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Stress and cognition: A user's guide to designing and interpreting studies

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ABSTRACT

Fueling the rapid growth in our understanding of how stress influences cognition, the number of studies examining the effects of stress on various cognitive processes has grown substantially over the last two decades. Despite this growth, few published guidelines exist for designing these studies, and divergent paradigm designs can diminish typical effects of stress or even reverse them. The goal of this review, therefore, is to survey necessary considerations (e.g., validating a stress induction), important considerations (e.g., specifying the timing of the stressor and cognitive task), and best practices (e.g., using Bayesian analyses) when designing a study that aims at least in part to examine the effects of acute stress on some cognitive process or function. These guidelines will also serve to help readers of these studies interpret what may otherwise be very confusing, anomalous results. Designing and interpreting studies with these considerations and practices in mind will help to move the field of stress and cognition forward by clarifying how, exactly, stress influences performance on a given cognitive task in a population of interest.

1. Introduction

Our understanding of the effects of stress on human cognitive functions and processes has grown tremendously over the past few decades. This growth has been driven by a steadily increasing number of papers published on the topic—both in absolute number and in the proportion of all papers published on stress—as illustrated in Fig. 1. Numerous studies have demonstrated fascinating, replicated effects; for example, stress after learning (i.e., post-encoding stress) actually enhances memory for the information learned previously (e.g., Cahill et al., 2003; Shields et al., 2017). A brief summary of the best-known effects of stress on cognition is provided in Table 1, and those interested in reading more about these effects are referred to excellent reviews and meta-analyses published on these topics (Gagnon and Wagner, 2016; Maren and Holmes, 2016; Meir Drexler et al., 2019; Shields et al., 2017, 2016b; Starcke and Brand, 2016, 2012; Wolf et al., 2016). At the same time, the progress of stress and cognition research has elucidated important factors that influence the effects of acute stress on cognition that, if left unconsidered, may impede progress in understanding these effects. In this brief review, I describe these factors, and provide guidelines for designing studies to best elucidate the effects of acute stress on cognitive processes of interest.

After the following short introduction, this review first describes factors that are crucial to consider when designing or interpreting a study aimed at elucidating the effects of acute stress on any cognitive process. Next, this review surveys important—though less important than the above—factors to consider when designing or interpreting these studies. Finally, this review will highlight cutting-edge and optimal approaches for determining the effects of acute stress on cognition before summarizing all of these recommendations and concluding.

1.1. Methods matter (of course)

Stress is as fickle as it is fascinating. This fact is perhaps best described by an excellent paper on how to define and measure stress (Epel et al., 2018). In addition to the difficulty in defining and measuring stress, when considering the effects of acute stress, numerous factors modulate the stress response and the biological mechanisms through which stress acts (Averill et al., 2018; Chida and Hamer, 2008; O'Connor et al., 2009; Stalder et al., 2016). And these biological mechanisms are many: Acute stress exerts its effects at least in part by altering the hypothalamic-pituitary-adrenal (HPA) axis, the sympathetic-adrenal-medullary (SAM) axis, the hypothalamic-pituitary-gonadal (HPG) axis, the immune system, and catecholaminergic activity, and these effects of stress can be modulated by factors such as sex, age, and stress appraisals (e.g., viewing a stressor as a challenge rather than a threat) (Denson et al., 2009; Dickerson and Kemeny, 2004; Laredo et al., 2015; Lennartsson et al., 2012a; Marsland et al., 2017; Shansky and Lipps, 2013; Shields et al., 2017). Moreover, and importantly, these sequelae of acute stress follow different timescales, entailing that biological effects of acute stress at any given timepoint post-stressor are unique interactions of the current state of each of these axes and

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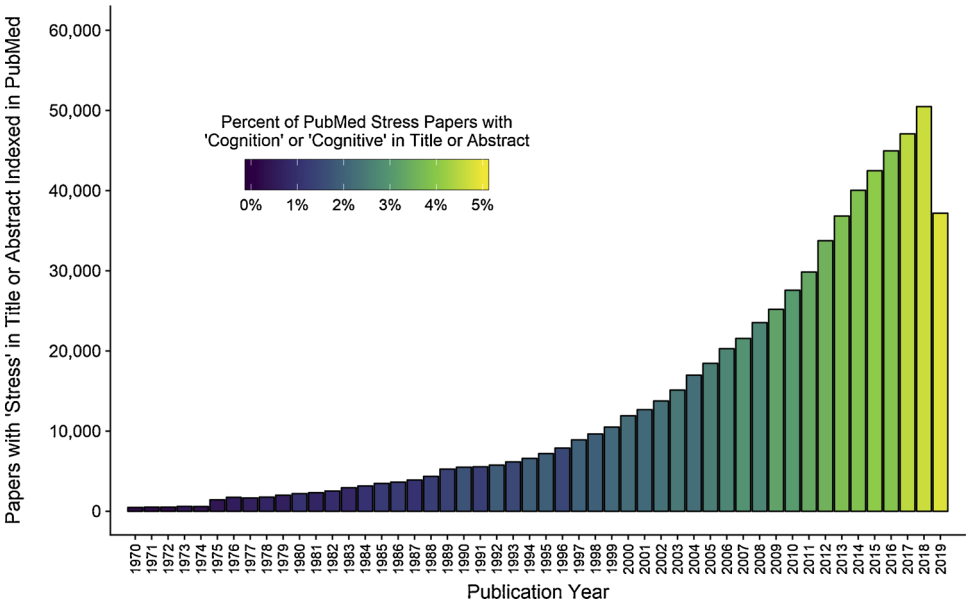

Fig. 1. Illustration of the growth in papers published on stress and cognition from 1970 to July 2019. Both the total number of papers mentioning stress and cognition and the proportion of stress and cognition to total stress papers have grown in recent years, illustrating the wealth of recent research aimed at elucidating the effects of stress on cognition.

Table 1
Summary of Well-Known Effects of Acute Stress on Cognitive Processes.

Cognitive Process	Effect of Acute Stress
Executive functions	
Working memory	↓
Response inhibition	↑
Interference control	↓
Cognitive flexibility	↓
Episodic memory	
Encoding	↑↓
Retention	↑
Retrieval	↓
Reactivation	–
Fear conditioning	
Learning	↑
Extinction	↑↓
Extinction Retrieval	↓
Decision-making	
Risk tasking	↑
Ambiguity tolerance	–
Accurate reward valuation	↓
Goal-directed over habitual	↓

Note: ↑represents a stress-induced increase, ↓represents a stress-induced decrease, and – represents a null effect.

systems listed above (e.g., Joëls et al., 2011; Slavich and Irwin, 2014). The complexity of stress, the stress response, and the neurobiological mechanisms through which it exerts its effects entails that any study aiming to determine how acute stress influences a given cognitive process should be careful to consider the particularities of stress—and how sample characteristics or study design may influence the stress response and thus the effects of stress.

Like stress, human cognition is intensely complex. Entire fields of research and centers exist solely to determine how individual aspects of human cognition—such as memory, language, attention, and executive function—work (Karr et al., 2018; Monaghan et al., 2017; Torralba et al., 2006; Yonelinas, 2013, 2002), and understanding the effects of stress on these aspects of cognition must therefore make use of this research. For example, memory is a broad construct, and although many studies examining the effects of stress on memory use standardized neuropsychological tests of memory (e.g., Hidalgo et al., 2014), measuring memory at this broad level can obfuscate effects on various phases of memory (e.g., encoding, retention, retrieval), effects on memory processes (e.g., recollection, familiarity), and subtle effects on memory that are only distinguishable by tests more sensitive to individual differences in healthy adults (Beckner et al., 2006; McCullough and Yonelinas, 2013; Wiemers et al., 2013a). These issues are not unique to memory, either—the same can be said of any cognitive process of interest. Therefore, to develop a complete understanding of the effects of stress on cognition, the complexity of cognitive processes and factors that influence their particularities should be considered.

The complexities of both stress and cognition highlight the fact that methodological considerations pertaining to both constructs should be weighed when designing studies aimed at determining the effects of acute stress on cognition. This is unlikely to be a point of contention for most—if not all—readers. Therefore, in what follows, I survey the methodological issues and considerations for designing acute stress and cognition studies. Of note, because this review focuses on paradigms for acute stress and cognition studies, this review often uses “stress” as a shorthand to refer to “acute stress” for brevity; other forms of stress (e.g., recent life stress) are noted as such.

2. Necessary factors to consider

Some factors are too important not to fully consider and address when designing or interpreting a study that aims to determine an effect of stress on some cognitive process. These issues and considerations include the need to validate the stress manipulation with a stress-specific biological measure in each study, control/assess relevant participant characteristics, provide sufficient but not excessive study acclimation time, avoid confounding stress effects on a task with task practice effects (or stress effects on retrieval of previously learned task strategies), avoid confounding stress effects on task performance with cognitive-task-induced fatigue, and use the most appropriate cognitive task. These factors are considered in detail below.

2.1. Validate the stress induction

Standardized acute stress induction protocols exist because stress can be difficult to manipulate: Stressful conditions such as uncontrollability or socio-evaluation—key factors of situations that govern the magnitude of the stress response (Dickerson and Kemeny, 2004; Koolhaas et al., 2011)—can be difficult to create within the lab (Allen et al., 2017; Kirschbaum et al., 1993; Roos et al., 2017b; Schwabe et al., 2008b). For example, minor variations in the Trier Social Stress Test (TSST), such as altering the gender composition of the evaluation panel

or instructing the evaluators to show negative rather than neutral behaviors and expressions, can significantly reduce cortisol responses to the stressor (Goodman et al., 2017). Further, although a group version of the TSST is a potent stress induction (von Dawans et al., 2011), care must be taken not to allow participants to interact, since a weak feeling of social cohesion can reduce stress responses to the group TSST (Häusser et al., 2012). Similarly, uncontrollability can be difficult to induce in the cold pressor test (CPT), and both uncontrollability and social-evaluative threat can be difficult to induce in the socially evaluated cold pressor test (SECPT), entailing that instructions and procedures for these stressors must be very precise to induce the typical stress response (Schwabe and Schächinger, 2018). Likewise, the Maastricht Acute Stress Test (MAST), imaging MAST (iMAST), Montreal Imaging Stress Test (MIST), and ScanStress paradigms all have very precise procedures that must be followed for proper stress induction (Dedovic et al., 2005; Quaedflieg et al., 2013a; Smeets et al., 2012; Streit et al., 2014). These considerations, and more, illustrate that acute stress inductions can be difficult to design and execute correctly; with minor protocol variation, a moderate-to-severe stress induction can become a mild stress induction—if it remains a stress induction at all.

Now, imagine that a study examining the effect of stress on some cognitive process finds a very unusual effect, but that study does not validate their stress induction with a stress-specific biomarker. Given how delicate stress inductions can be, it can be difficult to assess the cause of that effect. Was the stress induction really a stress induction, or was it so mild as to be more accurately labeled an arousal induction (and possibly at the peak of the Yerkes-Dodson curve)? Without validation from a stress-specific biomarker, answering that question can be extremely difficult. This difficulty makes it nearly impossible to interpret the results of such studies and integrate those results into prior empirical work on and theoretical models of stress and cognition. Therefore, to move our understanding of the effects of stress on cognition forward, it is imperative to validate a stress protocol using a stress-specific biomarker.

Biomarkers that are stress-specific are not as common as one might think (Allen et al., 2014). Crucially, arousal manipulations that are completely nonstressful produce increases in indices of sympathetic nervous system (SNS) activity, such as heart rate and salivary α-amylase (Nielson et al., 2005; Wiemers et al., 2013b). Despite this, however, SNS activity pre-, peri-, and post-stressor should be assessed if at all possible, as SNS activity is a critical component of the overall stress response (Allen et al., 2014). Widely accepted stress-specific biomarkers are glucocorticoid (e.g., cortisol) increases and proinflammatory cytokine increases (Dickerson and Kemeny, 2004; Marsland et al., 2017). There may be other stress-specific biomarkers, such as dehydroepiandrosterone or progesterone increases (Lennartsson et al., 2012b, 2012a; Shields et al., 2016a), but it remains to be shown that these hormones do not increase in response to arousal manipulations. Relevant blood-derived measures, such as adrenocorticotropin hormone (ACTH; Carpenter et al., 2007) and epinephrine (Ohira et al., 2008), may also add important information about stress and its effects on cognition.

The gold standard of stress-specific biomarkers is a cortisol increase (for reviews, see Allen et al., 2017, 2014; Goodman et al., 2017), and studies examining the effects of stress on cognition should plan to collect saliva samples to assay cortisol at the very least. Recommended saliva collection protocol for cortisol is passive drool (Granger et al., 2007b; Harmon et al., 2007), as this collection method has a number of advantages (e.g., minimizing influences from substances used to stimulate or collect saliva) and no disadvantages in compliant, awake adults (Granger et al., 2007b), and it avoids problems with sample recovery present in other collection methods (Harmon et al., 2007). To best capture the effect of stress on cortisol, the baseline sample should be collected immediately before introducing the stressor/control task and the post-manipulation sample should be collected between 21 min to 30 min post-manipulation onset (Dickerson and Kemeny, 2004)—though additional samples can be collected to better assess reactivity and recovery. To best preserve the samples, they should be stored at −20 °C or lower until assayed (Lewis, 2006), and the samples should not be repeatedly thawed and frozen again, as doing so can artificially lower cortisol concentrations (Gröschl et al., 2001). Importantly, because baseline cortisol values are inherently meaningful, both baseline and post-manipulation values should be reported for each group (rather than delta or baseline adjusted values alone) in order to ensure full transparency of results and allow readers to accurately determine the magnitude of a cortisol response.

Although biological stressor validation is a requirement, researchers should also assess baseline and post-manipulation negative affect, both as a manipulation check and to permit comparison of the strength of the manipulation with other studies that have assessed changes in negative affect from pre- to post-manipulation. Affect returns to baseline quicker than do hormones, so the post-manipulation assessment should ideally start no later than 1 min after stressor offset. Use of a common (e.g., Allen et al., 2014; Buchanan et al., 2006; Quaedflieg et al., 2013b; Schoofs et al., 2013; Shields et al., 2019d; Zoladz et al., 2018) standardized scale for affect assessment, such as the Current form of the Positive and Negative Affect Schedule (Watson et al., 1988), will best permit comparison with other studies. Additionally, inclusion of a measure more directly indexing subjective stress—such as the Primary Appraisal Secondary Appraisal scale (Gaab et al., 2005), the Stanford Acute Stress Reaction Questionnaire (Cardeña et al., 2000), or visual analogue scale items assessing subjective stress (e.g., Gaab et al., 2005)—is highly recommended.

2.2. Control/assess relevant participant characteristics

Various psychological and biological factors influence the biological and cognitive sequelae of acute stress. These considerations have led many researchers to use various sets of inclusion and exclusion criteria for studies of stress and cognition. Although these participant inclusion/exclusion criteria are not standardized, high-quality studies often exclude individuals who suffer from a psychiatric disorder (unless a particular disorder is a focus of the study), take psychotropic medication (e.g., antidepressants, stimulants) or medication(s) that can influence stress responses (e.g., immunosuppressants, beta-adrenergic inhalers, corticosteroids), habitually smoke, consume excessive amounts of caffeine (e.g., > 8 cups of coffee per day), have had severe sleep disturbance within the prior month (e.g., shift work, chronic insomnia), have an autoimmune or major health disorder (unless a particular disorder is a focus of the study), and are currently sick or have been sick over the past week, as well as women taking hormonal contraceptives or who are pregnant. These exclusion criteria exist because each of these factors modulates components of the stress response, such as noradrenergic activity, glucocorticoid activity, or immune system activity (e.g., Butt and Sultan, 2011; Childs and De Wit, 2009; Granger et al., 2007a; O'Connor et al., 2009; Rohleder et al., 2003; Schmid-Ott et al., 1998; Slavich and Irwin, 2014; Stalder et al., 2016), and failing to exclude participants with these conditions can therefore result in a different effect of stress than would typically be observed (e.g., Burke et al., 2005; Cackowski et al., 2014; Childs and de Wit, 2009; Minkel et al., 2014; Nielsen et al., 2014, 2013; Schmid-Ott et al., 1998). Additionally, most studies assess recent smoking, recent recreational drug and alcohol use, recent exercise, recent food intake, and women's phase of the menstrual cycle, and participants are usually further informed to avoid eating, drinking anything besides water, and exercise within two hours prior to the study. As above, those practices exist because each of the relevant factors can influence either stress responses (Child et al., 2011; Child and de Wit, 2009; Granger et al., 2007a; Shields et al., 2017; Zschucke et al., 2015) or measurement of salivary hormones (Granger et al., 2012; Schultheiss and Stanton, 2009). Best practices in this matter include verifying compliance with these inclusion/exclusion criteria at the beginning of the study session and further assessing oral

health in order to covary oral health in analyses of salivary analytes when appropriate. Following these guidelines will help to ensure that stress was induced successfully within the population of interest.

2.3. Provide sufficient but not excessive acclimation time

As is evident in the ubiquitous drop in cortisol from a sample taken upon arrival at the laboratory to a subsequent sample (e.g., Kirschbaum et al., 1993), participants are often stressed when arriving at a laboratory study. Therefore, to ensure that the control (i.e., non-stress) group is not stressed during the time in which the stress group would complete the stress task, a study acclimation period is recommended. This acclimation period should not be too long, however, so as to avoid boring participants through an excessively long study. The TSST, for example, shows the greatest effect on cortisol when participants have acclimated to the laboratory for between 16–30 min (Goodman et al., 2017). Similarly, the effects of stress on executive functions are strongest when the pre-stressor acclimation period is between 10–40 min (Shields et al., 2016b). In short, with too little acclimation, control participants may be stressed, but with too much acclimation, participants may get bored—and control participants therefore may also be impaired on cognitive tasks due to too little arousal. Therefore, studies examining the effects of stress on cognition should include an acclimation period (i.e., time in the laboratory filled with nonstressful, noncognitive tasks, such as filling out questionnaires unrelated to stress), ideally 16–30 min pre-stressor; the baseline saliva sample should be collected at the end of the acclimation period, followed by the start of the stress/control manipulation.

2.4. Do not have participants complete the same cognitive task pre- and post-manipulation

Practicing a cognitive task alters how stress influences it through at least two mechanisms. First, because stress impairs memory retrieval (Gagnon and Wagner, 2016; Shields et al., 2017), it is imperative to avoid conflating the effects of stress on a cognitive process of interest with the effect of stress on retrieval of task strategies by having stressed participants complete a task they completed previously. Even practicing memory retrieval moderates the effects of stress on memory retrieval (Smith et al., 2016). Second, performance on tasks usually reliant on executive control becomes automatic when those tasks are practiced or well-rehearsed tasks (Dulaney and Rogers, 1994), and—because stress strengthens automatic cognitive processing (Schwabe and Wolf, 2013)—the effect of stress on those tasks can reverse if the task is practiced or well-rehearsed (Arnsten, 2009).

Together, the effects of stress on retrieval (i.e., retrieval of task strategies) and the effects of task rehearsal on cognitive performance can alter the effects of stress on cognitive tasks. For example, studies that use between-subjects designs to examine the effects of stress on a task (i.e., comparing cognitive task performance post-manipulation) can obtain very different results from studies that use between-within designs to examine those effects (i.e., comparing changes in cognitive task performance from pre- to post-manipulation) (Dierolf et al., 2018; Roos et al., 2017a; Schwabe et al., 2013). Therefore, to determine the effect of stress on a particular cognitive task, the task of interest should be completed post-stressor and should not have been completed recently enough for practice effects to occur (i.e., within the past three to six months; Bartels et al., 2010; Burke et al., 2014).

However, within-subjects designs may be preferable to between-subjects designs in some circumstances, such as when a specific population of interest is difficult to recruit or when the task of interest has high inter-individual variability. If a within-subjects design is used, post-stressor and post-control cognitive task performance should not be should not be assessed on the same day due to the genomic effects of cortisol, which can persist for at least 4.5 h (Henckens et al., 2011, 2012; Shields et al., 2015). In addition, practice effects should at least be removed from collected data in within-subjects designs by having participants practice the task prior to completing it post-manipulation: Practice effects are strongest between the first and second assessments, and practicing tasks for 10 min appears to be sufficient to remove practice effects from collected data (Bartels et al., 2010; Beglinger et al., 2005; Thorndike, 1922). However, as noted above, the effects of stress on practiced tasks differ from the effects of stress on unpracticed tasks, so caution should be exercised when comparing results from designs using practiced tasks with results from designs using unpracticed tasks.

2.5. Avoid interactions between stress and cognitive fatigue

When designing experiments, it is important to keep in mind that—regardless of stress exposure—participants often experience cognitive fatigue: Completing a cognitive task can impair participants' ability to perform in subsequent tasks (Hagger et al., 2010). For example, completing a task reliant on executive functions impairs performance on a subsequent task reliant on executive function (Hagger et al., 2010; Schmeichel, 2007). Exactly why acute stress interacts with this effect is not entirely clear, but a meta-analysis found that studies examining the effects of acute stress on executive functions showed smaller effects of stress when participants completed multiple cognitive tasks in the same study (Shields et al., 2016b); similarly, mental fatigue produced by prior cognitive task completion also alters memory performance (Evers et al., 2013). This interaction between stress and mental fatigue may occur because mental fatigue alters cognitive and neuroendocrine function (Moreira et al., 2018; Smit et al., 2004), or possibly because stress contributes to fatigue (Hoppmann et al., 2015). Most likely, the effects of stress on cognitive task performance become watered down when multiple tasks are completed because the control group ends up mildly stressed, as sustained cognitive task performance is itself a stressor for many individuals (Compton et al., 2013; Evers et al., 2013). In any case, because the apparent effects of stress on at least some cognitive processes are weaker when more cognitive tasks are completed within a single study, studies aiming to understand the effects of stress on a particular cognitive process should ensure that participants' cognitive resources are not diminished by requiring them to complete other cognitive tasks. Relatedly, unless a cognitive function such as vigilance is the outcome, care should be taken to ensure that the cognitive task performed is not excessively long.[1]

2.6. Use an optimal cognitive task

Cognition is complex. Cutting-edge developments are made in its assessment every year. Although it may be tempting to use classic cognitive tasks in studies of stress and cognition, avoid this temptation in favor of utilizing contemporary cognitive assessments. These contemporary cognitive assessments are usually more sensitive and better at isolating processes of interest.

As an example of the greater sensitivity of contemporary assessments, although working memory was long thought to be spared with damage to the hippocampus—the famous patient H.M. showed no impairments on the digit span—more sensitive assessments of working memory show that damage to the hippocampus does in fact impair working memory sensitivity and capacity (Goodrich and Yonelinas, 2016; Warren et al., 2015; Yonelinas, 2013). As an example of the

[1] As for what would be "excessively long," cognitive fatigue reaches a peak and roughly plateaus approximately 30min post-task onset (Sauter et al., 2013; Warm et al., 2008), and the effects of stress on cognitive functions are often categorically different between different ~30min post-stressor epochs (Joëls et al., 2011; Schwabe et al., 2012; Shields et al., 2017). Therefore, if possible, use of tasks less than 30min in length is recommended. If this is not possible for other reasons (e.g., use of ERPs), time-dependent effects of stress on task performance should be explored.

improved ability to isolate processes of interest over recent years, a working memory task was developed that permits dissociation of component executive processes thought to contribute to working memory task performance: gating, updating, and maintenance (Rac-Lubashevsky and Kessler, 2016a, 2016b). Similarly, memory research has developed sophisticated paradigms that can be used for assessing the effects of stress on different phases of memory (Shields et al., 2017) and assessing different memory processes (e.g., recollection, familiarity) within types of memory (Yonelinas, 2002).

These examples serve to illustrate that research in cognitive science and cognitive psychology has much to offer the field of stress and cognition, and this research should be considered when selecting the cognitive task of interest. Therefore, the cognitive task used should be decided upon after reviewing the relevant cognitive literature and determining which task best isolates the cognitive process of interest and meets the two requirements described in the following paragraphs.

2.6.1. Avoid using a "dual task" unless dual-task performance is the construct of interest

Because stress influences multiple cognitive processes (e.g., Oei et al., 2012; Shields et al., 2016b; Starcke and Brand, 2016), it may be useful to assess the effects of stress on multiple cognitive processes concurrently. Unless dual-task performance is the construct of interest, however, these cognitive processes should not be assessed in two distinct tasks completed simultaneously. Stress exerts complex effects on dual-task performance, improving the ability to process concurrent tasks (Beste et al., 2013) but reducing the ability to shield against interference from a second task when one of the two is prioritized (Plessow et al., 2012). Therefore, depending upon the strategies participants use to complete a dual task (e.g., attempting to do well in both tasks, or attempting to do better in one at the expense of the other), stress may improve or impair performance on one or both tasks. Because of this issue, unless dual-task performance is the construct of interest, single cognitive tasks should be favored over dual cognitive tasks in stress studies.

2.6.2. Ensure the task shows sufficient performance variability in the population of interest

Different populations can have different cognitive abilities (e.g., Diamond and Lee, 2011; Mirelman et al., 2012; Pertzov et al., 2015; West, 2006), and care should be taken to ensure that the task used to assess cognitive performance does not suffer from floor or ceiling effects in the population of interest. For example, although it may be tempting to use standard neuropsychological tests (though see above), many of these tasks show little performance variability in healthy young adults (i.e., ceiling effects) (Beglinger et al., 2004), and are therefore less useful than more contemporary tasks for assessing the effects of stress on cognition in young adults (e.g., Hoffman and Al'Absi, 2004).

In short, the developments of cognitive science and cognitive psychology should be drawn upon when selecting the task to assess the cognitive process or function of interest in a study of stress and cognition.

3. Important factors to consider

Some factors are important to consider when designing or interpreting studies on stress and cognition, but are less crucial than those outlined above. In particular, these factors include the timing of the cognitive task relative to the stressor, accounting for potential sex differences, specifying and keeping consistent the time of day that the study is conducted, potential stressor-specific effects, and considering the severity of the stressor.

3.1. Timing of the stressor relative to the cognitive task alters stress effects

The biological stress response is time-dependent, with different components peaking and returning to baseline at different times post-stressor (Allen et al., 2014; Joëls et al., 2011; Slavich and Irwin, 2014). For example, after a stressor, SAM axis activity (e.g., norepinephrine) typically returns to near baseline levels before HPA axis activity (e.g., cortisol) peaks (Joëls et al., 2011), and HPA axis recovery is well underway before immune system activity (e.g., proinflammatory cytokines) peaks (Slavich and Irwin, 2014)—though the genomic effects of glucocorticoids persist after cortisol levels have returned to baseline (Henckens et al., 2011; Schwabe et al., 2012). Because these hormones and proteins influence cognitive processes (Donzis and Tronson, 2014; Henckens et al., 2012; Reichenberg et al., 2001), the time-dependent biological effects of stress entail that the length of time between stress onset/offset and assessment of cognitive performance matters, and should be carefully considered when designing a study examining stress and cognition.

A few studies to date have examined the time-dependent effects of stress on cognitive processes. Memory encoding, for example, is typically enhanced by stress that occurs immediately prior to or during encoding, whereas it is typically impaired by stress that occurs approximately 25 min or more before encoding (Joëls et al., 2011; Schwabe et al., 2012; Shields et al., 2017; Vogel and Schwabe, 2016; Zoladz et al., 2018). Relatedly, one study found that the effect of stress on memory retrieval became more impairing as the delay between stress and memory retrieval grew (Schwabe and Wolf, 2014). The effects of stress on risky decision-making also appear to be time-dependent: Stress decreases risk-taking during and immediately after stress exposure, but with a longer delay (i.e., greater than 10 min post-stressor offset), stress increases risk-taking (Bendahan et al., 2017; Pabst et al., 2013). Finally, the effects of stress on working memory may be time-dependent, as a meta-analysis indicated that greater delay between stress and working memory assessment was associated with a greater impairment (Shields et al., 2016b). These examples illustrate that the delay between stressor onset/offset and cognitive task performance should be considered as an important variable in studies of stress and cognition.

For cognitive processes where the time-dependent effects of stress are unknown, it may be useful to consider known biological mediators of the effects of stress. For example, stress modulates cognitive flexibility by influencing noradrenergic activity (Alexander et al., 2007). Because of this, a study assessing the effect of a stressor on cognitive flexibility might prefer to have participants complete the cognitive flexibility task within 5 min of stressor offset, so that noradrenergic activity has not yet returned to baseline (Joëls et al., 2011). Similar considerations could be made for other cognitive processes.

3.2. Numerous sex differences exist in stress and cognition effects

Stress affects males and females differently (e.g., Laredo et al., 2015; Liu et al., 2017; Trainor et al., 2013; for reviews, see Bangasser and Valentino, 2014; Cahill, 2012; Shansky, 2015; Shansky and Lipps, 2013). As might be expected, then, sex differences have been found in the effects of stress on numerous cognitive processes, including risky decision-making, memory encoding and consolidation, working memory, and cognitive flexibility (Felmingham et al., 2012; Kalia et al., 2018; Laredo et al., 2015; Nowacki et al., 2019; Schoofs et al., 2013; Schoofs and Wolf, 2009; Shields et al., 2016c; Starcke and Brand, 2012; Zoladz et al., 2018, 2014). These widespread sex differences in the effects of stress on cognitive function entail that, at minimum, studies should test for such differences in effects, or ensure that the discussion and conclusions strongly consider sex differences if the study did not have enough males and females to examine sex differences. Ideally, these widespread sex differences in the effects of stress on cognition entail that a study should include enough males and females to examine potential interactions between sex and stress on the cognitive process of interest. In addition, because sex hormones are likely contributors to many of these sex differences, it is important to assess women's

menstrual cycle phase (e.g., having women self-report the first day of their last period) and hormonal contraceptive usage. If possible, multiple groups of women should be assessed (e.g., with and without hormonal contraceptives), and although not always feasible, assessment of sex steroids (e.g., progesterone, testosterone) could potentially provide important information that spurs future research.

3.3. Time of day may influence the effects of stress on cognition

The time of day that a study begins might not seem to be an important consideration in most fields, but it is in studies of stress and cognition. Hormones and immune system processes through which stress exerts its effects exhibit strong diurnal rhythms—and baseline levels of these hormones processes can modulate the effects of stress on cognition (Lupien et al., 2007; Maheu et al., 2005; Shields et al., 2017). Because of this, most current studies of stress and cognition (see Shields et al., 2017) have participants complete the study between the early afternoon and early evening, as diurnal rhythms of these biological processes are more stable during this time (Deer et al., 2018; Matchock et al., 2007). If at all possible—unless time of day effects are of interest in a given study—future studies of stress and cognition should follow this guideline. If some participants must complete the study at different times of day, ensure that an equal number of participants complete the study in the morning for both stress and control groups, and consider exploring potential stress by time of day interactions in analyses. It should be noted, though, that examining how time of day modulates stress effects on cognition may be a fruitful avenue for future research.

3.4. Potential stressor-specific cognitive effects

It is also important to consider whether specific stressor protocols may produce unique effects on the cognitive process or function of interest (Roos et al., 2017b; Shields and Yonelinas, 2018). For example, a meta-analysis of stress and memory found that pain-based stressors impaired memory encoding regardless of the delay between stress and encoding, whereas non-pain-based stressors enhanced memory encoding with no delay between stress and encoding (Shields et al., 2017). This effect was observed across studies and therefore requires replication within an experiment. Still, one potential mechanism for this effect may be that pain-based stressors require constant response inhibition (i.e., suppressing a prepotent response)—namely, resisting the reflex (or, prepotent response) to remove one's arm from the painful ice water (Karsdorp et al., 2014)—and utilization of response inhibition impairs memory encoding (Chiu and Egner, 2015). Relatedly, a recent study and meta-analysis found that changing rooms between learning and stress abolished the standard post-encoding stress-induced enhancement of memory (Sazma et al., 2019a; Shields et al., 2017); therefore, post-encoding stress paradigms that require a room change may show less robust effects on memory than paradigms that do not. Finally, there is evidence indicating that the effects of stress on risky decision-making may also differ by stressor type, with men showing greater risk-taking than women following the cold pressor test, but women showing greater risk-taking following the socially evaluated cold pressor test (Nowacki et al., 2019). Although replication is needed for this unexpected result, this result again highlights the importance of considering potential stressor-specific effects on cognition. In short, research examining stressor-specific effects on cognition is nascent, but if a very unusual result is obtained, consideration that the stressor paradigm itself may have contributed to the result is warranted.

3.5. Stress severity may influence stress effects on cognition

Although most studies examining the effects of stress on cognition use paradigms inducing moderately severe stress (Shields et al., 2017), not all studies do so (e.g., Gagnon et al., 2018). In fact, these latter studies have found that stressors of different severities exert quantitatively and perhaps qualitatively different effects on cognition (Corbett et al., 2017; Hupbach and Fieman, 2012; Shields et al., 2016b, 2017; 2019d; 2019e). Relatedly, subtle protocol variations can dramatically alter stress responses (described above), entailing that unusual effects of stress may occur due to differences in stress severity. Of the standardized paradigms, according to a recent meta-analysis the TSST produces the largest cortisol response, the CPT produces the smallest (though the CPT is still a moderate stress induction by most definitions), and the SECPT falls in between (Shields et al., 2017). It should also be noted that although at the time of Shields et al.'s meta-analysis too few studies had incorporated the MAST, iMAST, MIST, or ScanStress paradigms to determine how these paradigms compared on a meta-analytic level to the TSST, CPT, or SECPT, individual studies provide support for the MAST being a stressor near or on par with the TSST in severity, while the iMAST, MIST, and ScanStress paradigms are closer to the SECPT or CPT in severity (Dedovic et al., 2005; Quaedflieg et al., 2013a; Smeets et al., 2012; Streit et al., 2014). Should an expected result fail to return significant despite adequate power, stress severity should be considered as a potential explanation.

4. Best practices

Although not required to design an easily interpretable study, the following constitutes a non-exhaustive overview of cutting-edge methods and best practices will help to move the field of stress and cognition forward.

4.1. Report associations between stress markers and cognition

Uncovering the hormonal and perhaps immunological mechanisms underpinning the effects of stress on cognition will be important for blocking the detrimental effects of stress on cognition—and mimicking the beneficial effects of stress on cognition without the occurrence of stress. Although some progress has been made on this front (e.g., Buchanan et al., 2006; Henckens et al., 2011; Meir Drexler et al., 2019; Rasmusson et al., 2004; Sazma et al., 2019b; Shields et al., 2019a; Sripada et al., 2014; van Stegeren et al., 2010), much about the biological correlates of stress effects is undocumented, because many published papers do not report associations between measured stress biomarkers and cognitive performance (e.g., Cackowski et al., 2014; Cousijn et al., 2012; Duncko et al., 2009; Finy et al., 2014; Giles et al., 2015; Hoffman and Al'Absi, 2004; Ishizuka et al., 2007; Lai et al., 2014; Luethi et al., 2008; Porcelli et al., 2008). Although a journal's word count limit is an obvious consideration for reporting additional analyses, if space is the limiting factor these associations should be reported in supplemental material at the very least. Documenting these associations will help to clarify how, exactly, stress influences these cognitive processes.

Some studies (e.g., Schwabe et al., 2008a) have examined potential categorical differences in the effects of stress on cognition by classifying people according to their cortisol responses. Because of this, the issue of categorizing individuals as cortisol responders vs. nonresponders should be discussed. In general, individuals should not be categorized (e.g., responder or nonresponder) according to values of a quantitative variable (e.g., changes in cortisol), as doing so can obscure true effects and produce spurious ones (MacCallum et al., 2002). However, this generality does not apply when there truly are latent classes underpinning data, as has been shown with cortisol responses (for cutoffs, see Miller et al., 2013). Supporting the utility of this approach, many studies have found differences in the effects of stress on cognition between cortisol responders and nonresponders (e.g., Buchanan and Tranel, 2008; Domes et al., 2002; Merz et al., 2010; Schwabe et al., 2008a; Smeets et al., 2006; Zoladz et al., 2011). As long as authors are mindful not to conflate stress with cortisol—since stress is more than a cortisol response—separating cortisol responders from nonresponders may help to advance our understanding of how cortisol influences cognition

within the context of a stressor. It should be noted, though, that non-responders may have extant HPA axis dysfunction or other factors preventing them from having normal stress responses (e.g., Childs and de Wit, 2009; Jansen et al., 1998; Nielsen et al., 2013; Petrowski et al., 2010), entailing that separating responders from nonresponders may not do much to clarify how an acute increase in cortisol influences cognition. Future research should address this issue in order to determine the reasons why responders sometimes differ from non-responders in cognitive performance. Nonetheless, to best elucidate how cortisol might associate with a cognitive variable of interest, studies should present correlations between cortisol and the cognitive variable of interest across all participants as well as results examining potential differences between cortisol responders and nonresponders.

4.2. Document every detail

Although many details of a study method may seem irrelevant, even seemingly irrelevant procedural details can dramatically alter stress responses and the effects of stress on cognition, as described above (e.g., Goodman et al., 2017; Sazma et al., 2019a). Moreover, some paradigm characteristics that are currently considered irrelevant likely play a crucial, as of yet unknown role in contributing to the effects of stress on cognitive performance. To best facilitate cumulative science, therefore, describe every detail of a study method if at all possible. Supplemental material is useful for including extra methodological detail if necessary. If space constraints are a limitation, preference should be given to documenting paradigm characteristics known or thought to influence stress or cognition, such as context, whether the gender of the experimenter and/or evaluator always differed from the participant or not, and—in a group setting—whether participants (including those participating in a different study sharing a room) could see other participants' computer screens (e.g., Goodman et al., 2017; Häusser et al., 2012; Shields et al., 2017).

4.3. Move beyond raw behavioral data

When attempting to understand the effects of stress on a specific cognitive process, it may be best to move beyond broad behavioral measures of cognitive performance and quantify the actual cognitive construct that is of interest. No performance task designed to assess cognitive function is process pure; performance on cognitive tasks is the result of both neurocognitive processes relevant to the construct of interest as well as processes that are irrelevant to the construct of interest (Calanchini et al., 2019). Generally, broad behavioral outcomes such as reaction time and accuracy alone cannot distinguish between the joint contribution of simultaneous neurocognitive processes, and as such, these measures can sometimes only crudely approximate the cognitive construct of interest (Farrell and Lewandowsky, 2018). Multiple excellent methodologies can overcome some of the limitations of broad behavioral outcomes. For example, computational cognitive modeling (e.g., McCullough and Yonelinas, 2013; Shields et al., 2019b, 2019c, Wiemers et al., 2013a), eye tracking (e.g., Herten et al., 2017; Macatee et al., 2017), mouse tracking (Shields et al., 2019d), electrophysiology (e.g., Alomari et al., 2015; Compton et al., 2013; Dierolf et al., 2018; Sänger et al., 2014; Weymar et al., 2012; Wirkner et al., 2013), functional magnetic resonance imaging (e.g., Porcelli et al., 2008; Qin et al., 2012; Weerda et al., 2010) and other neuroimaging methods (e.g., Kalia et al., 2018) have all been gainfully employed to better understand the effects of stress on cognition.

Another approach to moving beyond raw behavioral data is to use structural equation modeling to estimate latent factors underpinning performance on various cognitive tasks (e.g., Karr et al., 2018). Although this method has a number of advantages, to utilize it within a stress and cognition context would entail that multiple cognitive tasks would be completed post-stressor, which (as described above) weakens or alters the effects of stress on cognition. Because of this issue, estimating latent cognitive functions is not recommended when examining the effects of stress on those cognitive functions.

4.4. Ensure appropriate statistical power, and use Bayesian analysis

The effects of stress on most cognitive processes are, on average, small-to-moderate in magnitude (Shields et al., 2017, 2016b; Starcke and Brand, 2016). Although following the guidelines outlined in this review should produce stronger effects of stress on cognition, studies examining these effects should ensure that they have adequate statistical power by including large enough samples to detect small-to-moderate effect sizes with at least 80% power. Ensuring adequate statistical power will help enable publishing of null results, which is important if we are to understand the conditions in which stress does and does not influence cognition.

Related to the above, using Bayesian analyses—in addition to or in place of standard hypothesis testing—can help to further make sense of results by quantifying evidence in favor of the alternative and null hypotheses (Jeffreys, 1961). In the case of null hypotheses, these analyses can provide confidence in the null or suggest an appropriate level of uncertainty (e.g., Shields et al., 2019e). On the other hand, these analyses also help by quantifying evidence in favor of the alternative hypothesis: Even if a result is significant by standard convention, it may return a small Bayes factor, suggesting that replication is needed before firm confidence can be placed in a result.

4.5. Conduct and publish reproducible research

Much of what has been recommended in this review (e.g., use of a single cognitive task as an outcome, documenting every methods detail) touches on the issues of questionable research practices (QRPs) and reproducible results without directly addressing them. Although the field has made much progress in addressing issues that led to our reproducibility crisis (Nelson et al., 2018; Open Science Collaboration, 2015), because QRPs have been rampant in science (Head et al., 2015; John et al., 2012), and because the publishing process contributes to their use (Nosek et al., 2015; Smaldino and McElreath, 2016), methods that promote reproducible results should be highlighted and recommended. Coverage of these methods will be brief, as detailed reviews on them have been published elsewhere (e.g., Asendorpf et al., 2013; Munafò et al., 2017; Nelson et al., 2018; Nosek et al., 2018, 2015). The most powerful methods individual investigators can use to conduct and publish reproducible research are disclosing all methods used, pre-registering analytic plans and hypotheses, and—when possible—making code and data available. These methods decrease "researcher degrees of freedom," and use of them dramatically cuts the number of false-positive findings published (Nelson et al., 2018; Nosek et al., 2018; Simmons et al., 2011). Their use is also simple. Disclosing methods is as simple as listing everything used and stating that no other measures, manipulations, or conditions were part of the study used in the submitted manuscript, or, if there were others, what they are and why they were not included in the manuscript (e.g., the measures were not relevant to the current manuscript). Pre-registration of studies can be done through the Open Science Foundation (http://osf.io), which has comprehensive templates for pre-registration, and AsPredicted (http://AsPredicted.org), which asks researchers nine simple questions that document all details of the study relevant to pre-registration and produces a one-page output. Code and data—if one's IRB approval allows for sharing—can be easily uploaded to the Open Science Foundation, publishing platforms (such as Elsevier's Data in Brief), Mendeley Data, platforms that produce DOIs for data (such as Figshare, Zenodo, and Dryad), and personal websites. Use of these practices will contribute to increased research reproducibility in the field of stress and cognition, which will help further our understanding of these effects.

Table 2
Summary of Considerations for Designing and Interpreting Stress and Cognition Studies.

Necessary Considerations	Suggested Approaches
Every stress manipulation needs to be validated	Validate the stress induction by showing salivary cortisol (or blood cytokine levels) increase in the stress condition relative to the control condition; good practice also includes pre- and post-manipulation affect assessment
Numerous participant characteristics influence stress responses	Use common inclusion/exclusion criteria
Insufficient and excessive acclimation time alters stress effects	Provide an acclimation period of 16-30 min prior to the stress/control manipulation
Stress and cognitive task practice interact	Use a between-subjects design if possible and have participants only complete the cognitive task post-stressor; if a within-subjects design is preferable for other reasons, take steps to reduce the influence of practice effects (see Section 2.4)
Stress and cognitive fatigue interact	Assess effects on only one cognitive task, ideally keeping the task to a short duration
Many cognitive tasks are less than optimal	Browse recent cognitive science and cognitive psychology literature to identify cognitive tasks sensitive to the construct of interest; avoid using dual task paradigms; ensure the selected task shows sufficient variability in performance in the population of interest
Important Considerations	
The timing of the stressor relative to the cognitive task may modulate the effect	Identify the ideal window of time based upon either prior literature or the hypothesized mechanism(s) through which stress influences the cognitive process of interest
Numerous sex differences exist in stress effects on cognition	Include a sample large enough to examine sex differences; at minimum discuss possible sex differences in the manuscript if sample size is not large
The time of day a study begins may influence the effects of stress on cognition	Conduct the study between 12pm-5pm for all participants, and ensure time of day the study begins does not differ between stress and control groups
Aspects of a stressor may produce stressor-specific cognitive effects	Consider the stress paradigm and determine if any of its characteristics may influence the cognitive process of interest in a particular way
Stress severity moderates effects	Ensure that the stress manipulation produces cortisol or cytokine increases consistent with other stress manipulations of the intended severity
Best Practices	
Report associations between stress markers and cognition	If space constraints prohibit these analyses from being in the main text, include them in the supplemental material
Document every method detail	Include additional methods details in supplemental material if necessary
Move beyond simple behavioral data	Use computational cognitive modeling, eye tracking, mouse tracking, or electrophysiology
Ensure sufficient statistical power and use Bayesian analyses	Rely on prior work to obtain effect sizes and calculate required sample size to achieve 80% power; use programs such as R or JASP to conduct Bayesian analyses
Conduct and publish reproducible research	Disclose all methods used, pre-register your study hypotheses and analytic plan, and/or upload code and data used

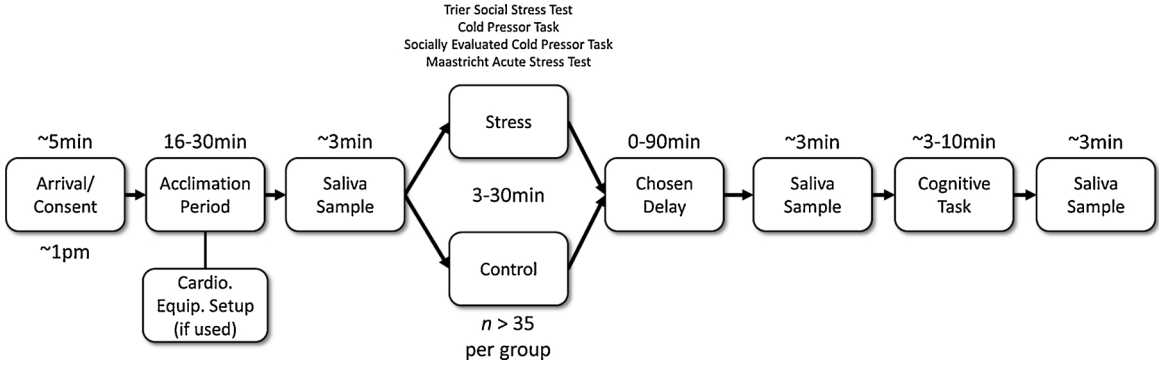

Fig. 2. An example study protocol meeting all necessary paradigm considerations and some recommended practices for stress and cognition studies. Preferably, at least two saliva samples should be taken post-stressor so that hormone values preceding and after the cognitive task can be examined in relation to cognitive task performance, though there should be a delay of at least ~20-25 min between stress onset and one of the two saliva samples in order to characterize cortisol reactivity. Ideally, SNS activity should be assessed via cardiovascular responses (with all equipment set up and on early on in the acclimation period) and/or quantifying salivary α-amylase from saliva samples immediately prior to and following the stressor. During the acclimation period, participants should complete nondemanding, nonstressful, noncognitive tasks, such as filling out personality questionnaires. Good practice also involves assessing women's menstrual cycle phase and hormonal contraceptive usage, as well as baseline and post-manipulation affect (immediately pre- and post-manipulation). During the chosen delay period, participants should either complete more nondemanding, nonstressful, noncognitive tasks, or they should be allowed to relax or read prescribed materials—such as popular science news articles—at their leisure. Each group should contain enough men and women to examine potential sex differences, and recruited participants should be subject to the inclusion/exclusion criteria outlined in the review (including these as prescreen items for the study can be helpful). If recent or lifetime stress is assessed, it is recommended that these constructs be assessed after the cognitive task is completed and the second saliva sample has been collected so as to not stress any of the control participants out by having them recount their stressful experiences. The second saliva sample should be collected at approximately the time of the peak cortisol response to a stressor, which may differ depending upon how long the stressor lasts (see Dickerson and Kemeny, 2004, for a discussion of peak cortisol response timing relative to stressor onset and offset). Modifications to this protocol will need to be made when assessing the effects of stress on certain cognitive processes (e.g., post-encoding stress paradigms), but this example protocol should serve as a useful guide for most studies.

5. Summary and conclusion

Due to the great work of many excellent scientists, our understanding of how stress influences how people think has made enormous progress over the past few decades (e.g., Butts et al., 2011; Cahill et al., 2003; Nowacki et al., 2019; Schoofs et al., 2009; Schwabe et al., 2012; Starcke and Brand, 2016; Wolf, 2018). Despite these advances, however, methodological differences have led to the emergence of conflicting results (e.g., Roos et al., 2017a; Schwabe et al., 2013). This review described numerous factors that can influence the effects of

stress on cognition and documented how to adjust for each of these factors. A summary of this information is presented in Table 2. Consideration of and adjustment for these factors will help to clarify the effects of stress on cognition and move this field of research forward. Careful consideration of these factors should also aid interpretation of results obtained in studies examining the effects of stress on various cognitive functions.

The literature reviewed suggests that in order to best assess the effects of acute stress on given cognitive processes, a study should include a large, relatively homogenous sample of both males and females representing the population of interest, an acclimation period of approximately 16–30 min, a between-subjects stress/control manipulation that is validated using a stress-specific biomarker, a chosen delay (or not) between stress onset and task performance, and a (somewhat short) task best representing the construct of interest that is sensitive to individual differences in the population of interest and completed only post-manipulation. An example paradigm meeting these requirements is depicted in Fig. 2. Although not every study of stress and cognition should necessarily follow this exact paradigm, it could serve as a useful guideline for researchers beginning to study the effects of stress on cognition.

In short, the effects of stress on cognition are many, and they are nuanced; many subtle factors can alter the effects of stress on cognitive functions. However, these nuanced effects reflect the multifaceted nature of stress and how difficult it can be to manipulate correctly within the lab. The field of stress and cognition is an exciting one, with an ever-growing number of papers being published on the subject each year. The hope of this review is to be a useful guideline for designing and interpreting such studies, so that the answer to the question, “How does acute stress affect how we think?” can be answered in full before long.

Declaration of Competing Interest

The author declares no conflict of interest in this work.

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