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What the Acute Stress Response Suggests about Memory

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Abstract

Research suggests that stress has immediate and long-term effects on attention and memory. Rather than disrupting memory formation and consolidation, acute stress has been shown to shift attention processes resulting in a tradeoff between prioritized and nonprioritized information. Both arousal and stress result in cognitive and neurobiological shifts that often support memory formation. When an acute stressor occurs, it can distort immediate attentional focus, increasing processing for high-priority features while reducing processing for extraneous features. The downstream cognitive consequences for this shift in attention are better memory for some features and poorer memory for others when compared to conditions of low stress. However, individual differences (e.g., sex, age, basal stress response, and stress reactivity) all impact the relationship between the acute stress response and memory. Although acute stress generally benefits memory formation, we suggest that forgetting and later recovery of stressful memories can better be understood by examining factors that influence the subjective experience of stress and stress reactivity.

Keywords: Stress; Trauma; Memories; Forgetting

1. What the acute stress response suggests about memory

Remembering events that are unpleasant, stressful, or even traumatic can be a difficult experience. Often, we may try to put these memories out of our minds. However, memories

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for stressful and/or traumatic experiences may be more resistant to forgetting. The persistence of vivid and distressing intrusive memories (c.f., Schacter, 2022) by clinical and nonclinical populations suggests that some may not be able to "put an event out of mind" or forget a stressful or traumatic event. Rather, stressful and/or traumatic experiences may foster more robust memories (Beckner, Tucker, Delville, & Mohr, 2006; Cahill, Gorski, & Le, 2003; Hu, Bergström, Gagnepain, & Anderson, 2006; Preuss & Wolf, 2009; Smeets et al., 2009) and disrupt inhibitory control mechanisms important for voluntary forgetting (Quaedflieg, Schneider, Daume, Engel, & Schwabe, 2020; Stramaccia, Meyer, Rischer, Fawcett, & Benoit, 2021). Further, involuntarily re-experiencing memories is a central feature of post-traumatic stress disorder (PTSD), which can develop after exposure to a traumatic event (e.g., Mary et al., 2020).

The goal of the present review is to examine the research on how acute stress impacts memory formation and consolidation. This review focuses on how biological responses impact cognitive processes in the context of an acute stressor. Although difficult to measure in a real-world context, acute stress may play a significant role in the experience of trauma (for review, see Epel et al., 2018). Laboratory research suggests that when acute stress occurs in the context of new learning, it has implications for attention, encoding, and storage of information that co-occurred during the stressor, and information directly associated with the stressor (for review, see Shields, Sazma, McCullough, & Yonelinas, 2017). Therefore, this research may help to shed light on the controversial topic of repressed memories of childhood sexual abuse (CSA), as the experience of this childhood trauma may accompany a physiological response to stress, depending on how that trauma is initially understood and experienced (Barnes et al., 2012; Brown, Harris, & Hepworth, 1995; Epstein & Bottoms, 2002; Kelly-Irving et al., 2013; McNally & Geraerts, 2009; Montez & Hayward, 2014). We will examine how acute stress at the time of new learning may influence later access to information. We will also briefly review research examining motivated forgetting in the context of an acute stress response. We will discuss why it is important to consider the memory formation and suppression literature in the context of the acute stress response when considering memories of stressful and traumatizing childhood experiences such as CSA. Finally, we will present a context in which to understand and interpret documented forgetting and later remembering of CSA (i.e., discontinuous memories) by weighing research on individual differences in the acute stress response, the relationship between chronic stress and stress reactivity, and research examining amnestic qualities associated with acute stress for contextual information.

Situations in which people encounter a (perceived) threat to their homeostasis will trigger an acute stress response (Goldstein & McEwen, 2002). Acute stress effects on attention and memory are driven by numerous neurotransmitters, hormones, and peptides, that are released in response to stressful events, and act directly, or indirectly on medial-temporal and prefrontal areas of the brain that are crucial for memory (Joëls & Baram, 2009). Importantly, the research examining acute stress on general memory processes has used a wide range of methodologies, examining different aspects of long-term memory, including memory for words, faces, and locations (for review, see Shields et al., 2017). We suggest that much can be gained by examining the scope of this research and considering it as it relates to the memory for childhood traumatic experiences. Further, while these memory tasks vary, they do provide

insight into just how acute stress may impact long-term episodic memory, and therefore, how acute stress may be considered in the context of formation, consolidation, and even voluntary forgetting of memories for childhood traumatic experiences (stored as long-term episodic memories).

2. Stress, attention, and encoding

Stress responses can be closely examined using standardized lab stressors, characterized by active, evocative situations that require instrumental behavior (e.g., spontaneous speeches, mental arithmetic; Blascovich & Tomaka, 1996). Lab stressors, while not as naturalistic as real-world stressors, are invaluable tools because they allow researchers to manipulate and understand contextual effects as well as physiological, cognitive, and affective responses as they unfold during a stressor. Studying the stress response in the laboratory involves assessing both physiological and behavioral responses to arbitrary short-term behavioral stimuli under artificial conditions that are seldom encountered in everyday life. Fortunately, there is consensus that physiological markers associated with an acute stress response in the context of real-world and laboratory stressors are similar (Kidd, Carvalho, & Steptoe, 2014; Steptoe, Cropley, & Joekes, 2000).

When an individual experiences an acute stressor, whether in the lab or in the real world, they likely undergo a biphasic stress response. The first phase is colloquially referred to as "fight-or-flight." During this phase, the body diverts energy and resources toward removing itself from the stressor (Godoy, Rossignoli, Delfino-Pereira, Garcia-Cairasco, & de Lima Umeoka, 2018). As Fig. 1 (adapted from Antoun, Edwards, Sweeting, & Ding, 2017, p. 3) illustrates, the immediate physiological impact of this phase includes the rapid activation of the sympathetic nervous system (SNS), releasing adrenaline, which in turn drives the activation of the amygdala, hippocampus, and prefrontal cortex and induces physiological changes (e.g., increased heart rate and change in blood pressure). The second phase, the "repair phase," begins approximately 20 min after the stressor has occurred (Kirschbaum, Pirke, & Hellhammer, 1993). The slower hypothalamic–pituitary–adrenal (HPA) axis releases glucocorticoids (cortisol in humans). Glucocorticoid actions enhance hippocampal plasticity and disrupt prefrontal functioning (Gagnon & Wagner, 2016).

The biphasic acute stress response has direct consequences for attention and memory (see Fig. 2 for a summary). Adrenaline and noradrenaline alter processes in the hippocampus, amygdala, and prefrontal cortex, which impact attention and encoding processes. This cognitive impact has been shown by research demonstrating that attention narrows during highly arousing scenarios (Easterbrook, 1959), as well as in research that has found an association between increased arousal and decreased short-term memory capacity (Sørensen et al., 2014). This research suggests that individuals who experience high levels of arousal may focus their attention on a subset of information, rather than attending to the event as a whole.

Researchers have provided evidence for attention narrowing using eye-tracking studies. In one study, Herten, Otto, and Wolf (2017) found that participants who underwent psychosocial stress induction had longer fixation times and more frequent fixations on items used by the

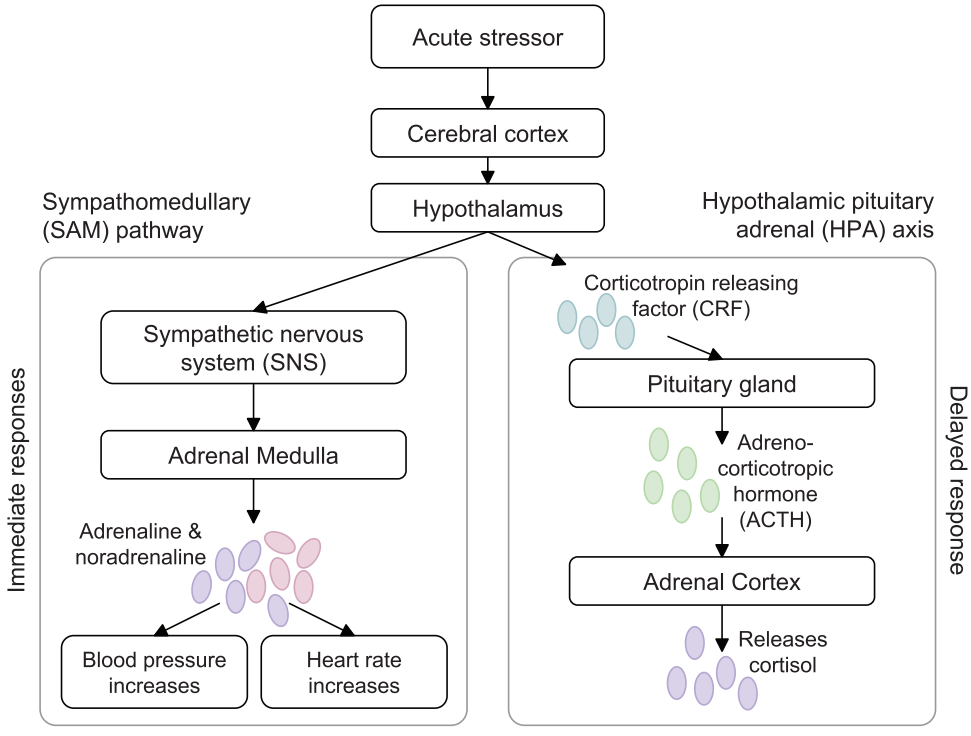

Fig. 1. Acute stress response pathways.

Note. Adapted from Antoun et al., 2017, p. 3. CC BY 4.0.

researchers during stress induction, compared to items that were present but not used. Further, they found that stressed participants spent less time fixating on the faces of the researchers performing the stress induction than nonstressed participants. Stress appeared to influence attention shifts toward relevant stimuli and away from information that was either irrelevant or uncomfortable for the participants. Similarly, researchers found that participants who underwent stress induction had better memory for items that were relevant to the stressor than for items that were irrelevant to the stressor (Shields et al., 2021).

Importantly, there is evidence to suggest that centrality to the stressor is not just about similarity or proximity to the stressor. Schwabe and Wolf (2010) induced stress by having participants submerge their hands in ice water. During this procedure, they played neutral words related to the stressor (e.g., water and cold) and neutral, negative, and positive words unrelated to the stressor. They found that stressed participants had poorer memory for words in all categories than nonstressed participants. They concluded that while words like "water" and "cold" were semantically related to the stressor, they may not have been deemed relevant to participants, who were busy focusing on another task—dealing with the pain of having their hand submerged in ice water. Importantly, this does not mean that individuals experiencing a stress response were able to *control* the information to which they attend. In one study,

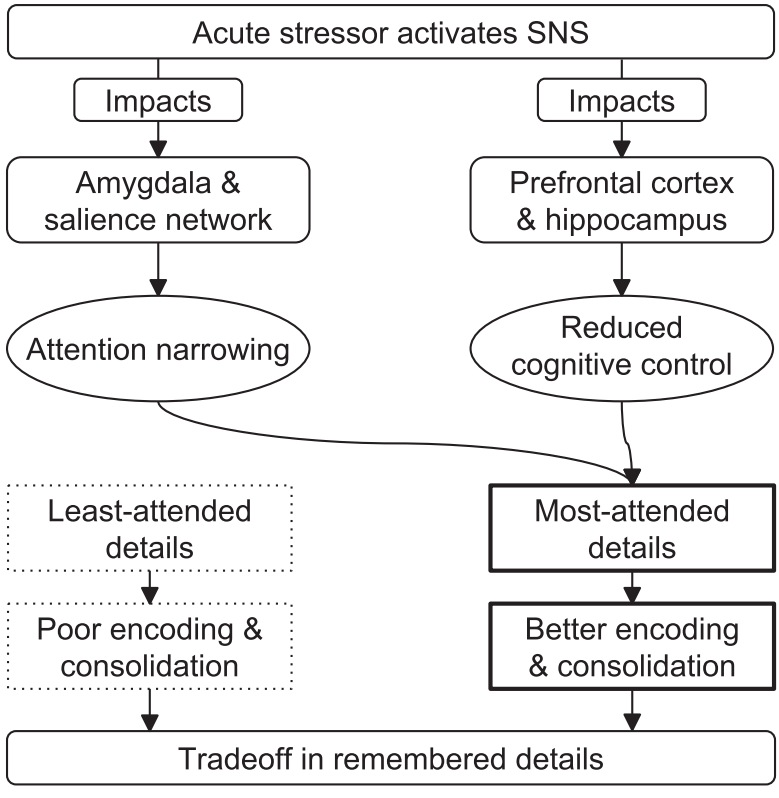

Fig. 2. Impact of acute stress on attention and memory processes.

stressed participants exhibited a decreased ability to ignore task-irrelevant information in a No-go Flanker task compared to nonstressed participants (Kan et al., 2021). While individuals may only be attending to a specific subset of information, they may have less control when deciding which subset to attend.

This impact of stress on attentional focus during an event may also have downstream consequences on memory for that event. Fig. 2 presents a schematic of the impact of the immediate acute stress response on the brain and cognition demonstrating how some information may be better remembered, while other information may be forgotten. Attention improves connections in the brain that preserve representations of information in the hippocampus. Therefore, the information to which individuals most attend during a stressful scenario will be better remembered than information that is less attended (e.g., Muzzio, Kentros, & Kandel, 2009). In parallel, the acute stress response has been shown to alter the functioning of frontal and temporal brain areas implemented in intentional memory control resulting in the poorer encoding of nonattended information (Hermans, Henckens, Joëls, & Fernández, 2014; Pruessner et al., 2008; Qin, Hermans, van Marle, Luo, & Fernández, 2009; Quaedflieg, Meyer, Smulders, & Smeets, 2015; Vogel, Kluen, Fernández, & Schwabe, 2018).

The impact of stress on cognitive processes like attention depends on an interaction between corticosteroid and noradrenergic mechanisms in the amygdala and hippocampus (Cadle & Zoladz, 2015; Schwabe, 2017) and stress reactivity changes as stress experiences accrue (Epel et al., 2018). With increasing numbers of acute stressors, long-term changes in the hippocampus emerge, likely impacting stress reactivity and the formation and accessibility of memories. What is clear from the extant literature is that stress upregulates activity in the amygdala, resulting in attentional shifts to emotional/arousing information and downregulation of activity in the cognitive control network (Quaedflieg et al., 2020). This dynamic regulation of specific neural regions has downstream consequences on what information may be better encoding. What remains unclear is how information is partition toward upward and downward regulation. However, this research does implicate a mechanism for highly salient memory of details associated with a childhood trauma, while also having no memory for other details.

3. Emotionality of stimuli

The emotional valence of stimuli has been found to interact with the acute stress experience, likely because of the upregulation of the salience network, which includes the amygdala. Acute stress may trigger the amplifying role of the amygdala (activated by emotional/arousing information) as the center of a widespread salience network, promoting hypervigilance toward threatening stimuli (Hermans et al., 2014). In support of this hypothesis, researchers found that, in the context of an acute stressor, stimuli designed to induce negative emotions were better remembered than stimuli designed to induce positive emotions (Kuhlmann, Kirschbaum, & Wolf, 2005; Kuhlmann, Piel, & Wolf, 2005, but see, Schwabe & Wolf, 2014; Smeets et al., 2009). In one study, researchers had participants undergo stress induction before viewing a series of pictures that depicted a stressful event which was either presented as reality (emotional event) or as a mock drill for a hospital (neutral event) (Payne et al., 2007). They found that participants who underwent stress induction had better memory for the emotional event, but poorer memory for the neutral event, than nonstressed participants, consistent with changes in both the salience network and top-down control.

The effect of stress on memory for emotionally valenced information can be seen using both behavioral and neuroscientific techniques. As one example, Wirkner, Weymar, Löw, and Hamm (2013) presented a series of negative, neutral, and positive pictures after inducing stress. After a delay, participants completed a recognition task that contained both old and new pictures, while their brain activity was measured using event-related potentials. Emotional pictures were better recognized than neutral pictures for stressed participants as compared to nonstressed participants. Further, acute preencoding stress enhanced the late centro-parietal old/new effect for negatively valenced pictures but not for neutral pictures.

While individuals may direct attention to emotional information when stressed, they also demonstrate enhanced retrieval of emotionally valenced information when stressed. For example, when participants encoded negative and neutral pictures immediately prior to a stress induction task, after a delay of 1 week, stressed participants recalled more pictures

and more details about the negative pictures than did nonstressed participants, though this difference was not found for neutral pictures (Cahill et al., 2003). As the pictures were not relevant to the stressor and were shown prior to stress induction, this indicates the emotional valence of the pictures had an impact on memory.

These results suggest that stress heightens the encoding and retrieval of negative and potentially traumatic memories, which is inconsistent with accounts of forgetting traumatic experiences. Further, it suggests that the experience of acute stress may result in highly accessible negative memories that may be difficult to put out of mind. Therefore, research on memory for negative information in the context of an acute stress response seemingly does not support the forgetting or repression of CSA. Rather, memories formed in the context of an acute stressor may be difficult to forget. There are several reasons why there may be inconsistencies in the literature on acute stress and memory and the literature documenting the forgetting of CSA. The research presented focuses on how specific physiological markers of an acute stress response impacts cognitive functioning. While there is reason to assume that individuals who experience real-world trauma experience similar physiological impacts of stress, there is only limited research examining the acute stress response in children. Additionally, the acute stress response is highly variable. Often in the laboratory, researchers employ numerous controls to study the stress response. For example, samples in which researchers examine the stress response are often restricted to male participants, as hormones produced by the ovaries may interview with the stress response. If female participants are included, researchers often restrict samples based on the use of hormonal birth control and/or menstrual cycle. This demonstrates a variability in the stress response that could have consequences on remembering traumatic experiences. We will return to this issue as we consider research that has documented forgetting and then later remembering of CSA.

4. Suppression and cognitive control

Thus far, this review has presented research that suggests that the acute stress response may result in attentional shifts that improve the encoding of negative information. This research suggests that traumatic experiences that accompany an acute stress response may be better remembered, rather than forgotten. However, there is a robust area of research that suggests that individuals may be able to exercise control over remembering, resulting in what has been termed suppression-induced forgetting (Anderson & Green, 2001), or voluntary forgetting. Suppression-induced forgetting has been studied via the Think/No-Think (T/NT) paradigm, where participants are cued to retrieve (Think) or suppress (No-Think) a target memory (Anderson & Green, 2001). Suppression is measured by the difference in memory performance on a later memory test between items that were repeatedly suppressed and items that were not suppressed (i.e., baseline items). Not only has suppression in the T/NT paradigm been shown to reduce direct memory access, but it also has been shown to reduce indirect memory expression, as measured by the number of intrusions across repeated suppression attempts (Benoit, Hulbert, Huddleston, & Anderson, 2015; Davidson, Hellerstedt, Jönsson, & Johansson, 2020; Hellerstedt, Johansson, & Anderson, 2016; Levy & Anderson,

2012; but see, Bulevich, Roediger, Balota, & Butler, 2006; Dieler, Plichta, Dresler, & Fallgatter, 2010; Wessel, Albers, Zandstra, & Heininga, 2020). Moreover, behavior outside of awareness, assessed with indirect memory tests (e.g., repetition priming), is also influenced by intentional suppression (for review, see Hu et al., 2017).

The phenomenon of memory suppression implicates control over memory whereby the individual actively tries to not think about specific information. This has direct relevance to how individuals may cope with traumatic events, suggesting that when trauma is remembered, an individual may actively try to avoid thinking about this to regulate their cognitive and emotional experience (see Thomas, Wulff, Landinez, & Bulevich, 2022). However, the few studies that have examined the impact of acute stress on one's ability to engage in this kind of control suggest that stress may actually disrupt this adaptive process. Quaedflieg et al. (2020) demonstrated that stress interfered with the active suppression of memories in the T/NT paradigm. They also discovered that the inability to control memories under stress was linked to altered theta activity in the right inferior parietal lobule and to changes in functional connectivity between the hippocampus and the right dorsolateral prefrontal cortex (DLPFC), associated with regulating and suppressing memory activity. In a follow-up study, the cue–target pairs consisted of more complex stimuli—the first half of a video clip as the cue and the second half as the target. With these stimuli, they found that suppression-induced forgetting was impaired in participants who showed a cortisol response to stress. These laboratory studies complement research examining the activation of the DLPFC in individuals with PTSD. In healthy individuals, attempts to prevent intrusive memories were associated with a significant reduction of the functional coupling between control and memory systems, compared to situations where a reminder did not trigger an intrusion. This decoupling was not found in individuals experiencing PTSD symptoms. (Mary et al., 2020).

Acute stress has been shown to impact prefrontal cortex functioning (Arnsten, 2009; Holmes & Wellman, 2009; Qin et al., 2009) and accompanying behavioral deficits in cognitive control and flexibility tasks, such as ignoring distractions (Plessow, Fischer, Kirschbaum, & Goschke, 2011) and generating compound remote associates (Alexander, Hillier, Smith, Tivarus, & Beversdorf, 2007). Stress has also been shown to impair attention switching (Elling et al., 2012) and overall "top-down" attentional control (Starcke & Brand, 2016). With control processes disrupted by an acute stressor, suppression may not be a viable explanation for voluntary or controlled forgetting of CSA.

5. Reconciling laboratory findings with documented forgetting of CSA

Although stress has been shown to result in robust memories, research has also demonstrated that individuals can forget traumatic childhood experiences, (e.g., Harvey & Herman, 1994; Schooler, Ambadar, & Bendiksen, 1997; Schooler, Bendiksen, & Ambadar, 1997). A central component of the repressed and recovered memory debate is that prior to recovery, the memory was inaccessible. Notwithstanding the arguments of encoding specificity (e.g., Tulving & Pearlstone, 1966) and appropriate retrieval cues (Wallace, 1978), researchers have provided evidence of corroboration for some

recovered memory reports, suggesting that for some individuals, memories for CSA were forgotten and later remembered. For example, Herman and Schatzow (1987) found that of 53 patients participating in group therapy for childhood abuse, 64% reported severe to moderate forgetting and 74% claimed to have strong corroboration (e.g., photos, diaries, and confessions). Schooler and colleagues (e.g., Schooler et al., 1997; Schooler et al., 1997; Shobe & Schooler, 2001) described corroborative evidence associated with several case studies of individuals who remembered long-forgotten memories of abuse. Further, Geraerts et al. (2007) found that continuous memories (e.g., no reported period of lack of access) of CSA were comparable to memories that were recalled unexpectedly out of the context of therapy in the likelihood of finding corroborative evidence. This research also aligns with studies demonstrating similar kinds of forgetting and later remembering of neutral autobiographical events (e.g., Read & Lindsay, 2000).

The research examining acute stress and cognitive processes associated with memory formation and voluntary forgetting would suggest that forgetting of CSA is unlikely. However, the laboratory research that we have discussed to this point has examined physiological responding to acute stressors under controlled conditions. Biomarkers associated with changes in the SNS and the HPA axis can be measured as evidence of a physiological response to an acute stressor. In real-world scenarios, such biomarkers cannot be measured and the experience of a physiological change in the context of an acute stressor such as CSA must be inferred. Research suggests that basal-level cortisol (i.e., resting level), variability in the acute stress response, and individual differences in stress response (e.g., age, sex) may influence attention and memory formation. Therefore, we briefly consider this literature as it may account for forgetting of traumatic experiences, such as CSA.

5.1. Variability in the stress response across the lifespan

While acute cortisol reactivity is important to understanding stress–encoding interactions, research suggests that basal cortisol may also play a role in memory robustness. For example, Gutchess, Alves, Paige, Rohleder, and Wolf (2019) found that higher levels of basal cortisol in older adults impaired encoding and retrieval processes for negative information. That is, older adults who had higher levels of cortisol at rest demonstrated disruption in encoding and retrieval. Shields et al. (2021) found that HPA axis activity in late childhood was inversely related to emotional memory in adolescence. That is, at low levels of prior HPA axis activity, memory-related hippocampus–amygdala functional connectivity was greater when encoding negative stimuli than when encoding neutral stimuli. For participants who experienced childhood stress resulting in higher prior HPA axis activity, encoding for negative stimuli was greater at low levels of cortisol than at high levels of cortisol. These results suggest that late childhood stress will have downstream consequences for memory formation and consolidation in the context of subsequent acute stressors. Importantly, the results with adolescents diverge from those found with older adults, and suggest that more research is needed to understand how maturation without age-related decline impacts increases in stress reactivity that emerges after early life stressors. Regardless, this research suggests that prior life stressors and age are important factors to consider when faced with potentially recovered memories of

CSA. These results suggest that age-related changes may occur in the relationship between biomarkers of the stress response and cognitive processes (see also Epel et al., 2018).

5.2. Sex

There are important sex differences in the acute stress response. For example, men tend to show greater overall changes in the physiological stress response as compared to women (Blascovich & Tomaka, 1996). However, often found sex differences are reduced when nonstress-related hormones and Body Mass Index (BMI) are controlled (Kassam, Koslov, & Mendes, 2009; Mendes, Mello, Ventura, Passarela, & Mari, 2008). While some of this work has been done in humans, greater control over specific physiological mechanisms is garnered by investigating these mechanisms in animal models. From work with mice, researchers have demonstrated that high estrogen levels have been shown to facilitate hippocampal-dependent spatial memory (Gresack & Frick, 2006; Luine & Frankfurt, 2020; Tuscher, Taxier, Schalk, Haertel, & Frick, 2019), potentially by increasing synapse-bearing dendritic spines (Gould, Woolley, Frankfurt, & McEwen, 1990; Vierk, Brandt, & Rune, 2014; Woolley, Gould, Frankfurt, & McEwen, 1990). However, high estrogen levels may also worsen hippocampus-dependent memory (Tanaka & Sokabe, 2013). Research has also demonstrated that male and female mice that were exposed to physical, emotional, and social stressors simultaneously had poorer spatial memory than mice in the control group. Importantly, female mice underwent stress induction either in early proestrus (associated with high levels of estradiol) or during estrus (associated with low physiological levels of estradiol). Mice experiencing stress induction during early proestrus had poor spatial memory, whereas those exposed to multiple stressors during estrus were protected (Hokenson et al., 2021). Although this work examining sex differences in how the acute stress response affects memories has primarily been studied in animal models, it is relevant to consider how the physiological response that accompanies an acute stressor may be influenced by hormones produced by the hypothalamus, pituitary, and ovaries.

The acute stress response is highly dependent on a wide range of co-occurring biological processes. Maturation, age-related cognitive decline, sex, and early life stressors have all been shown to influence cognitive processes (primarily memory) that occur in the context of an acute stressor. Further, research suggests that stressful experiences that initiate memory storage processes may also result in amnesia for subsequent information, resulting in an emotional-induced enhancement of memory at the cost of subsequently learned information (Cadle & Zoladz, 2015). Although the literature discussed encompasses a range of methodologies and spans work with humans and animals, the research suggests that there are factors that may contribute to forgetting even when a memory was formed in the context of an acute stressor.

Finally, it is relevant to consider how cognitive processes may be influenced by long-term exposure to stress (i.e., chronic stress). Chronic stress has been shown to cause dendritic shrinkage and debranching (Watanabe, Gould, & McEwen, 1992) and decrease the number of contacts made between neurons in the hippocampus (Sandi et al., 2003), which has been linked to disruptions in declarative memory (Tomar, Polygalov, & McHugh, 2021).

Greater levels of early life adversity are associated with poorer cognitive function in adulthood (Gold et al., 2021). Chronic stress causes decreased expression of cortisol receptors in the hippocampus (Herman, 2013) and neurodegeneration, which may lead to impaired hippocampus-dependent memory consolidation. This parallels findings from human clinical populations, showing that long-term elevated systemic cortisol seems to impair (emotional) memory consolidation (McEwen, 2016; Wirkner et al., 2017). Given that experiences of chronic stress are typically not reported in the recovered memory literature, we cannot examine the relationship between early and long-lasting life adversity and forgetting of childhood trauma. However, we suggest that researchers and clinicians should not discount experiences of disrupted and/or discontinuous memories for CSA by pointing to the acute stress literature, without considering the range of other stress-related factors that may impact memory formation and later access.

6. Conclusions

The primary goal of this brief review was to consider research on the acute stress response as it impacts memory formation and consolidation. We presented research that suggests that when acute stress occurs in the context of new learning, it influences attention, encoding, and storage of information that co-occurred during the stressor, and information directly associated with the stressor. Given the likelihood that experiences of childhood trauma and CSA occur in the context of the acute stress response, this review is relevant when considering phenomena, such as repressed and recovered memories. We also presented research that may help researchers and clinicians consider how disruption, forgetting, and later remembering of CSA may occur. While the acute stress response literature consistently demonstrates that memory for emotional information is enhanced when encoded in the context of stress, research also demonstrates that stress-enhanced encoding may accompany impaired memory for other co-occurring neutral or contextual information. Further, the physiological stress reactivity that results in these cognitive effects depends on age, sex, the experience of multiple stressors in close succession, and the presence or absence of chronic stress. Therefore, we urge caution in the evaluation of CSA memory reliability as the understanding of neurobiological factors in the formation and accessibility of memories continues to evolve.

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