[1] The authors do note that the Chajut and Algom (2003) study was only one of two studies included that had no paired study of biological reactivity. However, the potential for different conclusions in the 'response inhibition' domain based on the inclusion of this study is of concern given that it had the largest sample size and the largest effect size in the response inhibition EF domain.

[2] Notably, there are also similarities in timing imprecision for exogenous cortisol administration and acute stress studies linked to variable length in EF tasks.

course of the stressor paradigms (e.g., the Trier Social Stress Test [TSST], Socially-Evaluative Cold Pressor Task, [SECPT] etc.), which may introduce variability regarding the time course of onset and recovery in stress-system responsivity. For example, the standard TSST protocol employed in a number of the studies included in Shields et al.'s (2016) *meta*-analysis (e.g., Giles et al., 2015) is approximately 20 min long, while the SECPT protocol is 3 min in duration (Schwabe et al., 2013). The importance of precision in acute stress timing has been highlighted by other researchers given that the effects of stress-system activation involve a "*fine-tuned hormonal interplay of which time is of the essence*" (Joëls et al., 2011). Notably, challenges in timing precision also arise from the variable length of EF tasks that would be present across both exogenous cortisol administration and acute stress studies.

Taken together, we suggest that the timing moderator analyses in the Shields et al. (2016) meta-analysis, which do not consider stressor contexts (i.e., during or after stress), time since stressor offset, or the variable duration of stressor paradigms, may be unreliable. Future research examining these distinctions could help clarify the relevance of the context of EF assessment and lend further insight into how the timing characteristics of acute stressor paradigms influence EF performance.

5. Discussion

The goal of this commentary was to identify key methodological considerations for studies measuring the effects of acute stress on EF that limited the precision of the inferences that could be drawn from the Shields et al. (2016) meta-analysis. In particular, a more nuanced and neurally-informed approach to considering the roles of EF task, stressor paradigm selection, and the timing of the stressor in relation to the EF task might have led to considerably different conclusions. At minimum, additional discussion about the potential implications of the inclusion/exclusion decisions and the grouping of heterogeneous studies in the meta-analysis would be helpful for guiding a more precise interpretation of results. We note that these critiques are not specific to the Shields et al. (2016) review, but rather reflect imprecision in the acute stress field broadly. We hope that highlighting them in this commentary will invoke more precision and promote shared definitions in future work within the rapidly expanding field of acute stress research.

In considering the conclusions from this meta-analysis and looking to the future, there is a need for more research to incorporate additional biomarkers that we now know are linked to EF (e.g., fMRI, EEG, pupil diameter). This expanded view of the stress-linked neurobiological factors relevant to EF will help map how acute stress alters functioning of neural systems underpinning effective EF performance. For example, preliminary work has suggested that biomarkers of cognitive load (indexed by pupil diameter) predicted worse post-stressor sustained attention for participants with negative mood (Vinski and Watter, 2013), and negative affect, which often co-occurs with acute stress, alters both neural activation and response inhibition ability (Patterson et al., 2016).

Similarly, a consideration of how other stress response systems, such as the sympathetic and parasympathetic nervous systems, may be involved in the effects of acute stress on EF would be of substantial value. Autonomic nervous system function may be linked to EF based on shared top-down cognitive appraisal and regulatory resources that may involve overlapping and/or competing resource allocation during or following acute stress (reviewed in Graziano and Derefinko, 2013). Indeed, preliminary evidence suggests that the autonomic nervous system may play a central role in the effects of acute stress on EF. For example, Elzinga and Roelofs (2005) documented that sympathetic arousal could be the underlying mechanism directing EF over and above any effects the stressor may have on the HPA-axis. Similarly, our own research examining the effects of acute stress on stop-signal inhibition found that parasympathetic augmentation to the TSST predicted individual differences in EF impairment (Roos et al., 2017). The recommendation for considering multiple stress response systems is consistent with recent advice from the field of stress psychology that encourages the use of multiple biomarkers when seeking to understand the effects of interactive systems on behavior (Allen et al., 2014; American Psychological Association, 2016).

Taken together, there are numerous strengths of the Shields et al. (2016) review including its scope and ambition in examining EF across domains, thorough consideration of important moderating factors, and timeliness given the recent increase in research on this topic. The review's conclusions would have been even more impactful with a more precise and neurally-informed consideration of moderators with respect to EF task, stressor paradigm, and timing. Nonetheless, future work can benefit by accounting for these factors. Subsequent investigations incorporating biomarkers of various neurocognitive and physiological processes may be key to delineating a more complete and consistent account of acute stress effects on executive function.

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