Psychological Bulletin

The Effects of Acute Stress on Episodic Memory: A Meta-Analysis and Integrative Review

Grant S. Shields, Matthew A. Sazma, Andrew M. McCullough, and Andrew P. Yonelinas

The Effects of Acute Stress on Episodic Memory: A Meta-Analysis and Integrative Review

Grant S. Shields, Matthew A. Sazma, Andrew M. McCullough, and Andrew P. Yonelinas
University of California, Davis

A growing body of research has indicated that acute stress can critically impact memory. However, there are a number of inconsistencies in the literature, and important questions remain regarding the conditions under which stress effects emerge as well as basic questions about how stress impacts different phases of memory. In this meta-analysis, we examined 113 independent studies in humans with 6,216 participants that explored effects of stress on encoding, postencoding, retrieval, or postreactivation phases of episodic memory. The results indicated that when stress occurred prior to or during encoding it impaired memory, unless both the delay between the stressor and encoding was very short and the study materials were directly related to the stressor, in which case stress improved encoding. In contrast, postencoding stress improved memory unless the stressor occurred in a different physical context than the study materials. When stress occurred just prior to or during retrieval, memory was impaired, and these effects were larger for emotionally valenced materials than neutral materials. Although stress consistently increased cortisol, the magnitude of the cortisol response was not related to the effects of stress on memory. Nonetheless, the effects of stress on memory were generally reduced in magnitude for women taking hormonal contraceptives. These analyses indicate that stress disrupts some episodic memory processes while enhancing others, and that the effects of stress are modulated by a number of critical factors. These results provide important constraints on current theories of stress and memory, and point to new questions for future research.

Keywords: acute stress, memory, meta-analysis

Supplemental materials: http://dx.doi.org/10.1037/bul0000100.supp

Stress can have pronounced effects on our ability to remember past events. For example, as most students are aware, the acute stress brought about by taking an exam can often make it difficult to retrieve information that might otherwise be available. Indeed, a number of laboratory studies have now verified that acute social and/or physical stress can significantly impair memory retrieval (e.g., Kuhlmann, Piel, & Wolf, 2005; Roozendaal, 2002; Schwabe et al., 2009; Schwabe, Wolf, & Oitzl, 2010; Smeets, Otgaar, Candel, & Wolf, 2008). However, there is growing evidence that when acute stress (hereafter used interchangeably with stress, for brevity) is encountered shortly after information is learned (i.e., postencoding stress), stress can have beneficial effects on memory and can effectively rescue memories from the effects of forgetting (e.g., Andreano & Cahill, 2006; Beckner, Tucker, Delville, &

Mohr, 2006; Cahill, Gorski, & Le, 2003; Roozendaal, 2002; Smeets et al., 2008). Because we rely on memory in almost every aspect of daily life—such as in recognizing our friends and colleagues, remembering our grocery lists, and remembering to take daily medications—and many people experience stressful situations frequently, understanding how and when stress enhances or impairs memory has important implications for all of us.

The scientific literature on acute stress and memory has grown rapidly over the past 10 years, but there are a number of inconsistencies in the emerging literature (for earlier reviews, see e.g., de Kloet, Oitzl, & Joëls, 1999; Gagnon & Wagner, 2016; Joëls, Pu, Wiegert, Oitzl, & Krugers, 2006; Schwabe, Joëls, Roozendaal, Wolf, & Oitzl, 2012). For example, stress effects have been found to preferentially impact emotional memories in some studies (Cahill et al., 2003), but to have similar or even more pronounced effects on neutral memories in others (McCullough & Yonelinas, 2013). In addition, how stress impacts the process of encoding information into memory is particularly controversial, as there are some studies showing that stress impairs encoding (e.g., Maheu, Collicutt, Kornik, Moszkowski, & Lupien, 2005; Payne et al., 2007), but others showing that stress enhances encoding (e.g., Payne et al., 2007; Smeets, Giesbrecht, Jelicic, & Merckelbach, 2007). Because the experimental methods often differ considerably across these studies, it has been difficult to determine the factors that are responsible for the reported discrepancies. However, the large number of studies that have now been published affords us the opportunity to use meta-analytic methods to determine the

conditions under which stress improves or impairs memory and to identify the factors that moderate those effects.

How Does Stress Influence the Neural Substrates of Memory?

Stress influences multiple neural pathways and brain systems that are critical for episodic memory. For example, stress first exerts rapid effects in the brain by producing a surge in both dopaminergic and noradrenergic activity within the prefrontal cortex (Arnsten, 2009; Shansky & Lipps, 2013). Stress then acts through the sympathetic-adrenal-medullary (SAM) axis to upregulate peripheral adrenaline and noradrenaline (Allen, Kennedy, Cryan, Dinan, & Clarke, 2014; Joëls, Fernandez, & Roozendaal, 2011; Schwabe et al., 2012; Thoma, Kirschbaum, Wolf, & Rohleder, 2012). These hormones then stimulate afferents of the vagus nerve and ultimately influence the hippocampus, amygdala, and prefrontal cortex, among other regions (de Quervain, Aerni, & Roozendaal, 2007; Roozendaal, Okuda, de Quervain, & McGaugh, 2006; Schwabe et al., 2012; Schwabe & Wolf, 2011). On a slightly longer timescale of about 15 to 60 min, stress acts through the hypothalamic-pituitary-adrenal (HPA) axis, which upregulates production of glucocorticoids (cortisol in humans), among other hormones (Allen et al., 2014; Dickerson & Kemeny, 2004; Joëls et al., 2011; Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004; Schwabe et al., 2012). After upregulation from the adrenal glands, cortisol makes its way through circulation, freely crosses the blood–brain barrier, and can directly influence neural activity in the hippocampus, amygdala, and prefrontal cortex by binding to receptors located on neurons in those regions (Butts, Weinberg, Young, & Phillips, 2011; de Quervain, Roozendaal, & McGaugh, 1998; de Quervain, Roozendaal, Nitsch, McGaugh, & Hock, 2000; Patel, Katz, Karssen, & Lyons, 2008; Roozendaal, 2002; Yuen et al., 2009). On an even longer timescale, stress also acts to upregulate immune system production of inflammatory proteins (known as proinflammatory cytokines) through noradrenergic stimulation of immune system cells (Bierhaus et al., 2003; Slavich & Irwin, 2014). Proinflammatory cytokines can directly influence neural activity by binding to their receptors on neurons, or they can indirectly influence neural activity through stimulation of the vagus nerve (Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008; Raison, Capuron, & Miller, 2006). In either case, proinflammatory cytokines alter activity in the hippocampus, amygdala, and prefrontal cortex, among other regions (Audet, Jacobson-Pick, Wann, & Anisman, 2011; Harrison, Cercignani, Voon, & Critchley, 2015; T. K. Inagaki, Muscatell, Irwin, Cole, & Eisenberger, 2012; Zalcman et al., 1994).

These effects of stress on the brain do not occur in isolation. For example, the HPA axis and immune system regulate each other with feedback loops (Sapolsky, Rivier, Yamamoto, Plotsky, & Vale, 1987; Silverman & Sternberg, 2012). More importantly for this paper, glucocorticoids and noradrenaline critically interact to modulate activity within brain regions supporting memory, such as the hippocampus (de Quervain et al., 2007; Joëls et al., 2011, 2006; Roozendaal, Okuda, de Quervain, et al., 2006; Schwabe et al., 2012). Blocking noradrenergic activity, for example, blocks the effects of glucocorticoids on memory (de Quervain et al., 2007; Joëls et al., 2011; Schwabe et al., 2012).

Stress also exerts effects at the synaptic and molecular levels in the brain (Conrad, 2010; Conrad, Lupien, & McEwen, 1999; Diamond, Campbell, Park, Halonen, & Zoladz, 2007; Joëls et al., 2011, 2006; Roozendaal, 2002; Sandi & Pinelo-Nava, 2007). Stress prior to learning, for example, impairs long-term potentiation (LTP; thought to be critical for memory) in the hippocampus and elsewhere (Diamond et al., 2006; Maroun & Richter-Levin, 2003). In contrast, stress during learning enhances LTP (Conboy & Sandi, 2010). This time-dependent effect on hippocampal LTP is attributable at least in part to a biphasic effect of activity in the basolateral amygdala—a highly stress-responsive brain region (Schwabe et al., 2012)—on hippocampal plasticity (Akirav & Richter-Levin, 1999, 2002; Diamond et al., 2007). Importantly, this biphasic effect is mediated at least in part by noradrenaline and glucocorticoids (Akirav & Richter-Levin, 1999, 2002). That is, there are well-described pathways linking the biological effects of stress to synaptic and molecular changes in neurons related to memory.

Thus, stress impacts brain regions in several neural systems that are thought to be involved in memory (see below), including the amygdala, the hippocampus, and the prefrontal cortex, through a wide variety of routes. Moreover, some of these effects can be expected to occur within seconds after the stressor occurs, whereas other effects are expected to unfold over longer periods, up to hours after the stressor has passed.

Which Memory Processes Are Influenced by Stress?

In the current paper we focus on examining the effects of stress on episodic memory, which is the ability to remember past events as measured on tests such as recognition and free recall (for the effects of stress on other forms of human memory such as implicit memory and working memory, see e.g., Luethi, Meier, & Sandi, 2008; Shields, Sazma, & Yonelinas, 2016). In the discussion we will consider how these results in humans compare to results obtained in various animal learning tasks. In studies of episodic memory, stress can have different effects on memory depending on the phase of memory processing that the stressor impacts. For example, stress can impact the *encoding* of the initial event, the *retention* of the stored information (i.e., the postencoding period), and the *retrieval* of previously encoded information. In addition, recent studies have suggested that stress may impact memory if it occurs when memories are *reactivated* at some time between the initial encoding and the final retrieval phase. Note that although the different phases of memory may sometimes engage overlapping cognitive processes, these different phases need not all be influenced by stress in similar ways, and so it is important to separately examine studies focused on these different memory phases.

Episodic memory is critically dependent on a variety of separable memory processes supported by a network of brain regions, many of which are influenced by stress. Most critical is the hippocampus, which is essential for "binding" or associating the different features that make up an event (e.g., Eichenbaum, Otto, & Cohen, 1992; Scoville & Milner, 1957; Yonelinas, 2013; Zola-Morgan, Squire, & Amaral, 1986). The hippocampus is generally thought to support memory encoding by binding together object information it receives from the ventral "what" stream with the contextual information that it receives from the dorsal "where"

stream, as well as supporting the subsequent retrieval of those associations in tests of recognition and recall (Davachi, 2006; Diana, Yonelinas, & Ranganath, 2007; Eichenbaum, Yonelinas, & Ranganath, 2007; Mayes, Montaldi, & Migo, 2007; Norman & O'Reilly, 2003). In addition, the amygdala—which plays a key role in processing emotion—supports episodic memory for emotional events, and is either thought to form bindings between objects and emotions (Yonelinas & Ritchey, 2015) or to modulate the hippocampal binding of that information (McGaugh, 2004). Relatedly, the prefrontal cortex, in conjunction with other brain regions, is thought to be involved in supporting executive control processes that are important for encoding and retrieval. For example, memory encoding benefits from semantic elaboration as well as selective attention during encoding, both of which depend on the prefrontal cortex (Blumenfeld & Ranganath, 2007; Fletcher, Shallice, Frith, Frackowiak, & Dolan, 1998; Gershberg & Shimamura, 1995; Iidaka, Anderson, Kapur, Cabez, & Craik, 2000; Mangels, 1997; Parkin, 1997). In addition, memory retrieval benefits from prefrontal-dependent executive control processes that support organized memory search as well as memory monitoring (Dobbins, Foley, Schacter, & Wagner, 2002; Gershberg & Shimamura, 1995; Henson, Rugg, Shallice, & Dolan, 2000; Levy & Anderson, 2002; Incisa della Rocchetta & Milner, 1993). Thus, stress can influence episodic memory through its impact on the neural bases of binding, emotion, and/or executive function.

Current Theories of Stress and Memory

Several theories have been proposed to account for the effects of stress on memory including "consolidation," "dual-mode," "executive control," and "reconsolidation" accounts. One broad class of theories that has been useful in understanding the effects of stress on memory are consolidation theories (Cahill & McGaugh, 1998; Joëls et al., 2011, 2006; McGaugh, 2000, 2004, 2015). The main idea behind these theories is that recently encoded events are likely to be forgotten unless there is an active process of consolidation whereby the initial fragile memory traces formed by the encoding event are "stabilized" or "solidified" into long-term memories. This process is thought to be dependent on the medial temporal lobes and is assumed to be facilitated by stress.[1] If stress is experienced shortly after encoding it will aid in consolidating memory for recent information, resulting in slowed forgetting relative to conditions in which stress is not experienced. The process of consolidation is thought to be enhanced by the conjunctive effects of the noradrenergic and glucocorticoid responses to stress, specifically in the amygdala and the hippocampus (Joëls et al., 2011, 2006; McGaugh, 2000, 2015).

The most direct prediction about episodic memory from consolidation theory is that postencoding stress should facilitate consolidation of recently encoded events, and so it should slow forgetting. In addition, because of the role of the amygdala in supporting emotional memory, and its sensitivity to both the noradrenergic and corticosteroid responses to stress, it can also be expected that stress should have its greatest effects on memory for emotional or arousing materials (Cahill & McGaugh, 1998; Joëls et al., 2011; McGaugh, 2000, 2004, 2015). Thus, a second prediction from consolidation theory is that postencoding stress should preferentially benefit memory for arousing materials.

Although initial consolidation-based explanations of the effects of stress on memory focused on the effects of stress during the postencoding phase, a "dual-mode" model has been proposed in which the same consolidation processes that enhance memory retention also impact both memory encoding and memory retrieval (de Kloet et al., 1999; Diamond et al., 2007; Joëls et al., 2006; Schwabe et al., 2012). That is, there is assumed to be a fast-acting "memory formation mode" that can last up to 30 min after stress onset, followed by a slower "memory storage mode" that can last hours. During the initial period, fast-acting stress hormones (e.g., noradrenaline and "nongenomic" effects of cortisol) alter processing in the hippocampus, amygdala, and prefrontal cortex, which increases attention to and encoding of stress-relevant materials. This memory formation mode is assumed to compete with or suppress the retrieval of unrelated information (Cadle & Zoladz, 2015; Schwabe et al., 2012). In contrast, after a longer delay, glucocorticoids begin to exert slower, genomic effects (i.e., effects on and through changes in gene expression), initiating a "memory storage mode." These genomic glucocorticoid effects are thought to facilitate the consolidation of recently encoded memories and impair the ability to encode new information, thus reducing interference from novel information and further benefitting recently encoded memories.

The dual-mode theory predicts that postencoding stress should enhance memory because the encoded items will benefit from both the fast memory formation mode and the slower memory storage mode. In contrast, when stress occurs during or prior to retrieval, it should impair memory because both the initial memory formation mode and the slower memory storage mode inhibit retrieval. Moreover, this model predicts that stress will also impact encoding, but these effects will depend on the time delay between the stressor and the onset of the encoding phase. That is, if stress occurs immediately prior to or during encoding it should enhance memory because the study event occurs during the fast memory formation mode, and the slower memory storage mode will further consolidate those memories after they have been encoded. However, if the stressor precedes the study event by more than 20–30 min, then memory encoding should be impaired because the memory formation mode would have ended and been replaced by the memory storage mode, which inhibits new encoding. So, this theory predicts that stress immediately prior to or during encoding will enhance memory, particularly for materials related to the stressor, but as the delay between stress and encoding increases, the effects of stress should reverse, such that stress begins to impair memory.

Another account of how stress impacts memory assumes that stress impacts executive functions that are involved in memory encoding and retrieval (Gagnon & Wagner, 2016; Mather & Sutherland, 2011). We will refer to this as the "executive control" theory of stress and memory. Executive functions are known to support both effective memory encoding (Blumenfeld & Ranganath, 2007; Simons & Spiers, 2003; Spaniol et al., 2009) and successful memory retrieval (Dobbins et al., 2002; Gagnon &

---

[1] Note that stress-related consolidation is sometimes referred to as cellular or synaptic consolidation, which occurs within a few hours of encoding, and is distinct from "systems consolidation," which is said to involve the transfer of hippocampal memory traces to the cortex, which can occur gradually over many years (Dudai, 2004; Squire & Alvarez, 1995).

Wagner, 2016; Levy & Anderson, 2002; Simons & Spiers, 2003; Spaniol et al., 2009). Moreover, stress appears to impair various executive functions such as working memory, selective attention, and cognitive flexibility (Alexander, Hillier, Smith, Tivarus, & Beversdorf, 2007; Sänger, Bechtold, Schoofs, Blaszkewicz, & Wascher, 2014; Schoofs, Wolf, & Smeets, 2009; Shields, Bonner, & Moons, 2015; Shields, Sazma, et al., 2016), presumably through the catecholamine (e.g., noradrenaline) and glucocorticoid disruption of frontal lobe function (Arnsten, 2009; Shansky & Lipps, 2013). Together, these lines of evidence support the notion that stress may impact memory in part by impairing executive functions that influence encoding and retrieval (Gagnon & Wagner, 2016; Joëls et al., 2006; Mather & Sutherland, 2011; Schwabe et al., 2012).

Thus, by the executive control account, stress should generally impair memory when stress impacts the encoding phase or the retrieval phase by limiting the executive process necessary for effective encoding and retrieval. However, an important exception to this rule is that because stress is generally expected to shift attention toward threat-related stimuli while attenuating the processing of unrelated materials (Gagnon & Wagner, 2016; Mather & Sutherland, 2011), stress that occurs during or prior to encoding is expected to facilitate encoding of stress-related information at the cost of information that is unrelated to the stressor.

One additional account of stress and memory is "reconsolidation theory" (e.g., Schwabe, Nader, & Pruessner, 2014) which proposes that if an old memory is reactivated after it has already gone through an initial consolidation process, the reactivation will make that memory labile once again (similar to the initial postencoding period), and it will become susceptible to modification. Thus, if subjects are stressed shortly after reactivation (i.e., postreactivation) this should also benefit future memory for those items by allowing for another round of consolidation and enhancing that consolidation through stress. Although the mechanisms for this process are still debated (Besnard, Caboche, & Laroche, 2012; Schwabe et al., 2014), several studies have now examined the effects of postreactivation stress on memory.

Most theories of stress and memory assume that the effects of stress on memory are driven by independent and/or interactive effects of glucocorticoid stress hormones—cortisol in humans, corticosterone in rodents—and noradrenaline (Gagnon & Wagner, 2016; Het, Ramlow, & Wolf, 2005; Joëls et al., 2011; McGaugh, 2000; Schwabe et al., 2012). The consolidation theories emphasize the interactive effects of these hormones in the hippocampus and amygdala, whereas the executive control theories emphasize their role in the prefrontal cortex and other regions primarily underpinning executive functions, but which are also crucial to memory-related processing. Thus, from each of these perspectives one could expect that there might be a close relationship between the magnitude of the cortisol response to stress and the magnitude of the effect on memory, though the strength of this relationship may be reduced depending on the extent to which cortisol interacts with other stress hormones such as noradrenaline.

Understanding Discrepancies in Stress Effects on Memory

The above theories, despite their impressive breadth and clarity, may or may not be able to account for inconsistencies observed in the stress and memory literature. To understand these inconsistencies, it is important to examine the empirical literature to identify what factors have been proposed as moderators of stress effects on memory. This examination will thus help us conduct a meta-analysis that is sensitive to both theoretical predictions and predictions derived from empirical literature. For ease of understanding, we categorize factors that have been proposed to account for heterogeneity and inconsistency in effects of stress on memory as either *participant/sample variables* or *study design variables* in our review.

The participant/sample variables with the strongest evidence for moderating effects in the stress and memory literature are the following. First, a number of studies have indicated that stress can influence memory differently depending on the *sex* of the participants (Andreano & Cahill, 2006, 2009; McCullough & Yonelinas, 2013). In addition, there is evidence suggesting that *menstrual phase* (Andreano, Arjomandi, & Cahill, 2008), the use of *hormonal contraceptives* (Andreano & Cahill, 2009), and sex hormones (Barros, Tufik, & Andersen, 2015; T. Inagaki, Gautreaux, & Luine, 2010) influence memory or the effects of stress on memory. Similarly, multiple studies have suggested that effects of stress on memory differ as a function of *age* (Hidalgo et al., 2015; Hidalgo, Almela, Villada, & Salvador, 2014; Pulopulos et al., 2013). Finally, although not directly studied within the context of stress and memory, there is also reason to examine effects of whether a study excluded participants who smoked, used psychoactive medications, reported current illnesses, or had a BMI greater than 30, given literature indicating that these variables may alter stress-responsive physiological systems (Allen et al., 2014; Childs & De Wit, 2009; O'Connor et al., 2009).

The study design variables with the strongest evidence for moderating effects of stress include the following. One obvious design factor that varies across studies is the *stressor type*, (i.e., the manipulation used to induce stress). For example, common stressors include the Trier Social Stress Task (TSST), where participants give a speech and perform complex arithmetic in front of a stern panel of evaluators, the cold-pressor task (CPT), where participants immerse their nondominant arms in ice water, and the Socially-Evaluated Cold Pressor Task (SECPT), which is a hybrid task involving both ice water and social evaluation. There is some evidence that different stress induction procedures elicit reliably different physiological stress responses (Dickerson & Kemeny, 2004) and some indication that the TSST may produce a larger cortisol increase than the other methods (Skoluda et al., 2015).

In addition, the *delay* between the stressor and the specific memory phase varies widely between studies, and there is empirical evidence that suggests that the timing of stress in relation to learning or retrieval may be an important determinant of the effects of stress on memory (Schwabe & Wolf, 2014; Zoladz et al., 2011). For example, if stress acts in part through actions of cortisol, which is not expected to reach peak levels until approximately 20 min after stress is initiated (Dickerson & Kemeny, 2004), then the effects of stress on memory may depend upon the delay after the stressor. Moreover, because of the different time-dependent effects of cortisol described above (Joëls et al., 2011; Schwabe et al., 2012; Shields et al., 2015), the effects of stress may be quite different at different delay periods (Schwabe et al., 2012).

There is also evidence that the *valence* of the learned materials can impact the effects of stress on memory (Cahill et al., 2003).

That is, postencoding benefits of stress are sometimes found to be restricted to emotional materials (Cahill et al., 2003), other times to impact emotional and neutral materials (McCullough & Yonelinas, 2013), and yet in other cases to preferentially impact neutral materials (Yonelinas, Parks, Koen, Jorgenson, & Mendoza, 2011). In addition, there is evidence that stress may impair retrieval of negative information more so than neutral information (Gagnon & Wagner, 2016; Kuhlmann et al., 2005).

The *relevance* of the learned materials to the stressor may be another critical factor in accounting for heterogeneity in stress effects on memory. That is, some studies have found that stress enhances memory for information related to the experienced stressor, but not for unrelated information learned at the same time as the stress-relevant information (e.g., Smeets et al., 2007; Wiemers, Sauvage, Schoofs, Hamacher-Dang, & Wolf, 2013).

In addition, there is recent evidence to suggest that a change in spatial *context* between encoding and stress can impact the effects of stress. For example, Trammell and Clore (2014) found an impairing effect of postencoding stress on memory, which is in contrast to the typically observed enhancing effect. The primary methodological difference they proposed to explain the discrepant findings between their studies and others that found enhancements was that their participants changed contexts between learning and stress, whereas participants in most other studies experience stress in the same context as learning.

Further, the *type of memory test* (i.e., free recall, cued recall, or recognition) may influence the effects of stress on memory. Many studies have found effects of stress using recall tasks (Andreano & Cahill, 2006; Cahill et al., 2003), whereas others failed to observe effects on recall but observed enhanced recognition performance (McCullough & Yonelinas, 2013). One may also expect that different types of recognition processes, such as recollection and familiarity (Yonelinas, 2002), may be differentially impacted by stress, however, only a very small number of studies have included these measures (e.g., McCullough, Ritchey, Ranganath, & Yonelinas, 2015; McCullough & Yonelinas, 2013; Wiemers et al., 2013).

Similarly, the use of an *immediate recall* task may modulate effects of stress on encoding. One study experimentally manipulated the use of an immediate recall task and found that stress effects on encoding were only seen when an immediate recall task was not used (Wolf, 2012). Thus, there is reason to consider the use of an immediate recall task as a moderator of stress effects on encoding.

We also considered *time of day* as a potentially important moderator of stress effects on memory for two reasons. First, a meta-analysis of cortisol administration studies found that cortisol administration enhanced encoding when cortisol was administered in the afternoon, whereas cortisol administration impaired encoding when cortisol was administered in the morning (Het et al., 2005). Second, there is one empirical study showing that stress prior to encoding impaired subsequent memory when stress (and encoding) occurred in the morning but stress had no effect on encoding when experienced in the afternoon (Maheu et al., 2005).

There were several other variables that had not been directly implicated in previous studies, but that we felt might have some impact on the magnitude of stress effects on memory. These included variables related to learning, such as the *sensory modality* of stimulus presentation (i.e., visual/verbal/both), *material type* (pictures, words, narrative/slideshow, autobiographical), the study *list length*, the *duration of the encoding phase*, and *incidental versus intentional encoding*. Other potentially relevant variables included the *duration of the stressor*, *delay* between encoding and retrieval, whether there was a *context change between encoding and retrieval*, the number of *novel items* in the recognition test, and whether stress was manipulated *between or within subjects*.

How Do Stress-Induced Changes in Cortisol Relate to Memory?

Cortisol is an important component of the stress response, and there is considerable evidence that cortisol responses influence memory. As such, a number of models have proposed that the cortisol response is critically involved in producing the observed memory effects (Gagnon & Wagner, 2016; Joëls et al., 2011; Schwabe et al., 2012). These claims are based on the fact that stress produces increases in glucocorticoids via activation of the HPA axis (Allen et al., 2014; McEwen, 2007) coupled with animal work showing glucocorticoids can exert causal influences on memory (de Quervain et al., 1998; Joëls et al., 2011; Roozendaal, 2002). In addition, some studies have found that the magnitude of a person's cortisol response to postencoding stress is related to their memory performance (Andreano & Cahill, 2006; McCullough et al., 2015). Moreover, cortisol administration independently influences memory encoding and retrieval in ways that can parallel purported effects of stress (Het et al., 2005).

The role of cortisol in potentially mediating stress effects on memory has been used to explain why stress effects on memory are sometimes not observed in certain conditions. For example, males exhibit more robust cortisol responses to common laboratory stressors (Kirschbaum, Wüst, & Hellhammer, 1992), and they have sometimes been found to exhibit more pronounced stress effects on memory (e.g., Andreano & Cahill, 2006), presumably because of their larger cortisol responses. Moreover, some studies suggest that use of hormonal contraceptives dampens the stress-induced cortisol response (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999; Marinari, Leshner, & Doyle, 1976), and women taking hormonal contraceptives show altered stress effects on memory (Nielsen, Segal, Worden, Yim, & Cahill, 2013). These results suggest that the reduced sensitivity of women to the stress effects on memory may arise because of relatively smaller cortisol responses.

However, the relationship between cortisol and the observed memory effects of stress has not been systematically assessed, and there are reasons to suspect that it may reflect only part of the story, with other components of the stress response also playing critical roles. For example, stress effects on memory may also be driven by effects on hormones other than cortisol such as progesterone, estradiol, or DHEA (Barros et al., 2015; T. Inagaki et al., 2010; Sripada et al., 2013) or by immune system responses (Harrison, Doeller, Voon, Burgess, & Critchley, 2014; Reichenberg et al., 2001). Although there were too few studies reporting these other biological measures to support an analysis of these biomarkers, the cortisol analysis is useful in assessing the claim that cortisol plays an important role in mediating the effects of stress on memory, and it may provide insights into the neural mechanisms supporting those effects. We also note that neuroimaging studies are also useful in assessing the neural mechanisms, but at this point the number of such studies is also rather limited (e.g., Henckens,

Hermans, Pu, Joëls, & Fernández, 2009; Qin, Hermans, van Marle, & Fernández, 2012), and so these imaging results will not be considered in our analyses.

The Current Research

In the current paper, we conducted a meta-analysis of the human studies that have examined the effects of acute stress on episodic memory. To our knowledge such a meta-analysis has never been conducted. Such an analysis is important not only for understanding when stress will impact memory, but also to assess current theories of memory and stress. We addressed this gap by conducting a meta-analytic review, by systematically examining stress effects on each separate memory phase (e.g., encoding, postencoding, postreactivation and retrieval), as well as studies examining effects of stress on more than one memory phase. In addition, we attempted to elucidate potentially important moderators—outlined above—of stress effects on phases of memory using a metaregression approach. Finally, by examining the relationship between cortisol, stress, and memory we attempted to determine the role of cortisol in moderating the stress effects on memory.

Method

Study Selection and Inclusion Criteria

Literature review. To obtain studies for use in the meta-analysis, we performed an exhaustive search of the databases PsycINFO, PubMed, and Web of Science using the following search string: ((memory) AND (emotion OR positive OR negative OR neutral OR emotional) AND (encoding OR retrieval OR consolidation OR preencoding OR postencoding OR storage OR reconsolidation) AND (Recognition OR Recall) AND (Stress OR Stressful OR Stressor)).[2]

We concluded this search in October 2015. In this search, PubMed returned 267 results, PsycINFO returned 223 results, and Web of Science returned 469 results. References from relevant articles were reviewed, and studies that were potentially relevant were examined from those references. For all articles considered, we followed Dickerson and Kemeny (2004) in reviewing abstracts and examining full texts whenever an article had the potential to include a relevant effect (e.g., if a study incorporated or could have incorporated an acute stressor, given our search string, the full-text of the article was reviewed). The first three authors reviewed all articles that were selected to have their full text reviewed, and a decision that one of these articles did not meet our inclusion criteria and should be excluded from analyses was made by mutual agreement of the first three authors. Figure 1 depicts a flow diagram illustrating our review and inclusion process.

Inclusion criteria. Our nine inclusion criteria for this study were as follows: studies had to (a) experimentally manipulate (b) acute stress and assess effects on (c) human participants (d) without a known psychological/psychiatric disorder (e) who encoded, consolidated, reactivated, and/or retrieved memories within temporal proximity to the stressor or control task. (f) To ensure that acute stress was the primary manipulation rather than arousal, the stressor task used had to either be a previously validated stressor or include a biological measure of stress validation (e.g., cortisol, cytokine reactivity) that is not also sensitive to the effects of acute
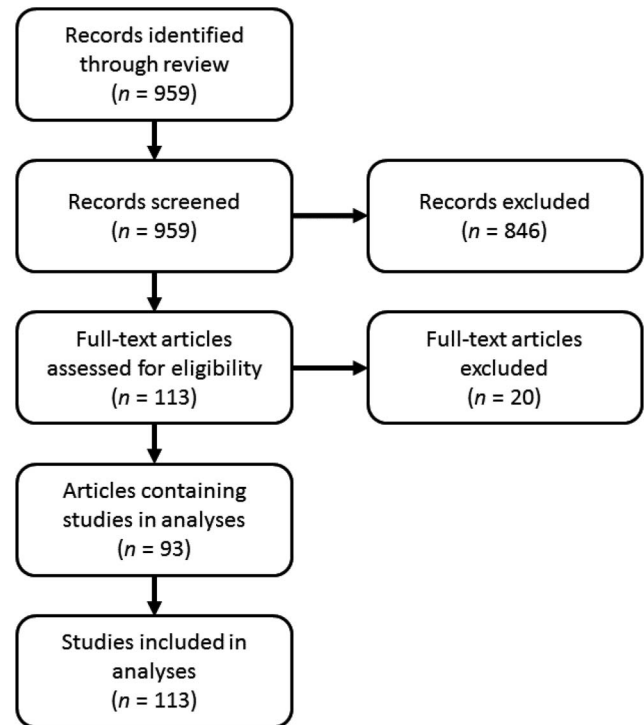

Figure 1. Flow diagram illustrating the process of our review, screening, and article selections.

arousal without stress. (g) Because stress hormones exert genomic effects on neural processes for hours after cessation of stress, the control condition could not have been subjected to a laboratory stressor on the same day as a memory procedure. This entails that if a study used a within-subjects, crossover design, the counterbalance of stress and control had to be separated by at least one day. (h) To separate effects of stress on long-term memory from stress effects on working memory, if encoding and retrieval were on the same day, a brief delay or interfering task had to separate memory encoding and retrieval. (i) Finally, because we were interested in assessing effects on memory accuracy rather than potential effects of stress on response bias, we only included recognition data if a study reported some bias-corrected measure

---

[2] We included this search term as an "AND" rather than an "OR" based upon pilot searching. Including this term as an "OR" term along with "memory" quadrupled our results in each database (e.g., PubMed went from 267 articles to 1,106), and by browsing the first few pages of each returned search we determined that almost if not all of these additional articles were irrelevant to our analyses (e.g., they were studies of stress and other cognitive processes that simply referenced stress effects on memory within the text). We found numerous articles that did not highlight or vary the timing of the stressor that nonetheless came up due to the description of the memory task within the methodology section (e.g., "Encoding took place . . ."). In short, we believe that this search string represented an efficient way of returning all relevant memory studies without returning irrelevant ones. Additionally, to ensure that our review was comprehensive, we conducted numerous nonexhaustive searches of Google Scholar using simple strings such as (stress AND memory) and similar variations. Further, we browsed references of numerous literature reviews of stress and memory to ensure we had not missed any studies referenced by prior reviews—we had not.

(i.e., d') or if a bias-corrected measure was unavailable, if no differences existed between groups in false alarms. To control for potential differences in learning between groups, if a study reported both proportion of total items recalled and proportion of immediate recall, we used proportion of immediate recall in analyses.[3] We chose these inclusion criteria to best isolate the relationship between acute stress and memory processes. Our inclusion criteria for studies given above implicitly defines what we designated a "study." Our final designation of a study was any independent (i.e., completely orthogonal) set of an experimental group and a control group that met the above inclusion criteria.

Definitions of stress in relation to memory phases. Because memory involves multiple phases (e.g., encoding, retention, and retrieval), and stress is thought to potentially influence these phases in different ways, we categorized studies in terms of the phase stress was expected to impact:

1. *Encoding* studies were those in which the stressor occurred prior to or during encoding.

2. *Postencoding (Retention)* studies were those in which the stressor occurred shortly after the encoding phase was competed.

3. *Retrieval* studies were those in which the stressor occurred shortly prior to or during retrieval.

Other studies however, used short enough retention intervals that stress (which can have effects that last for hours) was expected to impact more than one memory phase, and so were categorized as such:

4. *Postencoding/Retrieval* studies were those in which the stressor occurred shortly after encoding and there was a short enough interval between encoding and retrieval (<90 min) that the stressor likely impacted both the retention and retrieval phases.

5. *Encoding/Retention/Retrieval* studies (hereafter encoding/retrieval studies for brevity) were those in which the stressor occurred prior to or during encoding, and there was a short enough interval between encoding and retrieval (<90 min) that stress likely impacted encoding, retention, and retrieval.

Finally, a number of studies examined memory for information initially learned on one day, and subsequently reactivated on a later day with stress following the memory reactivation, and examined retrieval on a later day. These studies were categorized as such:

6. *Postreactivation* studies were those in which stressor onset occurred prior to or following memory reactivation, and neither encoding nor eventual retrieval of reactivated memories occurred on the same day as memory reactivation.

Selected studies. Our search and study inclusion criteria led to the incorporation of 113 studies, 108 of which were published in 88 peer-reviewed papers, and 5 of which were unpublished or reported in unpublished theses or dissertations. We chose to cite

the published paper if a study was presented in both a thesis/dissertation and a published paper. Of these 113 studies, 33 assessed effects of stress at encoding, 23 assessed effects of stress postencoding, 31 assessed effects on retrieval, 9 assessed effects on postencoding/retrieval, 15 assessed effects on encoding/retrieval, and 10 assessed effects of stress postreactivation.

Coding of Covariates and Moderators

We coded for a number of potential moderators of the effects of stress on memory, most of which were assessed because there was empirical or theoretical reason to believe the moderating effect would be significant. Two raters coded each study, and the agreement between raters was very good (89%). All discrepancies in study coding between raters were discussed and resolved. See Table 1 for a complete list of coded moderators.

Whenever possible, we incorporated the following information on moderators from explicit statements within the manuscript. If the manuscript did not explicitly state that information regarding a moderator but it could be inferred from their study protocol, we coded the moderator as it could be inferred (e.g., the manuscript did not state that participants either did or did not change contexts, but the study's stressor was one that required a room change—such as the Trier Social Stress Test without modifications—we coded the context as changed). Finally, when the information was not directly available in the manuscript or inferable from the protocol used, we emailed the corresponding authors of studies for that information. If that information was not obtainable, we did not include that study within a given moderator analysis.

Stressor type was coded as follows. Stressors were coded as "social" stressors if they included social evaluation but did not include pain (e.g., the Trier Social Stress Test). Stressors were coded as "pain" stressors if they included pain but did not include social evaluation (e.g., the Cold Pressor Task). Stressors were coded as "hybrid" stressors if they included both social evaluation and pain (e.g., the Socially Evaluated Cold Pressor Task). Stressors were coded as "other" if they included none of these characteristics (e.g., skydiving, mock prisoner of war stressor, threat of shock coupled with gruesome pictures).

Item type was coded as "words" if the items were presented as words or lists of words without accompanying pictures or other details to be remembered, "pictures," "narrative/slideshow" if items were presented as a narrative accompanied by pictures, or "self-related" if the items were autobiographical memories or personal questions. Finally, studied items were coded as "other" if the items were not any of the above.

The memory task was coded "integral" to the stressor if items studied were highly related to the stressor (e.g., personality words studied after a speech on one's personality to a critical evaluator panel) or if the stressor and memory task were indistinguishable to participants (e.g., face recognition for faces of the evaluators in the Trier Social Stress Test) and "nonintegral" otherwise. Sensory

---

[3] Numerous studies reported the proportion of studied items remembered at delayed recall to the number of studied items remembered at immediate recall. These scores thus entail that a score of 1.0 would imply that a participant remembered at the delayed recall test all of the items they remembered at the immediate recall test, thus controlling for individual differences in initial learning of the items.

Table 1
List of Moderators Considered in Analyses

Moderator	Variable type	Reference
Incidental or intentional encoding	Categorical (dummy-coded)	Incidental encoding
Exclusion of smokers	Categorical (dummy-coded)	Inclusion
Exclusion of women during menstrual period	Categorical (dummy-coded)	Inclusion
Exclusion hormonal contraceptives usage	Categorical (dummy-coded)	Inclusion
Exclusion of all illnesses	Categorical (dummy-coded)	Inclusion
Exclusion of all psychoactive medication	Categorical (dummy-coded)	Inclusion
Exclusion of BMI greater than 30	Categorical (dummy-coded)	Inclusion
Use of an immediate recall task postencoding	Categorical (dummy-coded)	No immediate recall
Stressor relevance of items (integral/nonintegral)	Categorical (dummy-coded)	Nonintegral
Context change between learning and stress	Categorical (dummy-coded)	No change
Context change between learning and retrieval	Categorical (dummy-coded)	No change
Stress manipulated between or within groups	Categorical (dummy-coded)	Between groups
Item valence	Categorical (dummy-coded)	Neutral items
Study material type	Categorical (contrast-coded)	
Sensory modality of study material presentation	Categorical (contrast-coded)	
Memory task type	Categorical (contrast-coded)	
Stressor type	Categorical (contrast-coded)	
Participant age	Continuous	
Percent male participants	Continuous	
Time of day study began	Continuous	
Study item list length	Continuous	
Number of novel items in a recognition task	Continuous	
Delay (hours) between item encoding and retrieval	Continuous	
Delay (min) between stress onset and encoding	Continuous	
Delay (min) between encoding and stress onset	Continuous	
Delay (min) between stress onset and retrieval	Continuous	
Delay (min) between reactivation and stress onset	Continuous	
Stressor duration (min)	Continuous	
Stress-induced Δ-cortisol (nmol/L)	Continuous	
Length of encoding phase (min)	Continuous	
Participant homogeneity	Continuous	

modality of item presentation was coded as "auditory" if the items were presented auditorily but not visually, "visual" if the items were presented visually but not auditorily, and "both" if the items were simultaneously presented auditorily and visually. Item valence was coded as "neutral," "positive," "negative" or "multiple" if more than one preceding valence type was included (notably, most studies with valenced materials will have positive, negative, and neutral item valence effects). We also considered criteria commonly reported by studies as a reason to exclude participants. All six of these study exclusion criteria we considered (i.e., excluded smokers, all illnesses, BMI greater than 30, women currently menstruating, hormonal contraceptive use, psychoactive medication use) were coded "excluded" if the study explicitly excluded the potential participants in question and coded "unreported or included" otherwise.[4] The "participant homogeneity" moderator represents the sum of the exclusion criteria moderators and is thus a variable ranging from 0–6 (with 6 having the strictest criteria and excluding the most participants).

Studied item valence was dummy-coded using neutral items as a reference group to examine potential differences in stress effects on positive items, negative items, or multiple valences relative to stress effects on neutral items. Stressor type, memory task type, studied item type, and sensory modality of item presentation were all contrast-coded to examine potential differences in stress effects without using one group of effects as a reference. All other categorical variables were dummy-coded with reference groups listed in Table 1.

Continuous variables considered as moderators were centered for analyses at the lowest obtained for each phase of memory and stress to make interpretation of the intercept (i.e., the effect size) equal to the effect of stress on memory at that lowest value of the covariate. Despite centering for analyses, graphs present uncentered data for ease of interpretability. If the average participant age was not given in the article, the median participant age was used if it was reported; if neither of these statistics were listed, the midpoint of the reported participant age range was used.

To assess stress effects on cortisol, we calculated the pretest-posttest-control group effect size (Morris, 2008) and converted from d to g using the correct transformation. We used the baseline samples as the pretest values and the peak reactivity samples (whichever value was the greatest in the stress group and the corresponding sample from the control group at this time) as the posttest values. This effect size provides an unbiased index of the effect of stress on the change in cortisol relative to the change in a control group, thus representing the effect size closest to how cortisol is analyzed in most studies.

The pretest–posttest correlation is required to calculate the variance of the pretest–posttest–control group effect size, and this

---

[4] Studies that only included males were assigned a value of "excluded" for the variable assessing exclusion of women during their menstrual period and the variable assessing exclusion of the use of hormonal contraceptives, even though the studies did not explicitly report these exclusions.

correlation was unknown to us given that no study reported this. As such, we set the pretest-posttest correlation at .3. Sensitivity analyses from .0 to .8 indicated no differences in stress effects on cortisol with high or low correlations used to derive the variance of the effects.

Because we were able to analyze stress effects on cortisol across all studies (e.g., stress at encoding, stress at retrieval, etc.), we chose to use the pretest-posttest-control group effect size, g^+_{ppc}. Because this effect size examines the difference from baseline to postmanipulation between groups (i.e., how change in cortisol over time differed between groups), it represents the best measure of effect size for determining stress effects on cortisol. However, because many studies did not provide enough information to derive g^+_{ppc} (e.g., they only reported Δ-cortisol), and reduced power greatly impacted our ability to detect cortisol effects on different memory phases (e.g., encoding, retrieval), we converted all cortisol values to nmol/L and used Δ-cortisol (posttest-pretest for the stress group) to examine cortisol effects on memory.[5] Of all studies considered in this meta-analysis, 78 provided enough information for us to extract g^+_{ppc} for cortisol, whereas 95 provided enough information for us to extract Δ-cortisol.[6]

Analytic Strategy

The effect size measure of interest was the standardized mean difference between stress and control groups. We used Hedges' g rather than Cohen's d as the effect size for analysis, given that the former is a relatively unbiased estimate of the population standardized mean difference effect size while the latter is a biased estimate. Whenever possible, we calculated Hedges' g from the means, standard deviations, and sample sizes presented in the article. If means and standard deviations were not reported and the design was between-studies, we used t or one-way F statistics—or p values resulting from tests of those two statistics—to calculate the effect size. If none of these statistics were reported, we emailed corresponding authors for these statistics. If we were unable to obtain the necessary statistics for a study from the corresponding author, that study was excluded from analysis. For within-studies designs, we converted effect size estimates and their variances into the between-study effect size metric following Morris and DeShon (2002).[7]

Given the multifaceted nature of memory, most studies often report more than one outcome (e.g., effects of stress on positive, negative, or neutral items; effects of stress on recall, cued recall, or recognition; etc.). Multiple outcomes are a problem for conventional meta-analytic methods, as averaging effect sizes within studies without accounting for their correlations can alter or obscure true effect size estimates (Borenstein, Hedges, Higgins, & Rothstein, 2009; Scammacca, Roberts, & Stuebing, 2014). Thus, we employed the meta-analytic technique of robust variance estimation, a random-effects metaregression that can account for dependence between effect size estimates (Hedges, Tipton, & Johnson, 2010; Tanner-Smith & Tipton, 2014). This technique robustly estimates effect size weights and standard errors for the given effects, allowing for multiple outcomes within studies (Hedges et al., 2010). We employed the robu() function of the robumeta package in **R**, version 3.2.2, to conduct our analyses of stress effects on memory, using the correlated weights given by Hedges et al. (2010) and using the small sample corrections

suggested by Tipton (2015). We did not use small-sample corrections in our analyses of stress effects on cortisol because we were able to examine effects across 78 studies. To account for dependency, ρ was set to the recommended .80 (Tanner-Smith & Tipton, 2014).[8] Because we were more interested in understanding factors that influence the effects of stress on memory than we were interested in understanding factors that contribute to heterogeneity in analyses, continuous moderator analyses do not separate continuous moderators into within- and between-study continuous moderators.

Degrees of freedom for all primary analyses were estimated using the Satterwaite approximation, where $df = 2/cv^2$ and cv represents the coefficient of variation, as simulation studies have indicated that this method of estimating degrees of freedom is most analytically valid with study set sizes under 40 using the RVE meta-analytic technique (Tipton, 2015). Because of how the degrees of freedom are estimated, if the degrees of freedom are less than four, there is a heightened risk of a Type I error and the analysis results cannot be trusted to represent population values (Tipton, 2015). However, because this estimation of degrees of freedom is extremely sensitive to outliers given a study set size such as in this meta-analysis (because degrees of freedom are divided by the coefficient of variation), one can be relatively confident that when degrees of freedom are greater than four, outlying studies are not driving observed significant effects.

In presenting our results, we discuss each effect sequentially and examine concurrent effects in a final model at the end of each section. We make exceptions to this rule when, by our examination of the data, two effects appear to be largely conflated and merit further attention before proceeding. For the forward stepwise regressions presented at the end of each subsection of our primary analyses, we chose a one-tailed test for these analyses a priori to ensure we had included all contributing moderators. We did not include methodological potential moderators with no a priori hypothesized effect or direction (e.g., study item list length) within these forward stepwise regression analyses. All of the effects considered in these stepwise regressions were hypothesized a priori to have an effect in an expected direction, justifying the use of a one-tailed test.

---

[5] Using g^+_{ppc} for analyses relating memory to cortisol did not alter any of the results.

[6] Three papers only reported effects of stress on cortisol averaged across their two stress and two control groups per paper. As such, to avoid giving extra weight to these studies, we considered them as single studies in analyses of stress effects on cortisol, leaving 78 studies for analyses of stress effects on cortisol.

[7] This conversion requires the correlation between performance in the stress condition with performance in the control condition if the stress/control condition is a within-subjects manipulation. None of the studies in this meta-analysis reported this correlation, so we set the correlation between these conditions at $r = .30$ (a moderate correlation) to account for measurement error and expected differences between the stress and control condition. Because so few of our studies used a within-subjects design (11.4%), sensitivity analyses setting the correlation between .00 and .80 and running the resultant meta-analysis showed that setting this correlation at .30 did not alter any of the reported results.

[8] Sensitivity analyses with values of ρ ranging from 0 to 1.0 evidenced no change in any effect size estimate greater in absolute value than 0.0005 across all effects of stress on memory phases, a change which was inconsequential for all analyses.

For all of the following analyses, a positive effect size indicates that stress *enhanced* memory relative to a control condition, whereas a negative effect size indicates that stress *impaired* memory relative to a control condition. In addition, because the outcome in these analyses is the standardized mean difference between groups (the effect size), a significant continuous moderator means that the effect size estimate depends upon levels of that continuous variable. In other words, if the coefficient for a continuous moderator is significant, it means that as the continuous variable increases or decreases, the effect of stress on memory relative to a control condition increases or decreases.

Results

Effects of Stress on Cortisol

Of the studies examining stress effects on memory, 78 studies included enough information to allow us to accurately derive the pretest–postest control group effect size for cortisol. This effect size allows us to best determine the stress-induced increase in cortisol relative to a control group. These cortisol analyses included 4,238 participants.

The overall effect of stress on increases in cortisol relative to a control group was strong and significant, $g_{ppc}^+ = 1.62$, $t(77) = 16.0$, $p < .001$, 95% CI$_g$ [1.42, 1.82] (see Figure 2). There was some heterogeneity in these effects, however, $\tau^2 = 0.53$, indicating that this effect likely differed as a function of moderators. To assess publication bias, we conducted Egger's test for funnel plot asymmetry (Egger, Davey Smith, Schneider, & Minder, 1997); interestingly, there was strong evidence for publication bias in these effects, $t(76) = 5.55$, $p < .001$, with estimates indicating that positive effects greater in magnitude were more likely to be published than effects weaker in magnitude. To address this concern, we conducted a trim and fill analysis (Duval, 2005; Duval & Tweedie, 2000a, 2000b) to estimate the number of missing studies and the correct effect size estimate. This analysis indicated that while the actual effect may be weaker than what was estimated, with 16 studies estimated to be missing ($SE = 5.81$) with effects less than the average reported effect, the estimated effect size including those estimated 16 studies was still strong and highly significant, $g^+ = 1.319$, $p < .001$. Thus, because our analysis focused on studies examining stress effects on memory, we took evidence for publication bias in stress effects on cortisol to imply that researchers often simply chose not to report the secondary analysis of stress effects on cortisol if it did not strengthen their papers, rather than a lack of a true effect.

We next examined potential moderators of stress effects on cortisol. As expected, age, $B = .036$, $t(73) = 2.73$, $p = .008$, percent male participants, $B = .010$, $t(76) = 4.41$, $p < .001$, and time of day, $B = .001$, $t(73) = 2.66$, $p = .010$, emerged as significant moderators of the effect of stress on cortisol, with stress effects on cortisol increasing as each of these variables increased. Interestingly, a significant quadratic effect emerged for time of day, $B_{\text{linear}} = -.002$, $B_{\text{quadratic}} < .001$, $t(72) = 2.60$, $p = .011$, with a relatively consistent effect of stress on cortisol before 1 p.m. that dramatically accelerated to large effects of stress on cortisol in the afternoon. We did not find any evidence for quadratic effects of age or percent male in moderating the effect of stress on cortisol, $ps > .242$. Although effects were all in the enhancing

direction, we did not find significant evidence that excluding participants who smoked regularly, took psychoactive medications, were currently sick, or had a BMI greater than 30 moderated the effects of stress on cortisol, $ps > .079$. In contrast, as expected, excluding women taking hormonal contraceptives, $B = .892$, $t(76) = 5.48$, $p < .001$, or excluding contraceptive-free women during their menstrual period, $B = 1.02$, $t(76) = 6.31$, $p < .001$, increased the effects of stress on cortisol. Thus, the relatively greater effect of stress on cortisol in men compared with women may be even greater when women taking hormonal contraceptives or during their menstrual period are included in analyses.

Finally, we examined stressor type as a moderator of stress effects on cortisol, as currently no meta-analysis has examined differences in cortisol responses to standardized laboratory stressors used in memory studies (Dickerson & Kemeny, 2004 examined cortisol responses to laboratory stressors but excluded the now common cold-pressor task). Thus, we examined how stress-induced cortisol increases in the Trier Social Stress Test (TSST), Cold-Pressor Task (CPT), Socially Evaluated Cold-Pressor Task (SECPT), Maastricht Acute Stress Test (MAST), and Fear Factor Stress Test (FFST) compared with each other. In our dataset for studies with complete cortisol data, 31 studies used the TSST, 19 used the CPT, 16 used the SECPT, 3 used the MAST, 2 used the FFST, and 7 used an unstandardized stressor. Because so few studies used the MAST and FFST, we do not present analyses of these stressors here. As expected, the TSST produced a greater cortisol increase ($g_{ppc}^+ = 1.931$, $p < .001$) than the CPT, $B = .830$, $t(72) = 3.94$, $p < .001$, and a marginally greater increase than the SECPT, $B = .532$, $t(72) = 1.98$, $p = .051$. The effect of the SECPT on cortisol ($g_{ppc}^+ = 1.399$, $p < .001$), however, did not significantly differ from the CPT ($g_{ppc}^+ = 1.101$, $p < .001$), $B = .299$, $t(72) = 1.28$, $p = .204$. Thus, the TSST reliably produced a larger increase in cortisol than did the SECPT or CPT, and no significant differences emerged between the SECPT and CPT.

Effects of Stress on Memory: Preliminary Analyses

Study characteristics. The final sample consisted of 113 studies—each of which is represented by *m*—assessing stress effects on memory in 6,216 participants. The Appendix presents a summary of each of these studies. There were 399 total effect sizes, each of which is represented by *k*. The number of effect sizes per study we obtained is relatively common in social science research (Scammacca et al., 2014) and is similar to the number of effect sizes per study seen in similar meta-analyses (Shields et al., 2015). Encoding stress effects were examined in 33 studies ($k = 131$) with 1,607 participants. Postencoding stress effects were examined in 23 studies ($k = 83$) with 1,668 participants. Retrieval stress effects were examined in 31 studies ($k = 102$) with 1,410 participants. Postencoding/retrieval stress effects were examined in 9 studies ($k = 15$) with 697 participants. Encoding/retrieval stress effects were examined in 16 studies ($k = 48$) with 1,148 participants. Finally, postreactivation stress effects were examined in 10 studies ($k = 20$) with 344 participants.

Assessment of publication bias. To assess publication bias, we conducted Egger's test for funnel plot asymmetry (Egger et al., 1997) in stress effects on each memory phase (see supplementary Figure 1). Egger's test returned nonsignificant for the overall study set, $t(111) = -0.20$, $p = .845$, postencoding, $t(21) = 1.05$, $p =$
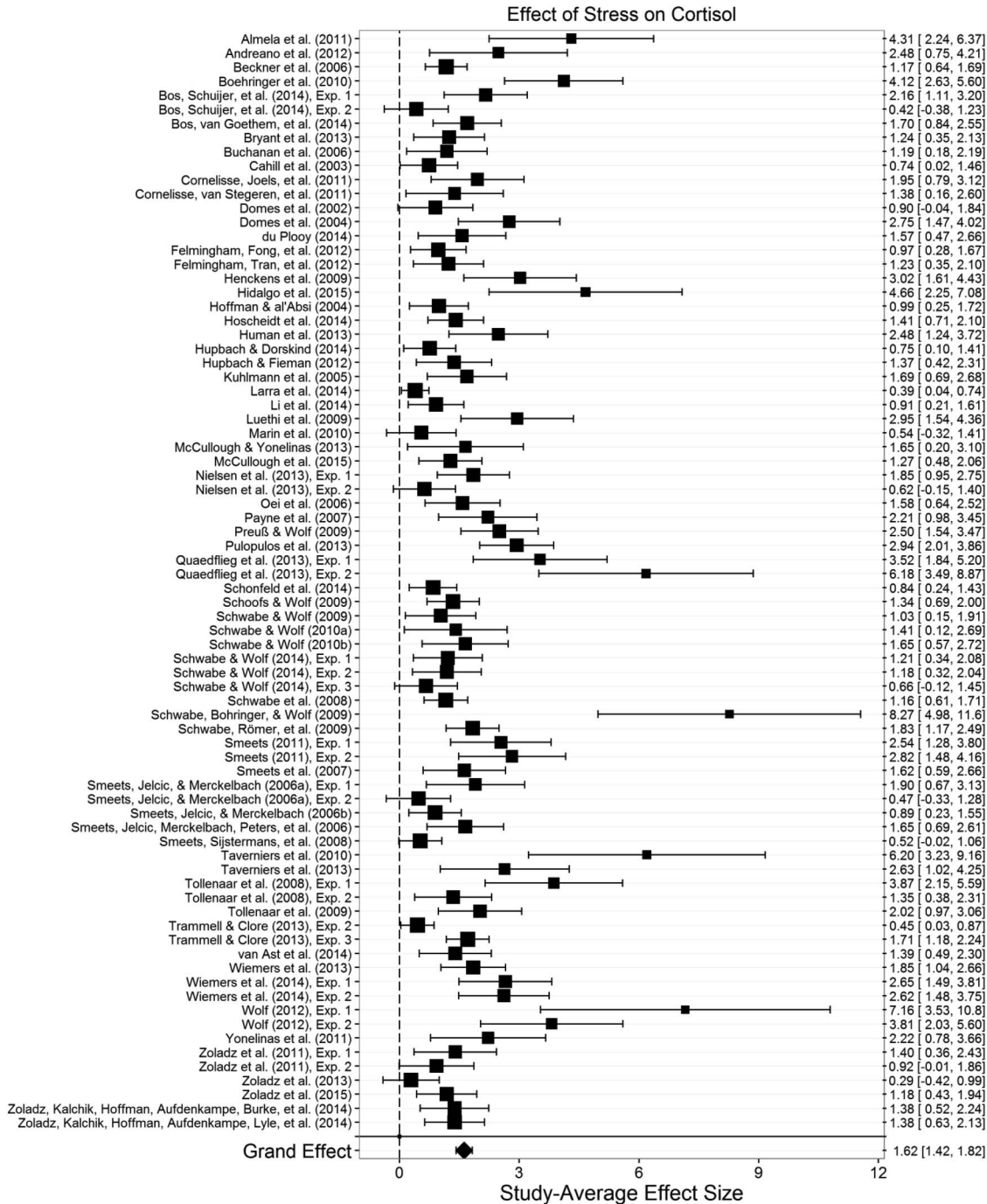
Effect of Stress on Cortisol

Study	Effect [95% CI]
Almela et al. (2011)	4.31 [2.24, 6.37]
Andreano et al. (2012)	2.48 [0.75, 4.21]
Beckner et al. (2006)	1.17 [0.64, 1.69]
Boehringer et al. (2010)	4.12 [2.63, 5.60]
Bos, Schuijer, et al. (2014), Exp. 1	2.16 [1.11, 3.20]
Bos, Schuijer, et al. (2014), Exp. 2	0.42 [-0.38, 1.23]
Bos, van Goethem, et al. (2014)	1.70 [0.84, 2.55]
Bryant et al. (2013)	1.24 [0.35, 2.13]
Buchanan et al. (2006)	1.19 [0.18, 2.19]
Cahill et al. (2003)	0.74 [0.02, 1.46]
Cornelisse, Joels, et al. (2011)	1.95 [0.79, 3.12]
Cornelisse, van Stegeren (2011)	1.38 [0.16, 2.60]
Domes et al. (2002)	0.90 [-0.04, 1.84]
Domes et al. (2004)	2.75 [1.47, 4.02]
du Plooy (2014)	1.57 [0.47, 2.66]
Felmingham, Fong, et al. (2012)	0.97 [0.28, 1.67]
Felmingham, Tran, et al. (2012)	1.23 [0.35, 2.10]
Henckens et al. (2009)	3.02 [1.61, 4.43]
Hidalgo et al. (2015)	4.66 [2.25, 7.08]
Hoffman & al'Absi (2004)	0.99 [0.25, 1.72]
Hoscheidt et al. (2014)	1.41 [0.71, 2.10]
Human et al. (2013)	2.48 [1.24, 3.72]
Hupbach & Dorskind (2014)	0.75 [0.10, 1.41]
Hupbach & Fieman (2012)	1.37 [0.42, 2.31]
Kuhlmann et al. (2005)	1.69 [0.69, 2.68]
Larra et al. (2014)	0.39 [0.04, 0.74]
Li et al. (2014)	0.91 [0.21, 1.61]
Luethi et al. (2009)	2.95 [1.54, 4.36]
Marin et al. (2010)	0.54 [-0.32, 1.41]
McCullough & Yonelinas (2013)	1.65 [0.20, 3.10]
McCullough et al. (2015)	1.27 [0.48, 2.06]
Nielsen et al. (2013), Exp. 1	1.85 [0.95, 2.75]
Nielsen et al. (2013), Exp. 2	0.62 [-0.15, 1.40]
Oei et al. (2006)	1.58 [0.64, 2.52]
Payne et al. (2007)	2.21 [0.98, 3.45]
Preuß & Wolf (2009)	2.50 [1.54, 3.47]
Pulopulos et al. (2013)	2.94 [2.01, 3.86]
Quaedflieg et al. (2013), Exp. 1	3.52 [1.84, 5.20]
Quaedflieg et al. (2013), Exp. 2	6.18 [3.49, 8.87]
Schonfeld et al. (2014)	0.84 [0.24, 1.43]
Schoofs & Wolf (2009)	1.34 [0.69, 2.00]
Schwabe & Wolf (2009)	1.03 [0.15, 1.91]
Schwabe & Wolf (2010a)	1.41 [0.12, 2.69]
Schwabe & Wolf (2010b)	1.65 [0.57, 2.72]
Schwabe & Wolf (2014), Exp. 1	1.21 [0.34, 2.08]
Schwabe & Wolf (2014), Exp. 2	1.18 [0.32, 2.04]
Schwabe & Wolf (2014), Exp. 3	0.66 [-0.12, 1.45]
Schwabe et al. (2008)	1.16 [0.61, 1.71]
Schwabe, Bohringer, & Wolf (2009)	8.27 [4.98, 11.6]
Schwabe, Römer, et al. (2009)	1.83 [1.17, 2.49]
Smeets (2011), Exp. 1	2.54 [1.28, 3.80]
Smeets (2011), Exp. 2	2.82 [1.48, 4.16]
Smeets et al. (2007)	1.62 [0.59, 2.66]
Smeets, Jelcic, & Merckelbach (2006a), Exp. 1	1.90 [0.67, 3.13]
Smeets, Jelcic, & Merckelbach (2006a), Exp. 2	0.47 [-0.33, 1.28]
Smeets, Jelcic, & Merckelbach (2006b)	0.89 [0.23, 1.55]
Smeets, Jelcic, Merckelbach, Peters, et al. (2006)	1.65 [0.69, 2.61]
Smeets, Sijstermans, et al. (2008)	0.52 [-0.02, 1.06]
Taverniers et al. (2010)	6.20 [3.23, 9.16]
Taverniers et al. (2013)	2.63 [1.02, 4.25]
Tollenaar et al. (2008), Exp. 1	3.87 [2.15, 5.59]
Tollenaar et al. (2008), Exp. 2	1.35 [0.38, 2.31]
Tollenaar et al. (2009)	2.02 [0.97, 3.06]
Trammell & Clore (2013), Exp. 2	0.45 [0.03, 0.87]
Trammell & Clore (2013), Exp. 3	1.71 [1.18, 2.24]
van Ast et al. (2014)	1.39 [0.49, 2.30]
Wiemers et al. (2013)	1.85 [1.04, 2.66]
Wiemers et al. (2014), Exp. 1	2.65 [1.49, 3.81]
Wiemers et al. (2014), Exp. 2	2.62 [1.48, 3.75]
Wolf (2012), Exp. 1	7.16 [3.53, 10.8]
Wolf (2012), Exp. 2	3.81 [2.03, 5.60]
Yonelinas et al. (2011)	2.22 [0.78, 3.66]
Zoladz et al. (2011), Exp. 1	1.40 [0.36, 2.43]
Zoladz et al. (2011), Exp. 2	0.92 [-0.01, 1.86]
Zoladz et al. (2013)	0.29 [-0.42, 0.99]
Zoladz et al. (2015)	1.18 [0.43, 1.94]
Zoladz, Kalchik, Hoffman, Aufdenkampe, Burke, et al. (2014)	1.38 [0.52, 2.24]
Zoladz, Kalchik, Hoffman, Aufdenkampe, Lyle, et al. (2014)	1.38 [0.63, 2.13]
Grand Effect	1.62 [1.42, 1.82]

Study-Average Effect Size

Figure 2. Effect of stress on cortisol. Size of the square indicates the relative weight assigned to that study in the analysis. Error bars represent the 95% confidence interval of the effect. This meta-analysis indicates that stress significantly increased cortisol from baseline to postmanipulation relative to a control condition.

.307, postencoding/retrieval, $t(7) = 0.31$, $p = .765$, encoding/retrieval, $t(14) = -0.22$, $p = .827$, and postreactivation, $t(8) = 0.79$, $p = .450$ indicating a lack of evidence for publication bias in these effects. There was, however, evidence for publication bias in stress effects on encoding, $t(31) = -2.46$, $p = .020$, and in stress effects on retrieval, $t(29) = -3.27$, $p = .003$. Estimates indicate

that impairing effects of stress on both encoding and retrieval were published disproportionately more than null or enhancing effects of stress.

The significant evidence for publication bias in stress effects on retrieval prompts a concern that if more null effects of stress on retrieval would have been published these studies might have

reduced the effect to a trivial or negligible size. To examine this, we conducted trim and fill analyses (Duval, 2005; Duval & Tweedie, 2000a, 2000b) for stress effects on encoding and retrieval. The trim and fill analysis for stress effects on encoding did not estimate any missing studies (estimated missing = 0; SE = 3.49), indicating that Egger's test for publication bias may have overestimated publication bias for stress effects on encoding. The trim and fill analysis for stress effects on retrieval estimated that one unpublished study was missing from analyses of stress effects on retrieval (estimated missing = 1; SE = 3.51). Although the actual effect may be weaker than what was estimated, the estimated effect of stress on retrieval including the estimated one missing study was still significant, p = .002. Thus, despite some evidence for publication bias, the trim and fill analyses indicate that we can be confident that the effects of stress on encoding and retrieval are true effects. Moreover, the lack of evidence for publication bias in stress effects on most memory phases provides confidence that the observed effects of stress on memory processes are true effects.

Achieved power analysis. To ensure that we had appropriate power to detect effects, we conducted power analyses for our random effects meta-analyses (Valentine, Pigott, & Rothstein, 2010).[9] As shown in Table 2, our analyses were extremely well powered, with almost all analyses obtaining approximately .90 power to detect even small effects (i.e., $|g|$ = .20) and all analyses obtaining .90 power or greater to detect medium effects (i.e., $|g|$ = .50). Thus, nearly all of our analyses had sufficient power to detect even subtle effects of stress on memory.

Primary Analyses

Encoding. The overall effect of stress on encoding[10] (m = 33, k = 131, N = 1,607) was not significant, g^+ = −.109, $t(31.5)$ = −1.28, p = .211, 95% CI_g [−.282, .065] (see Figure 3). There was, however, some heterogeneity in these effects, τ^2 = 0.21, indicating that this null effect likely differed as a function of moderators. Thus, we explored the effects of moderators expected a priori to play an important role in the effects of stress on encoding. In the interest of assisting future researchers with study design, the effects of all potential moderators of stress effects on encoding are displayed in Table 3.

We first examined whether the delay between stress onset and encoding (hereafter stress-encoding delay) moderated the effect of

stress on encoding, given a strong theoretical reason to expect this effect (Schwabe et al., 2012). As hypothesized, the stress-encoding delay moderated the effects of stress on encoding, B = −.0167, $t(7.5)$ = −3.06, p = .017 (see Figure 4). This slope estimate represents the effect of each minute of the stress-encoding delay, and the intercept represents the effect of stress on encoding when there is no stress-encoding delay. This analysis thus indicates that with no delay between stress onset and the encoding task, stress nonsignificantly enhances encoding, g^+ = .186, p = .143. As the delay increases, however, the effect of stress on encoding becomes progressively more negative, and at just over 11 min poststressor onset, the effect of stress on encoding begins to impair memory, rather than enhance it. Further, the effect of stress on encoding becomes a significant impairing effect with a stress-encoding delay of approximately 22 min.

We next examined whether the relevance of encoding material to the stressor (i.e., integral or nonintegral) moderated the effect of stress on encoding, given prior work suggesting that stress at encoding may enhance, rather than impair, memory when the stimuli are relevant to the stressor (e.g., Wiemers, Sauvage, Schoofs, Hamacher-Dang, & Wolf, 2013). As expected, the relevance of the encoded material to the stressor moderated the effect of stress on encoding, $t(6.5)$ = 2.68, p = .034 (see Figure 4). When the materials encoded were integral to the stressor, stress tended to enhance encoding, g^+ = .334, $t(4.8)$ = 1.89, p = .119, but when the materials encoded were nonintegral (i.e., not relevant) to the stressor, stress tended to impair encoding, g^+ = −.180, $t(28.3)$ = −2.02, p = .053.

By examining our data, however, we noted that it is very difficult to disentangle the effects of item relevance from the stress-encoding delay (see Figure 5). That is, an examination of Figure 5 shows that almost all studies that included stressor-relevant items included only very short delays between stress and encoding. Thus, at a study-average level, it is unclear whether the beneficial effects of stress that were observed were attributable to a short stress-encoding delay or to the relevance of the items to the stressor. However, although we graphically depict everything at the study-average level for ease of visual interpretation, our statistics operate at the level of individual effects and suggest that both the stress-encoding delay and the relevance of the learned items to the stressor independently moderate stress effects on encoding. We return to this issue below when discussing how to maximize stress effects on encoding.

We also examined whether the time of day the study was conducted moderated the effect of stress on encoding, given previous work suggesting that the effect of cortisol administration on memory encoding depended upon the time of day the study began

Table 2
Power Analyses Describing Achieved Power to Detect Effects of Stress on Different Phases of Memory in a Two-Tailed Test, Rounded to Two Decimals

	Achieved power to detect a		
Effect of stress on	Small effect (i.e., $|g|$ = .20)	Medium effect (i.e., $|g|$ = .50)	Large effect (i.e., $|g|$ = .80)
Encoding	.95	1	1
Postencoding	.95	1	1
Retrieval	.93	1	1
Postencoding/retrieval	.70	1	1
Encoding/retrieval	.85	1	1
Reactivation	.26	.90	1

---

[9] We used the average sample size for the stress and control groups as the "typical" sample size per group as well as the observed heterogeneity (τ^2) to demonstrate the actual power of our analyses.

[10] In the main analyses of 'encoding' we included studies in which the stressor occurred during encoding as well as studies in which stress occurred just prior to encoding. The difference between these studies was assessed by examining the effect of the time delay between stress and encoding. In addition, a secondary analysis was conducted in which we analyzed these two types of studiers separately (see supplementary material). Those results were found to be consistent with the main analysis, with the exception that the significance levels of the effects were generally reduced given the reduced samples.
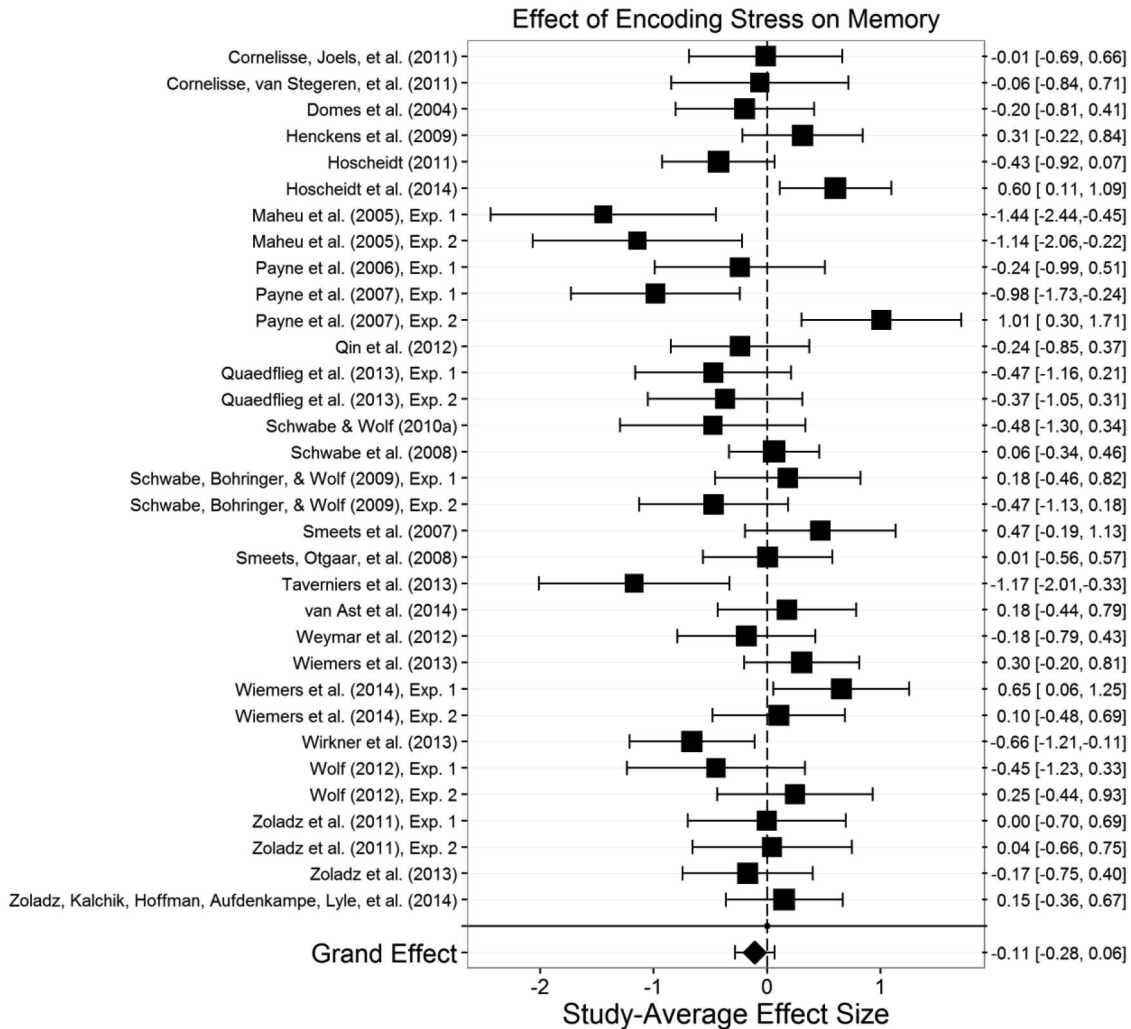
Figure 3. Effect of stress on encoding. Size of the square indicates the relative weight assigned to that study in the analysis. Error bars represent the 95% confidence interval of the effect. Points to the left of zero indicate a study-average impairment of encoding, and points to the right of zero indicate a study-average enhancement of encoding. This meta-analysis indicated that stress did not significantly influence encoding across all studies and paradigms.

(Het et al., 2005). Surprisingly, time of day did not moderate stress effects on encoding, either by a linear, $B < -.001$, $t(5.6) = -0.32$, $p = .762$, or quadratic function, $B_{quadratic} < -.001$, $t(2.4) = -2.41$, $p = .117$. Coding time of day as a categorical variable (i.e., morning and afternoon) rather than as a continuous variable did not alter these results ($p = .652$).

Additionally, we examined whether the stress-induced increase in cortisol moderated the effect of stress on encoding (cf. Het et al., 2005). Contrary to our expectations, the stress-induced cortisol increase did not moderate stress effects on encoding, $B = -.022$, $t(4.6) = -0.92$, $p = .404$. Because Het et al. found that the effects of cortisol depended upon the time of day that cortisol was administered, we controlled for time of day as well as examined a potential interaction with time of day. Neither of these changes revealed any association between stress-induced cortisol increases and the effect of stress on encoding. There was marginal evidence

for an inverted-U moderating effect of stress-induced cortisol increases on stress effects on encoding, $B_{linear} = .066$, $B_{quadratic} = -.004$, $t(3.6) = -2.72$, $p = .059$, with small and large stress-induced cortisol increases tending to impair encoding more than moderate increases. However, closer examination of these data revealed this effect was driven by an outlier, as reflected in the low *df*—because *df* are sensitive to variability, they are less than four in this case, and because the *df* for the quadratic effect are less than 4, there is a twofold increase in the likelihood of making a Type I error. The quadratic effect was no longer close to significance after removing this single outlier ($p = .443$).

We further examined whether the use of an immediate recall task moderated the effect of stress on encoding (cf. Wolf, 2012). Surprisingly, the use of an immediate recall task did not moderate the effects of stress at encoding, $t(11.6) = -0.29$, $p = .778$. Because Wolf (2012) found evidence for this effect in recall with

Table 3

Potential Moderators of Encoding Stress Effects on Memory

Moderator	B	df	p
Incidental or intentional encoding	.094	22.9	.570
Exclusion of smokers	.166	23.5	.318
Exclusion of women during menstrual period	−.141	29.5	.422
Exclusion hormonal contraceptives usage	−.144	22.9	.453
Exclusion of all illnesses	−.022	27.4	.902
Exclusion of all psychoactive medication	.052	14.1	.804
Exclusion of BMI greater than 30	−.077	17.1	.676
Use of an immediate recall task postencoding	−.056	11.6	.778
Stressor relevance of items (integral/nonintegral)	**.514**	**6.5**	**.034**
Context change between stress and learning	.055	26.1	.727
Context change between learning and retrieval	.056	11.2	.806
Stress manipulated between or within groups	NA		
Item valence (compared with neutral)			
Negative	.040	26.7	.840
Positive	.008	13.0	.957
Study material type			
Pictures	−.063	19.8	.646
Words	.119	18.6	.382
Narrative/slideshow	−.217	8.6	.430
Autobiographical	NA		
Other	.156	3.9	.595
Sensory modality of study material presentation			
Verbal	.108	2.4	.601
Visual	.015	6.6	.908
Verbal + Visual	−.123	7.7	.418
Memory task type			
Free recall	−.196	22.3	.078
Cued recall	.140	6.8	.219
Recognition	.056	21.6	.554
Stressor type			
Socio-evaluative	.087	7.2	.580
Pain	.195	1.3	.320
Hybrid (socio-evaluative & pain)	−.108	8.4	.417
Other	−.174	2.7	.614
Participant age	−.032	10.8	.391
Percent male participants	−.004	20.1	.061
Time of day study began	<−.001	5.6	.762
Study item list length	<.001	5.1	.718
Number of novel items in a recognition task	<.001	1.5	.999
Delay (hours) between item encoding and retrieval	−.002	4.1	.093
Delay (min) between stress onset and encoding	**−.017**	**7.5**	**.017**
Stressor duration (min)	−.002	4.6	.836
Stress-induced Δ-cortisol (nmol/L)	−.022	4.5	.404
Length of encoding phase (min)	.033	8.5	.182
Participant homogeneity	−.013	15.2	.828

Note. Significant ($p < .05$) moderators are shown in boldface font. B represents the change in the effect size for every one-unit change in the moderator. For dummy-coded categorical variables, B represents the difference between estimated effects for each group; for contrast-coded categorical variables, B represents the difference between the group in question and the average estimated effect. If $df < 4$, there is a twofold greater risk of making a Type I error. The listed p value represents the significance of the moderator in question. When there were not enough studies to estimate an effect, NA is listed in the column for B.

preencoding stress, we next restricted the analysis to studies in which stress occurred prior to encoding (rather than including stress-during-encoding studies in the analysis) and focused on only studies with recall as the retrieval task. However, even in this restricted analysis, the use of an immediate recall task did not moderate the effect of stress on encoding, $t(9.8) = 0.43$, $p = .679$.

We examined potential effects of age, gender, item valence, stressor type, delay between encoding and retrieval, and mem-

ory task type across all stress effects on encoding because of the literature's consistent consideration of these variables as methodologically relevant. Of these potential moderators, none emerged as significant moderators of stress effects on encoding ($ps > .05$).

Because of the differential effects of stress on encoding as a function of the stress-encoding delay, we examined whether any other potential moderators altered the relation between the stress-encoding delay and the effect of stress on memory. In these analyses, we found that as the percent of male participants increased, the effect of the stress-encoding delay increased in magnitude, $t(9.3) = −2.35$, $p = .043$ (i.e., the effect of stress on encoding become more negative as the delay between stress and encoding became longer). This result was also obtained by contrasting studies that excluded women taking hormonal contraceptives ($p = .035$; see supplementary Figure 2) or studies that excluded women during their menstrual period ($p = .017$) with studies that included those participants. Moreover, hybrid pain/socioevaluative stressors moderated the relation between the stress-encoding delay and stress effects on encoding, $t(9.3) = 3.57$, $p = .006$—though we should note that there were not enough studies using a pain-based stressor without a social-evaluative component to examine the effects of pain-only stressors on the relation between the stress-encoding delay and stress effects on encoding. If the stressor was a hybrid (pain and social evaluation) stressor such as the SECPT, stress during encoding did not produce an encoding enhancement at short delays but produced a general impairment ($g^+ = −.341$), $t(6.3) = −3.49$, $p = .012$—unlike a nonhybrid stressor, which enhanced encoding at no delay ($g^+ = .383$, $t(7.5) = 4.25$, $p = .003$). In addition, for nonhybrid stressors, each minute increase in the stress-encoding delay significantly alters the estimated effect of stress on encoding by $B = −.026$, $p = .007$. However, if the stressor was a hybrid stressor, the stress-encoding delay was not a significant moderator of the effects of stress on encoding, $B = .006$, $p = .437$, and the difference between the slopes for hybrid and nonhybrid stressors was significant ($p = .006$).

For the benefit of future research, we will attempt to highlight the conditions necessary to produce the biggest stress effects on encoding. We used a forward stepwise regression approach to determine all simultaneously significant moderators ($p < .05$, one-tailed) in one model, including the largest effect at each step. In this model, we found that the significant moderators were the stress-encoding delay by stressor type interaction and the relevance of studied items to the stressor.

Considering all simultaneously significant moderators controllable by the experimenter in one model, the biggest reliably obtained enhancing effect of stress on encoding should be obtained when encoding happens during a nonhybrid stressor and the items encoded are relevant to the stressor. The estimated effect size with these moderators at their specified conditions is moderate and significant, $g^+ = 0.592$, $t(4.0) = 4.60$, $p = .010$, 95% CI_g [0.233, 0.951]. A sample size of 88 (44 stress, 44 control) is necessary to achieve 80% power to detect this effect in a two-tailed test. Similarly, the largest reliable impairing effect of stress on encoding will be obtained by manipulating those same variables within the ranges observed
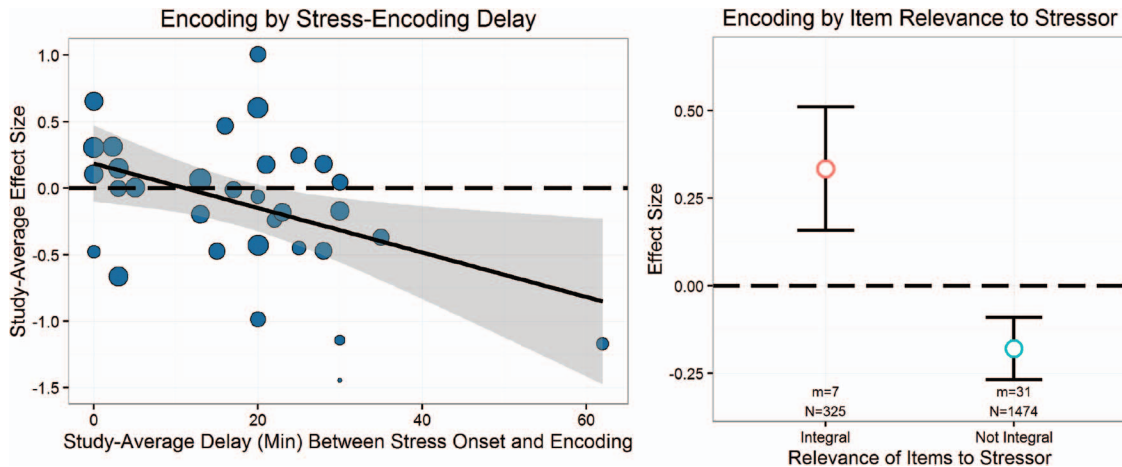
Figure 4. Significant moderators of stress effects on encoding. Size of circles in the continuous plot indicates the relative weight given to that study in the analysis. Effects of stress on encoding were moderated by the stress-encoding delay as well as the relevance of the learned items to the stressor. See the online article for the color version of this figure.

in our study.[11] That is, using a nonhybrid stressor, a stress-encoding delay of approximately 35 min, and studied items that were unrelated to the stressor, the estimated stress-induced impairment in encoding is moderate and significant, $g^+ = -0.473$, $t(4.0) = -2.82$, $p = .048$, 95% CI_g $[-0.941, -0.005]$. A sample size of 134 (67 stress, 67 control) is necessary to achieve 80% power to detect this effect in a two-tailed test.

In sum, stress prior to encoding tended to decrease memory unless the delay between stress and encoding was very short or if the materials were stressor-relevant, in which case stress appeared to improve performance. One exception to this pattern was seen with the hybrid socioevaluative/pain stressor—with these stressor paradigms, stress impaired encoding even at a short delay. In addition, the effect of the stress-encoding delay was reduced in magnitude in studies that included females taking hormonal contraceptives or females that were tested during their menstrual period.

Postencoding. The overall effect of postencoding stress on memory ($m = 23$, $k = 83$, $N = 1,668$) was significant, $g^+ = .206$, $t(21.5) = 2.22$, $p = .037$, 95% CI_g $[.013, .399]$ (see Figure 6), such that postencoding stress generally enhanced memory. There was some heterogeneity in these effects, $\tau^2 = 0.26$, indicating that this enhancing effect likely differed as a function of moderators. The effects of each potential moderator on postencoding stress effects are displayed in Table 4.

We first examined whether participant sex would moderate postencoding stress effects, given previous reports of sex differences in postencoding stress effects (e.g., McCullough & Yonelinas, 2013). Although the percentage of male participants did not moderate postencoding stress effects, $B = -.001$, $t(10.6) = -0.48$, $p = .639$, excluding hormonal contraceptive use did moderate postencoding stress effects on memory, $t(20.6) = 2.58$, $p = .018$ (Figure 7; see also supplementary Figure 3 for more detail). Studies that excluded hormonal contraceptive use showed a significant memory enhancing effect of postencoding stress, $g^+ = .444$, $t(11.1) = 3.34$, $p = .007$, whereas studies that did not exclude hormonal contraceptive use did not show a significant

memory enhancing effect of postencoding stress, $g^+ = -.021$, $t(10.1) = -0.18$, $p = .863$. These effects did not extend to whether the study excluded women during their menstrual period, $p = .541$, or—as noted above—to the percentage of male participants in the study. Thus, it appears hormonal contraceptive use in females is a critical factor that negates the memory-enhancing effects of postencoding stress.

We then examined whether the valence of items moderated effects of postencoding stress on memory, given prior studies suggesting that postencoding stress enhances memory for negatively valenced information to a greater extent than neutral information (e.g., Cahill, Gorski, & Le, 2003). Surprisingly, although we found that negatively valenced materials predicted a slightly greater effect of postencoding stress than neutral materials, $B = .089$, this difference was not significant, $t(17.4) = 0.51$, $p = .618$. Similarly, stress effects on memory for positively valenced items did not differ from neutral items, $t(3.8) = -0.57$, $p = .601$. Thus, postencoding stress enhanced memory regardless of emotional valence.

We next examined whether a context change between encoding and the stressor would moderate postencoding stress effects (cf. Trammell & Clore, 2014). As expected, a change of context from encoding to the stressor moderated postencoding stress effects, $t(11.0) = -3.95$, $p = .002$ (see Figure 7). If the context between learning and stress remained constant, the memory-enhancing effect of postencoding stress was significant, $g^+ = .380$, $t(14.6) = 4.04$, $p = .001$, whereas with a change of context, the effect of postencoding stress on memory was nonsignificant and tended toward an impairment, $g^+ = -.196$, $t(5.6) = -1.76$, $p = .131$. Thus, the current results indicate that a change of context between

---

[11] The effects of a hybrid stressor on encoding do not differ as a function of any moderator. Thus, the use of a nonhybrid can produce a reliably bigger impairment in encoding with a long stress-encoding delay.
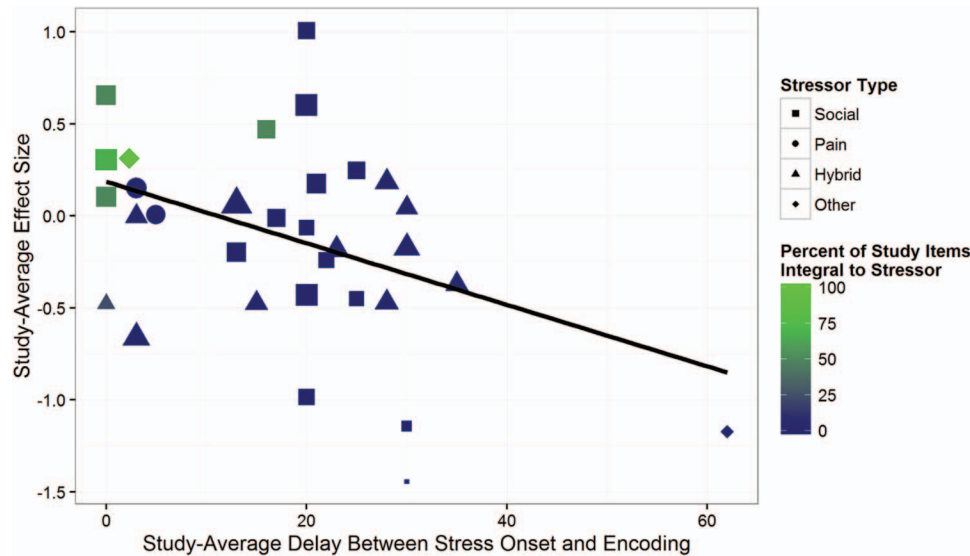
Figure 5. Depiction of the interrelations of stressor type, item relevance, and stress-encoding delay. Size of the dots indicates the relative weight given to each study in the analysis. This graph illustrates that at a study-average level it is difficult to disentangle item relevance to the stressor from delay, especially once stressor type is also taken into account. See the online article for the color version of this figure.

encoding and stress onset can dramatically reduce—and may even reverse—the memory-enhancing effects of postencoding stress.[12]

Additionally, we examined whether the stress-induced increase in cortisol was related to the effects of postencoding stress on memory. Contrary to our expectations, the stress-induced cortisol increase did not moderate postencoding stress effects in a linear, $B = -.005$, $t(3.4) = -0.20$, $p = .855$ or quadratic function, $B_{linear} = -.086$, $B_{quadratic} = .006$, $t(5.2) = 1.51$, $p = .189$.

We also examined potential effects of age, stressor type, memory task type, and time of day the study began because of the literature's consistent consideration of these variables as methodologically relevant. Of these moderators, only time of day emerged as a significant moderator of postencoding stress effects, $B = .001$, $t(4.0) = 4.18$, $p = .014$ (all other $ps > .088$). These results indicate that the memory-enhancing effect of postencoding stress increases as the time of day the study begins is later, and there was no evidence for a quadratic effect of time of day, $B_{linear} = .001$, $B_{quadratic} < .001$, $t(3.2) = 0.48$, $p = .663$—although it should be noted the latest any study started analysis was 3 p.m. (see Figure 7). Thus, postencoding stress enhances memory more in the afternoon than in the morning.

For the benefit of future research, we will attempt to highlight the conditions necessary to produce the biggest postencoding stress effect. We used a forward stepwise regression approach to determine all simultaneously significant moderators ($p < .05$, one-tailed) in one model, including the largest effect at each step. Considering all moderators simultaneously significant together, our analyses indicate that the biggest reliably obtained effect of postencoding stress would be obtained if a study began at 1 p.m., excluded women taking hormonal contraceptives, and kept the context constant (i.e., no change of rooms, odors, etc.) between encoding and the stressor. The estimated effect size with these conditions is strong and significant, $g^+ = 0.689$, $t(9.5) = 4.85$,

$p < .001$, 95% CI_g [0.370, 1.009]. A sample size of 70 (35 stress, 35 control) is necessary to achieve 80% power to detect this effect in a two-tailed test. Thus, to obtain a stress-induced enhancement of postencoding processes, future researchers should begin a study in the afternoon, exclude females using hormonal contraceptives, and keep the context constant between the learning phase and the stressor.

In sum, postencoding stress generally enhances memory. The enhancing effect of postencoding stress is stronger if the stressor and encoding task occur later in the day, and if analyses are restricted to only men or to women not using hormonal contraceptives. The enhancing effect of postencoding stress can be

---

[12] Even though the delay between learning and the stressor did not moderate the effects of postencoding stress on memory (see Table 4), it could be possible that changing contexts reduced the effects of stress on memory because it introduced a longer delay between study and the stressor. However, when we controlled for the encoding-stressor delay, context change remained a significant moderator of postencoding stress effects on memory, $B = -.635$, $t(6.2) = -4.29$, $p = .005$, whereas the encoding-stressor delay was not a significant moderator, $B = .006$, $t(2.6) = 1.56$, $p = .229$, indicating that the context effects were not due to increased delays. One other potential concern was that because the reported experimental methods often did not explicitly mention whether the rooms were change between the encoding phase and the stressor we had to infer whether such changes occurred (i.e., the standard Trier Social Stress Test requires a room change, but this was often not stated explicitly in the methods sections). To address this, we conducted a secondary analysis that included only studies that explicitly stated that either they changed contexts (4 studies) between learning and stress or they did not (7 studies)—11 studies in total. Context change was a significant moderator of postencoding stress effects, $t(5.4) = -4.92$, $p = .004$, such that studies with a constant context between encoding and stress significantly enhanced memory ($g^+ = .529$, $p = .018$), whereas studies with a changed context between encoding and stress did not, $g^+ = -.217$, $p = .107$).
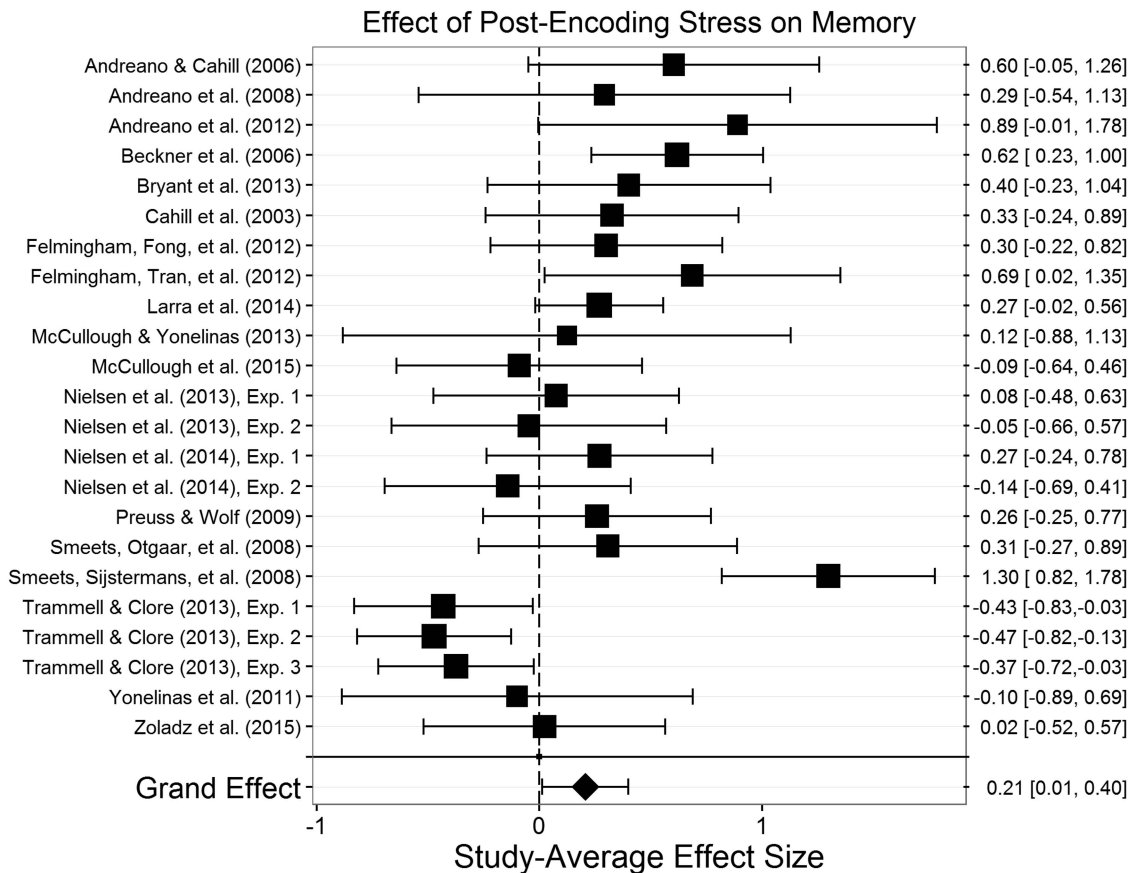
Effect of Post-Encoding Stress on Memory

Figure 6. Effect of postencoding stress on memory. Size of the square indicates the relative weight assigned to that study in the analysis. Error bars represent the 95% confidence interval of the effect. Points to the left of zero indicate a study-average impairment in memory, and points to the right of zero indicate a study-average enhancement in memory. This meta-analysis indicated that postencoding stress significantly enhanced memory across all studies and paradigms.

significantly reduced, and was effectively eliminated, with a change in context between encoding and the stressor.

Retrieval. The overall effect of stress on retrieval ($m = 31$, $k = 102$, $N = 1,410$) was significant, $g^+ = -.215$, $t(28.8) = -3.39$, $p = .002$, 95% CI_g [$-.346$, $-.085$] (see Figure 8), such that stress impaired memory retrieval. There was low heterogeneity across these effects, $\tau^2 = 0.11$, indicating that the impairing effect of stress on retrieval are relatively consistent across various conditions. Nonetheless, we explored the effects of moderators expected a priori to play an important role in the effects of stress on retrieval. The effects of all potential moderators on stress effects on retrieval are displayed in Table 5.

We first examined whether item valence moderated the effects of stress on retrieval, given previous experimental evidence (Kuhlmann et al., 2005). As expected, stress impaired retrieval of negatively valenced items ($g^+ = -.303$, $df = 20.5$, $p = .005$) to a marginally greater degree than neutral items ($g^+ = -.136$, $df = 21.3$, $p = .0499$), $t(24.8) = -1.91$, $p = .068$ (see Figure 9). Similarly, retrieval stress effects on positive items ($g^+ = -.385$, $df = 5.7$, $p < .001$) were significantly more impairing than neutral items, $t(7.1) = -2.87$, $p = .024$ (see Figure 9). Thus, stress

impaired retrieval of emotionally valenced items more than neutral items.

We next examined whether the delay between stress onset and retrieval (i.e., the stress-retrieval delay) moderated the effect of stress on retrieval, given empirical evidence suggesting this delay as a moderator (Schwabe & Wolf, 2014). Unexpectedly, the stress-retrieval delay did not moderate the effects of stress on retrieval, $B = -.003$, $t(3.5) = -0.50$, $p = .646$. Coding this analysis as the delay between stress offset—rather than onset—and retrieval (cf. Schwabe & Wolf, 2014) did not alter this result; that is, the stress offset to retrieval delay did not moderate effects of stress on retrieval either, $p = .311$.

We also examined whether the stress-induced cortisol increase moderated the effects of stress on retrieval. Contrary to our expectations, the stress-induced cortisol increase did not moderate the effects of stress on retrieval, $B = -.001$, $t(6.4) = -0.04$, $p = .968$, nor was there any evidence for a quadratic moderating effect, $B_{\text{linear}} = -.047$, $B_{\text{quadratic}} = .003$, $t(6.0) = 0.98$, $p = .363$. Controlling for all variables—individually or concurrently—that influenced the cortisol response to stress in our data (see section 4.1) did not alter the lack of effect of stress-induced cortisol increases on stress effects on retrieval.

Table 4

Potential Moderators of Postencoding Stress Effects on Memory

Moderator	B	df	p
Incidental or intentional encoding	.281	4.3	.241
Exclusion of smokers	−.107	2.6	.528
Exclusion of women during menstrual period	.131	15.3	.541
Exclusion hormonal contraceptives usage	**.464**	**20.7**	**.018**
Exclusion of all illnesses	.297	1.2	.320
Exclusion of all psychoactive medication	.361	11.7	.105
Exclusion of BMI greater than 30	.088	1.2	.533
Use of an immediate recall task postencoding	.078	7.9	.672
Context change between learning and stress	**−.576**	**11.0**	**.002**
Context change between learning and retrieval	NA		
Stress manipulated between or within groups	NA		
Item valence (compared with neutral)			
Negative	.089	17.4	.618
Positive	−.141	3.8	.601
Study material type			
Pictures	−.209	14.5	.153
Words	−.333	2.9	.191
Narrative/Slideshow	.100	7.9	.554
Autobiographical	NA		
Other	.442	2.5	.241
Sensory modality of study material presentation			
Verbal	.297	2.8	.083
Visual	−.119	8.2	.276
Verbal + Visual	−.179	5.1	.110
Memory task type			
Free recall	−.098	6.1	.544
Cued recall	NA		
Recognition	.098	6.1	.544
Stressor type			
Socio-evaluative	.003	1.2	.985
Pain	−.063	1.9	.694
Hybrid (socio-evaluative & pain)	NA		
Other	.060	1.1	.841
Participant age	.040	2.1	.088
Percent male participants	−.001	10.6	.639
Time of day study began	**.001**	**4.0**	**.014**
Study item list length	−.002	1.6	.194
Number of novel items in a recognition task	.001	3.0	.800
Delay (hours) between item encoding and retrieval	<.001	1.1	.863
Delay (min) between encoding and stress onset	−.007	1.9	.299
Stressor duration (min)	−.011	2.1	.479
Stress-induced Δ-cortisol (nmol/L)	−.005	3.4	.855
Length of encoding phase (min)	−.016	1.6	.387
Participant homogeneity	.142	6.7	.083

Note. Significant ($p < .05$) moderators are shown in boldface font. B represents the change in the effect size for every one-unit change in the moderator. For dummy-coded categorical variables, B represents the difference between estimated effects for each group; for contrast-coded categorical variables, B represents the difference between the group in question and the average estimated effect. If $df < 4$, there is a twofold greater risk of making a Type I error. The listed p value represents the significance of the moderator in question. When there were not enough studies to estimate an effect, NA is listed in the column for B.

We examined potential effects of age, sex, exclusion of hormonal contraceptives, exclusion of women during their menstrual period, stressor type, memory task, the delay between encoding and retrieval, and time of day the study began, because of the literature's consistent consideration of these variables as methodologically relevant. We did not have enough studies with a different context at encoding and retrieval to examine potential moderating effects of this context change on stress effects on retrieval. Of these variables, only exclusion of contraceptives emerged as a

moderator of stress effects on retrieval (all other $ps > .185$), $t(8.1) = -3.44$, $p = .009$ (Figure 9; see supplementary Figure 4 for more detail). If a study examining stress effects on retrieval did not exclude women taking hormonal contraceptives, the effect of stress on retrieval was negligible, $g^+ = .101$, $t(5.2) = 1.15$, $p = .302$. If, however, a study examining stress effects on retrieval excluded women taking hormonal contraceptives, the impairing effect of stress on retrieval was significant, $g^+ = -.294$, $t(23.2) = -4.14$, $p < .001$.

To identify the conditions necessary to produce the biggest stress effect on retrieval we used a forward stepwise regression to determine all simultaneously significant moderators ($p < .05$, one-tailed). Considering all simultaneously significant moderators together, we found that the biggest reliably obtained effect of stress on retrieval would occur if a study examined effects on positively- or negatively valenced items and excluded women taking hormonal contraceptives. The estimated effect size with these conditions is moderate and significant, $g^+ = -0.388$, $t(19.7) = -4.51$, $p < .001$, 95% CI_g [−0.567, −0.208]. A sample size of 202 (101 stress, 101 control) is necessary to achieve 80% power to detect this effect in a two-tailed test. The effect of stress on retrieval of neutral items with these conditions ($g^+ = -0.229$) is significantly smaller $t(25.0) = 2.24$, $p = .034$. We should also note that all studies of stress effects on retrieval with negative items included a mix of neutral items in the study list, and all studies with positive items included a mix of negative and neutral items in the study list. As such, our data cannot address whether valence itself moderates stress effects on retrieval, or whether emotional valence coupled with neutral items moderates stress effects on retrieval. Thus, to obtain a stress-induced impairment on retrieval, future researchers should use a study list with a mixture of emotional and neutral items, examine effects on emotionally valenced items, and exclude women taking hormonal contraceptives.

In sum, stress prior to retrieval generally led to a decrease in memory. These effects were larger for negative and positive materials than neutral materials, and these effects were reduced in magnitude if women who were taking contraceptives were included in the study.

Postencoding/retrieval. We considered stress to affect both postencoding and retrieval processes (i.e., postencoding/retrieval) if stress onset occurred within 60 min postencoding and stress offset occurred within 90 min of retrieval. For all studies fitting these criteria, stress onset occurred within 20 min of encoding, and retrieval occurred within 35 min of stress offset. The overall effect of stress on postencoding/retrieval ($m = 9$, $k = 15$, $N = 697$) was not significant, $g^+ = .004$, $t(7.7) = 0.03$, $p = .974$, 95% CI_g [−.279, .287] (see Figure 10). This null effect is not surprising, given the prior analyses revealing that postencoding stress generally enhances memory whereas retrieval stress impairs memory. There was low heterogeneity in these effects, $\tau^2 = 0.09$, indicating that this nonsignificant effect likely did not differ as a function of moderators. Nonetheless, we explored the effects of moderators expected a priori to play an important role in postencoding or retrieval stress effects as well as potential methodologically relevant moderators. In the interest of assisting future researchers with study design, the effects of all potential moderators of stress effects on postencoding/retrieval are displayed in Table 6.

We examined whether the delay between stress onset and retrieval, sex (including hormonal contraceptive use), age, memory
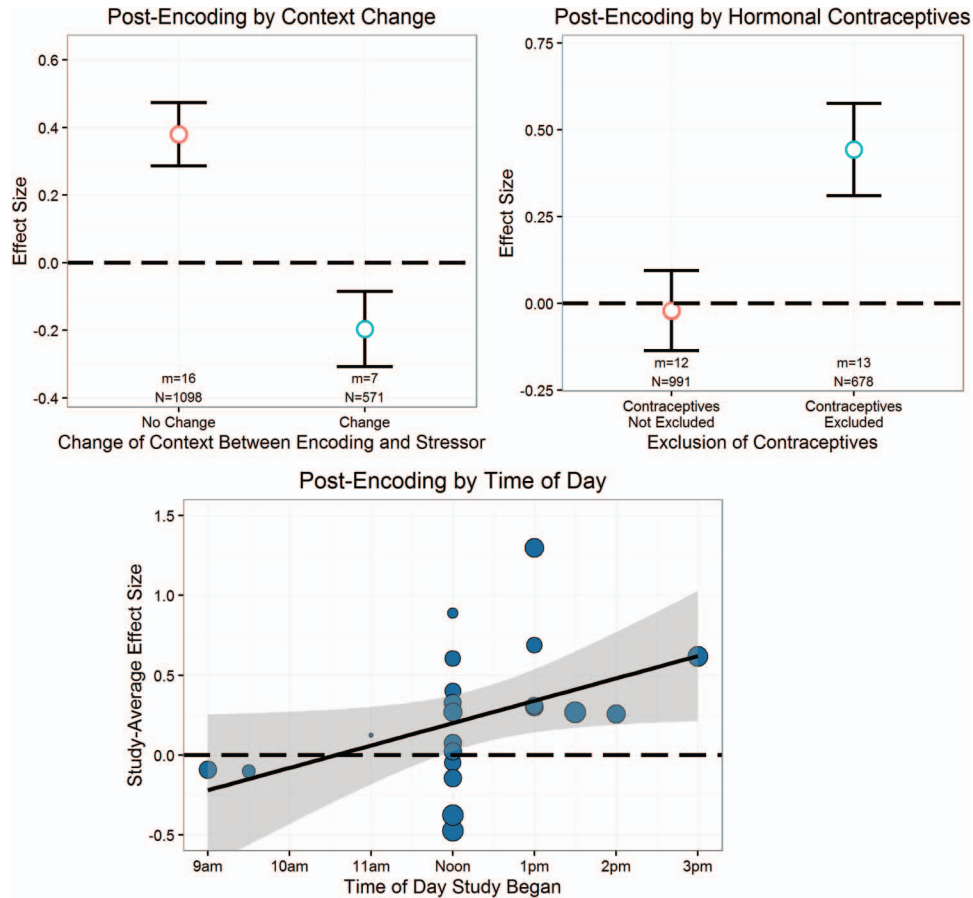
Figure 7. Significant moderators of postencoding stress effects. Size of circles in the continuous plot indicates the relative weight given to that study in the analysis. Effects of postencoding stress were moderated by whether the encoding task and stressor were conducted in the same physical context, the inclusion of hormonal contraceptives, and the time of day the study began. See the online article for the color version of this figure.

task type, stress-induced cortisol increase, study-item valence, a context change between learning and stress, stressor type, delay between encoding and retrieval, and the time of day the study began moderated the effect of stress on postencoding/retrieval. None of these potential moderators, however, influenced effects of stress on postencoding/retrieval, $ps > .174$, although we should note that with only nine studies examining stress effects on postencoding/retrieval, we likely lacked the power necessary to detect any subtle moderating effects.

In sum, we did not find any reliable effect of stress on memory when the stressor impacted both the postencoding period and the retrieval period. This is broadly consistent with the results described above, in that when stress selectively impacts the postencoding period it enhances memory, whereas when stress selectively impacts retrieval it impairs memory; thus, stress has no overall effect when it influences both postencoding and retrieval processes.

Encoding/retrieval. We considered stress to affect encoding, postencoding, and retrieval processes (i.e., encoding/retrieval) when stress offset occurred prior to or during encoding and within 90 min of retrieval. For all such studies, stress offset ranged from 35 min before encoding to during encoding, and retrieval occurred

with 65 min of stress offset (with encoding and retrieval being separated by 60 min at most). The overall effect of stress on encoding/retrieval ($m = 16$, $k = 48$, $N = 1,148$) was marginally significant and negative, $g^+ = -.185$, $t(14.2) = -2.02$, $p = .062$, 95% CI$_g$ [$-.382$, $.011$] (see Figure 11). There was some heterogeneity in these effects, $\tau^2 = 0.12$, indicating that this impairing effect might differ as a function of moderators. The effects of all potential moderators on stress effects on encoding/retrieval are displayed in Table 7.

The only variable found to modulate the negative effect of stress on encoding/retrieval with $df > 4$ was the delay in hours between encoding and retrieval, $B = .554$, $t(5.2) = 3.59$, $p = .015$, indicating that the impairing effect of stress became smaller as the delay between encoding and retrieval increased (see Figure 12). Additional analyses indicated that the biggest stress-induced impairment of encoding/retrieval would be obtained if there was a zero-minute delay (e.g., only a very brief interfering task) between stress and retrieval, $g^+ = -0.312$, $t(7.8) = -3.39$, $p = .010$, 95% CI$_g$ [-0.525, -0.098]. A sample of 314 participants (157 stress, 157 control) is necessary to achieve 80% power to detect this effect in a two-tailed test.
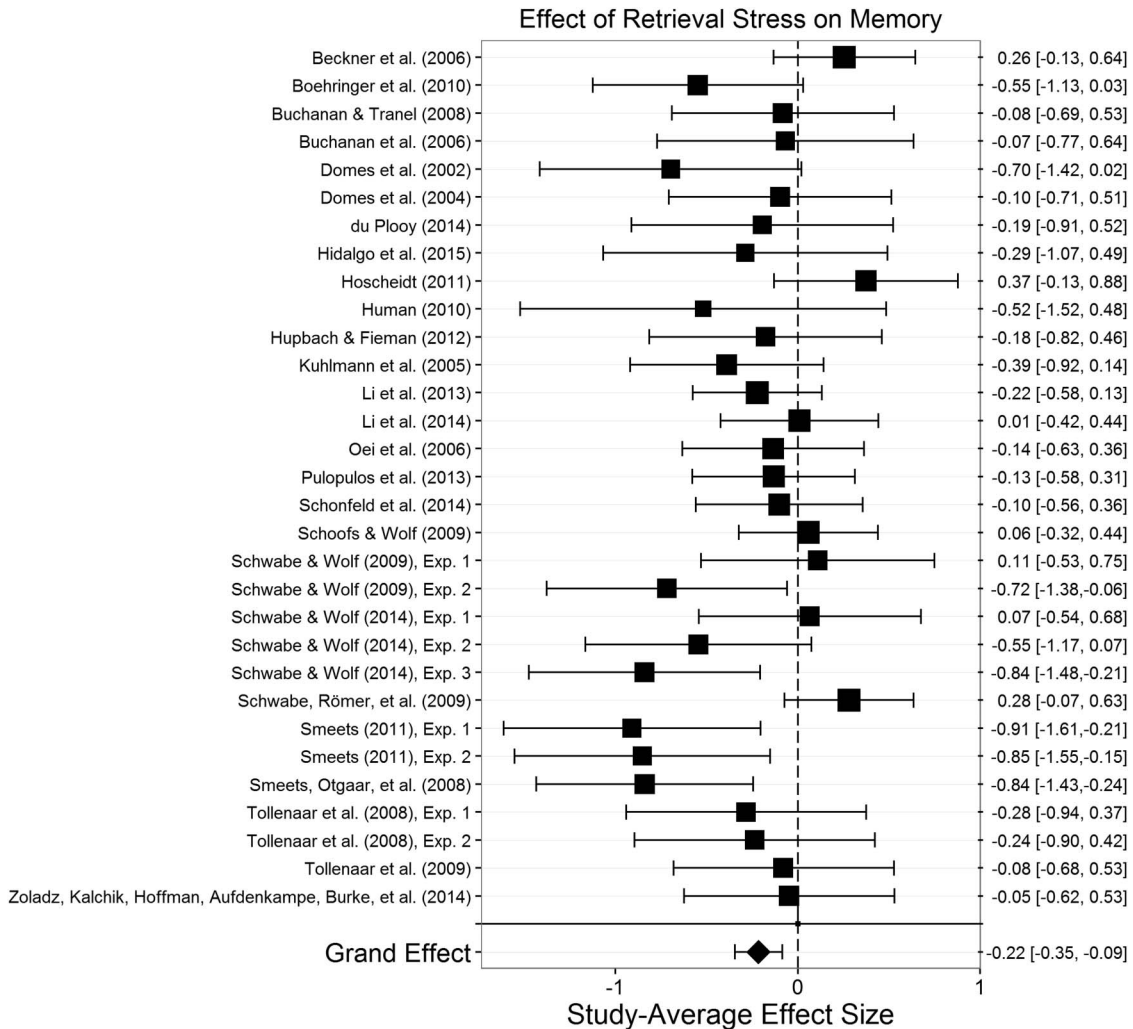
Effect of Retrieval Stress on Memory

Study	Effect Size [95% CI]
Beckner et al. (2006)	0.26 [-0.13, 0.64]
Boehringer et al. (2010)	-0.55 [-1.13, 0.03]
Buchanan & Tranel (2008)	-0.08 [-0.69, 0.53]
Buchanan et al. (2006)	-0.07 [-0.77, 0.64]
Domes et al. (2002)	-0.70 [-1.42, 0.02]
Domes et al. (2004)	-0.10 [-0.71, 0.51]
du Plooy (2014)	-0.19 [-0.91, 0.52]
Hidalgo et al. (2015)	-0.29 [-1.07, 0.49]
Hoscheidt (2011)	0.37 [-0.13, 0.88]
Human (2010)	-0.52 [-1.52, 0.48]
Hupbach & Fieman (2012)	-0.18 [-0.82, 0.46]
Kuhlmann et al. (2005)	-0.39 [-0.92, 0.14]
Li et al. (2013)	-0.22 [-0.58, 0.13]
Li et al. (2014)	0.01 [-0.42, 0.44]
Oei et al. (2006)	-0.14 [-0.63, 0.36]
Pulopulos et al. (2013)	-0.13 [-0.58, 0.31]
Schonfeld et al. (2014)	-0.10 [-0.56, 0.36]
Schoofs & Wolf (2009)	0.06 [-0.32, 0.44]
Schwabe & Wolf (2009), Exp. 1	0.11 [-0.53, 0.75]
Schwabe & Wolf (2009), Exp. 2	-0.72 [-1.38, -0.06]
Schwabe & Wolf (2014), Exp. 1	0.07 [-0.54, 0.68]
Schwabe & Wolf (2014), Exp. 2	-0.55 [-1.17, 0.07]
Schwabe & Wolf (2014), Exp. 3	-0.84 [-1.48, -0.21]
Schwabe, Römer, et al. (2009)	0.28 [-0.07, 0.63]
Smeets (2011), Exp. 1	-0.91 [-1.61, -0.21]
Smeets (2011), Exp. 2	-0.85 [-1.55, -0.15]
Smeets, Otgaar, et al. (2008)	-0.84 [-1.43, -0.24]
Tollenaar et al. (2008), Exp. 1	-0.28 [-0.94, 0.37]
Tollenaar et al. (2008), Exp. 2	-0.24 [-0.90, 0.42]
Tollenaar et al. (2009)	-0.08 [-0.68, 0.53]
Zoladz, Kalchik, Hoffman, Aufdenkampe, Burke, et al. (2014)	-0.05 [-0.62, 0.53]
Grand Effect	-0.22 [-0.35, -0.09]

Study-Average Effect Size

Figure 8. Effect of retrieval stress on memory. Size of the square indicates the relative weight assigned to that study in the analysis. Error bars represent the 95% confidence interval of the effect. Points to the left of zero indicate a study-average impairment in retrieval, and points to the right of zero indicate a study-average enhancement in retrieval. This meta-analysis indicated that stress significantly impaired retrieval across all studies and paradigms.

In sum, stress on average impairs memory when the stressor impacts the encoding, postencoding, and retrieval periods. This impairment is greatest at a short delay between stress offset and retrieval and attenuates as that delay increases. The results are broadly consistent with the earlier results, in the sense that stress during retrieval and during encoding sometimes impaired memory, whereas postencoding stress usually enhanced memory.

Postreactivation. We considered stress to affect postreactivation processes when stress followed reactivation of a memory no more than 60 min postreactivation, and when learning, reactivation, and each retrieval phases took place on separate days. The overall effect of postreactivation stress on memory ($m = 10$, $k = 20$, $N = 344$) was not significant, $g^+ = .154$, $t(8.9) = 0.66$, $p = .526$, 95% CI$_g$ [−0.375, 0.683] (see Figure 13). There was approximately moderate heterogeneity in these effects, $\tau^2 = 0.82$, indicating that this nonsignificant effect likely differed as a func-

tion of moderators. We should note, however, that with only 10 postreactivation studies we did not have the power to fully test these moderating effects. The effects of all potential moderators on postreactivation stress effects on memory are displayed in Table 8.

We first examined whether stress-induced cortisol increases moderated the effect of postreactivation stress on memory. We found a marginal quadratic relation between stress-induced cortisol increases and postreactivation stress effects on memory, $B_{linear} = −.887$, $t(2.4) = −3.63$, $p = .053$, $B_{quadratic} = .095$, $t(2.1) = 4.08$, $p = .052$, with small and large postreactivation stress-induced cortisol increases tending to enhance memory, but moderate cortisol increases tending to impair memory. However, because the *df* are less than 4 in the above analyses, there is a twofold greater risk of making a Type I error.

We next examined whether a change in context between memory reactivation and stress moderated the effect of postreactivation

Table 5
Potential Moderators of Retrieval Stress Effects on Memory

Moderator	B	df	p
Incidental or intentional encoding	−.035	11.2	.810
Exclusion of smokers	−.238	26.8	.075
Exclusion of women during menstrual period	−.070	27.0	.623
Exclusion hormonal contraceptives usage	**−.395**	**8.1**	**.009**
Exclusion of all illnesses	−.023	27.9	.861
Exclusion of all psychoactive medication	−.150	9.1	.352
Exclusion of BMI greater than 30	.091	13.3	.532
Use of an immediate recall task postencoding	−.108	22.2	.379
Context change between learning and retrieval	.243	1.1	.261
Stress manipulated between or within groups	.216	8.5	.114
Item valence (compared with neutral)			
Negative	−.166	24.8	.068
Positive	**−.248**	**7.1**	**.024**
Study material type			
Pictures	−.059	3.5	.411
Words	**−.214**	**20.0**	**.025**
Narrative/Slideshow	.243	2.6	.192
Autobiographical	.039	7.7	.427
Other	−.008	8.7	.922
Sensory modality of study material presentation			
Verbal	−.002	3.9	.993
Visual	−.101	2.8	.593
Verbal + Visual	.102	1.2	.790
Memory task type			
Free recall	.023	18.7	.792
Cued recall	−.052	6.0	.684
Recognition	.028	15.4	.789
Stressor type			
Socio-evaluative	−.030	18.8	.704
Pain	−.152	3.4	.386
Hybrid (socio-evaluative & pain)	−.213	11.3	.152
Other	NA		
Participant age	−.002	1.4	.817
Percent male participants	<.001	15.4	.966
Time of day study began	<.001	8.8	.537
Study item list length	−.002	3.6	.451
Number of novel items in a recognition task	<.001	7.7	.984
Delay (hours) between item encoding and retrieval	<.001	1.1	.875
Delay (min) between stress onset and retrieval	−.003	3.5	.646
Stressor duration (min)	.014	21.0	.142
Stress-induced Δ-cortisol (nmol/L)	−.001	6.4	.968
Length of encoding phase (min)	−.018	1.9	.784
Participant homogeneity	−.053	12.5	.238

Note. Significant ($p < .05$) moderators are shown in boldface font. B represents the change in the effect size for every one-unit change in the moderator. For dummy-coded categorical variables, B represents the difference between estimated effects for each group; for contrast-coded categorical variables, B represents the difference between the group in question and the average estimated effect. If $df < 4$, there is a twofold greater risk of making a Type I error. The listed p value represents the significance of the moderator in question. When there were not enough studies to estimate an effect, NA is listed in the column for B.

stress on memory, and found that a change in context between memory reactivation and stress moderated the effect of postreactivation stress on memory, $t(3.8) = 5.28$, $p = .007$, such that a change in context between reactivation and stress onset was associated with an enhancement of memory, $g^+ = 1.01$, $t(2.0) = 18.3$, $p = .003$, whereas a constant context was associated with a nonsignificant impairment in memory, $g^+ = −.191$, $t(5.9) = −0.87$, $p = .419$. However, because df are less than four in the above contrast, there is a twofold greater risk of making a Type I error.

In addition, it is important to note that all studies that employed a context change between reactivation and stress without a change of context between encoding and reactivation were conducted in the same laboratory using the same paradigm, thus holding numerous factors constant, whereas none of the studies with a constant context between reactivation and stress used the same paradigm. Thus, it is unclear whether a change in context between reactivation and stress actually produced an enhancing effect of postreactivation stress on memory or if another methodological factor was responsible.

We also examined the effects of sex, the exclusion of women taking hormonal contraceptives, and the exclusion of women during their menstrual period, and found that none of these variables influenced the effects of postreactivation stress on memory, $ps > .717$. Finally, we examined the delay between reactivation and stress onset, the delay between encoding and retrieval, participant age, time of day, item valence, memory task type, and stressor type, given the tendency in the stress and memory literature to treat these variables as methodologically relevant. None of these variables were found to be significant moderators, $ps > .175$.

In sum, we did not find meta-analytic evidence for an overall postreactivation stress effect on memory or any reliable moderators of this potential stress effect. However, given the small number of reactivation studies that have been published, and the notable heterogeneity in observed effects (cf. Table 2 demonstrating the relatively low power of these analyses), we hesitate to draw any strong conclusions about postreactivation stress effects (or lack thereof) until more research has been conducted.

Discussion

In this meta-analysis, we examined the effects of acute social and physical stressors on episodic memory. We assessed the results of 113 studies, which included a total of 6,216 participants, that examined the effects of stress on encoding, postencoding, retrieval, and postreactivation phases. We explored the effects of several variables that previous studies indicated might be critical, as well as several methodological factors that were available in most studies. In general, we found that whether stress enhanced or impaired memory critically depended upon whether stress occurred during the encoding, retention, or retrieval phase. Moreover, there was evidence that the timing of the stressor, the context in which the stressor was experienced, and the nature of the studied materials played critical roles in determining how stress impacted the different memory phases. There was little evidence that the effects of stress on memory were modulated by the overall cortisol changes produced by stress in individual studies, but the effects of stress on memory were generally reduced in women taking hormonal contraceptives. We first discuss the effects of stress on cortisol, and we then describe the effects of stress on each phase of memory. We then consider those results in light of current theories of stress and memory, and highlight questions for further research.

Stress and Cortisol

Given that cortisol is thought to provide an index of stress and has been proposed to moderate the effects of stress on memory, we first assessed the ability of acute laboratory stressors to elicit cortisol responses. We found that stress significantly increased
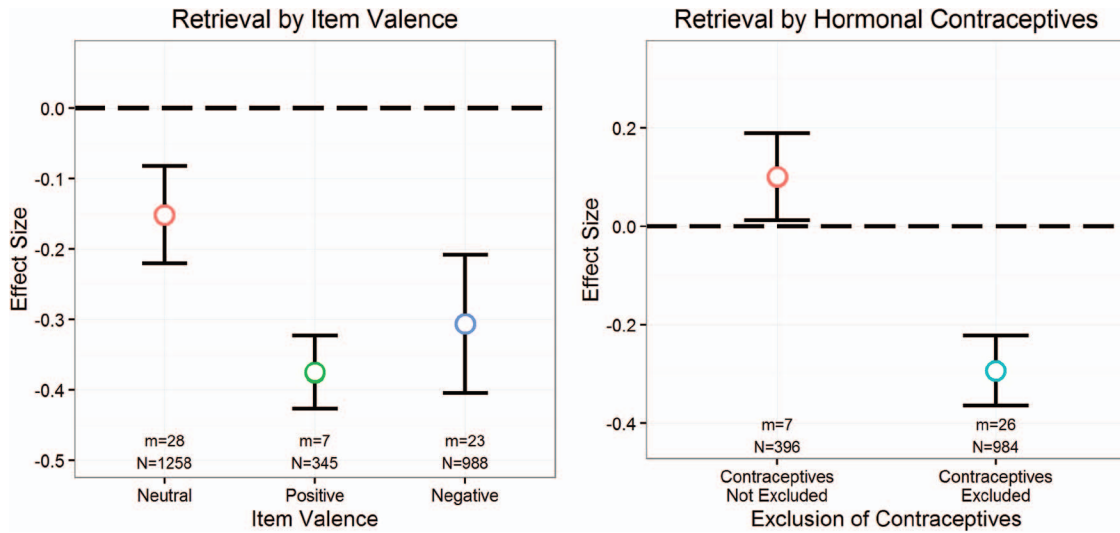
Figure 9. Significant moderators of stress effects on retrieval. Effects of stress on retrieval were greater for negative and positive items than for neutral items. In addition, effects of stress on retrieval were greater when women using hormonal contraceptives were excluded from the study. See the online article for the color version of this figure.

cortisol, and the TSST elicited a larger cortisol increase than either the CPT or SECPT—which is consistent with individual studies that have compared these stressors (Giles, Mahoney, Brunyé, Taylor, & Kanarek, 2014; Schwabe, Haddad, & Schachinger, 2008; Skoluda et al., 2015). Moreover, studies that performed the stress manipulation in the afternoon showed a significantly larger cortisol increase than studies that began in the morning—consistent with an earlier meta-analysis (Dickerson & Kemeny, 2004).

Additionally, our analyses indicated that stress elicited larger cortisol responses as the percentage of males included in the study increased, and revealed larger cortisol responses in studies that excluded women who were taking hormonal contraceptives or women in their menstrual phase. This is consistent with previous work showing that men exhibit consistently higher cortisol responses to laboratory stressors than women (Kirschbaum et al., 1992) and that both hormonal contraceptives and menstrual cycle
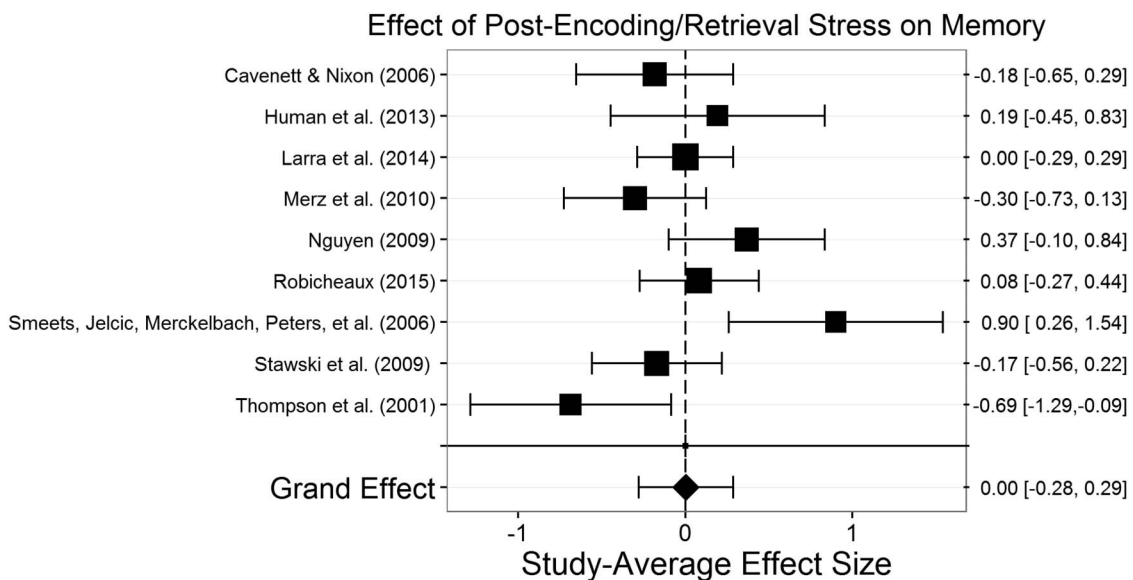
Figure 10. Effects of postencoding/retrieval stress on memory. Size of the square indicates the relative weight assigned to that study in the analysis. Error bars represent the 95% confidence interval of the effect. Points to the left of zero indicate a study-average impairment in memory, and points to the right of zero indicate a study-average enhancement in memory. This meta-analysis indicated that stress that impacted both the postencoding and retrieval phases of memory did not influence memory across all studies and paradigms.

Table 6

Potential Moderators of Postencoding/Retrieval Stress Effects on Memory

Moderator	B	df	p
Incidental or intentional encoding	−.435	4.3	.146
Exclusion of smokers	NA		
Exclusion of women during menstrual period	**.627**	**3.3**	**.048**
Exclusion hormonal contraceptives usage	.644	1.4	.274
Exclusion of all illnesses	NA		
Exclusion of all psychoactive medication	NA		
Exclusion of BMI greater than 30	NA		
Use of an immediate recall task postencoding	.158	3.9	.557
Context change between learning and stress	−.046	1.8	.823
Context change between learning and retrieval	−.507	1.5	.248
Stress manipulated between or within groups	−.578	1.6	.155
Item valence (compared with neutral)			
Negative	−.018	1.6	.938
Positive	NA		
Study material type			
Pictures	NA		
Words	−.195	5.1	.452
Narrative/Slideshow	NA		
Autobiographical	.030	3.7	.895
Other	.165	1.6	.731
Sensory modality of study material presentation			
Verbal	−.348	4.3	.267
Visual	.348	4.3	.267
Verbal + Visual	NA		
Memory task type			
Free recall	−.216	5.8	.450
Cued recall	NA		
Recognition	.216	5.8	.450
Stressor type			
Socio-evaluative	.152	4.7	.479
Pain	.049	1.4	.669
Hybrid (socio-evaluative & pain)	NA		
Other	−.403	1.3	.246
Participant age	−.098	3.4	.203
Percent male participants	.003	4.1	.697
Time of day study began	−.002	1.6	.175
Study item list length	.004	2.1	.371
Number of novel items in a recognition task	.026	1.0	.569
Delay (hours) between item encoding and retrieval	−.031	3.6	.961
Delay (min) between encoding and stress onset	−.021	1.9	.459
Delay (min) between stress onset and retrieval	.001	1.7	.975
Stressor duration (min)	.008	4.8	.584
Stress-induced Δ-cortisol (nmol/L)	.100	2.4	.222
Length of encoding phase (min)	−.010	1.4	.271
Participant homogeneity	.302	2.9	.063

Note. Significant ($p < .05$) moderators are shown in boldface font. *B* represents the change in the effect size for every one-unit change in the moderator. For dummy-coded categorical variables, *B* represents the difference between estimated effects for each group; for contrast-coded categorical variables, *B* represents the difference between the group in question and the average estimated effect. If $df < 4$, there is a twofold greater risk of making a Type I error. The listed *p* value represents the significance of the moderator in question. When there were not enough studies to estimate an effect, NA is listed in the column for *B*.

phase blunt or modulate cortisol responses to stress (Kirschbaum et al., 1999; Marinari et al., 1976). The finding that excluding women during their menstrual period increased effects of stress on cortisol may seem surprising because of the suppressive effects of estrogen on cortisol responses (Ycaza Herrera & Mather, 2015) and because estrogen is low during the menstrual period. One possible explanation for this effect is that progesterone increases

stress responses (Sakaki & Mather, 2012) and excluding women in the menstrual phase may yield a larger proportion of women in the luteal—high progesterone—phase of their cycle.

In sum, to the extent that cortisol provides an index of the stress response, it appears that the stress induction methods used in studies of stress and memory consistently produce stress responses. These analyses therefore showed that acute stress inductions in the stress and memory literature are successful, and thus put us in a position to make valid claims about effects of acute stress on memory.

Encoding

Overall, there was no significant main effect of stress on encoding, but several factors significantly influenced whether stress enhanced or impaired memory encoding. Two of the strongest moderators of stress effects on encoding were the delay between stress and encoding (i.e., the stress-encoding delay) and the relevance of the learned information to the stressor (i.e., stressor relevance). In general, the analyses indicated that stress impaired encoding unless the items learned were related to the stressor and the stress-encoding delay was very short, in which case stress appeared to enhance encoding.

To our knowledge, only two human studies have directly manipulated the stress-encoding delay to examine time-dependence effects of preencoding stress. The first of these (Zoladz et al., 2011) found weak support for the idea that the stress-encoding delay plays an important role. Zoladz et al. found a memory enhancement for only positive items (and no effect for negative or neutral items) in a no delay condition and an impairment in only negative items in a 30 min delay condition. The second study (Quaedflieg, Schwabe, Meyer, & Smeets, 2013), however, failed to find any behavioral differences in stress effects on encoding between the immediate and 30 min delay conditions. Importantly, however, both of these studies employed a hybrid socioevaluative/pain stressor, and our results indicate that this stressor type—or any pain-based stressor, see below—may have prevented actual effects of the stress-encoding delay from emerging. Future studies testing this possibility are needed.

We found evidence that the stressor type appeared to moderate the stress-encoding delay effect. That is, in general, as the delay between stress and encoding increased, the effects of stress became more negative. However, this pattern was not observed in studies that used the hybrid socioevaluative/pain stressors (i.e., the SECPT), where stress appeared to lead to a general impairment in encoding, regardless of delay. When interpreting this finding it is important to note that we did not have enough power to determine whether this effect was specific to hybrid stressors, or whether stressors that only included pain, such as the CPT, also showed this effect, as only two studies of stress at encoding used a stressor involving pain without social evaluation. Thus, one possibility is that pain-based stressors might be unique. For example, they may require continuous response inhibition (i.e., not retracting one's arm from painful ice water), and this inhibition may have a general impairing effect on memory encoding regardless of delay (Chiu & Egner, 2015). Alternatively, it is possible that the combination of the social and physical stress itself is what is producing disruptive effects on memory even at short stress-encoding delays. Future
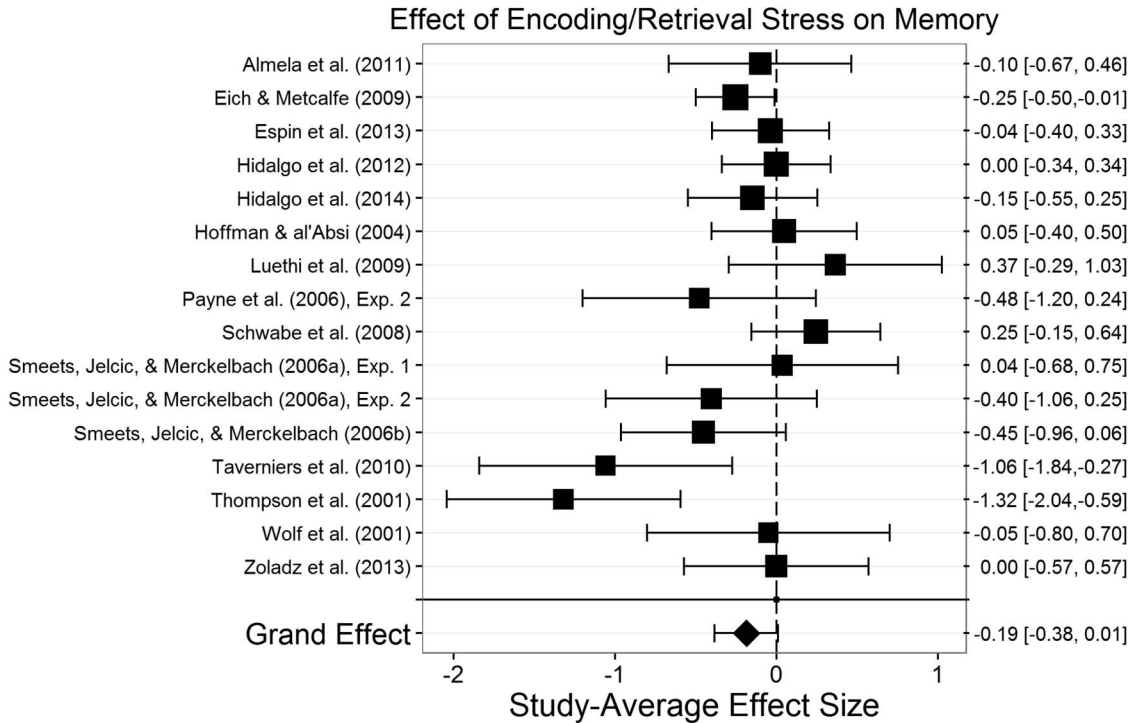
Figure 11. Effect encoding/retrieval stress on memory. Size of the square indicates the relative weight assigned to that study in the analysis. Error bars represent the 95% confidence interval of the effect. Points to the left of zero indicate a study-average impairment in memory, and points to the right of zero indicate a study-average enhancement in memory. This meta-analysis indicated that stress that impacted encoding, postencoding, and retrieval phases of memory marginally impaired memory across all studies and paradigms.

research contrasting the delay effects of different stressors will be important in resolving this issue.

The significant moderating effect of stressor relevance is consistent with several previous studies that directly examined this factor. For example, Wiemers et al. (2013) and Smeets, Giesbrecht, Jelicic, and Merckelbach (2007) both found that stress prior to or during encoding enhanced memory only for information relevant to the stressor. These results indicate that stressor relevance is important in determining the effects of stress on encoding.

Sex and exclusion of hormonal contraceptives significantly moderated stress-encoding delay effects, although sex was no longer a significant moderator once the exclusion of hormonal contraceptives was controlled (data not shown). These results are consistent with prior research indicating that sex hormones modulate memory encoding processes; namely, that testosterone (Ackermann et al., 2012; van Wingen, Mattern, Verkes, Buitelaar, & Fernández, 2008) and estradiol (Kramár, Babayan, Gall, & Lynch, 2013; Srivastava et al., 2011) enhance memory encoding. Because hormonal contraceptive use decreases both estradiol and testosterone (Graham & Milad, 2013; Liening, Stanton, Saini, & Schultheiss, 2010), it is not surprising that hormonal contraceptives might blunt stress effects on encoding.

Surprisingly, stress-induced cortisol increases did not significantly moderate stress effects on encoding, nor did we find any evidence for the effects of time-of-day. This contrasts with an earlier meta-analysis that found a direct effect of cortisol administration and time-of-day on memory encoding (Het et al., 2005).

In our analyses, controlling for the time of day only weakened the nonsignificant effect of cortisol further (data not shown). We expound upon the lack of observed cortisol effects in the Theoretical Integration section below. In addition, we did not observe a moderating effect of using an immediate recall task in stress effects on encoding, as was expected from a prior study (Wolf, 2012).

The valence of study items (i.e., positive, negative, neutral) is commonly assessed as a potential moderator of stress effects on encoding (e.g., van Ast, Cornelisse, Meeter, & Kindt, 2014; Wolf, 2012; Zoladz et al., 2011), but in this meta-analysis we failed to find any evidence that stress influenced encoding items of one valence more than another. One explanation for this lack of effect might be that oftentimes studies present participants with a mixed list of neutral and emotional items together. It is possible that mixing item valences diminishes any interaction between stress and item valence, obscuring effects. To assess this possibility further, we controlled for the valences included in the study list (data not shown) and found that it did not alter the results, nor did the study list valences interact with memory for specific item valence in producing stress effects on encoding.

In sum, stress prior to encoding led to a decrease in memory unless the delay between the stressor and encoding was very short and the materials were relevant to the stressor. In addition, the effect of the stress-encoding delay was reduced in women on hormonal contraceptives and in women who were menstruating, highlighting the potential importance of sex hormones in the stress

Table 7

Potential Moderators of Encoding/Retrieval Stress Effects on Memory

Moderator	B	df	p
Incidental or intentional encoding	−.076	3.0	.764
Exclusion of smokers	.269	5.3	.130
Exclusion of women during menstrual period	.038	13.7	.848
Exclusion hormonal contraceptives usage	−.025	12.6	.897
Exclusion of all illnesses	.074	5.0	.775
Exclusion of all psychoactive medication	.328	12.1	.085
Exclusion of BMI greater than 30	−.060	1.3	.938
Use of an immediate recall task postencoding	.029	13.3	.888
Context change between stress and learning	−.181	4.2	.381
Context change between learning and retrieval	NA		
Stress manipulated between or within groups	−.068	8.0	.771
Item valence (compared with neutral)			
Negative	.066	3.6	.611
Positive	.213	2.3	.180
Study material type			
Pictures	NA		
Words	NA		
Narrative/Slideshow	NA		
Autobiographical	NA		
Other	NA		
Sensory modality of study material presentation			
Verbal	.017	8.7	.898
Visual	−.192	2.7	.389
Verbal + Visual	.175	5.0	.274
Memory task type			
Free recall	.062	6.0	.554
Cued recall	NA		
Recognition	−.043	3.1	.774
Stressor type			
Socio-evaluative	.126	4.7	.422
Pain	NA		
Hybrid (socio-evaluative & pain)	.368	1.5	.181
Other	−.494	2.9	.159
Participant age	−.005	2.0	.489
Percent male participants	−.003	8.8	.377
Time of day study began	−.001	5.2	.210
Study item list length	**−.017**	**2.3**	**.024**
Number of novel items in a recognition task	−.009	1.5	.644
Delay (hours) between item encoding and retrieval	**.554**	**5.2**	**.015**
Delay (min) between stress onset and encoding	−.001	1.3	.556
Delay (min) between stress onset and retrieval	<−.001	1.4	.999
Stressor duration (min)	−.001	1.3	.748
Stress-induced Δ-cortisol (nmol/L)	−.026	2.1	.475
Length of encoding phase (min)	−.014	1.4	.907
Participant homogeneity	.051	10.2	.476

Note. Significant (*p* < .05) moderators are shown in boldface font. *B* represents the change in the effect size for every one-unit change in the moderator. For dummy-coded categorical variables, *B* represents the difference between estimated effects for each group; for contrast-coded categorical variables, *B* represents the difference between the group in question and the average estimated effect. If *df* < 4, there is a twofold greater risk of making a Type I error. The listed *p* value represents the significance of the moderator in question. When there were not enough studies to estimate an effect, NA is listed in the column for *B*.

effects on encoding. Surprisingly, we found no moderating effects of cortisol reactivity or time of day (despite our finding that time of day significantly influenced cortisol reactivity). In addition, we found no moderating effects of item valence, or the use of an immediate recall task, despite prior work suggesting that these variables may moderate stress effects on encoding. This suggests that the effects of these moderators may be relatively specific to precise experimental conditions, rather than generalizing across studies of stress.

Postencoding Stress

Postencoding stress led to a general increase in episodic memory, but there were several important moderators of this effect. Most notably, when the stress manipulation was administered in the same context as the initial encoding phase, stress led to an increase in memory, whereas in studies in which the context was changed between encoding and stress (e.g., moving to another room) stress did not improve memory. This is consistent with a recent report that found that postencoding stress did not benefit memory across a series of experiments, each of which involved a context change between the encoding phase and the stressor (Trammell & Clore, 2014; see also McCullough et al., 2015). Most other studies in the literature that found an enhancing effect of postencoding stress on memory did not have a context change between encoding and stress. No published studies have directly examined the effects of changing the context of the postencoding stressor, so studies that directly examine the effects of changing context within a single experiment will be important in verifying the importance of context changes on stress and memory.

Another significant moderator of the postencoding effects was time of day, which indicated that studies that began in the afternoon showed a larger effect than studies that began in the morning. As described earlier, time of day can affect the cortisol response; what is interesting here is that time of day did not interact with cortisol change, so in these postencoding stress studies, time of day seems to have some other effect on memory in addition to or independent of any effect it may have on the cortisol response. In addition, contrary to our expectations, cortisol change in response to stress did not moderate the effects of postencoding stress on memory (either linearly or quadratically).

Despite no association of postencoding stress effects with effects of stress on cortisol, we found that contraceptive use was a significant moderator of the stress effects on memory. If a study excluded women taking hormonal contraceptives, memory was significantly better for the postencoding stress group, whereas studies that did not exclude women taking hormonal contraceptives did not find beneficial effects of stress on memory. Participant sex was not found to moderate postencoding stress effects on memory, so contraceptive use appears to be a critical factor for whether studies find an enhancing effect of postencoding stress on memory or not, rather than participant sex. Contraceptive use also did not interact with cortisol change, suggesting that dampening the cortisol response in itself (see analyses of stress effects on cortisol) does not fully explain the contraceptive effects. Instead, this suggests that contraceptives may act through pathways aside from cortisol to dampen the stress effects on memory. One means by which this might happen is via effects of estradiol, which both increases as a result of stress (Lennartsson, Kushnir, Bergquist, Billig, & Jonsdottir, 2012) and enhances memory retention (T. Inagaki et al., 2010). Because hormonal contraceptives reduce ovarian estradiol output, leading to decreased levels of estradiol (Graham & Milad, 2013), hormonal contraceptives may blunt effects of postencoding stress because of their effects on estradiol (see also Barros et al., 2015). Conversely, estradiol exerts opposing effects on cortisol (Ycaza Herrera & Mather, 2015), which
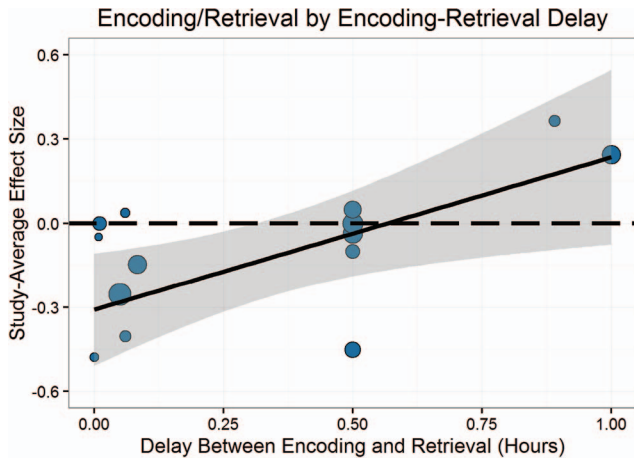
Figure 12. Delay between encoding and retrieval moderated effects of stress on memory when the stressor occurred within a timeframe to influence encoding, postencoding, and retrieval phases. With less time between encoding and retrieval, stress at encoding/retrieval impaired memory to a greater extent than with more time. See the online article for the color version of this figure.

may be a mechanism through which hormonal contraceptives modulate effects of stress on memory.

Contrary to expectations, effects of postencoding stress on memory did not differ for negative, positive, or neutral materials. Thus, although it is possible additional experimental conditions moderate the extent to which valence plays a role, we did not find evidence for valence being an important moderator of postencoding stress effects on memory when considered across experiments.

In sum, postencoding stress generally improved memory. However, this effect appeared to be eliminated when the context between encoding and stress was changed, indicating that postencoding stress may be limited to improving memory for items that were encoded in the same spatial context as the stressor. Although the effects of postencoding stress on memory were not related to cortisol reactivity or sex, the beneficial effects of stress were reduced when women taking hormonal contraceptives were included, and when the study was conducted in the morning rather than the afternoon, suggesting some hormonal or immune system sensitivity of the stress effects. Finally, contrary to expectations, material valence did not emerge as a significant moderator of postencoding stress effects on memory.

Retrieval

Stress generally impaired memory retrieval, and these effects were largest for emotional materials and in studies in which women taking oral contraceptives were excluded. The finding that stress impaired retrieval of negative and positive items more so than neutral items is consistent with a recent review that highlighted the relatively greater impairing effect of stress on retrieving emotional material (Gagnon & Wagner, 2016).

Similar to effects observed for encoding and postencoding stress, studies that excluded women taking oral contraceptives showed a greater effect of stress on memory retrieval. Specifically, when a study did not exclude women taking hormonal contraceptives from participating, there was no significant impairing effect of stress on retrieval. In contrast, if a study excluded women taking hormonal contraceptives, stress significantly impaired retrieval. Like results discussed in previous sections, this result implies that sex hormones may be more important for stress effects on retrieval
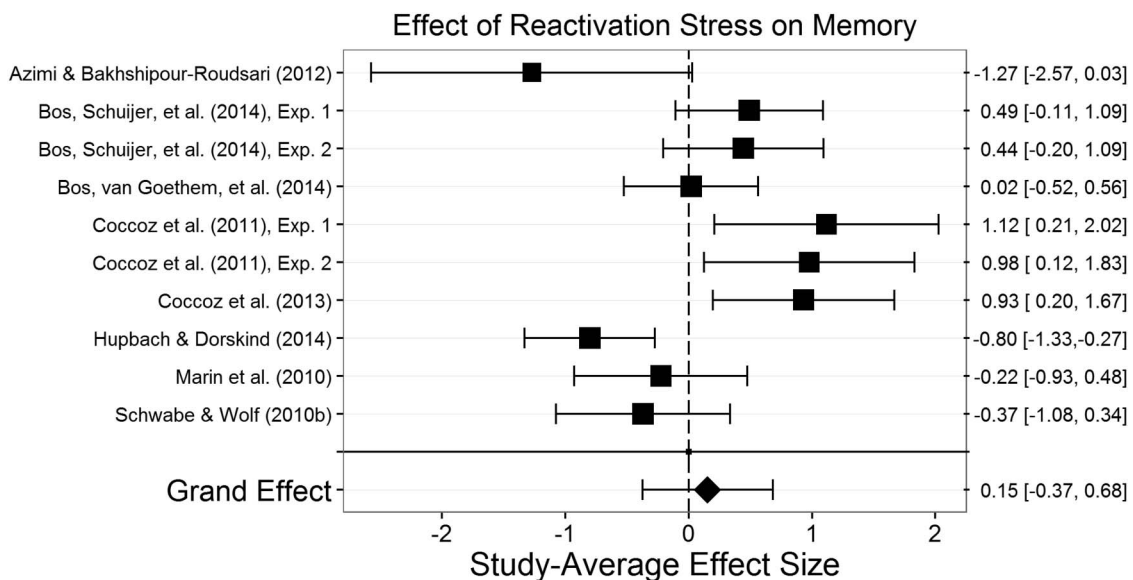

Figure 13. Effects of reactivation stress on memory. Size of the square indicates the relative weight assigned to that study in the analysis. Error bars represent the 95% confidence interval of the effect. Points to the left of zero indicate a study-average impairment in memory, and points to the right of zero indicate a study-average enhancement in memory. This meta-analysis indicated that postreactivation stress did not influence memory across all studies and paradigms.

Table 8
Potential Moderators of Reactivation Stress Effects on Memory

Moderator	B	df	p
Incidental or intentional encoding	.051	6.7	.923
Exclusion of smokers	.629	7.9	.205
Exclusion of women during menstrual period	−.145	1.6	.893
Exclusion hormonal contraceptives usage	−.030	1.7	.977
Exclusion of all illnesses	.128	4.0	.769
Exclusion of all psychoactive medication	.951	1.6	.125
Exclusion of BMI greater than 30	NA		
Use of an immediate recall task postencoding	.567	1.6	.224
Stressor relevance of items (integral/nonintegral)	NA		
Context change between reactivation and stress	**1.196**	**3.8**	**.007**
Context change between learning and retrieval	.421	1.4	.604
Stress manipulated between or within groups	NA		
Item valence (compared with neutral)			
Negative	.346	6.3	.554
Positive	.535	4.0	.360
Study material type			
Pictures	NA		
Words	.289	3.8	.298
Narrative/Slideshow	NA		
Autobiographical	−.786	1.6	.183
Other	.497	5.1	.220
Sensory modality of study material presentation			
Verbal	NA		
Visual	.162	4.1	.502
Verbal + Visual	.338	1.2	.182
Memory task type			
Free recall	.129	6.8	.598
Cued recall	−.156	6.8	.526
Recognition	NA		
Stressor type			
Socio-evaluative	NA		
Pain	.296	6.5	.324
Hybrid (socio-evaluative & pain)	−.296	6.5	.324
Other	NA		
Participant age	.370	3.8	.176
Percent male participants	.006	2.3	.718
Time of day study began	.002	1.8	.202
Study item list length	.007	2.1	.247
Number of novel items in a recognition task	NA		
Delay (hours) between item encoding and retrieval	−.006	3.5	.083
Delay (min) between reactivation and stress onset	−.109	1.5	.207
Stressor duration (min)	−.037	1.7	.224
Stress-induced Δ-cortisol (nmol/L)	−.001	2.8	.994
Length of encoding phase (min)	−.014	1.4	.655
Participant homogeneity	.379	2.5	.124

Note. The context change between reactivation and stress should not be trusted; see the main text for an explanation. Significant ($p < .05$) moderators are shown in boldface font. B represents the change in the effect size for every one-unit change in the moderator. For dummy-coded categorical variables, B represents the difference between estimated effects for each group; for contrast-coded categorical variables, B represents the difference between the group in question and the average estimated effect. If $df < 4$, there is a twofold greater risk of making a Type I error. The listed p value represents the significance of the moderator in question. When there were not enough studies to estimate an effect, NA is listed in the column for B.

than previously thought, highlighting the importance of considering these hormones when examining stress effects on memory.

We found that the delay between stress onset and retrieval (i.e., the stress-retrieval delay) did not moderate stress effects on retrieval. This result did not differ when we examined the delay between stress offset, rather than onset, and retrieval. This contrasts with a recent study that found that the negative effects of stress on retrieval were greatest at 90 min poststressor (Schwabe & Wolf, 2014). However, we note that all the other studies considered in this meta-analysis tested retrieval within 40 min of stressor offset; thus, more research with longer delays between stress and retrieval would be important before making strong claims about this potential moderator.

In sum, stress exerted a relatively consistent impairing effect on memory retrieval. Although this effect did not seem to be related to stress-induced cortisol responses, the impairing effect of stress on retrieval was greater when information to be retrieved was emotionally charged, as well as when considering only men or women not taking hormonal contraceptives.

Postencoding/Retrieval

When the stressor was expected to influence both the postencoding period and the retrieval phase, we found no significant effect of stress, and there was no indication that the effects were moderated by any other variables. Although the lack of effects could be attributable to the relatively smaller number of studies of this type (nine studies), the results are consistent with the main conclusions described above in that postencoding stress generally increased memory whereas retrieval stress generally reduced memory. Thus, it makes sense that there would be no overall effect of stress in studies in which the stressor impacts both postencoding and retrieval phases.

Encoding/Retrieval

Although we did not have strong a priori hypotheses for stress effects on memory when a single stressor was expected to impact the encoding, postencoding, and retrieval phases of memory, 16 such studies were reported and stress tended to impair memory. However, the impairing effects of stress were found to reverse in studies with longer delays between encoding and retrieval. The detrimental effects of stress that were observed are consistent with the fact that stress generally impairs retrieval and can impair encoding processes, both of which would be impacted in these designs. Why the detrimental effects decreased in magnitude with longer encoding-retrieval delays is not entirely clear, but it could be that using shorter delays effectively reduced any positive effects produced by postencoding stress.

Postreactivation

We found no overall significant effects of postreactivation stress on memory. Although we found some preliminary evidence for potentially important moderators of postreactivation stress effects, our lack of power limited our ability to detect small effects in these analyses (see Table 2). Thus, future research should continue to examine what effects, if any, postreactivation stress has on memory.

Relating the Current Results to Studies of Nonhuman Animals

The effects of stress have been studied quite extensively in rats and mice, and these studies have provided a rich body of knowledge about the neuromodulatory mechanisms that are influenced

by stress (for thorough reviews see Conrad, 2010; Diamond et al., 2007; Finsterwald & Alberini, 2014; Joëls et al., 2011; Roozendaal, 2002; Sandi & Pinelo-Nava, 2007). Relating those results to human episodic memory, however, is made somewhat difficult because of the inherent differences between tasks typically used to assess memory in animal and human studies. Nonetheless, it is worth considering how the current findings relate to those generally reported in the animal literature.

At the broadest level, the animal literature is consistent in showing that postencoding stress or coadministration of glucocorticoids and a noradrenergic agonist generally benefits memory, whereas stress or a similar pharmacological manipulation during retrieval impairs memory (Finsterwald & Alberini, 2014; Roozendaal, 2002; Roozendaal, Okuda, de Quervain, et al., 2006). Moreover, this literature has also found stress during encoding may benefit memory—especially for stress-related information (e.g., the location of an escape from a threatening situation)—if stress and learning occur in the same context (e.g., within the same space and close proximity within time) (Conboy & Sandi, 2010; Joëls et al., 2006). As far as we are aware, there have not been studies that have examined the effects of varying context between the study materials and the postencoding stressor, although in general stress or glucocorticoid administration often occurs outside of the learning apparatus (e.g., Roozendaal, Okuda, Van der Zee, & McGaugh, 2006). Given that the postencoding effects in humans appear to only occur when the study event occurs in the same spatial context as the stressor, it would seem important to determine whether the same holds in nonhuman animal postencoding stress paradigms as well.

One factor that has been examined at some length in the human literature, but that to our knowledge has not been directly manipulated in the same way within the animal literature, is the impact of the emotional valence of the encoding materials. Based largely on animal studies pointing to a role of the amygdala in producing stress effects (e.g., Akirav & Richter-Levin, 1999, 2002; Diamond et al., 2007; Joëls et al., 2011; Roozendaal & McGaugh, 1997; Roozendaal, Okuda, de Quervain, et al., 2006; Schwabe et al., 2012), it was expected that stress effects on memory would be larger for emotional then neutral materials, but this expectation was not strongly supported by the human literature. The only emotion effect that was consistently observed was that the impairing effects of stress at retrieval were larger for positive and marginally larger for negative materials than for neutral materials. Future studies in nonhuman animals aiming to determine whether stress has comparable effects on memory for emotionally salient compared with neutral materials will be important.

The animal results also somewhat parallel the current human results in the sense that stress effects are often larger in male than female rodents (Andreano & Cahill, 2009). This seems important, suggesting that sex or sex hormone effects in stress and memory may be quite general. Notably, we found that effects of hormonal contraceptives were consistently seen in studies examining effects of stress during the encoding, postencoding, and retrieval phases of memory, with the use of hormonal contraceptives dampening effects of stress on each of these phases. Thus, consistent with animal literature (Barros et al., 2015; Graham & Milad, 2013; Harburger, Pechenino, Saadi, & Frick, 2008; Kramár et al., 2013), our results suggest that sex hormones may be important modulators of memory and stress effects on memory.

In sum, despite differences in paradigms and species, there are a number of behavioral similarities observed in the human and nonhuman studies of stress and memory. However, there are several findings emerging in the human literature that have not yet been directly examined in the animal literature and these seem to be important targets of future research.

General Discussion and Theoretical Integration

The effects of stress on memory are complex, and it is clear that whether stress impairs or enhances memory depends on a number of factors. Although the results of this meta-analysis provide support for a number of the predictions of existing theories of stress and memory, no single theory seems capable of accounting for all these results without extension or modification. For example, consolidation theory (Cahill & McGaugh, 1998; McGaugh, 2000, 2015) is in good agreement with some aspects of the postencoding stress studies, but it fails to account for other important aspects of those studies, and it fails to provide accounts for impairing effects of stress on encoding or retrieval. The primary prediction of the consolidation account is that postencoding stress will enhance memory (McGaugh, 2000), and this prediction was strongly supported. In addition, although the consolidation account does not make explicit predictions about the effect of stress on encoding processes per se, to the extent that stress occurred during or immediately prior to encoding one may expect increased cortisol levels to persist into the postencoding period, and thus to result in enhancements in memory. Thus, the consolidation model is broadly consistent with the finding that encoding stress can improve memory if it occurs immediately prior to encoding. However, it is not clear from this account why encoding stress leads to a decrease in memory when it occurs much earlier than the encoding phase. In addition, one of the core predictions of this approach is that stress should enhance memory for emotional materials more so than neutral memories (Cahill & McGaugh, 1998; Joëls et al., 2011; McGaugh, 2000, 2004, 2015). Overall, there was no effect of emotionality on postencoding stress, with some studies showing an advantage for emotional materials, others showing the opposite, and still others showing no difference. One possibility, however, is that the manipulations of stress and emotion conducted in the studies of human memory were not sufficiently powerful to lead to consolidation. Thus, it is possible that the consolidation process does accurately account for traumatic memory formation for emotional events that precede stress, but it does not account for the types of memory typically examined in laboratory studies of human memory.

Consolidation theory also fails to provide an explanation for why postencoding stress would only impact memory when the stressor occurred in the same spatial context as the study materials. This theory, which is tied to neurobiological mechanisms, predicts that stress should benefit memory when it occurs shortly after the encoding phase, as stress and the associated hormonal changes facilitate long-term potentiation of recently encoded events (McGaugh, 2000), and thus there is no clear reason why a change in context would negate those neurobiological effects.

Another limitation of the consolidation account is that it says little about the impairing effects of stress on memory that were observed when stress occurred prior to encoding or retrieval (McGaugh, 2000), as few clear predictions regarding stress effects on

encoding and retrieval processes are provided by consolidation theory alone. Given the consistency of those results, this shortcoming seems critical. One could argue that the consolidation model was not intended to account for the effects of encoding or retrieval stress. In fact, it has been most directly tested in animal studies of memory, and only recently applied to understand human episodic memory in postencoding stress effects (e.g., Cahill et al., 2003). The consolidation model has been quite successful in accounting for a variety of results from the animal literature (Joëls et al., 2011; McGaugh, 2000; Roozendaal, 2002), so the problems that it has accounting for human episodic memory should not necessarily be seen as undermining the utility of that model to account for the animal literature.

The executive control theory of stress and memory (Gagnon & Wagner, 2016) can account naturally for a number of the results from studies examining the effects of stress on encoding and retrieval, but it runs into other problems with those studies, and it fails to account for the effects of postencoding stress. That is, if stress generally limits executive control process and draws attention toward the stress-related materials (Mather & Sutherland, 2011), stress should generally reduce both encoding of information not relevant to the stressor and memory retrieval, as was generally observed. Moreover, at time of encoding, to the extent that stress draws attention toward stress-relevant materials, stress should enhance memory encoding for stress relevant materials, as was also observed. In addition, the executive control account can also explain why effects of stress on encoding become more impairing with a longer delay between stress and encoding, because stress effects on executive functions such as working memory become more impairing with a longer delay as well (Shields, Sazma, et al., 2016).

The executive control theory does not make strong a priori predictions about the effects of the emotionality of the materials, but it could account for the finding that stress at time of retrieval seems to impair memory for emotional materials more so than neutral materials. That is, the effects of stress on executive processes, such as monitoring and retrieval orientation, may be more critical in the case of emotionally charged items than in the case of neutral items. For example, emotional stimuli require more executive control to ignore than do neutral items (e.g., Shields, Kuchenbecker, Pressman, Sumida, & Slavich, 2016), and divided attention manipulations at test can reduce recognition memory for emotional more than neutral materials, suggesting that executive processes are more critical for emotional than neutral materials during retrieval (Maddox, Naveh-Benjamin, Old, & Kilb, 2012). Nonetheless, further work aimed at determining whether the effects of stress on retrieval reflect alteration in executive processes would be useful.

One potential problem for the executive control account is that to the extent that recall is more heavily dependent on executive control than is recognition (Gagnon & Wagner, 2016), the encoding and retrieval deficits should have been larger for recall than recognition, and there was little evidence for this. However, it should be acknowledged that most studies that have directly contrasted recall and recognition had their recognition tests follow the free recall tests, and any carry-over effects may be expected to make the recognition and recall results more similar. Thus, additional studies that directly contrast the magnitude of the recall and recognition effects will be important in testing the executive con-

trol theories. In addition, only a small number of studies have examined the differential effects of stress on the processes that contribute to recall and recognition such as recollection and familiarity, thus future studies examining whether stress differentially impacts these different forms of episodic memory will also be important (McCullough et al., 2015; McCullough & Yonelinas, 2013). Regardless of the recall/recognition results, the executive function theories are limited in that they do not provide an explanation for why postencoding stress would improve memory. Given the consistency of those results, they point to an important limitation of the executive control approach.

The dual-mode model (Cadle & Zoladz, 2015; Diamond et al., 2007; Joëls et al., 2011; Schwabe et al., 2012) can account for a number of the encoding, postencoding, and retrieval effects of stress. Indeed, this model incorporates aspects of the traditional consolidation model and the executive control approaches. For example, because both the memory formation mode and memory storage mode are expected to enhance memory for recently encoded information (Schwabe et al., 2012), postencoding stress should improve memory, as was found. Moreover, because both modes lead to an inhibition of memory retrieval processes (Cadle & Zoladz, 2015; Schwabe et al., 2012), stress before or during retrieval should impair memory, as was also observed. In addition, in line with our current findings, this model predicts that stress prior to encoding will impair encoding unless the stress-encoding delay is very short, in which case the memory formation mode is expected to lead to an improvement in memory for stress-relevant materials (Schwabe et al., 2012).

However, as with the traditional consolidation models, the dual-mode approach predicts that the encoding and postencoding effects of stress should be greatest for emotional materials (Schwabe et al., 2012), and there was little direct support for this prediction in either the encoding or postencoding stress studies. Moreover, how the dual-mode theory would account for the finding that stress only improved memory when it occurred in the same spatial context as the study materials is also not clear. We note that Joëls et al. (2006) suggested that context changes may play an important role in determining the effects of stress on encoding. That is, they argued that for stress to enhance memory encoding, it may be necessary for the study materials to be presented while both noradrenergic activity and glucocorticoid activity is high. Thus, if subjects are stressed in one context then moved to another context to encode materials into memory, stress will no longer facilitate encoding, as noradrenergic activity will have presumably returned to baseline. Although it is not obvious how such an account could explain the fact that postencoding benefits of stress are eliminated when the study-stress context changes (as the delay between encoding and stress is the same in these two types of studies), it is possible that the model could be modified to account for these results.

Another somewhat troubling aspect of the results for the dual-mode models is that stress at retrieval was found to impair negative and positive materials more than neutral materials. One potential account is to argue that the inhibition of retrieval processes produced by the "memory formation" mode may impair the ability to simultaneously retrieve information, especially information closely related to current circumstances (Cadle & Zoladz, 2015; Schwabe et al., 2012). However, this explanation does not account for why stress impairs retrieval of positive information more than neutral information.

These inconsistencies notwithstanding, the dual-mode model is notable in that it successfully predicted many of the effects we observed within the meta-analysis (Schwabe et al., 2012). Whether the dual-mode model can be extended to account for the context-dependent effect of postencoding stress or the effects of valence has yet to be seen, but this model's unusual a priori predictions, such as a significant effect of the stress-encoding delay (Diamond et al., 2007; Joëls et al., 2011; Schwabe et al., 2012), were supported by our data. We also note that the model has received support from various animal studies as well (Akirav & Richter-Levin, 1999, 2002; Diamond et al., 2007).

Perhaps one of the more surprising and theoretically challenging findings of the current review was that postencoding stress improved memory only when the stressor occurred in the same context as the study event. Although as suggested above there may be ways in which existing models (e.g., Joëls et al., 2006) can be modified to account for these results, another possibility is that the postencoding stress effects may not rely on consolidation mechanisms per se, but rather may reflect changes in postencoding interference, or shifts in experimental context (Sazma, McCullough, & Yonelinas, 2016). For example, postencoding stress may benefit memory for the study list because it reduces the episodic encoding of information occurring after the study list, and so it reduces interference. Such a reduction in interference would be expected to be particularly important when the stressor and the study event shared the same context. That is, because episodic memory tasks like recall and recognition require the retrieval of item-context bindings (i.e., subjects must indicate if an item was encountered in the specified study context), increasing the number of items encoded in that context should increase the amount of interference. However, if the stress/control manipulation occurs in a different context than the study event, then stress should have much less of an impact on interference, as the current results seem to suggest. This contextual binding account is also consistent with the finding that postencoding stress benefits memory for both emotional and neutral materials, as long as they occur in the same context as the stressor, which was another finding that was problematic for the consolidation accounts. A related possibility is that stress may act to produce a shift in mental context, and this shift acts to isolate the earlier study list from the interfering effects of information encoded after the stressor. In this way, changing physical context by shifting rooms, or changing mental context by inducing stress may act to reduce retroactive interference, and effectively slow forgetting. We acknowledge that these accounts are entirely speculative, and so future studies that aim to contrast these different explanations of postencoding stress will be critical, particularly if existing models (e.g., Joëls et al., 2006) cannot be extended to account for this context-dependent postencoding stress effect.

An unexpected finding was the lack of association of cortisol with stress effects on memory. Stress-induced increases in cortisol were not associated with stress effects on memory when stress occurred prior to or during encoding, shortly after encoding, or before retrieval. This result is at odds with prior work that has found that pharmacological manipulations of glucocorticoids produce effects on memory similar to those of stress (Het et al., 2005; Joëls et al., 2011; Roozendaal, 2002; Roozendaal & McGaugh, 1997; Schwabe et al., 2012), or that that blocking actions of glucocorticoids can block effects of stress on memory (de Quer-

vain et al., 1998; Roozendaal, 2002; Schwabe et al., 2012; Vogel, Fernández, Joëls, & Schwabe, 2016). There are a number of potential reasons that may explain why cortisol was not found to be related to memory in the current review. For example, nonlinearities in the cortisol-memory relationship may have obscured any noticeable effects in the meta-analysis. That is, there is evidence from animal and human studies that stress-induced cortisol increases may have inverted U-shape effects on memory performance, and so averaging across subjects may mask such a relationship. Similarly, the cortisol-memory relationship may be obscured because of interactions with other factors, such as arousal. That is, glucocorticoids critically interact with noradrenaline to contribute to stress effects on memory (Joëls et al., 2011; Roozendaal & McGaugh, 1997; Schwabe et al., 2012; van Stegeren, Roozendaal, Kindt, Wolf, & Joëls, 2010). For example, administration of a noradrenergic antagonist blocks the enhancing memory-enhancing effects of postlearning glucocorticoid administration (Roozendaal & McGaugh, 1997). It is possible that various paradigms influence noradrenaline and cortisol to varying degrees, and these differences would be held constant within a study but not across studies, which could mask associations of cortisol with memory—because cortisol interacts with noradrenaline to contribute to effects of stress on memory. Thus, by not concurrently examining noradrenergic activity across studies, correlations of stress effects on cortisol with stress effects on memory may fail to return significant despite cortisol playing a role in effects of stress on memory.

Our data thus suggest that for understanding the biological basis of stress effects on memory, it may be important to look beyond manipulations or measures of cortisol alone and instead to measuring or simultaneously manipulating multiple hormones and immune system processes influenced by stress. Indeed, our data suggest that sex hormones may play an important role in modulating stress effects on memory, and there is evidence to suggest that other stress-responsive hormones, such as DHEA (Sripada, Welsh, Marx, & Liberzon, 2014; Yabuki et al., 2015), and immune system processes (Harrison et al., 2014; Reichenberg et al., 2001) may influence memory. Thus, we suggest future stress and memory research consider factors other than cortisol alone when considering the biological level of analysis of stress effects on memory.

Conclusion

In this meta-analysis, we examined the results of studies assessing effects of acute stress on memory. Because encoding, postencoding, retrieval, and postreactivation phases of memory all differ, many of the effects of stress were selective to specific phases of memory. For example, effects on encoding were strongly moderated by item relevance to the stressor and by the delay between stress and encoding, but these variables did not moderate stress effects on other memory phases. Nonetheless, some general trends did emerge. Overall, postencoding stress tended to enhance memory, whereas stress at retrieval impaired memory, and stress at encoding could enhance or impair memory depending upon key moderators. Similarly, hormonal contraceptive use blunted effects of stress during the encoding, postencoding period, retrieval phases, indicating that sex hormones play an important role in stress effects on memory. Surprisingly, effects of stress on cortisol

did not predict effects of stress on memory during any memory phase, indicating that stress may act through pathways in addition to cortisol to influence memory.

It is our hope that by synthesizing the wealth of stress and memory data that have been collected to date, we can aid future stress and cognition researchers in designing effective studies that test critical theories, while minimizing the noise from additional factors. By quantitatively elucidating factors that modulate stress effects on encoding, postencoding, retrieval, and postreactivation phases of memory, we hope that our meta-analysis will be useful in helping researchers achieve this goal.

References

Ackermann, S., Spalek, K., Rasch, B., Gschwind, L., Coynel, D., Fastenrath, M., . . . de Quervain, D. J.-F. (2012). Testosterone levels in healthy men are related to amygdala reactivity and memory performance. *Psychoneuroendocrinology, 37,* 1417–1424. http://dx.doi.org/10.1016/j.psyneuen.2012.01.008

Akirav, I., & Richter-Levin, G. (1999). Biphasic modulation of hippocampal plasticity by behavioral stress and basolateral amygdala stimulation in the rat. *The Journal of Neuroscience, 19,* 10530–10535. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10575049

Akirav, I., & Richter-Levin, G. (2002). Mechanisms of amygdala modulation of hippocampal plasticity. *The Journal of Neuroscience, 22,* 9912–9921. http://www.ncbi.nlm.nih.gov/pubmed/12427848

Alexander, J. K., Hillier, A., Smith, R. M., Tivarus, M. E., & Beversdorf, D. Q. (2007). Beta-adrenergic modulation of cognitive flexibility during stress. *Journal of Cognitive Neuroscience, 19,* 468–478. http://dx.doi.org/10.1162/jocn.2007.19.3.468

Allen, A. P., Kennedy, P. J., Cryan, J. F., Dinan, T. G., & Clarke, G. (2014). Biological and psychological markers of stress in humans: Focus on the Trier Social Stress Test. *Neuroscience and Biobehavioral Reviews, 38,* 94–124. http://dx.doi.org/10.1016/j.neubiorev.2013.11.005

Almela, M., Hidalgo, V., Villada, C., Espín, L., Gómez-Amor, J., & Salvador, A. (2011). The impact of cortisol reactivity to acute stress on memory: Sex differences in middle-aged people. *Stress, 14,* 117–127. http://dx.doi.org/10.3109/10253890.2010.514671

Andreano, J. M., Arjomandi, H., & Cahill, L. (2008). Menstrual cycle modulation of the relationship between cortisol and long-term memory. *Psychoneuroendocrinology, 33,* 874–882. http://dx.doi.org/10.1016/j.psyneuen.2008.03.009

Andreano, J. M., & Cahill, L. (2006). Glucocorticoid release and memory consolidation in men and women. *Psychological Science, 17,* 466–470. http://dx.doi.org/10.1111/j.1467-9280.2006.01729.x

Andreano, J. M., & Cahill, L. (2009). Sex influences on the neurobiology of learning and memory. *Learning & Memory, 16,* 248–266. http://dx.doi.org/10.1101/lm.918309

Andreano, J., Waisman, J., Donley, L., & Cahill, L. (2012). Effects of breast cancer treatment on the hormonal and cognitive consequences of acute stress. *Psycho-Oncology, 21,* 1091–1098. http://dx.doi.org/10.1002/pon.2006

Arnsten, A. F. T. (2009). Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience, 10,* 410–422. http://dx.doi.org/10.1038/nrn2648

Audet, M.-C., Jacobson-Pick, S., Wann, B. P., & Anisman, H. (2011). Social defeat promotes specific cytokine variations within the prefrontal cortex upon subsequent aggressive or endotoxin challenges. *Brain, Behavior, and Immunity, 25,* 1197–1205. http://dx.doi.org/10.1016/j.bbi.2011.03.010

Azimi, Z., & Bakhshipour-Roudsari, A. (2012). The impairing role of stress on autobiographical memory reconsolidation. *Zahedan Journal of Research in Medical Sciences, 14,* 51–55. Retrieved from http://www.zjrms.ir/article-1-2007-en.html

Barros, L. A., Tufik, S., & Andersen, M. L. (2015). The role of progesterone in memory: An overview of three decades. *Neuroscience and Biobehavioral Reviews, 49,* 193–204. http://dx.doi.org/10.1016/j.neubiorev.2014.11.015

Beckner, V. E., Tucker, D. M., Delville, Y., & Mohr, D. C. (2006). Stress facilitates consolidation of verbal memory for a film but does not affect retrieval. *Behavioral Neuroscience, 120,* 518–527. http://dx.doi.org/10.1037/0735-7044.120.3.518

Besnard, A., Caboche, J., & Laroche, S. (2012). Reconsolidation of memory: A decade of debate. *Progress in Neurobiology, 99,* 61–80. http://dx.doi.org/10.1016/j.pneurobio.2012.07.002

Bierhaus, A., Wolf, J., Andrassy, M., Rohleder, N., Humpert, P. M., Petrov, D., . . . Nawroth, P. P. (2003). A mechanism converting psychosocial stress into mononuclear cell activation. *Proceedings of the National Academy of Sciences of the United States of America, 100,* 1920–1925. http://dx.doi.org/10.1073/pnas.0438019100

Blumenfeld, R. S., & Ranganath, C. (2007). Prefrontal cortex and long-term memory encoding: An integrative review of findings from neuropsychology and neuroimaging. *The Neuroscientist, 13,* 280–291. http://dx.doi.org/10.1177/1073858407299290

Boehringer, A., Schwabe, L., & Schachinger, H. (2010). A combination of high stress-induced tense and energetic arousal compensates for impairing effects of stress on memory retrieval in men. *Stress, 13,* 444–453. http://dx.doi.org/10.3109/10253891003725256

Borenstein, M., Hedges, L. V., Higgins, J. P. T., & Rothstein, H. R. (2009). *Introduction to meta-analysis.* Chichester, UK: Wiley. http://dx.doi.org/10.1002/9780470743386

Bos, M. G. N., Jacobs van Goethem, T. H., Beckers, T., & Kindt, M. (2014). Cortisol response mediates the effect of post-reactivation stress exposure on contextualization of emotional memories. *Psychoneuroendocrinology, 50,* 72–84. http://dx.doi.org/10.1016/j.psyneuen.2014.07.030

Bos, M. G. N., Schuijer, J., Lodestijn, F., Beckers, T., & Kindt, M. (2014). Stress enhances reconsolidation of declarative memory. *Psychoneuroendocrinology, 46,* 102–113. http://dx.doi.org/10.1016/j.psyneuen.2014.04.011

Bryant, R. A., McGrath, C., Felmingham, K. L., Brewin, C., Gregory, J., Lipton, M., . . . Schachinger, H. (2013). The roles of noradrenergic and glucocorticoid activation in the development of intrusive memories. *PLoS ONE, 8,* e62675. http://dx.doi.org/10.1371/journal.pone.0062675

Buchanan, T. W., & Tranel, D. (2008). Stress and emotional memory retrieval: Effects of sex and cortisol response. *Neurobiology of Learning and Memory, 89,* 134–141. http://dx.doi.org/10.1016/j.nlm.2007.07.003

Buchanan, T. W., Tranel, D., & Adolphs, R. (2006). Impaired memory retrieval correlates with individual differences in cortisol response but not autonomic response. *Learning & Memory, 13,* 382–387. http://dx.doi.org/10.1101/lm.206306

Butts, K. A., Weinberg, J., Young, A. H., & Phillips, A. G. (2011). Glucocorticoid receptors in the prefrontal cortex regulate stress-evoked dopamine efflux and aspects of executive function. *Proceedings of the National Academy of Sciences of the United States of America, 108,* 18459–18464. http://dx.doi.org/10.1073/pnas.1111746108

Cadle, C. E., & Zoladz, P. R. (2015). Stress time-dependently influences the acquisition and retrieval of unrelated information by producing a memory of its own. *Frontiers in Psychology, 6,* 910. http://dx.doi.org/10.3389/fpsyg.2015.00910

Cahill, L., Gorski, L., & Le, K. (2003). Enhanced human memory consolidation with post-learning stress: Interaction with the degree of arousal at encoding. *Learning & Memory, 10,* 270–274. http://dx.doi.org/10.1101/lm.62403

Cahill, L., & McGaugh, J. L. (1998). Mechanisms of emotional arousal and lasting declarative memory. *Trends in Neurosciences, 21,* 294–299. http://dx.doi.org/10.1016/S0166-2236(97)01214-9

Cavenett, T., & Nixon, R. D. V. (2006). The effect of arousal on memory for emotionally-relevant information: A study of skydivers. *Behaviour Research and Therapy, 44,* 1461–1469. http://dx.doi.org/10.1016/j.brat.2005.11.002

Childs, E., & de Wit, H. (2009). Hormonal, cardiovascular, and subjective responses to acute stress in smokers. *Psychopharmacology, 203,* 1–12. http://dx.doi.org/10.1007/s00213-008-1359-5

Chiu, Y.-C., & Egner, T. (2015). Inhibition-induced forgetting: When more control leads to less memory. *Psychological Science, 26,* 27–38. http://dx.doi.org/10.1177/0956797614553945

Coccoz, V., Maldonado, H., & Delorenzi, A. (2011). The enhancement of reconsolidation with a naturalistic mild stressor improves the expression of a declarative memory in humans. *Neuroscience, 185,* 61–72. http://dx.doi.org/10.1016/j.neuroscience.2011.04.023

Coccoz, V., Sandoval, A. V., Stehberg, J., & Delorenzi, A. (2013). The temporal dynamics of enhancing a human declarative memory during reconsolidation. *Neuroscience, 246,* 397–408. http://dx.doi.org/10.1016/j.neuroscience.2013.04.033

Conboy, L., & Sandi, C. (2010). Stress at learning facilitates memory formation by regulating AMPA receptor trafficking through a glucocorticoid action. *Neuropsychopharmacology, 35,* 674–685. http://dx.doi.org/10.1038/npp.2009.172

Conrad, C. D. (2010). A critical review of chronic stress effects on spatial learning and memory. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 34,* 742–755. http://dx.doi.org/10.1016/j.pnpbp.2009.11.003

Conrad, C. D., Lupien, S. J., & McEwen, B. S. (1999). Support for a bimodal role for type II adrenal steroid receptors in spatial memory. *Neurobiology of Learning and Memory, 72,* 39–46. http://dx.doi.org/10.1006/nlme.1998.3898

Cornelisse, S., Joëls, M., & Smeets, T. (2011). A randomized trial on mineralocorticoid receptor blockade in men: Effects on stress responses, selective attention, and memory. *Neuropsychopharmacology, 36,* 2720–2728. http://dx.doi.org/10.1038/npp.2011.162

Cornelisse, S., van Stegeren, A. H., & Joels, M. (2011). Implications of psychosocial stress on memory formation in a typical male versus female student sample. *Psychoneuroendocrinology, 36,* 569–578. http://dx.doi.org/10.1016/j.psyneuen.2010.09.002

Dantzer, R., O'Connor, J. C., Freund, G. G., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: When the immune system subjugates the brain. *Nature Reviews Neuroscience, 9,* 46–56. http://dx.doi.org/10.1038/nrn2297

Davachi, L. (2006). Item, context and relational episodic encoding in humans. *Current Opinion in Neurobiology, 16,* 693–700. http://dx.doi.org/10.1016/j.conb.2006.10.012

de Kloet, E. R., Oitzl, M. S., & Joëls, M. (1999). Stress and cognition: Are corticosteroids good or bad guys? *Trends in Neurosciences, 22,* 422–426. http://dx.doi.org/10.1016/S0166-2236(99)01438-1

de Quervain, D. J-F., Aerni, A., & Roozendaal, B. (2007). Preventive effect of beta-adrenoceptor blockade on glucocorticoid-induced memory retrieval deficits. *The American Journal of Psychiatry, 164,* 967–969. http://dx.doi.org/10.1176/appi.ajp.164.6.967

de Quervain, D. J-F., Roozendaal, B., & McGaugh, J. L. (1998). Stress and glucocorticoids impair retrieval of long-term spatial memory. *Nature, 394,* 787–790. http://dx.doi.org/10.1038/29542

de Quervain, D. J-F., Roozendaal, B., Nitsch, R. M., McGaugh, J. L., & Hock, C. (2000). Acute cortisone administration impairs retrieval of long-term declarative memory in humans. *Nature Neuroscience, 3,* 313–314. http://dx.doi.org/10.1038/73873

Diamond, D. M., Campbell, A. M., Park, C. R., Halonen, J., & Zoladz, P. R. (2007). The temporal dynamics model of emotional memory processing: A synthesis on the neurobiological basis of stress-induced amnesia, flashbulb and traumatic memories, and the Yerkes-Dodson law. *Neural Plasticity, 2007,* 60803.

Diamond, D. M., Campbell, A. M., Park, C. R., Woodson, J. C., Conrad, C. D., Bachstetter, A. D., & Mervis, R. F. (2006). Influence of predator stress on the consolidation versus retrieval of long-term spatial memory and hippocampal spinogenesis. *Hippocampus, 16,* 571–576. http://dx.doi.org/10.1002/hipo.20188

Diana, R. A., Yonelinas, A. P., & Ranganath, C. (2007). Imaging recollection and familiarity in the medial temporal lobe: A three-component model. *Trends in Cognitive Sciences, 11,* 379–386. http://dx.doi.org/10.1016/j.tics.2007.08.001

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130,* 355–391. http://dx.doi.org/10.1037/0033-2909.130.3.355

Dobbins, I. G., Foley, H., Schacter, D. L., & Wagner, A. D. (2002). Executive control during episodic retrieval: Multiple prefrontal processes subserve source memory. *Neuron, 35,* 989–996. http://dx.doi.org/10.1016/S0896-6273(02)00858-9

Domes, G., Heinrichs, M., Reichwald, U., & Hautzinger, M. (2002). Hypothalamic-pituitary-adrenal axis reactivity to psychological stress and memory in middle-aged women: High responders exhibit enhanced declarative memory performance. *Psychoneuroendocrinology, 27,* 843–853. http://dx.doi.org/10.1016/S0306-4530(01)00085-3

Domes, G., Heinrichs, M., Rimmele, U., Reichwald, U., & Hautzinger, M. (2004). Acute stress impairs recognition for positive words–association with stress-induced cortisol secretion. *Stress, 7,* 173–181. http://dx.doi.org/10.1080/10253890412331273213

Dudai, Y. (2004). The neurobiology of consolidations, or, how stable is the engram? *Annual Review of Psychology, 55,* 51–86. http://dx.doi.org/10.1146/annurev.psych.55.090902.142050

du Plooy, C. P. (2014). *The effects of acute stress on retrieval of visual and spatial material.* University of Cape Town. Retrieved from http://www.bme.uct.ac.za/sites/default/files/image_tool/images/150/THESIS_DuPlooy_2015_PhD.pdf

Duval, S. (2005). The trim and fill method. In H. R. Rothstein, A. J. Sutton, & M. Borenstein (Eds.), *Publication bias in meta-analysis: Prevention, assessment, and adjustments* (pp. 127–144). New York, NY: Wiley.

Duval, S., & Tweedie, R. (2000a). A nonparametric "trim and fill" method of accounting for publication bias in meta-analysis. *Journal of the American Statistical Society, 95,* 89–98.

Duval, S., & Tweedie, R. (2000b). Trim and fill: A simple funnel-plot-based method of testing and adjusting for publication bias in meta-analysis. *Biometrics, 56,* 455–463. http://dx.doi.org/10.1111/j.0006-341X.2000.00455.x

Egger, M., Davey Smith, G., Schneider, M., & Minder, C. (1997). Bias in meta-analysis detected by a simple, graphical test. *BMJ (Clinical Research Ed.), 315,* 629–634. http://dx.doi.org/10.1136/bmj.315.7109.629

Eich, T. S., & Metcalfe, J. (2009). Effects of the stress of marathon running on implicit and explicit memory. *Psychonomic Bulletin & Review, 16,* 475–479. http://dx.doi.org/10.3758/PBR.16.3.475

Eichenbaum, H., Otto, T., & Cohen, N. J. (1992). The hippocampus—What does it do? *Behavioral & Neural Biology, 57,* 2–36. http://dx.doi.org/10.1016/0163-1047(92)90724-I

Eichenbaum, H., Yonelinas, A. P., & Ranganath, C. (2007). The medial temporal lobe and recognition memory. *Annual Review of Neuroscience, 30,* 123–152. http://dx.doi.org/10.1146/annurev.neuro.30.051606.094328

Espin, L., Almela, M., Hidalgo, V., Villada, C., Salvador, A., & Gomez-Amor, J. (2013). Acute pre-learning stress and declarative memory: Impact of sex, cortisol response and menstrual cycle phase. *Hormones and Behavior, 63,* 759–765. http://dx.doi.org/10.1016/j.yhbeh.2013.03.013

Felmingham, K. L., Fong, W. C., & Bryant, R. A. (2012). The impact of progesterone on memory consolidation of threatening images in women.

Psychoneuroendocrinology, 37, 1896–1900. http://dx.doi.org/10.1016/j.psyneuen.2012.03.026

Felmingham, K. L., Tran, T. P., Fong, W. C., & Bryant, R. A. (2012). Sex differences in emotional memory consolidation: The effect of stress-induced salivary alpha-amylase and cortisol. *Biological Psychology, 89,* 539–544. http://dx.doi.org/10.1016/j.biopsycho.2011.12.006

Finsterwald, C., & Alberini, C. M. (2014). Stress and glucocorticoid receptor-dependent mechanisms in long-term memory: From adaptive responses to psychopathologies. *Neurobiology of Learning and Memory, 112,* 17–29. http://dx.doi.org/10.1016/j.nlm.2013.09.017

Fletcher, P. C., Shallice, T., Frith, C. D., Frackowiak, R. S. J., & Dolan, R. J. (1998). The functional roles of prefrontal cortex in episodic memory. II. Retrieval. *Brain: A Journal of Neurology, 121,* 1249–1256. http://dx.doi.org/10.1093/brain/121.7.1249

Gagnon, S. A., & Wagner, A. D. (2016). Acute stress and episodic memory retrieval: Neurobiological mechanisms and behavioral consequences. *Annals of the New York Academy of Sciences, 1369,* 55–75. http://dx.doi.org/10.1111/nyas.12996

Gershberg, F. B., & Shimamura, A. P. (1995). Impaired use of organizational strategies in free recall following frontal lobe damage. *Neuropsychologia, 33,* 1305–1333. http://dx.doi.org/10.1016/0028-3932(95)00103-A

Giles, G. E., Mahoney, C. R., Brunyé, T. T., Taylor, H. A., & Kanarek, R. B. (2014). Stress effects on mood, HPA axis, and autonomic response: Comparison of three psychosocial stress paradigms. *PLoS ONE, 9,* e113618. http://dx.doi.org/10.1371/journal.pone.0113618

Graham, B. M., & Milad, M. R. (2013). Blockade of estrogen by hormonal contraceptives impairs fear extinction in female rats and women. *Biological Psychiatry, 73,* 371–378. http://dx.doi.org/10.1016/j.biopsych.2012.09.018

Harburger, L. L., Pechenino, A. S., Saadi, A., & Frick, K. M. (2008). Post-training progesterone dose-dependently enhances object, but not spatial, memory consolidation. *Behavioural Brain Research, 194,* 174–180. http://dx.doi.org/10.1016/j.bbr.2008.07.014

Harrison, N. A., Cercignani, M., Voon, V., & Critchley, H. D. (2015). Effects of inflammation on hippocampus and substantia nigra responses to novelty in healthy human participants. *Neuropsychopharmacology, 40,* 831–838. http://dx.doi.org/10.1038/npp.2014.222

Harrison, N. A., Doeller, C. F., Voon, V., Burgess, N., & Critchley, H. D. (2014). Peripheral inflammation acutely impairs human spatial memory via actions on medial temporal lobe glucose metabolism. *Biological Psychiatry, 76,* 585–593. http://dx.doi.org/10.1016/j.biopsych.2014.01.005

Hedges, L. V., Tipton, E., & Johnson, M. C. (2010). Robust variance estimation in meta-regression with dependent effect size estimates. *Research Synthesis Methods, 1,* 39–65. http://dx.doi.org/10.1002/jrsm.5

Henckens, M. J. A. G., Hermans, E. J., Pu, Z., Joëls, M., & Fernández, G. (2009). Stressed memories: How acute stress affects memory formation in humans. *The Journal of Neuroscience, 29,* 10111–10119. http://dx.doi.org/10.1523/JNEUROSCI.1184-09.2009

Henson, R. N. A., Rugg, M. D., Shallice, T., & Dolan, R. J. (2000). Confidence in recognition memory for words: Dissociating right prefrontal roles in episodic retrieval. *Journal of Cognitive Neuroscience, 12,* 913–923. http://dx.doi.org/10.1162/08989290051137468

Het, S., Ramlow, G., & Wolf, O. T. (2005). A meta-analytic review of the effects of acute cortisol administration on human memory. *Psychoneuroendocrinology, 30,* 771–784. http://dx.doi.org/10.1016/j.psyneuen.2005.03.005

Hidalgo, V., Almela, M., Villada, C., & Salvador, A. (2014). Acute stress impairs recall after interference in older people, but not in young people. *Hormones and Behavior, 65,* 264–272. http://dx.doi.org/10.1016/j.yhbeh.2013.12.017

Hidalgo, V., Pulopulos, M. M., Puig-Perez, S., Espin, L., Gomez-Amor, J., & Salvador, A. (2015). Acute stress affects free recall and recognition of pictures differently depending on age and sex. *Behavioural Brain Research, 292,* 393–402. http://dx.doi.org/10.1016/j.bbr.2015.07.011

Hidalgo, V., Villada, C., Almela, M., Espín, L., Gómez-Amor, J., & Salvador, A. (2012). Enhancing effects of acute psychosocial stress on priming of non-declarative memory in healthy young adults. *Stress, 15,* 329–338. http://dx.doi.org/10.3109/10253890.2011.624224

Hoffman, R., & Al'Absi, M. (2004). The effect of acute stress on subsequent neuropsychological test performance. *Archives of Clinical Neuropsychology, 19,* 497–506. http://dx.doi.org/10.1016/j.acn.2003.07.005

Hoscheidt, S. M. (2011). *Impairing and enhancing effects of psychosocial stress on episodic memory and eyewitness report.* University of Arizona. Retrieved from http://hdl.handle.net/10150/202762.

Hoscheidt, S. M., LaBar, K. S., Ryan, L., Jacobs, W. J., & Nadel, L. (2014). Encoding negative events under stress: High subjective arousal is related to accurate emotional memory despite misinformation exposure. *Neurobiology of Learning and Memory, 112,* 237–247. http://dx.doi.org/10.1016/j.nlm.2013.09.008

Human, R. (2010). *The effects of acute psychological stress on working memory and verbal declarative memory.* Cape Town, South Africa: University of Cape Town. Retrieved from http://www.dpru.uct.ac.za/sites/default/files/image_tool/images/117/Robyn.Human_.pdf

Human, R., Thomas, K. G., Dreyer, A., Amod, a. R., Wolf, P. S., & Jacobs, W. J. (2013). Acute psychosocial stress enhances visuospatial memory in healthy males. *South African Journal of Psychology, 43,* 300–313. http://dx.doi.org/10.1177/0081246313496913

Hupbach, A., & Dorskind, J. M. (2014). Stress selectively affects the reactivated components of a declarative memory. *Behavioral Neuroscience, 128,* 614–620. http://dx.doi.org/10.1037/bne0000006

Hupbach, A., & Fieman, R. (2012). Moderate stress enhances immediate and delayed retrieval of educationally relevant material in healthy young men. *Behavioral Neuroscience, 126,* 819–825. http://dx.doi.org/10.1037/a0030489

Iidaka, T., Anderson, N. D., Kapur, S., Cabez, R., & Craik, F. I. (2000). The effect of divided attention on encoding and retrieval in episodic memory revealed by positron emission tomography. *Journal of Cognitive Neuroscience, 12,* 267–280. http://dx.doi.org/10.1162/089892900562093

Inagaki, T., Gautreaux, C., & Luine, V. (2010). Acute estrogen treatment facilitates recognition memory consolidation and alters monoamine levels in memory-related brain areas. *Hormones and Behavior, 58,* 415–426. http://dx.doi.org/10.1016/j.yhbeh.2010.05.013

Inagaki, T. K., Muscatell, K. A., Irwin, M. R., Cole, S. W., & Eisenberger, N. I. (2012). Inflammation selectively enhances amygdala activity to socially threatening images. *NeuroImage, 59,* 3222–3226. http://dx.doi.org/10.1016/j.neuroimage.2011.10.090

Incisa della Rocchetta, A., & Milner, B. (1993). Strategic search and retrieval inhibition: The role of the frontal lobes. *Neuropsychologia, 31,* 503–524. http://dx.doi.org/10.1016/0028-3932(93)90049-6

Joëls, M., Fernandez, G., & Roozendaal, B. (2011). Stress and emotional memory: A matter of timing. *Trends in Cognitive Sciences, 15,* 280–288. http://dx.doi.org/10.1016/j.tics.2011.04.004

Joëls, M., Pu, Z., Wiegert, O., Oitzl, M. S., & Krugers, H. J. (2006). Learning under stress: How does it work? *Trends in Cognitive Sciences, 10,* 152–158. http://dx.doi.org/10.1016/j.tics.2006.02.002

Kirschbaum, C., Kudielka, B. M., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. *Psychosomatic Medicine, 61,* 154–162. http://dx.doi.org/10.1097/00006842-199903000-00006

Kirschbaum, C., Wüst, S., & Hellhammer, D. (1992). Consistent sex differences in cortisol responses to psychological stress. *Psychosomatic Medicine, 54,* 648–657. http://dx.doi.org/10.1097/00006842-199211000-00004

Kramár, E. A., Babayan, A. H., Gall, C. M., & Lynch, G. (2013). Estrogen promotes learning-related plasticity by modifying the synaptic cytoskeleton. *Neuroscience, 239,* 3–16. http://dx.doi.org/10.1016/j.neuroscience.2012.10.038

Kudielka, B. M., Schommer, N. C., Hellhammer, D. H., & Kirschbaum, C. (2004). Acute HPA axis responses, heart rate, and mood changes to psychosocial stress (TSST) in humans at different times of day. *Psychoneuroendocrinology, 29,* 983–992. http://dx.doi.org/10.1016/j.psyneuen.2003.08.009

Kuhlmann, S., Piel, M., & Wolf, O. T. (2005). Impaired memory retrieval after psychosocial stress in healthy young men. *The Journal of Neuroscience, 25,* 2977–2982. http://dx.doi.org/10.1523/JNEUROSCI.5139-04.2005

Larra, M. F., Schulz, A., Schilling, T. M., Ferreira de Sá, D. S., Best, D., Kozik, B., & Schächinger, H. (2014). Heart rate response to post-learning stress predicts memory consolidation. *Neurobiology of Learning and Memory, 109,* 74–81. http://dx.doi.org/10.1016/j.nlm.2013.12.004

Lennartsson, A.-K., Kushnir, M. M., Bergquist, J., Billig, H., & Jonsdottir, I. H. (2012). Sex steroid levels temporarily increase in response to acute psychosocial stress in healthy men and women. *International Journal of Psychophysiology, 84,* 246–253. http://dx.doi.org/10.1016/j.ijpsycho.2012.03.001

Levy, B. J., & Anderson, M. C. (2002). Inhibitory processes and the control of memory retrieval. *Trends in Cognitive Sciences, 6,* 299–305. http://dx.doi.org/10.1016/S1364-6613(02)01923-X

Li, S., Weerda, R., Guenzel, F., Wolf, O. T., & Thiel, C. M. (2013). ADRA2B genotype modulates effects of acute psychosocial stress on emotional memory retrieval in healthy young men. *Neurobiology of Learning and Memory, 103,* 11–18. http://dx.doi.org/10.1016/j.nlm.2013.03.006

Li, S., Weerda, R., Milde, C., Wolf, O. T., & Thiel, C. M. (2014). Effects of acute psychosocial stress on neural activity to emotional and neutral faces in a face recognition memory paradigm. *Brain Imaging and Behavior, 8,* 598–610. http://dx.doi.org/10.1007/s11682-013-9287-3

Liening, S. H., Stanton, S. J., Saini, E. K., & Schultheiss, O. C. (2010). Salivary testosterone, cortisol, and progesterone: Two-week stability, interhormone correlations, and effects of time of day, menstrual cycle, and oral contraceptive use on steroid hormone levels. *Physiology & Behavior, 99,* 8–16. http://dx.doi.org/10.1016/j.physbeh.2009.10.001

Luethi, M., Meier, B., & Sandi, C. (2008). Stress effects on working memory, explicit memory, and implicit memory for neutral and emotional stimuli in healthy men. *Frontiers in Behavioral Neuroscience, 2,* 5. http://dx.doi.org/10.3389/neuro.08.005.2008

Luethi, M., Meier, B., & Sandi, C. (2009). Stress effects on working memory, explicit memory, and implicit memory for neutral and emotional stimuli in healthy men. *Frontiers in Behavioral Neuroscience, 2,* 5.

Maddox, G. B., Naveh-Benjamin, M., Old, S., & Kilb, A. (2012). The role of attention in the associative binding of emotionally arousing words. *Psychonomic Bulletin & Review, 19,* 1128–1134. http://dx.doi.org/10.3758/s13423-012-0315-x

Maheu, F. S., Collicutt, P., Kornik, R., Moszkowski, R., & Lupien, S. J. (2005). The perfect time to be stressed: A differential modulation of human memory by stress applied in the morning or in the afternoon. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 29,* 1281–1288. http://dx.doi.org/10.1016/j.pnpbp.2005.08.012

Mangels, J. A. (1997). Strategic processing and memory for temporal order in patients with frontal lobe lesions. *Neuropsychology, 11,* 207–221. http://dx.doi.org/10.1037/0894-4105.11.2.207

Marin, M. F., Pilgrim, K., & Lupien, S. J. (2010). Modulatory effects of stress on reactivated emotional memories. *Psychoneuroendocrinology, 35,* 1388–1396. http://dx.doi.org/10.1016/j.psyneuen.2010.04.002

Marinari, K. T., Leshner, A. I., & Doyle, M. P. (1976). Menstrual cycle status and adrenocortical reactivity to psychological stress. *Psychoneuroendocrinology, 1,* 213–218. http://dx.doi.org/10.1016/0306-4530(76)90011-1

Maroun, M., & Richter-Levin, G. (2003). Exposure to acute stress blocks the induction of long-term potentiation of the amygdala-prefrontal cortex pathway in vivo. *The Journal of Neuroscience, 23,* 4406–4409. http://www.ncbi.nlm.nih.gov/pubmed/12805280

Mather, M., & Sutherland, M. R. (2011). Arousal-biased competition in perception and memory. *Perspectives on Psychological Science, 6,* 114–133. http://dx.doi.org/10.1177/1745691611400234

Mayes, A., Montaldi, D., & Migo, E. (2007). Associative memory and the medial temporal lobes. *Trends in Cognitive Sciences, 11,* 126–135. http://dx.doi.org/10.1016/j.tics.2006.12.003

McCullough, A. M., Ritchey, M., Ranganath, C., & Yonelinas, A. (2015). Differential effects of stress-induced cortisol responses on recollection and familiarity-based recognition memory. *Neurobiology of Learning and Memory, 123,* 1–10. http://dx.doi.org/10.1016/j.nlm.2015.04.007

McCullough, A. M., & Yonelinas, A. P. (2013). Cold-pressor stress after learning enhances familiarity-based recognition memory in men. *Neurobiology of Learning and Memory, 106,* 11–17. http://dx.doi.org/10.1016/j.nlm.2013.06.011

McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews, 87,* 873–904. http://dx.doi.org/10.1152/physrev.00041.2006

McGaugh, J. L. (2000). Memory—A century of consolidation. *Science, 287,* 248–251. http://dx.doi.org/10.1126/science.287.5451.248

McGaugh, J. L. (2004). The amygdala modulates the consolidation of memories of emotionally arousing experiences. *Annual Review of Neuroscience, 27,* 1–28. http://dx.doi.org/10.1146/annurev.neuro.27.070203.144157

McGaugh, J. L. (2015). Consolidating memories. *Annual Review of Psychology, 66,* 1–24. http://dx.doi.org/10.1146/annurev-psych-010814-014954

Merz, C. J., Wolf, O. T., & Hennig, J. (2010). Stress impairs retrieval of socially relevant information. *Behavioral Neuroscience, 124,* 288–293. http://dx.doi.org/10.1037/a0018942

Morris, S. B. (2008). Estimating effect sizes from pretest-posttest-control group designs. *Organizational Research Methods, 11,* 364–386. http://dx.doi.org/10.1177/1094428106291059

Morris, S. B., & DeShon, R. P. (2002). Combining effect size estimates in meta-analysis with repeated measures and independent-groups designs. *Psychological Methods, 7,* 105–125. http://dx.doi.org/10.1037/1082-989X.7.1.105

Nguyen, D. T. H. (2009). *The effects of psychosocial stress and social support on memory.* San Jose, CA: San Jose State University. Retrieved from http://scholarworks.sjsu.edu/etd_theses

Nielsen, S. E., Ahmed, I., & Cahill, L. (2014). Postlearning stress differentially affects memory for emotional gist and detail in naturally cycling women and women on hormonal contraceptives. *Behavioral Neuroscience, 128,* 482–493. http://dx.doi.org/10.1037/a0036687

Nielsen, S. E., Segal, S. K., Worden, I. V., Yim, I. S., & Cahill, L. (2013). Hormonal contraception use alters stress responses and emotional memory. *Biological Psychology, 92,* 257–266. http://dx.doi.org/10.1016/j.biopsycho.2012.10.007

Norman, K. A., & O'Reilly, R. C. (2003). Modeling hippocampal and neocortical contributions to recognition memory: A complementary-learning-systems approach. *Psychological Review, 110,* 611–646. http://dx.doi.org/10.1037/0033-295X.110.4.611

O'Connor, M.-F., Bower, J. E., Cho, H. J., Creswell, J. D., Dimitrov, S., Hamby, M. E., . . . Irwin, M. R. (2009). To assess, to control, to exclude: Effects of biobehavioral factors on circulating inflammatory markers. *Brain, Behavior, and Immunity, 23,* 887–897. http://dx.doi.org/10.1016/j.bbi.2009.04.005

Oei, N. Y. L., Everaerd, W. T. A. M., Elzinga, B. M., van Well, S., & Bermond, B. (2006). Psychosocial stress impairs working memory at high loads: An association with cortisol levels and memory retrieval. *Stress, 9,* 133–141. http://dx.doi.org/10.1080/10253890600965773

Parkin, A. J. (1997). Normal age-related memory loss and its relation to

frontal lobe dysfunction. In P. Rabbitt (Ed.), *Methodology of frontal and executive function* (pp. 171–194). New York, NY: Psychology Press.

Patel, P. D., Katz, M., Karssen, A. M., & Lyons, D. M. (2008). Stress-induced changes in corticosteroid receptor expression in primate hippocampus and prefrontal cortex. *Psychoneuroendocrinology, 33,* 360–367. http://dx.doi.org/10.1016/j.psyneuen.2007.12.003

Payne, J. D., Jackson, E. D., Hoscheidt, S., Ryan, L., Jacobs, W. J., & Nadel, L. (2007). Stress administered prior to encoding impairs neutral but enhances emotional long-term episodic memories. *Learning & Memory, 14,* 861–868. http://dx.doi.org/10.1101/lm.743507

Payne, J. D., Jackson, E. D., Hoscheidt, S., Ryan, L., Jacobs, W. J., & Nadel, L. (2007). Stress administered prior to encoding impairs neutral but enhances emotional long-term episodic memories. *Learning & Memory, 14,* 861–868. http://dx.doi.org/10.1101/lm.743507

Payne, J. D., Jackson, E. D., Ryan, L., Hoscheidt, S., Jacobs, J. W., & Nadel, L. (2006). The impact of stress on neutral and emotional aspects of episodic memory. *Memory, 14,* 1–16. http://dx.doi.org/10.1080/09658210500139176

Preuß, D., & Wolf, O. T. (2009). Post-learning psychosocial stress enhances consolidation of neutral stimuli. *Neurobiology of Learning and Memory, 92,* 318–326. http://dx.doi.org/10.1016/j.nlm.2009.03.009

Pulopulos, M. M., Almela, M., Hidalgo, V., Villada, C., Puig-Perez, S., & Salvador, A. (2013). Acute stress does not impair long-term memory retrieval in older people. *Neurobiology of Learning and Memory, 104,* 16–24. http://dx.doi.org/10.1016/j.nlm.2013.04.010

Qin, S., Hermans, E. J., van Marle, H. J. F., & Fernández, G. (2012). Understanding low reliability of memories for neutral information encoded under stress: Alterations in memory-related activation in the hippocampus and midbrain. *The Journal of Neuroscience, 32,* 4032–4041. http://dx.doi.org/10.1523/JNEUROSCI.3101-11.2012

Quaedflieg, C. W. E. M., Schwabe, L., Meyer, T., & Smeets, T. (2013). Time dependent effects of stress prior to encoding on event-related potentials and 24h delayed retrieval. *Psychoneuroendocrinology, 38,* 3057–3069. http://dx.doi.org/10.1016/j.psyneuen.2013.09.002

Raison, C. L., Capuron, L., & Miller, A. H. (2006). Cytokines sing the blues: Inflammation and the pathogenesis of depression. *Trends in Immunology, 27,* 24–31. http://dx.doi.org/10.1016/j.it.2005.11.006

Reichenberg, A., Yirmiya, R., Schuld, A., Kraus, T., Haack, M., Morag, A., & Pollmächer, T. (2001). Cytokine-associated emotional and cognitive disturbances in humans. *Archives of General Psychiatry, 58,* 445–452. http://dx.doi.org/10.1001/archpsyc.58.5.445

Robicheaux, T. R. (2015). *Stress and eyewitness memory: Timing of stressor and association with cortisol stress responding.* University of Nebraska. Retrieved from http://digitalcommons.unl.edu/psychdiss

Roozendaal, B. (2002). Stress and memory: Opposing effects of glucocorticoids on memory consolidation and memory retrieval. *Neurobiology of Learning and Memory, 78,* 578–595. http://dx.doi.org/10.1006/nlme.2002.4080

Roozendaal, B., & McGaugh, J. L. (1997). Basolateral amygdala lesions block the memory-enhancing effect of glucocorticoid administration in the dorsal hippocampus of rats. *European Journal of Neuroscience, 9,* 76–83. http://dx.doi.org/10.1111/j.1460-9568.1997.tb01355.x

Roozendaal, B., Okuda, S., de Quervain, D. J-F., & McGaugh, J. L. (2006). Glucocorticoids interact with emotion-induced noradrenergic activation in influencing different memory functions. *Neuroscience, 138,* 901–910. http://dx.doi.org/10.1016/j.neuroscience.2005.07.049

Roozendaal, B., Okuda, S., Van der Zee, E. A., & McGaugh, J. L. (2006). Glucocorticoid enhancement of memory requires arousal-induced noradrenergic activation in the basolateral amygdala. *Proceedings of the National Academy of Sciences of the United States of America, 103,* 6741–6746. http://dx.doi.org/10.1073/pnas.0601874103

Sakaki, M., & Mather, M. (2012). How reward and emotional stimuli induce different reactions across the menstrual cycle. *Social and Personality Psychology Compass, 6,* 1–17. http://dx.doi.org/10.1111/j.1751-9004.2011.00415.x

Sandi, C., & Pinelo-Nava, M. T. (2007). Stress and memory: Behavioral effects and neurobiological mechanisms. *Neural Plasticity, 2007,* 78970.

Sänger, J., Bechtold, L., Schoofs, D., Blaszkewicz, M., & Wascher, E. (2014). The influence of acute stress on attention mechanisms and its electrophysiological correlates. *Frontiers in Behavioral Neuroscience, 8,* 353.

Sapolsky, R., Rivier, C., Yamamoto, G., Plotsky, P., & Vale, W. (1987). Interleukin-1 stimulates the secretion of hypothalamic corticotropin-releasing factor. *Science, 238,* 522–524. http://dx.doi.org/10.1126/science.2821621

Sazma, M. A., McCullough, A. M., & Yonelinas, A. P. (2016). The importance of context for post-encoding stress effects on memory: An alternative to consolidation theory. Poster Presented at CNS in New York, NY.

Scammacca, N., Roberts, G., & Stuebing, K. K. (2014). Meta-analysis with complex research designs: Dealing with dependence from multiple measures and multiple group comparisons. *Review of Educational Research, 84,* 328–364. http://dx.doi.org/10.3102/0034654313500826

Schönfeld, P., Ackermann, K., & Schwabe, L. (2014). Remembering under stress: Different roles of autonomic arousal and glucocorticoids in memory retrieval. *Psychoneuroendocrinology, 39,* 249–256. http://dx.doi.org/10.1016/j.psyneuen.2013.09.020

Schoofs, D., & Wolf, O. T. (2009). Stress and memory retrieval in women: No strong impairing effect during the luteal phase. *Behavioral Neuroscience, 123,* 547–554. http://dx.doi.org/10.1037/a0015625

Schoofs, D., Wolf, O. T., & Smeets, T. (2009). Cold pressor stress impairs performance on working memory tasks requiring executive functions in healthy young men. *Behavioral Neuroscience, 123,* 1066–1075. http://dx.doi.org/10.1037/a0016980

Schwabe, L., Bohringer, A., Chatterjee, M., & Schachinger, H. (2008). Effects of pre-learning stress on memory for neutral, positive and negative words: Different roles of cortisol and autonomic arousal. *Neurobiology of Learning and Memory, 90,* 44–53. http://dx.doi.org/10.1016/j.nlm.2008.02.002

Schwabe, L., Haddad, L., & Schachinger, H. (2008). HPA axis activation by a socially evaluated Cold-Pressor Test. *Psychoneuroendocrinology, 33,* 890–895. http://dx.doi.org/10.1016/j.psyneuen.2008.03.001

Schwabe, L., Joëls, M., Roozendaal, B., Wolf, O. T., & Oitzl, M. S. (2012). Stress effects on memory: An update and integration. *Neuroscience and Biobehavioral Reviews, 36,* 1740–1749. http://dx.doi.org/10.1016/j.neubiorev.2011.07.002

Schwabe, L., Nader, K., & Pruessner, J. C. (2014). Reconsolidation of human memory: Brain mechanisms and clinical relevance. *Biological Psychiatry, 76,* 274–280. http://dx.doi.org/10.1016/j.biopsych.2014.03.008

Schwabe, L., Römer, S., Richter, S., Dockendorf, S., Bilak, B., & Schächinger, H. (2009). Stress effects on declarative memory retrieval are blocked by a β-adrenoceptor antagonist in humans. *Psychoneuroendocrinology, 34,* 446–454. http://dx.doi.org/10.1016/j.psyneuen.2008.10.009

Schwabe, L., & Wolf, O. T. (2009). The context counts: Congruent learning and testing environments prevent memory retrieval impairment following stress. *Cognitive, Affective & Behavioral Neuroscience, 9,* 229–236. http://dx.doi.org/10.3758/CABN.9.3.229

Schwabe, L., & Wolf, O. T. (2010a). Learning under stress impairs memory formation. *Neurobiology of Learning and Memory, 93,* 183–188. http://dx.doi.org/10.1016/j.nlm.2009.09.009

Schwabe, L., & Wolf, O. T. (2010b). Stress impairs the reconsolidation of autobiographical memories. *Neurobiology of Learning and Memory, 94,* 153–157. http://dx.doi.org/10.1016/j.nlm.2010.05.001

Schwabe, L., & Wolf, O. T. (2011). Stress-induced modulation of instrumental behavior: From goal-directed to habitual control of action. *Behavioural Brain Research, 219,* 321–328. http://dx.doi.org/10.1016/j.bbr.2010.12.038

Schwabe, L., & Wolf, O. T. (2014). Timing matters: Temporal dynamics of stress effects on memory retrieval. *Cognitive, Affective, & Behavioral Neuroscience, 14,* 1041–1048. http://dx.doi.org/10.3758/s13415-014-0256-0

Schwabe, L., Wolf, O. T., & Oitzl, M. S. (2010). Memory formation under stress: Quantity and quality. *Neuroscience and Biobehavioral Reviews, 34,* 584–591. http://dx.doi.org/10.1016/j.neubiorev.2009.11.015

Scoville, W. B., & Milner, B. (1957). Loss of recent memory after bilateral hippocampal lesions. *Journal of Neurology, Neurosurgery, & Psychiatry, 20,* 11–21. http://dx.doi.org/10.1136/jnnp.20.1.11

Shansky, R. M., & Lipps, J. (2013). Stress-induced cognitive dysfunction: Hormone-neurotransmitter interactions in the prefrontal cortex. *Frontiers in Human Neuroscience, 7,* 123. http://dx.doi.org/10.3389/fnhum.2013.00123

Shields, G. S., Bonner, J. C., & Moons, W. G. (2015). Does cortisol influence core executive functions? A meta-analysis of acute cortisol administration effects on working memory, inhibition, and set-shifting. *Psychoneuroendocrinology, 58,* 91–103. http://dx.doi.org/10.1016/j.psyneuen.2015.04.017

Shields, G. S., Kuchenbecker, S. Y., Pressman, S. D., Sumida, K. D., & Slavich, G. M. (2016). Better cognitive control of emotional information is associated with reduced pro-inflammatory cytokine reactivity to emotional stress. *Stress, 19,* 63–68. http://dx.doi.org/10.3109/10253890.2015.1121983

Shields, G. S., Sazma, M. A., & Yonelinas, A. P. (2016). The effects of acute stress on core executive functions: A meta-analysis and comparison with cortisol. *Neuroscience and Biobehavioral Reviews, 68,* 651–668. http://dx.doi.org/10.1016/j.neubiorev.2016.06.038

Silverman, M. N., & Sternberg, E. M. (2012). Glucocorticoid regulation of inflammation and its functional correlates: From HPA axis to glucocorticoid receptor dysfunction. *Annals of the New York Academy of Sciences, 1261,* 55–63. http://dx.doi.org/10.1111/j.1749-6632.2012.06633.x

Simons, J. S., & Spiers, H. J. (2003). Prefrontal and medial temporal lobe interactions in long-term memory. *Nature Reviews Neuroscience, 4,* 637–648. http://dx.doi.org/10.1038/nrn1178

Skoluda, N., Strahler, J., Schlotz, W., Niederberger, L., Marques, S., Fischer, S., . . . Nater, U. M. (2015). Intra-individual psychological and physiological responses to acute laboratory stressors of different intensity. *Psychoneuroendocrinology, 51,* 227–236. http://dx.doi.org/10.1016/j.psyneuen.2014.10.002

Slavich, G. M., & Irwin, M. R. (2014). From stress to inflammation and major depressive disorder: A social signal transduction theory of depression. *Psychological Bulletin, 140,* 774–815. http://dx.doi.org/10.1037/a0035302

Smeets, T. (2011). Acute stress impairs memory retrieval independent of time of day. *Psychoneuroendocrinology, 36,* 495–501. http://dx.doi.org/10.1016/j.psyneuen.2010.08.001

Smeets, T., Giesbrecht, T., Jelicic, M., & Merckelbach, H. (2007). Context-dependent enhancement of declarative memory performance following acute psychosocial stress. *Biological Psychology, 76,* 116–123. http://dx.doi.org/10.1016/j.biopsycho.2007.07.001

Smeets, T., Jelicic, M., & Merckelbach, H. (2006a). Stress-induced cortisol responses, sex differences, and false recollections in a DRM paradigm. *Biological Psychology, 72,* 164–172. http://dx.doi.org/10.1016/j.biopsycho.2005.09.004

Smeets, T., Jelicic, M., & Merckelbach, H. (2006b). The effect of acute stress on memory depends on word valence. *International Journal of Psychophysiology, 62,* 30–37. http://dx.doi.org/10.1016/j.ijpsycho.2005.11.007

Smeets, T., Jelicic, M., Merckelbach, H., Peters, M., Fett, A., Taverniers, J., . . . Dautzenberg, J. (2006). Enhanced memory performance on an internal-internal source monitoring test following acute psychosocial stress. *Behavioral Neuroscience, 120,* 1204–1210. http://dx.doi.org/10.1037/0735-7044.120.6.1204

Smeets, T., Otgaar, H., Candel, I., & Wolf, O. T. (2008). True or false? Memory is differentially affected by stress-induced cortisol elevations and sympathetic activity at consolidation and retrieval. *Psychoneuroendocrinology, 33,* 1378–1386. http://dx.doi.org/10.1016/j.psyneuen.2008.07.009

Smeets, T., Sijstermans, K., Gijsen, C., Peters, M., Jelicic, M., & Merckelbach, H. (2008). Acute consolidation stress enhances reality monitoring in healthy young adults. *Stress: The International Journal on the Biology of Stress, 11,* 235–245. http://dx.doi.org/10.1080/10253890701754076

Spaniol, J., Davidson, P. S. R., Kim, A. S. N., Han, H., Moscovitch, M., & Grady, C. L. (2009). Event-related fMRI studies of episodic encoding and retrieval: Meta-analyses using activation likelihood estimation. *Neuropsychologia, 47,* 1765–1779. http://dx.doi.org/10.1016/j.neuropsychologia.2009.02.028

Squire, L. R., & Alvarez, P. (1995). Retrograde amnesia and memory consolidation: A neurobiological perspective. *Current Opinion in Neurobiology, 5,* 169–177. http://dx.doi.org/10.1016/0959-4388(95)80023-9

Sripada, R. K., Marx, C. E., King, A. P., Rajaram, N., Garfinkel, S. N., Abelson, J. L., & Liberzon, I. (2013). DHEA enhances emotion regulation neurocircuits and modulates memory for emotional stimuli. *Neuropsychopharmacology, 38,* 1798–1807. http://dx.doi.org/10.1038/npp.2013.79

Sripada, R. K., Welsh, R. C., Marx, C. E., & Liberzon, I. (2014). The neurosteroids allopregnanolone and dehydroepiandrosterone modulate resting-state amygdala connectivity. *Human Brain Mapping, 35,* 3249–3261. http://dx.doi.org/10.1002/hbm.22399

Srivastava, D. P., Waters, E. M., Mermelstein, P. G., Kramár, E. A., Shors, T. J., & Liu, F. (2011). Rapid estrogen signaling in the brain: Implications for the fine-tuning of neuronal circuitry. *The Journal of Neuroscience, 31,* 16056–16063. http://dx.doi.org/10.1523/JNEUROSCI.4097-11.2011

Stawski, R. S., Sliwinski, M. J., & Smyth, J. M. (2009). The effects of an acute psychosocial stressor on episodic memory. *European Journal of Cognitive Psychology, 21,* 897–918. http://dx.doi.org/10.1080/09541440802333042

Tanner-Smith, E. E., & Tipton, E. (2014). Robust variance estimation with dependent effect sizes: Practical considerations including a software tutorial in Stata and SPSS. *Research Synthesis Methods, 5,* 13–30. http://dx.doi.org/10.1002/jrsm.1091

Taverniers, J., Taylor, M. K., & Smeets, T. (2013). Delayed memory effects after intense stress in Special Forces candidates: Exploring path processes between cortisol secretion and memory recall. *Stress, 16,* 311–320. http://dx.doi.org/10.3109/10253890.2012.721824

Taverniers, J., Van Ruysseveldt, J., Smeets, T., & von Grumbkow, J. (2010). High-intensity stress elicits robust cortisol increases, and impairs working memory and visuo-spatial declarative memory in Special Forces candidates: A field experiment. *Stress, 13,* 323–333. http://dx.doi.org/10.3109/10253891003642394

Thoma, M. V., Kirschbaum, C., Wolf, J. M., & Rohleder, N. (2012). Acute stress responses in salivary alpha-amylase predict increases of plasma norepinephrine. *Biological Psychology, 91,* 342–348. http://dx.doi.org/10.1016/j.biopsycho.2012.07.008

Thompson, L. A., Williams, K. L., L'Esperance, P. R., & Cornelius, J. (2001). Context-dependent memory under stressful conditions: The case of skydiving. *Human Factors, 43,* 611–619. http://dx.doi.org/10.1518/001872001775870377

Tipton, E. (2015). Small sample adjustments for robust variance estimation with meta-regression. *Psychological Methods, 20,* 375–393. http://dx.doi.org/10.1037/met0000011

Tollenaar, M. S., Elzinga, B. M., Spinhoven, P., & Everaerd, W. A. M. (2008). The effects of cortisol increase on long-term memory retrieval during and after acute psychosocial stress. *Acta Psychologica, 127,* 542–552. http://dx.doi.org/10.1016/j.actpsy.2007.10.007

Tollenaar, M. S., Elzinga, B. M., Spinhoven, P., & Everaerd, W. (2009). Autobiographical memory after acute stress in healthy young men. *Memory, 17,* 301–310. http://dx.doi.org/10.1080/09658210802665845

Trammell, J. P., & Clore, G. L. (2014). Does stress enhance or impair memory consolidation? *Cognition and Emotion, 28,* 361–374. http://dx.doi.org/10.1080/02699931.2013.822346

Valentine, J. C., Pigott, T. D., & Rothstein, H. R. (2010). How many studies do you need? A primer on statistical power for meta-analysis. *Journal of Educational and Behavioral Statistics, 35,* 215–247. http://dx.doi.org/10.3102/1076998609346961

van Ast, V. A., Cornelisse, S., Meeter, M., & Kindt, M. (2014). Cortisol mediates the effects of stress on the contextual dependency of memories. *Psychoneuroendocrinology, 41,* 97–110. http://dx.doi.org/10.1016/j.psyneuen.2013.12.007

van Stegeren, A. H., Roozendaal, B., Kindt, M., Wolf, O. T., & Joëls, M. (2010). Interacting noradrenergic and corticosteroid systems shift human brain activation patterns during encoding. *Neurobiology of Learning and Memory, 93,* 56–65. http://dx.doi.org/10.1016/j.nlm.2009.08.004

van Wingen, G., Mattern, C., Verkes, R. J., Buitelaar, J., & Fernández, G. (2008). Testosterone biases automatic memory processes in women towards potential mates. *NeuroImage, 43,* 114–120. http://dx.doi.org/10.1016/j.neuroimage.2008.07.002

Vogel, S., Fernández, G., Joëls, M., & Schwabe, L. (2016). Cognitive adaptation under stress: A case for the mineralocorticoid receptor. *Trends in Cognitive Sciences, 20,* 192–203. http://dx.doi.org/10.1016/j.tics.2015.12.003

Weymar, M., Schwabe, L., Löw, A., & Hamm, A. O. (2012). Stress sensitizes the brain: Increased processing of unpleasant pictures after exposure to acute stress. *Journal of Cognitive Neuroscience, 24,* 1511–1518. http://dx.doi.org/10.1162/jocn_a_00174

Wiemers, U. S., Sauvage, M. M., Schoofs, D., Hamacher-Dang, T. C., & Wolf, O. T. (2013). What we remember from a stressful episode. *Psychoneuroendocrinology, 38,* 2268–2277. http://dx.doi.org/10.1016/j.psyneuen.2013.04.015

Wiemers, U. S., Sauvage, M. M., & Wolf, O. T. (2014). Odors as effective retrieval cues for stressful episodes. *Neurobiology of Learning and Memory, 112,* 230–236. http://dx.doi.org/10.1016/j.nlm.2013.10.004

Wirkner, J., Weymar, M., Löw, A., & Hamm, A. O. (2013). Effects of pre-encoding stress on brain correlates associated with the long-term memory for emotional scenes. *PLoS ONE, 8.* http://dx.doi.org/10.1371/journal.pone.0068212

Wolf, O. T. (2012). Immediate recall influences the effects of pre-encoding stress on emotional episodic long-term memory consolidation in healthy young men. *Stress, 15,* 272–280. http://dx.doi.org/10.3109/10253890.2011.622012

Wolf, O. T., Schommer, N. C., Hellhammer, D. H., McEwen, B. S., & Kirschbaum, C. (2001). The relationship between stress induced cortisol levels and memory differs between men and women. *Psychoneuroendocrinology, 26,* 711–720. http://dx.doi.org/10.1016/S0306-4530(01)00025-7

Yabuki, Y., Shinoda, Y., Izumi, H., Ikuno, T., Shioda, N., & Fukunaga, K. (2015). Dehydroepiandrosterone administration improves memory deficits following transient brain ischemia through sigma-1 receptor stimulation. *Brain Research, 1622,* 102–113. http://dx.doi.org/10.1016/j.brainres.2015.05.006

Ycaza Herrera, A., & Mather, M. (2015). Actions and interactions of estradiol and glucocorticoids in cognition and the brain: Implications for aging women. *Neuroscience and Biobehavioral Reviews, 55,* 36–52. http://dx.doi.org/10.1016/j.neubiorev.2015.04.005

Yonelinas, A. P. (2002). The Nature of Recollection and Familiarity: A Review of 30 Years of Research. *Journal of Memory and Language, 46,* 441–517. http://dx.doi.org/10.1006/jmla.2002.2864

Yonelinas, A. P. (2013). The hippocampus supports high-resolution binding in the service of perception, working memory and long-term memory. *Behavioural Brain Research, 254,* 34–44. http://dx.doi.org/10.1016/j.bbr.2013.05.030

Yonelinas, A. P., Parks, C. M., Koen, J. D., Jorgenson, J., & Mendoza, S. P. (2011). The effects of post-encoding stress on recognition memory: Examining the impact of skydiving in young men and women. *Stress, 14,* 136–144. http://dx.doi.org/10.3109/10253890.2010.520376

Yonelinas, A. P., & Ritchey, M. (2015). The slow forgetting of emotional episodic memories: An emotional binding account. *Trends in Cognitive Sciences, 19,* 259–267. http://dx.doi.org/10.1016/j.tics.2015.02.009

Yuen, E. Y., Liu, W., Karatsoreos, I. N., Feng, J., McEwen, B. S., & Yan, Z. (2009). Acute stress enhances glutamatergic transmission in prefrontal cortex and facilitates working memory. *Proceedings of the National Academy of Sciences of the United States of America, 106,* 14075–14079. http://dx.doi.org/10.1073/pnas.0906791106

Zalcman, S., Green-Johnson, J. M., Murray, L., Nance, D. M., Dyck, D., Anisman, H., & Greenberg, A. H. (1994). Cytokine-specific central monoamine alterations induced by interleukin-1, −2 and −6. *Brain Research, 643,* 40–49. http://dx.doi.org/10.1016/0006-8993(94)90006-X

Zoladz, P. R., Clark, B., Warnecke, A., Smith, L., Tabar, J., & Talbot, J. N. (2011). Pre-learning stress differentially affects long-term memory for emotional words, depending on temporal proximity to the learning experience. *Physiology & Behavior, 103,* 467–476. http://dx.doi.org/10.1016/j.physbeh.2011.01.016

Zoladz, P. R., Kalchik, A. E., Hoffman, M. M., Aufdenkampe, R. L., Burke, H. M., Woelke, S. A., . . . Talbot, J. N. (2014). Brief, pre-retrieval stress differentially influences long-term memory depending on sex and corticosteroid response. *Brain and Cognition, 85,* 277–285. http://dx.doi.org/10.1016/j.bandc.2014.01.010

Zoladz, P. R., Kalchik, A. E., Hoffman, M. M., Aufdenkampe, R. L., Lyle, S. M., Peters, D. M., . . . Rorabaugh, B. R. (2014). ADRA2B deletion variant selectively predicts stress-induced enhancement of long-term memory in females. *Psychoneuroendocrinology, 48,* 111–122. http://dx.doi.org/10.1016/j.psyneuen.2014.06.012

Zoladz, P. R., Peters, D. M., Cadle, C. E., Kalchik, A. E., Aufdenkampe, R. L., Dailey, A. M., . . . Rorabaugh, B. R. (2015). Post-learning stress enhances long-term memory and differentially influences memory in females depending on menstrual stage. *Acta Psychologica, 160,* 127–133. http://dx.doi.org/10.1016/j.actpsy.2015.07.008

Zoladz, P. R., Warnecke, A. J., Woelke, S. A., Burke, H. M., Frigo, R. M., Pisansky, J. M., . . . Talbot, J. N. (2013). Pre-learning stress that is temporally removed from acquisition exerts sex-specific effects on long-term memory. *Neurobiology of Learning and Memory, 100,* 77–87. http://dx.doi.org/10.1016/j.nlm.2012.12.012

Zola-Morgan, S., Squire, L. R., & Amaral, D. G. (1986). Human amnesia and the medial temporal region: Enduring memory impairment following a bilateral lesion limited to field CA1 of the hippocampus. *The Journal of Neuroscience, 6,* 2950–2967. Retrieved from http://www.jneurosci.org/content/6/10/2950.short

(Appendix follows)

Appendix

Studies in the Final Sample

Study	Phase of memory studied	k	Study design	N	Study-average g	Age	% male participants
Almela et al. (2011)	Encoding/Retrieval	2	Within-subjects	30	−.1	62.09	50
Andreano and Cahill (2006)	Postencoding	2	Between-subjects	82	.604	21.5	50
Andreano et al. (2008)	Postencoding	3	Between-subjects	64	.292	24	0
Andreano et al. (2012)	Postencoding	2	Between-subjects	20	.89	39.9	0
Azimi and Bakhshipour-Roudsari (2012)	Reactivation	3	Between-subjects	20	−1.271	20.4	0
Beckner et al. (2006)	Postencoding Retrieval	4	Between-subjects	157	Postencoding: .618 Retrieval: .255	18.77	35.6
Boehringer et al. (2010)	Retrieval	3	Between-subjects	51	−.549	24.57	100
Bos, Schuijer, et al. (2014), Exp. 1	Reactivation	3	Between-subjects	43	.491	21.3	48.83
Bos, Schuijer, et al. (2014), Exp. 2	Reactivation	3	Between-subjects	36	.444	21.51	50
Bos, van Goethem, et al. (2014)	Reactivation	2	Between-subjects	51	.02	21.84	45.1
Bryant et al. (2013)	Postencoding	4	Between-subjects	78	.402	19.78	50
Buchanan and Tranel (2008)	Retrieval	2	Between-subjects	40	−.082	20	50
Buchanan et al. (2006)	Retrieval	4	Between-subjects	30	−.069	18.9	53.33
Cahill et al. (2003)	Postencoding	4	Between-subjects	48	.327	20.1	29.17
Cavenett and Nixon (2006)	Postencoding/Retrieval	4	Between-subjects	70	−.184	26.47	64.29
Coccoz et al. (2011), Exp. 1	Reactivation	1	Between-subjects	20	1.116	23	42.5
Coccoz et al. (2011), Exp. 2	Reactivation	1	Between-subjects	22	.977	23	100
Coccoz et al. (2013)	Reactivation	1	Between-subjects	30	.931	24	
Cornelisse, Joels, et al. (2011)	Encoding	1	Between-subjects	32	−.013	21.75	100
Cornelisse, van Stegeren, et al. (2011)	Encoding	4	Between-subjects	77	−.065	20.44	50
Domes et al. (2002)	Retrieval	1	Between-subjects	32	−.697	47.3	0
Domes et al. (2004)	Encoding Retrieval	12	Between-subjects	60	Encoding: −.197 Retrieval: −.097	27.1	100
du Plooy (2014)	Retrieval	8	Between-subjects	60	−.195	19.97	50
Eich and Metcalfe (2009)	Encoding/Retrieval	1	Between-subjects	261	−.254	36.37	54.02
Espin et al. (2013)	Encoding/Retrieval	2	Between-subjects	119	−.036	19.33	26.89
Felmingham, Fong, et al. (2012)	Postencoding	2	Between-subjects	56	.301		0
Felmingham, Tran, et al. (2012)	Postencoding	4	Between-subjects	80	.688	29	50
Henckens et al. (2009)	Encoding	1	Within-subjects	18	.312	22	100
Hidalgo et al. (2012)	Encoding/Retrieval	1	Within-subjects	46	0	21.56	37
Hidalgo et al. (2014)	Encoding/Retrieval	2	Within-subjects	67	−.147	41.6	50.71
Hidalgo et al. (2015)[see note]	Retrieval	4	Between-subjects	50	−.287	22.5	50
Hoffman and al'Absi (2004)	Encoding/Retrieval	2	Within-subjects	25	.048	24.8	40
Hoscheidt (2011)	Encoding Retrieval	4	Between-subjects	90	Encoding: −.429 Retrieval: .375	19	44.44
Hoscheidt et al. (2014)	Encoding	1	Between-subjects	68	.602	19	55.88
Human (2010)	Retrieval	2	Between-subjects	18	−.519	20.22	50
Human et al. (2013)	Postencoding/Retrieval	1	Between-subjects	36	.193	20.5	100
Hupbach and Dorskind (2014)	Reactivation	1	Between-subjects	58	−.801		100
Hupbach and Fieman (2012)	Retrieval	2	Between-subjects	75	−.177	27.5	50
Kuhlmann et al. (2005)	Retrieval	5	Within-subjects	19	−.39	24.58	100
Larra et al. (2014)	Postencoding and Postencoding/Retrieval	2	Between-subjects	206	Postencoding: .269 Postencoding/ Retrieval: ~0	23	48.54
Li et al. (2013)	Retrieval	2	Within-subjects	42	−.222	23.63	100
Li et al. (2014)	Retrieval	2	Within-subjects	27	.01	24.25	100
Luethi et al. (2009)	Encoding/Retrieval	2	Between-subjects	35	.366	23.4	100
Maheu et al. (2005), Exp. 1	Encoding	2	Between-subjects	19	−1.444	22.5	100
Maheu et al. (2005), Exp. 2	Encoding	2	Between-subjects	20	−1.143	22.5	100
Marin et al. (2010)	Reactivation	2	Between-subjects	32	−.225	22.09	50
McCullough and Yonelinas (2013)	Postencoding	16	Between-subjects	38	.124	19.35	50
McCullough et al. (2015)	Postencoding	4	Between-subjects	49	−.09	24.2	100
Merz et al. (2010)	Postencoding/Retrieval	3	Within-subjects	29	−.301	23.17	48.2
Nguyen (2009)	Postencoding/Retrieval	1	Between-subjects	72	.368	20.86	39
Nielsen et al. (2013), Exp. 1	Postencoding	3	Between-subjects	49	.076	20.37	0
Nielsen et al. (2013), Exp. 2	Postencoding	3	Between-subjects	41	−.047	20.37	0

(Appendix continues)

Appendix (*continued*)

Study	Phase of memory studied	k	Study design	N	Study-average g	Age	% male participants
Nielsen et al. (2014), Exp. 1	Postencoding	2	Between-subjects	60	.27	20.1	0
Nielsen et al. (2014), Exp. 2	Postencoding	2	Between-subjects	49	−.142	20.3	0
Oei et al. (2006)	Retrieval	2	Within-subjects	20	−.135	21.86	100
Payne et al. (2006), Exp. 1	Encoding	8	Between-subjects	56	−.24		50
Payne et al. (2006), Exp. 2	Encoding/Retrieval	8	Between-subjects	61	−.477		50
Payne et al. (2007), Exp. 1	Encoding	3	Between-subjects	31	−.984		42.11
Payne et al. (2007), Exp. 2	Encoding	3	Between-subjects	34	1.006		42.11
Preuß and Wolf (2009)	Postencoding	3	Between-subjects	58	.259	23.6	51.72
Pulopulos et al. (2013)	Retrieval	8	Between-subjects	76	−.132	64.63	50
Qin et al. (2012)	Encoding	1	Between-subjects	40	−.239	22.18	100
Quaedflieg et al. (2013), Exp. 1	Encoding	4	Between-subjects	32	−.475	21.25	100
Quaedflieg et al. (2013), Exp. 2	Encoding	4	Between-subjects	32	−.371	21.25	100
Robicheaux (2015)	Postencoding/Retrieval	2	Between-subjects	128	.084		28.91
Schönfeld et al. (2014)	Retrieval	4	Between-subjects	72	−.101	23.2	50
Schoofs and Wolf (2009)	Retrieval	3	Within-subjects	36	.058	24.47	0
Schwabe and Wolf (2009), Exp. 1	Retrieval	1	Between-subjects	36	.11	25.1	50
Schwabe and Wolf (2009), Exp. 2	Retrieval	1	Between-subjects	36	−.718	25.1	50
Schwabe and Wolf (2010a)	Encoding	16	Between-subjects	48	−.478	23.6	50
Schwabe and Wolf (2010b)	Reactivation	3	Between-subjects	32	−.369	23.3	50
Schwabe and Wolf (2014), Exp. 1	Retrieval	2	Between-subjects	40	.066	23.61	50
Schwabe and Wolf (2014), Exp. 2	Retrieval	2	Between-subjects	40	−.546	23.61	50
Schwabe and Wolf (2014), Exp. 3	Retrieval	2	Between-subjects	40	−.84	23.61	50
Schwabe et al. (2008)	Encoding Encoding/Retrieval	9	Between-subjects	96	Encoding: .061 Encoding/ Retrieval: .245	23.3	50
Schwabe and Wolf (2009), Exp. 1	Encoding	3	Between-subjects	36	.183	25	50
Schwabe and Wolf (2009), Exp. 2	Encoding	3	Between-subjects	36	−.471	25	50
Schwabe, Römer, et al. (2009)	Retrieval	2	Within-subjects	44	.282	23.7	100
Smeets (2011), Exp. 1	Retrieval	2	Between-subjects	38	−.91	19.9	44.73
Smeets (2011), Exp. 2	Retrieval	2	Between-subjects	38	−.852	19.9	44.73
Smeets et al. (2007)	Encoding	4	Between-subjects	52	.469	23.08	25
Smeets, Jelicic, and Merckelbach (2006a), Exp. 1	Encoding/Retrieval	4	Between-subjects	58	.037	19.91	50
Smeets, Jelicic, and Merckelbach (2006a), Exp. 2	Encoding/Retrieval	4	Between-subjects	92	−.403	19.74	50
Smeets, Jelicic, and Merckelbach (2006b)	Encoding/Retrieval	6	Between-subjects	60	−.452	19.65	50
Smeets, Jelicic, Merckelbach, Peters, et al. (2006)	Postencoding/Retrieval	1	Between-subjects	40	.901	19.2	100
Smeets, Otgaar, et al. (2008)	Encoding Postencoding Retrieval	6	Between-subjects	90	Encoding: .005 Postencoding: .308 Retrieval: −.839	20.6	5.8
Smeets, Sijstermans, et al. (2008)	Postencoding	1	Between-subjects	80	1.297	20.3	40
Stawski et al. (2009)	Postencoding/Retrieval	1	Between-subjects	100	−.17	18.94	26
Taverniers et al. (2010)	Encoding/Retrieval	1	Between-subjects	27	−1.057	27.4	100
Taverniers et al. (2013)	Encoding	1	Between-subjects	24	−1.171	27.04	100
Thompson et al. (2001)	Postencoding/Retrieval Encoding/Retrieval	3	Within-subjects	16	Postencoding/ Retrieval: −.7 Encoding/ Retrieval: −1.32	26	87.5
Tollenaar et al. (2008), Exp. 1	Retrieval	4	Between-subjects	35	−.284	21.34	100
Tollenaar et al. (2008), Exp. 2	Retrieval	4	Between-subjects	35	−.236	21.34	100
Tollenaar et al. (2009)	Retrieval	4	Between-subjects	40	−.081	21.7	100
Trammell and Clore (2014), Exp. 1	Postencoding	1	Between-subjects	97	−.431	18.97	39.18
Trammell and Clore (2014), Exp. 2	Postencoding	1	Between-subjects	131	−.472	18.47	47.3
Trammell and Clore (2014), Exp. 3	Postencoding	1	Between-subjects	127	−.374	18.88	45.67
van Ast et al. (2014)	Encoding	8	Between-subjects	40	.175	22	100
Weymar et al. (2012)	Encoding	2	Between-subjects	40	−.183	24.5	100
Wiemers et al. (2013)	Encoding	3	Between-subjects	60	.305	23.87	50
Wiemers et al. (2014), Exp. 1	Encoding	2	Between-subjects	44	.654	24.12	47.7
Wiemers et al. (2014), Exp. 2	Encoding	2	Between-subjects	45	.103	24.12	48.9
Wirkner et al. (2013)	Encoding	3	Between-subjects	52	−.662	23	55.77
Wolf (2012), Exp. 1	Encoding	3	Between-subjects	24	−.45	24	100

(*Appendix continues*)

Appendix (*continued*)

Study	Phase of memory studied	*k*	Study design	*N*	Study-average *g*	Age	% male participants
Wolf (2012), Exp. 2	Encoding	3	Between-subjects	32	.246	24.84	100
Wolf et al. (2001)	Encoding/Retrieval	2	Between-subjects	58	−.05	24.09	50
Yonelinas et al. (2011)	Postencoding	8	Between-subjects	50	−.099	25.2	50
Zoladz et al. (2011), Exp. 1	Encoding	6	Between-subjects	31	−.003	19.68	27.78
Zoladz et al. (2011), Exp. 2	Encoding	6	Between-subjects	31	.044	19.68	27.78
Zoladz et al. (2013)	Encoding Encoding/ Retrieval	18	Between-subjects	97	Encoding: −.172 Encoding/ Retrieval: ∼ 0	19.18	50
Zoladz et al. (2015)	Postencoding	12	Between-subjects	52	.023	20.3	51.92
Zoladz, Kalchik, Hoffman, Aufdenkampe, Burke, et al. (2014)	Retrieval	12	Between-subjects	93	−.047	19.45	50
Zoladz, Kalchik, Hoffman, Aufdenkampe, Lyle, et al. (2014)	Encoding	4	Between-subjects	120	.151	19.7	50

Note. The data for older participants in Hidalgo et al. (2015) were originally presented in another study included in our analyses. As such, only younger participants from this study were included in our meta-analyses.