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Commentary

Balancing precision with inclusivity in meta-analyses: A response to Roos and colleagues (2017)

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ABSTRACT

Roos and colleagues' commentary on our recent meta-analysis examining acute stress effects on executive functions is an important delineation of the limits of meta-analyses with heterogeneous outcomes. In this response, we wish to both clarify the inferences we feel are appropriate given the analyses and address the methodological concerns they raise. Additionally, we present new analyses that answer questions raised in their commentary. We first discuss the classification of a broad array of tasks that depend upon the same construct (e.g., inhibition) and note that this allows for inferences regarding the process that underpins all of those tasks, but this does not entail that all tasks dependent upon that construct will evidence the same effect of stress. Second, we argue that requiring that a study present a significant effect of stress on cortisol for inclusion in analyses is too stringent for a number of reasons (e.g., some studies using validated stressor paradigms correctly do not assay cortisol for budgetary reasons) and we present analyses showing that even when studies that did not present a cortisol response were removed, the initially observed effects still held. Finally, we address concerns raised regarding analyses of time-dependent effects by presenting new analyses that help to allay those concerns. In sum, we applaud Roos and colleagues' exortation for greater methodological and conceptual rigor in studies of stress and executive function, and the additional analyses prompted by their questions help to clarify observed effects and further the field of stress and executive function research.

1. Introduction

Roos et al.'s (2017b) commentary on our meta-analysis of acute stress effects on executive function (Shields et al., 2016b) is an appropriately-timed commentary noting the limits of the precision of inferences that can be made in our and any meta-analysis examining multiple outcomes. In this response, we wish to both clarify the inferences we feel are appropriate given the analyses and address the methodological concerns they raise. Although many of the examples presented by Roos and colleagues in their commentary (e.g., reclassifying the Stroop task) are, in fact, examples in service of broader points they wish to make, we will examine them in detail when possible in order to determine whether altering analyses in ways Roos and colleagues suggest fundamentally alters the conclusions warranted by our meta-analysis. In addition, by examining these examples in detail, we hope to make a broader point as well, which is to show that the decisions made in our original meta-analysis were not arbitrary; instead, our decisions were made based upon findings and suggestions from prior literature.

At its core, Roos and colleagues' article is an exhortation for greater methodological and conceptual rigor in studies of acute stress effects on executive functions. We applaud this position and agree with it wholeheartedly. Presumably, greater methodological and conceptual rigor would help to explain discrepant results and further clarify the effects of acute stress on executive functions (see also Schaller, 2016). Indeed, our analyses (Shields et al., 2016b) found that the precision in which a given study examined the effects of acute stress on executive function tasks was a significant predictor of effects of stress on working memory and cognitive flexibility. Thus, we agree that methodological and conceptual precision should be a high priority in these studies and meta-analyses of them.

Ideally, we would benefit most from a meta-analysis of specific stressor paradigms on specific executive function tasks. Each stressor paradigm used is unique in its characteristics (Kirschbaum et al., 1993; Schwabe et al., 2008; Shields and Slavich, 2017) and even demands on cognition (e.g., the cold-pressor test requires consistent response inhibition to withhold removing one's arm from ice water, whereas the arithmetic task in the Trier Social Stress Test taxes working memory). Similarly, each executive function task contains elements not common to other tasks designed to assess the same construct (Friedman et al.,

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2008, 2006; Friedman and Miyake, 2004; Miyake et al., 2001, 2000; Testa et al., 2012). However, the literature examining acute stress effects on executive functions is nascent and thus too small to support such analyses. As such, when we conducted this meta-analysis, as with any meta-analysis, we had to balance being inclusive enough to permit analyses and being precise enough to make claims about the constructs we analyzed. For stressor paradigms, we attempted to achieve that balance by only including stressor paradigms that were a previously validated stressor (e.g., the Trier Social Stress Test), contained components sufficient to elicit a stress response (i.e., a task requiring motivated performance with socio-evaluative threat; Dickerson and Kemeny, 2004), or included a biological measure of stress validation (e.g., cortisol, cytokine reactivity) that is not also sensitive to the effects of acute arousal without stress. For executive function tasks, we followed classification of tasks made in prior research (e.g., Diamond, 2013), such as from factor analytic work showing that tasks with varying characteristics—e.g., the stop signal task and Stroop task—load on the same latent factor (Friedman et al., 2008, 2006; Friedman and Miyake, 2004; Miyake et al., 2001, 2000; Miyake and Friedman, 2012). We elaborate on these decisions and discuss their potential limitations below.

2. On heterogeneity in executive function tasks

Because the tasks included in our meta-analysis of each executive function are heterogeneous, we do not claim to determine effects of acute stress on all factors contributing to performance in each class of executive function tasks. For example, performance on the relatively simple change detection working memory task is well described by a model estimating parameters for attention, capacity, and guessing bias (Rouder et al., 2008); however, the extent to which performance on this task is sensitive to variation in attention, capacity, or guessing presumably differs from another working memory task, such as the OSPAN. Additionally, performance on the OSPAN incorporates a number of neurocognitive processes unassessed in the change detection task, such as word knowledge (Unsworth et al., 2005). Thus, although stress likely exerts different effects on these tasks depending upon what neurocognitive component processes they require and to what extent those processes are required for performance, stress—presumably—should exert the same effects on the underlying processes that it affects (e.g., capacity). As such, although the tasks included are heterogeneous, we balanced inclusivity and precision in task selection by including all tasks thought to rely primarily on the same underlying executive function factor for performance in part. This entails that although we cannot make claims about the effects of stress on any given task relying on that executive function, we can make claims about the effects of stress on that executive function itself.

Roos and colleagues note that many terms included within our search string only partly overlap with inhibition. However, tasks that assess each of the quoted terms (e.g., sustained attention) have been found to load on the same latent factor as response inhibition tasks (Friedman and Miyake, 2004; for reviews, see Diamond, 2013; Miyake and Friedman, 2012). Thus, although these tasks are distinct and performance on them is unique, performance on them is also partly underpinned by the same construct—inhibition—and this construct was what we examined the effects of acute stress on. Similarly, our categorization of tasks as response or cognitive inhibition followed prior literature in classifying the tasks. Although the tasks assigned to each construct are variable, effects of stress on the particular construct of interest is what emerges from an analysis of all of the tasks together.

Roos and colleagues also suggest that the Stroop color reading task might be better classified as cognitive inhibition, and, as such, should not have been included with the response inhibition tasks in analyses. To make this argument, they cite work showing that performance on the Stroop task is associated with different neural activity from other response inhibition tasks, including the stop-signal task and the go/no-go task (Cieslik et al., 2015). However, importantly, Cieslik and colleagues also conducted a conjunction analysis and found overlap between all of the aforementioned tasks in activation of the anterior insula and right inferior frontal junction. Moreover, a stronger case can be made for including the Stroop task with response inhibition tasks. In particular, in Miyake et al.'s (2000) paradigmatic factor analysis of the structure of executive function, the Stroop and Stop-Signal Task both loaded on the latent factor of inhibition, and did so with approximately the same factor loadings (see also Friedman and Miyake, 2004). Thus, there is evidence that the Stroop task is properly classified as a response inhibition task. Despite differences in characteristics from other response inhibition tasks—leading to different patterns of activation—neural and behavioral evidence converge to show that performance on the Stroop task underpinned in part by response inhibition, and including it in analyses of response inhibition allows the effect of stress on response inhibition to emerge from the aggregate.

Finally, even if it is concluded that the Stroop task requires both response and cognitive inhibition and therefore it should not be included in analyses of response vs. cognitive inhibition, the results remain essentially the same. In particular, after excluding the Stroop task, inhibition type (response compared to cognitive) remained a significant moderator of acute stress effects on inhibition, $B = .418$, $p = .024$. Similarly, acute stress marginally enhanced response inhibition even without the Stroop included in analyses, $g^{+} = .193$, $p = .069$ (note that the lack of significance here is primarily due to a lack of power; after excluding studies with the Stroop task, there were only four studies left in the response inhibition category). Thus, inclusion of the Stroop task with the response inhibition studies was not responsible for the moderating effect of inhibition type or the enhancing effect of acute stress on response inhibition in our meta-analysis.

In closing their statement on task heterogeneity within our meta-analysis, Roos and colleagues admonish a more nuanced interpretation of our results based upon neurocognitive findings: "For example,… acute stressors might be expected to have greater effects on Stop Signal… compared to Go/No-Go inhibition performance" (p. 7). We acknowledge that this may certainly be the case; as we outlined in our first paragraph in this section, stress may differentially influence performance on measures in the same class of executive function tasks depending upon the neurocognitive component processes each task requires. However, because we do not have the statistical power to examine effects of acute stress on individual tasks, we did not and are not making claims about the effects of stress on individual tasks (e.g., the Go/No-Go or the Stop Signal). Instead, we made claims about the effects of acute stress on the underlying construct thought to contribute to performance across these tasks (e.g., response inhibition).

3. On requiring cortisol as a benchmark to establish a successful stress induction

Because of the cost of cortisol assays, some researchers may rely on prior validation for a stressor paradigm or a theoretical framework for stress induction despite not being able to conduct assays of cortisol in the actual study of interest. Roos and colleagues suggest that our choice to include studies in which the biological stress response was not explicitly confirmed by a significant stress-induced cortisol response within that exact study was a poor decision due to a lack of conceptual precision in stress. We respond to this in the following ways.

First, we believe that requiring a study show a significant cortisol response for inclusion is too stringent. Due to chance alone, it is possible for a successful stress induction to fail to return a significant cortisol response (i.e., a type II error). Similarly, due to differences in HPA axis regulation, some individuals—such as women taking hormonal contraceptives, those with psychiatric disorders (e.g., schizophrenia, ADHD), abstinent alcoholics, those with tinnitus, or those who suffered from childhood maltreatment—evidence blunted or completely absent cortisol responses to stress (Hébert and Lupien, 2007;

Jansen et al., 1998; Kirschbaum et al., 1995; Lovallo et al., 2000; MacMillan et al., 2009; Randazzo et al., 2008). These responses are blunted not because the participants are not stressed, but for biological reasons. Thus, because cortisol responses do not always accompany a successful stress induction, requiring a study show a significant cortisol response for inclusion in analyses is too stringent.

Roos and colleagues suggest that because we did not specifically examine the subpopulations mentioned above, these blunting effects are irrelevant. However, this is misguided for two reasons. First, to our knowledge, no studies of stress and executive functions excluded abstinent alcoholics, individuals who suffered from childhood maltreatment but did not have a psychological/psychiatric disorder, or individuals suffering from tinnitus. Moreover, many studies of stress and executive functions did not have formal health-related exclusion criteria and thus did not exclude individuals with psychiatric, psychological, or medical disorders that are associated with blunted cortisol responses to stress. As such, many studies presumably included participants who would not show a cortisol response to stress despite being stressed. Second, our decision to include these studies is theoretical: cortisol responses do not always accompany a successful stress induction (either because of a Type II error or sample constituency), so requiring a study to show a significant cortisol response even if a study uses a previously validated stressor or paradigm theoretically thought to induce a stress response is too stringent. This point may be a simple disagreement on the relative importance of inclusivity and precision in analyses. However, as we describe below, studies that validated their stressor with a significant cortisol response did not differ from studies that did not, making this disagreement more theoretically than practically important.

Because we collected cortisol reactivity data for each study, we were able to test whether studies that validated their stress inductions by presenting a significant stress-induced cortisol response differed in their effects on executive function from studies that did not present cortisol or did not have a significant effect of stress on cortisol. We presented these results within footnote #1 in our published meta-analysis. Notably, studies that confirmed their stress inductions with a cortisol response relative to a control condition did not differ from studies that did not confirm their stress inductions in that way across all studies, $t(30.7) = -1.07$, $p = .295$, studies examining working memory, $t(15.6) = -0.35$, $p = .734$, and studies examining inhibition, $t(14.8) = -0.61$, $p = .549$. Not enough studies of cognitive flexibility reported cortisol for us to reliably examine this contrast in only studies of cognitive flexibility. Although the above are null effects, the analysis across all studies (i.e., all 53) was not null primarily due to a lack of power. Thus, studies with stress paradigms not validated by a significant stress-induced cortisol response within the study itself did not differ from those without a cortisol response, as long as the paradigm had been previously validated or contained characteristics sufficient to induce a stress response.

Roos and colleagues criticize the inclusion of one study (Chajut and Algom, 2003) in the meta-analysis in particular, stating that "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" (p. 8). Roos and colleagues argue that the stressor paradigm Chajut and Algom does not actually contain social-evaluative stress because it does not meet the criteria for social evaluation defined by Dickerson and Kemeny (2004) in their meta-analysis. However, we believe that this is not true. Within Chajut and Algom's study, participants in the stress condition were told that they would be able to compare their performance in the difficult task to normative data, which we believe satisfies Dickerson and Kemeny's third criterion: "presence of a negative social comparison (the real or potential outperformance by a confederate or other participant)" (p. 361). Although the comparison to normative data were optional in Chajut and Aglom's study, so is knowing normative data against which to compare one's SAT scores, and taking the SAT was identified by a number of high school students as the most severe stressor they had experienced in the previous year (Shields & Slavich, unpublished results). As such, although interpretation of Dickerson and Kemeny's criterion is debatable, we believe that this study satisfied the criteria for socio-evaluative stress and should thus be included in analyses.

In addition, and more importantly, removing Chajut and Algom (2003) from our analyses did not impact the results. Inhibition type (response vs. cognitive) remained a significant moderator of acute stress effects on inhibition, $B = .420$, $p = .010$, and acute stress still significantly enhanced response inhibition, $g^{+} = .198$, $p = .025$. Thus, including Chajut and Algom in analyses was not responsible for the enhancing effects of acute stress on response inhibition that we observed.

Similarly, removing the only other study (Steinhauser et al., 2007) that did not use either a biological measure of validation within the study or a stressor that was previously biologically validated did not impact the results: stress still significantly impaired cognitive flexibility, $g^{+} = -.344$, $p = .043$. Thus, including studies that contained a stressor with characteristics theoretically believed to induce a stress response but that did not use a previously validated stressor or validate their stressor within their study was not responsible for the effects we observed in our analyses.

Roos and colleagues also note that there is considerable variability in cortisol responses to stress and state that an individual study's cortisol response might not be the best predictor of an individual study's effect of stress on executive function, suggesting that examining correlations of a cortisol response and executive function within a single study provides a clearer picture of any association between the two. We agree with this, and also note that there are a number of individual differences moderating how stress influences executive functions (e.g., Elzinga and Roelofs, 2005; Shamosh and Gray, 2007)—which is important to stress-related outcomes (Quinn and Joormann, 2015; Shields et al., 2017). However, if cortisol is exerting a main (i.e., non-interactive) effect on executive functions, this effect should track both within and across studies. As we stated within our initial meta-analysis, the lack of association we observed suggests other psychological or hormonal factors may interact with cortisol responses to influence executive functions.

In sum, requiring a study present a successful cortisol response for inclusion in our meta-analysis would have reduced our statistical power unnecessarily, given both the aforementioned reasons why a study may not include a significant cortisol response and the lack of difference between studies with a significant cortisol response and studies without. As such, we believe that our inclusion criteria for stressor paradigms appear to appropriately balance inclusivity and precision.

4. Precision in timing of acute stress effects on executive functions

Roos and colleagues suggest that our analyses of the timing of acute stress effects on executive functions are unreliable due to the nature of the endogenous stress response. In particular, they provide two criticisms.

Their first criticism is that because the timecourse of stress effects (Dickerson and Kemeny, 2004; Lennartsson et al., 2012a, 2012b; McEwen, 2007; Shields et al., 2016a) differs from exogenous administration—which is typically given as a single bolus (Shields et al., 2015)—we cannot make reliable claims about the timecourse of acute stress effects on executive function like we can of cortisol administration. However, in these analyses we are not claiming to make claims about the timecourse of effects of *stress-induced cortisol* but about the timecourse of effects of *stress* on executive functions. The fact that cortisol (and other stress-responsive hormones and proteins) shows a graded and time-dependent release is irrelevant to making claims about the timecourse of effects of *stress* on executive function, and these graded releases are consistent both within and across studies relative to

stressor onset or offset (e.g., Dickerson and Kemeny, 2004).

Their second criticism is that stressor paradigms differ in how long they take to complete, making comparison across paradigms difficult.[1] This is an excellent point. Within our paper, we examined the delay between stress *onset* and executive function testing; this examines the timecourse of effects of stress on executive function after stress began—making the initial time-dependent effects of stress (e.g., nongenomic and genomic effects) occur at relatively similar times across studies, since the initial stress response is consistent across studies whenever the stress-inducing qualities of a stressor begin. Presumably, however, the offset (i.e., the end) of the stressor relative to the task is also important because the time-dependent effects of stress (e.g., the relative contribution of nongenomic and genomic effects) might differ if a stressor lasted longer. As such, to ensure the timing results we obtained were not due to this confound, we controlled for the delay between stress offset and executive function testing as well as the type of stressor both separately and together. In these analyses, the delay between stress onset and executive function testing remained the same—significant for working memory ($Bs \geq -.009$ and $\leq -.006$, $ps \leq .033$) and nonsignificant for both inhibition ($Bs \geq -.007$ and $\leq -.005$, $ps \geq .109$) and cognitive flexibility ($Bs \geq -.001$ and $\leq .006$, $ps \geq .468$).

It is important to note that we do not make claims about the lack of observed effects for inhibition and cognitive flexibility. There were too few studies and too much variability across studies for us to conduct analyses of the timecourse of stress effects on cognitive flexibility, response inhibition, or cognitive inhibition with a reasonable level of confidence in the results. In addition, and even more importantly, the moderating effect of time here is associational (across studies) rather than experimentally manipulated. Thus, although we are reasonably confident that acute stress is related to working memory performance over time in the way we observed, we believe that the timecourse of acute stress effects on each executive function should be examined within a single experiment.

Roos and colleagues also suggest that effects of acute stress on executive functions during stress may qualitatively differ from effects of acute stress on executive functions after the stressor has passed. In fact, this was a question we considered prior to conducting our meta-analysis.[2] However, there are currently not enough studies to conduct such an analysis with any reasonable amount of confidence in the results. We encourage future researchers to examine this possibility by experimentally manipulating the timing of the stressor relative to completion of an executive function task.

[1] As Roos and colleagues point out, comparing timing across studies of exogenous cortisol administration is subject to the same type of limitation as comparing timing across studies of acute stress: some executive function tasks take longer than others to complete (this is especially true when the study also examines effects on ERPs, which can produce extremely long executive function tasks). As such, the relative contribution of genomic and nongenomic glucocorticoid effects to task performance differs not only across studies of stress and executive function, but also across studies of cortisol administration and executive function.

[2] Roos and colleagues state that the study with the greatest enhancing effect of stress on response inhibition (Chajut and Algom, 2003) assessed executive function during, rather than after the acute stress induction. However, this not clearly described in Chajut and Algom's Procedure section. In particular, Chajut and Algom state, "The participants were tested individually in a dimly lit room. The three 'psychometric' tests [which were listed above in the manipulation of stress section] comprised the first part of the experiment. The tests were fully computerized, with item difficulty, time constraints, and the instructions regarding personal relevance differing for participants in the high-stress and the low-stress conditions. The participant entered responses to all of the items in all of the tests by pressing the appropriate key on the computer keyboard. After conclusion of the three tasks, the [Stroop task was completed]…" (p. 237). It is certainly possible that the stressor was not clearly demarcated and the participants thought that they would be evaluated on their Stroop task performance as well; however, as stated, the Stroop task came after the cessation of the stressor within their paradigm.

5. Discussion

Roos and colleagues commentary is in general an appropriate reminder of the difficulties and limitations of meta-analyses with heterogeneous outcomes assessing the same construct (see also Scammacca et al., 2014). We agree with Roos and colleagues in that greater precision would be beneficial: analyzing how stress affects specific tasks would be ideal for making claims about those tasks (Cooper, 1998). However, as in our original paper, we argue not that stress will exert uniform effects on all tasks considered in the same analysis (e.g., response inhibition tasks) but that stress exerts effects we found on processes underlying performance across all tasks considered in the same analysis (e.g., response inhibition). Thus, while acute stress may enhance response inhibition, it may impair performance on some response inhibition tasks that also rely on other neurocognitive processes impaired by stress—such as working memory, cognitive flexibility, or some nonexecutive processes.

We also do not claim that stress always exerts the same effects on each executive function; there are numerous moderators of these effects, both ones we discussed within our meta-analysis and ones not yet known. For example, a recent study of stress effects on response inhibition (Roos et al., 2017a) found impairing effects of stress on one response inhibition task (the stop-signal task), whereas an earlier study found enhancing effects of stress on that same task (Schwabe et al., 2013). No moderators in our meta-analysis can explain this discrepancy, illustrating that there is still much work to be done in understanding effects of stress on executive functions. However, across all paradigms and situations, our meta-analysis showed the main (i.e., unmoderated) effects of acute stress on executive functions and uncovered some moderators of those main effects.

Calls for more precision in studies of stress and cognition as well as a greater integration of a biologically-informed theoretical framework can only be applauded. Similarly, reminders of the limited precision of inferences possible from meta-analyses including multiple outcomes is important, as it is important not to overinterpret any results. Roos et al. (2017b) commentary is valuable for all of these reasons and more. In this reply, we hope to have clarified the inferences made possible by our meta-analysis as well as addressed Roos and colleagues' stated analytical and methodological concerns. As we hope to have made clear through this commentary, we do not believe work required to understand stress effects on executive functions is close to finished—there are a number of additional questions unanswered and unknown moderators to be determined—but hope that our meta-analysis spurs further research. Indeed, as some of the effects we discovered raised questions, we have used the results of the meta-analysis to develop novel studies that we are conducting within our lab currently. It is our hope that other researchers follow in this vein, ideally with the methodological rigor and conceptual precision called for by Roos and colleagues.

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