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Acute stress, memory, and the brain

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1. Introduction

Four decades of research have focused on the relationship between acute stress and memory. A quick search on PsychINFO yields over 400 peer-reviewed articles on the topic. Although progress has been made towards understanding the relationship between stress and memory, it remains a complicated topic. Research continues to work on understanding this important relationship, yet the public clamors for answers due to a wide range of possibilities for application. Educators are interested in capitalizing on potentially positive stress effects on learning and retention. The criminal justice system strives to improve interpretation of eyewitness reports by applying conclusions from the scientific community to jury instructions and general policy recommendations. However, with limited scientific consensus regarding the influence of stress on memory, these conclusions may be premature. The Editors of the Special Issue on Acute Stress, Memory and the Brain were motivated to highlight the complexity surrounding research focused on acute stress and memory. This Special Issue is both important and timely, as it follows on the heels of recent meta-analyses and general reviews (see, Gagnon & Wagner, 2016; Shields, Sazma, & Yonelinas, 2016). This Special Issue is geared towards focusing on how acute stress affects different process associated with declarative memory formation.

Recent studies and reviews suggest that existing prominent models of stress and memory are unable to account for the complexity of reported findings in the literature. Critically, research suggests that no one theory is capable of accounting for the overall pattern of results (see Shields et al., 2016). The Editors of this Special Issue argue that there are three important factors that must be considered when developing a more comprehensive model to account for the cognitive and neurocognitive effects of stress on declarative memory. The Editors argue that full consideration of these factors, and how they may interact, will allow researchers to develop testable theoretical models for the relationship of acute stress and declarative memory.

- Consider the time course of the physiological stress response.
- Consider interactions between the physiological time course and the different underlying processes associated with encoding, consolidation, and retrieval.
- Consider moderating factors, such as stimuli valence, sex, and age.

2. Time course of the physiological stress response

These first two considerations continue to receive much attention from researchers; however, it is only recent research that has begun to tease apart exactly how the multi-staged-stress response interacts with early and later processes of memory. Most of this work has focused on episodic memory processes.

The stress response is characterized by different phases of hormone release, with the first phase beginning immediately after the hypothalamus detects the presence of a threat (Everly & Lating, 2013). The hypothalamus stimulates the adrenal medulla via the autonomic nerves, causing the adrenal medulla to excrete the catecholamines, epinephrine and norepinephrine. Catecholamines prompt the “fight-or-flight” response that prepares the body to take defensive action. These hormones cannot cross the blood-brain barrier to exert direct effects on neural activity, but instead stimulate the solitary nucleus in the medulla, which then stimulates the basolateral amygdala (BLA; Williams & Clayton, 2001 Chap. xvi). The BLA modulates encoding and consolidation of information, particularly emotional information (McGaugh, Cahill, & Roozendaal, 1996). During a brief period after the onset of stress (< 10 min), memory retrieval may also be enhanced (e.g., Hupbach & Fieman, 2012) or otherwise unaffected (e.g., Schönfeld, Ackermann, & Schwabe, 2014), though literature examining memory immediately post-stress is sparse. Most of the research in this area has focused on stress reactivity as measured by the hypothalamic pituitary adrenal (HPA) axis. The influence of hormonal reactivity in association with the Sympathetic-Adrenal-Medullary Pathway (SAM) has been less well studied.

The second phase of the stress response is longer and occurs via a different mechanism, referred to as the HPA axis (Everly & Lating, 2013). While the hypothalamus activates the adrenal medulla during the first phase of the stress response, it simultaneously secretes corticotropin-releasing factor (CRF) that stimulates the pituitary gland. The pituitary gland then releases adrenocorticotropic hormone (ACTH) into the bloodstream, triggering the synthesis and release of the human stress hormone, cortisol, from the adrenal cortex. The release of cortisol from the adrenal cortex is gradual, reaching peak levels in the blood between 20 and 40 min after the initial detection of a threat. The magnitude of the HPA axis response to stress varies greatly at the individual level (e.g., Kudielka, Buske-Kirschbaum, Hellhammer, &

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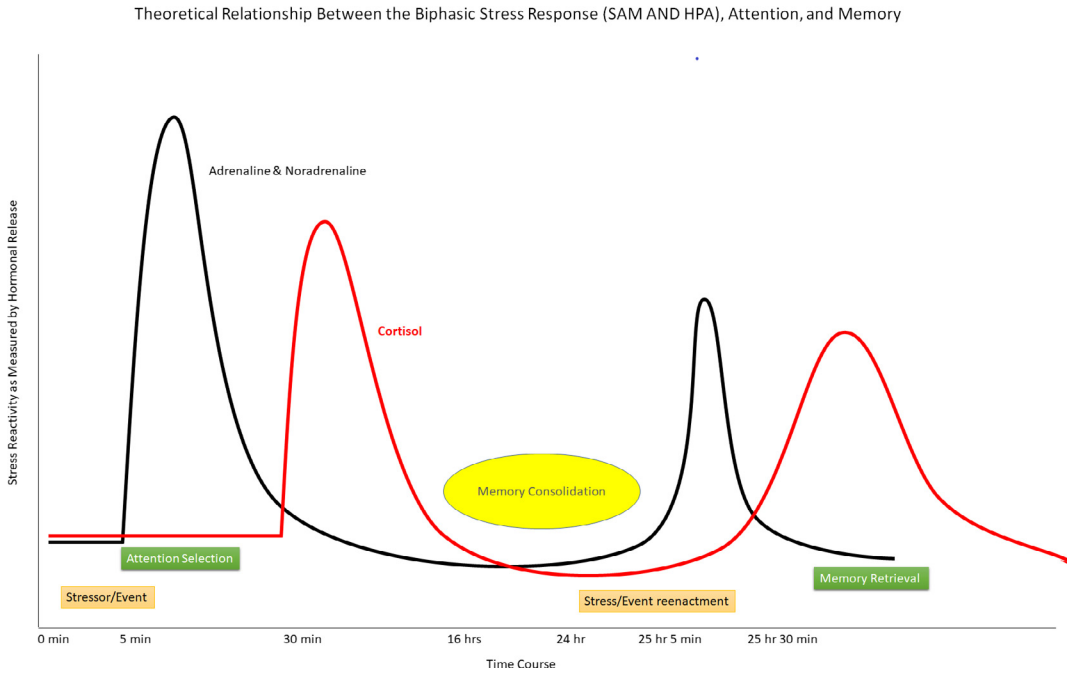

Fig. 1. The theoretical relationship between the biphasic stress response (SAM and HPA), attention, and memory.

Kirschbaum, 2004; Uhart, Chong, Oswald, Lin, & Wand, 2006), but is the largest and most reliable in stress paradigms involving social evaluation (Dickerson & Kemeny, 2004; Skoluda et al., 2015).

Cortisol crosses the blood-brain barrier to exert both positive and negative effects on the brain. In particular, cortisol binds heavily to glucocorticoid and mineralocorticoid receptors in the amygdala and hippocampus (Robinson, Laird, Glahn, Lovallo, & Fox, 2010), both of which are implicated in memory formation and retrieval. Increased occupation of glucocorticoid receptors in the amygdala and hippocampus perpetuates the enhancement of memory formation that begins during the first phase of the stress response (Schwabe, Joëls, Roozendaal, Wolf, & Oitzl, 2012). However, the prioritization of neural pathways involved in encoding and consolidation comes at the cost of retrieval. Once cortisol levels peak after stress, memory retrieval is generally impaired for up to three hours (for a review see Gagnon & Wagner, 2016).

3. Examining the time course of memory in the context of stress

As illustrated by Fig. 1, the time course of stress reactivity associated with each pathway is different. Information attended to during the immediate stress response (early in the time course) should be enhanced. Information attended to later in the stress response may not show this same benefit. Similarly, retrieval that occurs in the context of a delayed stress response (such as a witness not accustomed to public speaking having to testifying in a courtroom, or a student taking a high stake exam) may impair retrieval. Whereas retrieval that occurs early in the stress response may be facilitated.

4. The present volume

It is apparent from the above discussion that the stress-memory relationship is dependent on a host of factors. As such, the Editors sought to include review papers and original articles on this topic in the present Special Issue.

This Special Issue includes two review papers that make important progress in synthesizing the human and rodent literatures on the relationship between long-term memory and physiological stress (both induced via implementation of naturally stressful external factors and mimicked via oral or intravenous drug administration). Specifically, Sazma and colleagues examined the effects of post-encoding stress on episodic memory and Goldfarb examined stress-induced enhancements in long-term memory. A third review paper by Tsai and colleagues tackles important questions from a different approach, in that they consider the effects of psychological responses to stress and how this interacts with learning and memory processes.

In addition to these review papers, the Special Issue includes a variety of original articles. These papers demonstrate the importance of considering the different aspects of memory processes, as well as independently assessing the immediate (Phase 1) and delayed (Phase 2) stress response. Furthermore, the Editors consider the effects of biological factors that appear to yield predictable individual differences, including *BclI* polymorphism and cortisol response (responders, non-responders), as well as the valence of the stimuli used (negative, neutral). Together, these factors seem to contribute to the complexities of the stress-memory relationship and highlight the need for additional research in which these factors are considered. The factors identified across this Special Issue are discussed below - and, we certainly don't see this list as exhaustive.

4.1. Memory processes

Three articles included within this Special Issue demonstrate the importance of independently considering distinct memory processes. Specifically, in this issue, differential effects of stress appeared when memories were categorized as recollection-based vs. familiarity-based (i.e., Wiemers et al.) and as item-based vs. source-based (i.e., Smith et al.). Differential effects of stress on memory were also observed when hit and false alarm rates were individually considered (as opposed to d-prime; i.e., Shermohammed et al.).

Wiemers and colleagues found differential effects of pre-encoding stress on recollection-based memories and familiarity-based memories. Specifically, they demonstrated a positive correlation between pre-encoding Phase 1 stress response (i.e., blood pressure) and recollection memory, and a negative correlation between pre-encoding perceived stress (as indicated by self-report) and recollection memory. For familiarity-based memory, they reported a negative correlation with pre-encoding Phase 2 stress response (i.e., magnitude of cortisol response).

Relatedly, Smith and colleagues examined the effects of stress during retrieval of item and source information under conditions of retrieval practice or study practice. They report differential effects of stress on item memory and source memory under conditions of retrieval practice. Furthermore, Shermohammed and colleagues employed a paradigm in which incidental encoding of neutral and negative images occurred during the Phase 2 stress response. A subsequent old-new recognition test revealed that stress modulated the rate of false alarms, as opposed to hits, and that the direction of this modulation was dependent on stimulus valence (discussed below). Together, these findings highlight the importance of independently considering each memory process.

4.2. Biological factors

This Special Issue also presents articles that highlight the need for continued research on the manner in which biological individual differences modulate the stress-memory relationship. Zoldaz and colleagues presented evidence in which encoding that occurred during Phase 1 of the stress response resulted in enhanced long-term memory, while encoding during Phase 2 resulted in impaired long-term memory. This effect was specific to males – a finding that is consistent with the idea that there may be differences across sexes. Most notable, however, was the effect of the *Bcl*I polymorphism, which has been associated with lower basal cortisol concentrations and greater glucocorticoid receptor sensitivity to corticosteroids (among other things, including somatic disorders and elevated risk of psychopathology). The stress manipulation - independent of its timing relative to encoding - enhanced memory in non-carriers of the *Bcl*I polymorphism, while impairing memory in carriers of the *Bcl*I polymorphism. Sorting participants on the basis of this polymorphism is an exciting direction for future stress and memory research.

Additionally, other recent work suggests that some individuals display increased concentrations of cortisol in response to a stressor, while other individuals do not undergo significant physiological changes under the same conditions. These individuals are referred to as responders and nonresponders, respectively. Critically, such physiological differences have downstream effects on cognitive processes. For instance, in this issue, Smeets and colleagues demonstrated that it is not overall stress that predicts a potentially maladaptive shift in behavior from goal-directed to habitual control. Instead, cortisol response predicts this behavior, as evidenced by a lack of behavioral shift in nonresponders. In line with previous work, this reinforces the need for future research to explicitly investigate and/or control for individual physiological differences in cortisol responsivity.

4.3. Stimuli valence

As discussed above, the literature suggests that stress-memory effects also may differ depending on the valence of the information being remembered. This was further explored in this Special Issue. For instance, Wiemers and colleagues found no significant effects of stress (regardless of phase) on memory for neutral stimuli. Effects only emerged for emotionally arousing stimuli. As mentioned above, Shermohammed and colleagues also found that stress differentially affected memory based on stimulus valence. Specifically, they found that encoding of negative and neutral images during Phase 2 stress response impaired later recognition memory for negative pictures, while enhancing recognition memory for neutral pictures. Together, these studies highlight the importance of considering stimuli valence.

4.4. Other complexities

The Editors also present articles that further highlight the complexities of understanding the relationship between acute stress and memory. First, research on acute stress and memory often focuses on memory in isolation. However, memory interacts with a variety of other cognitive processes, including decision-making. In this Special Issue, Byrne and colleagues investigated the way that Phase 2 stress response modulates memory and decision-making interactions during an uncertainty-based decision-making task in which participants must learn the optimal immediate and long-term decisions to increase ultimate reward. They found that the group experiencing a Phase 2 stress response during this task (confirmed by elevated cortisol levels) performed better than control participants.

The Editors also considered other, perhaps more ecologically valid sources of stress. As demonstrated across this Special Issue, research on acute stress and memory typically employs stress inductions that reliably induce both psychological and physiological stress responses, including the Trier Social Stress Test (TSST) and the Socially Evaluated Cold-Pressor Test (SECPT). However, stress can be induced in a variety of ways. In this Special Issue, Paige and colleagues investigated the effects of lying - an inherently stressful experience - on subsequent memory performance. They found that lying about having previously seen stimuli resulted in poorer source monitoring for that information after a delay in older adults. These results are consistent with the idea that a Phase 1 stress response - induced by the act of lying - may have facilitated encoding of these items. Furthermore, this article highlights the importance of considering the manner in which the stress-memory relationship may change across the lifespan. While this study did not directly assess psychological and physiological stress response, this and other naturalistic stress inductions are an exciting avenue for future research.

The works included in the present Special Issue advance our current understanding of the complexities surrounding the manner in which stress interacts with memory. Together, they highlight the importance of considering a variety of factors in future research.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bandc.2019.04.004.

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