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The Effects of Stress on Prospective Memory: A Systematic Review

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Prospective memory (PM) refers to mnemonic processes, which are directed to the future. It has been subdivided into different stages of processing: the planning, retention, performance, and evaluation phase. Moreover, PM can be time- or event-based. It is well known that retrospective memory (RM) can be affected by stress as seen in patients with posttraumatic stress disorder (PTSD). However, data on the effects of stress on PM are rare. In this review, available behavioral studies of PM are reviewed with respect to its vulnerability to stress. Based on the available data, we suggest that stress may have enhancing or disturbing effects on PM, depending on (a) the stressor characteristics, (b) whether PM is time- or event-based, and (c) which phase of processing is affected. Studies in healthy adults indicate rather an increase of PM in response to acute stress (average effect size of $d = .10$). In contrast, studies in PTSD patients found a deteriorating effect of the disorder on PM performance (average effect size of $d = -.58$). We discuss the putative clinical relevance of a better knowledge of the relationship between stress and PM for the diagnosis and therapy of PTSD.

Keywords: planning, intentions, neuroendocrinology, stress axis, cortisol

Stress and its influence on cognitive performance and psychological health has become a central issue in the last decades. Acute and chronic stress is known to be associated with a range of cognitive disabilities and mental as well as somatoform disorders. This has been shown by numerous studies in particular for explicit retrospective memory (RM) and in patients with posttraumatic stress disorder (PTSD; for review see Het, Ramlow, & Wolf, 2005; Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; Wingenfeld & Wolf, 2015; Wolf, 2009). The present review addresses studies that are concerned with the issue of stress influences on prospective memory (PM), that is, future-directed memory. Although PM functions play a key role in our everyday life (e.g., we daily have to plan actions, events, or meetings, try to realize intentions, and bring them into action), data on the modulation of PM are still rare. The focus of the review lies on the effects of stress on PM of healthy human subjects, but also includes the available data on PM in clinical samples. Several psychological disorders (e.g., anxiety disorders and affective disorders such as major depression) are known to be associated with an aberrant psychobiological stress response (Yehuda, 2002; Yehuda, Teicher, Trestman, Levengood, & Siever, 1996). However, in this review we only refer to PTSD since the disorder (a) results from the influence of acute stress and (b) has obtained the role of a paradigmatic model in psychobiological research on the influence of stress on brain functions and behavior. Given the extended knowledge about the influences of stress on RM, we also refer in the Discussion to data on the influence of RM, where they are needed to understand the modulation of PM by stress. Since everyday life demands are strongly based on PM functions, we propose that a better knowledge about the modulation of PM by stress may be of high relevance for the scientific and practical evaluation of stress influences on cognitive functions and psychological health. We assume that PM is comparably vulnerable to acute stress as RM. In particular, we hypoth-

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esize that acute stress may have enhancing or disturbing effects on PM in healthy adults and patients with PTSD depending on (Hypothesis 1) the stressor characteristics, (Hypothesis 2) whether PM is time- or event-based, and (Hypothesis 3) which phase of PM processing is directly affected by the stressor. Before presenting the results of our review, we provide relevant information on the current knowledge of the psychobiological stress response and the neuropsychological concept of PM. We then present the results of our systematic review including effect sizes where possible. Based on these results, we discuss enhancing and disturbing modulatory effects of stress on PM and their putative clinical relevance for the diagnosis and therapy of PTSD.

The Biological Stress Reaction

The stress reaction can be conceptualized as the adaptive response of an organism aiming at the reinstantiation of homeostasis (Cannon, 1929; Goldstein & Kopin, 2007; Goldstein & McEwen, 2002). This adaptive response is physiologically based on two functionally distinct stress axes. These are the sympathetic-adrenal-medullary-system (SAM) and the hypothalamic-pituitary-adrenal axis HPA system (e.g., de Kloet, Joëls, & Holsboer, 2005). Although the SAM and the HPA systems work parallel after the confrontation with a stressor, the two systems differ from each other with respect to the progress and speed of processing, as well as their biological influences on the organism. The SAM system is referred to as the fast stress axis, which allows within a few seconds for the release of epinephrine from chromaffin cells. It builds up the basis for the "flight and fight reaction." The HPA axis has a slower stress response. In response to stress exposure the hypothalamus produces corticotrophin-releasing factor (CRF), which in turn stimulates the pituitary gland to release adrenocorticotropin (ACTH; Holsboer & Ising, 2010). ACTH stimulates the synthesis of the glucocorticoid cortisol from the adrenal gland. Via a negative feedback loop into the central nervous system, cortisol mainly contributes to the regulation of the stress response (Kirschbaum & Hellhammer, 1994).

The Concept of PM

PM refers to mnemonic processes, which are directed to the future. These include the planning, the maintenance (i.e., retention), and the performance (i.e., realization; execution) of deferred intentions (Brandimonte, Einstein, & McDaniel, 1996; Ellis, 1996; McDaniel & Einstein, 2007; Simons, Schölvinck, Gilbert, Frith, & Burgess, 2006). Accordingly, PM has been subdivided into differential consecutive stages of processing. These are (a) the planning phase, (b) the retention interval, and (c) the performance phase (Ellis, 1996; see Figure 1). Brandimonte et al. (1996) proposed that the planning phase is the first stage of PM, which comprises the formation and encoding of future-directed intentions and plans. The following retention interval is conceptualized as a delay between planning and performance of intentions. During this interval, intended actions are maintained and postponed to a future point in time. The retention interval is the critical phase during PM since intentions and plans need to be protected against interference (Brandimonte et al., 1996; Brandimonte, Ferrante, Feresin, & Delbello, 2001). In cases where the retention of an intended action is successful, the performance phase of PM may follow. During the performance phase an action, which had been planned earlier, is retrieved, initiated, and put into action. However, Brandimonte et al. (1996) proposed a further stage of PM, during which the evaluation of planning, maintaining, and executing an action as well as the result of the action occurs. According to this point of view, PM always includes the recapitulation of mnemonic subprocesses and the evaluation of results from actions, which had been planned and executed earlier.

Note, PM does not need to be successful (see Figure 1). The action executed during the performance phase may be remembered incorrectly such that it can be completely or at least partially wrong. Importantly, the executed action can be wrong with respect to diverse aspects. These are mainly the time point of initiation, the content and structure of the action, the planned order of actions, or a combination thereof.

Besides the differential stages of PM processing, event- and time-based PM have been distinguished (Brandimonte et al., 1996; Einstein & McDaniel, 1990; Kliegel, Martin, McDaniel,

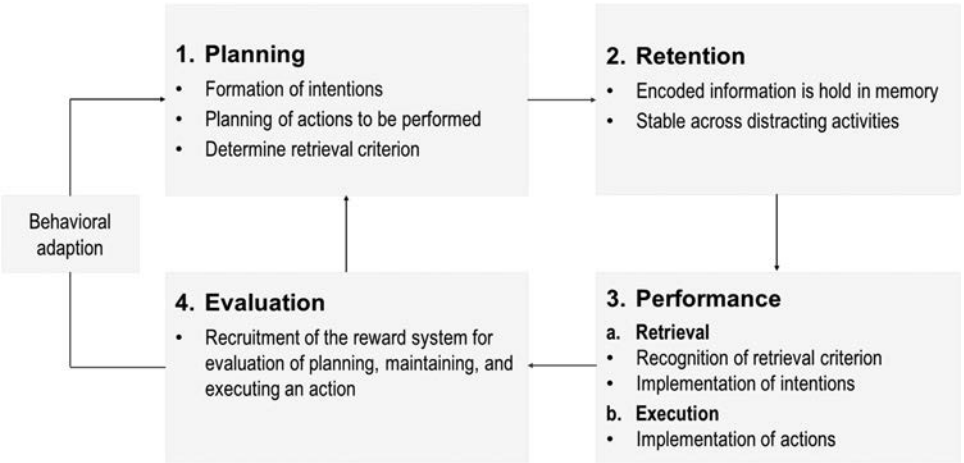


Figure 1. The different successive phases of PM and their functional specificity. It is likely that the effect of stress on PM depends on the time point of stress induction. Stress induction before the planning, the retention, or the performance phase may differentially enhance or compromise actual PM performance. In the case that stress is induced only before the evaluation phase, the effect will mainly be observed in future situations with PM demands.

& Einstein, 2001). According to this distinction, event- and time-based PM are considered to represent two different forms of PM, which are based on differential cognitive (e.g., Kliegel et al., 2001) and neurofunctional processes (e.g., Cheng, Wang, Xi, Niu, & Fu, 2008; Okuda et al., 2007). Event-based PM is supposed to depend mainly on external cues. Based on this assumption, a specific event acts as the reminder for the performance of an action planned earlier. For example, the planning and realization of the intake of medicine after lunch relies on event-based PM processes. In contrast, time-based PM is assumed to possess only a weak dependence from external cues. Rather, it is based on a priori planned time points and/or time periods. The planning and realization of the intake of medicine at 12 a.m. is an example for time-based PM processes. The differentiation between event- and time-based PM is supported by the findings of neuroimaging studies of Okuda et al. (2007) and Cheng et al. (2008). The authors concordantly reported differential frontal brain regions to be either associated with time- or event-based PM. In particular, they found that the right superior frontal gyrus, the anterior medial frontal lobe, and the anterior cingulate gyrus involved time-based PM processes. In contrast, the left superior frontal gyrus was associated with the processing of event-based PM. Note, however, that on the behavioral level in everyday life the distinction between time- and event-based PM may sometimes be difficult due to the frequent entanglement of the two forms of PM. For example, time-based PM may sometimes also be triggered by external cues (i.e., events) such as the alarm of a clock.

For an ongoing PM task paradigm, underlying cognitive strategies for PM have been modeled in a "multiprocess framework" (McDaniel & Einstein, 2000). The multiprocess framework supposes two cognitive strategies of PM. These are either strategic monitoring or spontaneous retrieval. Strategic monitoring describes a mainly top-down controlled memory process, which holds the intention active in mind and scans the environment for the "PM cue" (e.g., the cue that elicits the execution of an intention; i.e., Guynn, 2003). Spontaneous retrieval means that the retrieval of an intention is triggered spontaneous by a PM cue (McDaniel & Einstein, 2000). In consequence, spontaneous retrieval is mainly associated with bottom-up processes (Cona, Scarpazza, Sartori, Moscovitch, & Bisiacchi, 2015; McDaniel & Einstein, 2000; Scullin, McDaniel, & Shelton, 2013). Scullin et al. (2013) modified the multiprocess framework by introducing a dynamic component. Thus, in ongoing experimental tasks the underlying PM

strategies were either strategic monitoring or spontaneous retrieval but in the modified framework the processes are understood dynamically depending on the context.

Although there is evidence that PM is strongly associated with executive functions, working memory (WM), and RM (e.g., Addis, Wong, & Schacter, 2007; Schacter, Addis, & Buckner, 2007), it is widely accepted as a distinct cognitive function (Graf & Uttl, 2001). This view is supported for example by clinical studies in neurological patients, which demonstrated selective disturbances of PM in the presence of relatively intact executive and memory functions (West, McNerney, & Krauss, 2007).

Stress and Memory Processing in Patients With PTSD

A classic clinical explanatory model for the formation of psychological disorders is the diathesis–stress model (Zubin & Spring, 1977). The model assumes that a psychological disorder is the result of a combination of acute or chronic stress that is appraised as a threat, and a person's predisposition for the development of a psychological disease (Lazarus & Folkman, 1984). According to this account, stress plays a critical role in the genesis—and most likely also for the maintenance—of psychological disorders. In this context a differentiation between acute and chronic stress needs to be considered. Acute stress may be caused by threats of the recent past or expected demands in the recent future. In patients with Posttraumatic stress disorder (PTSD), the acute physiological stress response is mainly characterized by diminished levels of cortisol (i.e., Elzinga et al., 2008; Mason, 1968; Yehuda et al., 1996) caused by an acute and traumatic stress situation. Normally, when being confronted with a stressor, a high release of cortisol occurs in the organism. Cortisol then inhibits the neural structures, which are involved in the regulation of cortisol release (in particular the hypothalamus and the pituitary gland). This functional circuit is referred to as a negative feedback loop. It has been suggested that this negative feedback loop is interrupted in patients with PTSD, and that this is due to the low basal cortisol level associated with the disorder (Yehuda, 2002). Interestingly, studies using the dexamethasone suppression test (DST) demonstrated a normal function of the negative feedback loop as well as the HPA axis in patients with PTSD (Yehuda, 2002). The DST allows for the investigation of the physiological stress response by the application of a low dose of a synthetic cortisol (dexamethasone). The data thus suggest that patients with PTSD have an intact negative feedback loop and HPA axis, but that the organism is unable to activate the functional circuit of the HPA axis autonomously (Yehuda, 2002).

Chronic stress may result from sustained exposure to stressors and/or from a single exposure to uncontrollable acute traumatic stress. While acute everyday stress can normally be managed without negative influences on a person's health, chronic stress frequently leads to aberrations of the physiological stress response and associated psychological disorders such as PTSD (Miller, Chen, & Zhou, 2007). It is likely that alterations of the slow HPA axis rather than deviant responses of the SAM system are relevant in PTSD since the regulating function of the negative feedback loop by cortisol is more related to the HPA axis than the SAM system (Yehuda et al., 1996). PTSD is well known to be accompanied by cognitive and affective disturbances, in particular in the memory domain. It is therefore likely that PM processing is also affected in patients with PTSD.

Method

According to the aims of the review we had six main inclusion criteria and four main exclusion criteria for relevant studies.

Inclusion criteria:

(1) For healthy adults only studies that refer to PM were included into this review. However, since clinical studies on PM are very rare and studies found a significant association between PM and episodic future memory (i.e., Terrett et al., 2015), for studies with PTSD patients we also included studies that relied on episodic future memory.
(2) Only studies of adults were included. Studies on children were excluded from this review since developmental aspects of the biological stress reaction in children (Elmlinger, Kühnel, & Ranke, 2002; Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004) and their relevance to the influence of stress on PM would be a topic of a separate review.
(3) For studies that report a psychoendocrinological marker, we review only published data concerning the stress hormone cortisol. Although there is evidence that other hormones and neurotransmitters (e.g., endorphins and epinephrine; i.e., Cahill & Alkire, 2003) may also play crucial roles in the psychoendocrino-

logical processing of stress, cortisol is known to be the key player in the biological stress response. Cortisol is also well known to affect memory functions, due to the wide distribution of cortisol binding glucocorticoid receptors (GC-receptors) to neural substrates that are associated with memory functions in the brain (i.e., Dedovic, Duchesne, Andrews, Engert, & Pruessner, 2009; Lupien et al., 2007; Wingenfeld & Wolf, 2015; Wolf, 2009). Moreover, cortisol is the stress modulator that has most intensely been investigated to date. Studies on other neuromodulators involved in the processing of stress are only referred to in cases where there are relevant for the understanding of cortisol functions in psychoneuroendocrinological responses to acute stress.

(4) Only studies of patients with PTSD were included in the review. PTSD has become a paradigmatic model of chronic alterations of the HPA axis and their influences on PM performance. Typically, patients with PTSD show consistently reduced cortisol levels caused by a dysfunction of the HPA axis (i.e.Yehuda, 2002, 2006; Yehuda et al., 1996). The picture of alterations of the HPA axis in PTSD patients (compared to other psychiatric disorders) is consistent (i.e., Meewisse, Reitsma, de Vries, Gersons, & Olff, 2007; Peeters, Nicolson, & Berkhof, 2004; Yehuda et al., 1996).
(5) All studies of patients with PTSD included to this review were controlled clinical trials.
(6) The patients investigated in the studies did not have confounding comorbid disorders other than depression such as, for example, personality disorders.

Exclusion criteria:
Studies were excluded when:

(1) they exclusively used indirect memory measures (e.g., electroencephalography without behavioral data);
(2) they administered synthetic glucocorticoids (e.g., dexamethasone) or included participants who received pharmacological treatment;
(3) they included patients with comorbid disorders other than depression (e.g., borderline personality disorder, psychotic disorders etc.);
(4) participants of the study met criteria for traumatic brain injury.

Literature Research

Keywords that were used in the systematic bibliographic research were *prospective memory*, *future memory*, *planning phase*, *retention phase*, *retrieval phase*, *execution phase*, *implementation of intentions*, *retrospective memory*, *consolidation*, *retrieval*, *stress*, *cortisol*, *posttraumatic stress disorder*, *PTSD*, *monitoring*, *spontaneous retrieval* and combinations thereof. The databases used for literature research were PubMed, PsycINFO, PubPsych, and Google Scholar. Publication years included in the bibliographic research ranged from 2005 to 2016. For studies on the influence of stress on PM in healthy adults, bibliographic research ranged from 2005 to 2016, and from 2013 to 2016 for studies on PM in patients with PTSD. Literature research took place from October 2015 until December 2016. The review does not require an ethical approval.

Calculation of Effect Sizes

Effect sizes were calculated for overall PM performance and for time- and event-based PM separately. Our analysis of effect sizes relied on Hedges' g (g_{Hedges}), which describes the difference of means of an experimental group and a control group standardized by a pooled standard deviation (*SD*; Hedges & Olkin, 1985). Positive g_{Hedges} indicate better, negative g_{Hedges} indicate deteriorating PM in response to stress. As suggested by Cohen (1988), an effect size of .20 was classified as small, .50 as moderate, and .80 as large. Where available, calculations of effect sizes were based on means and *SD* of means. To control for overestimation of the effect sizes, each g_{Hedges} was adjusted and converted to Cohen's d value with respect to Hedge's formula (Hedges & Olkin, 1985). In two cases effect sizes were calculated on the basis on correlational coefficients. For these studies, Fischer's z was adjusted and converted into Cohens d. Especially in the clinical studies, ds were available for multiple PM measures (e.g., PM specificity). In cases where more than one measure of the same dependent variable was available, ds were first calculated for each single variable and then averaged to one d called PM performance. For time- and event dependent PM we also calculated separate ds.

Statistical Analysis of Effect Sizes

Average effect sizes were calculated for PM for each study reported. A confidence interval of 95% was defined as the level of significance (95% CI). To investigate whether the effect sizes were consistent across studies all ds were summarized in a weighted average effect size, with its standard deviation and 95% CI. To test whether the ds share a common effect size, the Cochran Q test was consulted to prove homogeneity. A significant result indicates heterogeneous distributions of ds, which points out significant differences between the studies. To identify putative mediating factors of the ds

origin, they were attributed to the factors included in our hypotheses: (a) stressor characteristics, (b) PM type, and (c) PM phase. This differentiation could be accomplished only for the studies with healthy participants. In the studies of PTSD patients, too little studies addressing each factor were available. For the studies of nonclinical samples, we pooled measures of overall PM performance after the induction of acute experimental stress procedures (Glienke & Piefke, 2016; Nater et al., 2006; Walser, Fischer, Goschke, Kirschbaum, & Plessow, 2013) with PM measures without experimentally induced stress (Ihle et al., 2014; Ihle, Schnitzspahn, Rendell, Luong, & Kliegel, 2012; Nakayama, Takahashi, & Radford, 2005). Moreover, we pooled measures (a) of time-based PM (Glienke & Piefke, 2016; Ihle et al., 2014; Nater et al., 2006), (b) event based PM (Glienke & Piefke, 2016; Nakayama et al., 2005; Nater et al., 2006; Walser et al., 2013), and (c) across time- and event-based PM types (Glienke & Piefke, 2016; Ihle et al., 2012). Because studies that differentiated between phases of PM are very rare we could not include this factor into the statistical analyses. To test for a publication bias we used a significance test according to Begg (1994). A Spearman correlation (r_s) was applied to test to assess standardized effect sizes and their variances. A publication bias is indicated by a significant positive (r_s). Publication biases were calculated separately for nonclinical and clinical samples.

Results

Stress Influences on PM

Histological studies show evidence for the existence of cortisol-binding glucocorticoid receptors in some neural structures, which have repeatedly been associated with PM. These are mainly the frontal pole and medial temporal lobe (Bremner, 2006; de Quervain et al., 2003; Kremen et al., 2010). Given these neuroanatomical and neurobiological conditions, it is reasonable to assume that the release of cortisol will change PM performance. Research of the last years found diverging impact of stress on PM in healthy participants. For instance, in overall PM, Ihle et al. (2012) reported better PM performance under lower stress levels, whereas Glienke and Piefke (2016) found enhanced overall PM for significant increased cortisol levels.

Stress influence on event-dependent PM. As displayed in Table 1, Nakayama and colleagues (2005); Nater et al. (2006) and Walser et al. (2013) reported event-dependent PM performance to be unaffected by an alteration in stress level. Within the real-life related paradigm of Glienke and Piefke (2016) an improvement in event-dependent PM was found after an acute increase in cortisol after laboratory stress exposure.

Stress influence on time-dependent PM. In behavioral studies, Nater et al. (2006) and Glienke and Piefke (2016) reported better time-based PM performance after the induction of psychosocial stress. In another study, Ihle et al. (2014) found that a relaxation intervention decreased subjective and physiological stress levels but had no implication on time-based PM.

Both, histological studies as well as behavioral and psychophysiological research in humans indicate that stress most likely alters PM processing.

Analysis of Effect Sizes for the Nonclinical Samples

As shown in Table 1 we found a broad range of effects sizes from $d = -1.42$ (Ihle et al., 2012) to $d = .61$ (Nater et al., 2006) across all studies in healthy adults. The integration of effect sizes revealed an average weighted effect size of $d = .10$ ($.01 \leq d \leq .18$) for all studies. The homogeneity analysis showed a higher Q (22.11) than the critical value of χ^2 distribution ($\chi^2_8 = 11.42$; $p > .05$), indicating heterogeneous distributions of *d*s. This indicates that the *d*s do not share a common underlying effect size and may therefore result from the investigation of different populations, the use of different study designs, stressors, and tasks, or combinations thereof. As depicted in Table 2, results of laboratory studies applying standardized acute experimental stressors show average effect sizes ($p < .05$), which are significantly different from zero. In contrast, for studies that measured different forms of basal stress, a significant heterogeneity of distributions and a nonsignificant average effect size was evident. When separating time- and event- dependent PM, we also found significant average effect sizes and homogeneity of distributions for each type of PM. Moreover, there was a significant heterogeneity and a non-

Table 1
Summary of Descriptive Facets and Average Effect Sizes of PM Performance for the Included Nonclinical Studies

Study	Number of subjects (age in years = Mean)	Subjects	Stress induction method	Stress measurement	Type of PM	PM task	PM phase affected by stress	Stress effect on PM	Hedges *g/d*
Nakayama et al. (2005)	34 ♂ (*M* = 19.00)	Healthy subjects	—	Basal salivary cortisol	Event-based PM	Ongoing task paradigm	—	No association between cortisol and event-based PM	$d^{\text{event-based PM}} = .21$
Nater et al. (2006)	20 ♂ (*M* = 24.45)	Healthy subjects	TSST (cross-over design)	Induced salivary cortisol	Time-based PM & event- PM	Ongoing task paradigm	—	Stress improved time-based PM	$d^{\text{event-based PM}} = .26$ $d^{\text{time-based PM}} = .61$
Ihle et al. (2012)	8 ♂; 12 ♀ (*M* = 22.50) 9 ♂; 10 ♀ (*M* = 68.20)	Young and old healthy subjects	—	Subjective rating	No difference	Everyday PM task	Retrieval and execution phase	Lower stress levels resulted in better PM	$d^{\text{PM performance}} = -1.42$
Walser et al. (2013)	Controls 20 ♂; 21♀ (*M* = 21.96) Stressed 20 ♂; 21♀ (*M* = 21.96)	Healthy subjects	TSST	Induced salivary cortisol	Event-based PM	Ongoing task paradigm	Retrieval and execution phase	Stress had no effect on event-based PM	$d^{\text{event-based PM}} = .01$
Ihle et al. (2014)	3 ♂; 30 ♀ (*M* = 20.80) 12 ♂; 29 ♀ (*M* = 65.20)	Young and old healthy subjects	—	Subjective rating and blood pressure	Time-based PM	Ongoing task paradigm	—	Stress had no effect on time-based PM	$d^{\text{time-based PM}} = .11$
Glienke & Piefke (2016)	Controls 24 ♂ (*M* = 23.64) Stressed 25 ♂ (*M* = 23.95)	Healthy subjects	SECPT	Induced salivary cortisol	Time-based PM & event-based PM	Computerized real life-related PM task	Planning phase	Stress improved time-based PM & event-based PM	$d^{\text{PM performance}} = .34$ $d^{\text{event-based PM}} = .17$ $d^{\text{time-based PM}} = .27$

Note. Hedges *g/d* = (average) effect size of a particular study. TSST = trier social stress test; PM = Prospective Memory.

Table 2
Results of Pooled Averaged Effect Sizes

	Significance test for average effect size		Analysis of effect size heterogeneity		
	d	95% CI	χ^2	*df*	Q
Stress characteristics					
Acute stress ($n = 6$)	.24*	($.19 \leq d \leq .28$)	5.47	5	2.59
Basal stress ($n = 3$)	−.21	($-.44 \leq d \leq .02$)	5.88	2	14.82*
PM type					
Time-based PM ($n = 3$)	.28*	($.21 \leq d \leq .34$)	2.6	2	1.57
Event-based PM ($n = 4$)	.13*	($.10 \leq d \leq .16$)	6.64	3	.55
Overall PM ($n = 2$)	−.35	($-.76 \leq d \leq .07$)	.80	1	14.65*

Note. Integration was done with respect to our hypotheses for healthy adults.* Indicate that the average effect size is significantly different to 0 or the *d*s of one class of data has a heterogeneous distribution. PM = Prospective Memory.

significant average effect size for studies that did not differ between time and event-dependent PM. This indicates that the differentiation of effect sizes for time- and event-based PM may explain in part the differential effects of stress on PM reported in published studies. Data from studies that did not distinguish between time- and event-dependent only indicate that stress may have differential effects on overall PM. The test of publication biases showed a significant negative correlation of the standard effect sizes and their variance ($r = -.25$; $p < .05$), indicating the absence of any publication bias.

Cognitive Disturbances Associated With PTSD

Numerous researchers investigated RM in patients with PTSD (for a review, see Buckley, Blanchard, & Neill, 2000). Studies of retrospective real life memory consistently reported that PTSD is associated with impaired autobiographical memory (e.g., Beblo et al., 2006; LaGarde, Doyon, & Brunet, 2010; McNally, 2006; Piefke et al., 2008). These findings are of particular interest since autobiographical memory deeply integrates retrospective and PM processes (Schacter et al., 2007). As displayed in Table 3, a similar picture of alterations in patients with PTSD is detectable for PM performance. All authors reported difficulties in PM performance for PTSD patients (Brown et al., 2013, 2014; Kleim, Graham, Fihosy, Stott, & Ehlers, 2014; McFarland et al., 2016; Scott et al., 2016).

Studies on the impact of stress on different phases of PM in patients with PTSD have not been published yet.

Analysis of Effect Sizes for the Clinical Samples

As shown in Table 3 we found a broad range of effects sizes for the effect of PTSD on PM from $d = -1.99$ (Brown et al., 2013) to $d = .14$ (Brown et al., 2014). The integration of effect sizes revealed an average weighted effect size of $d = -.58$ ($-.67 \leq d \leq -.49$) for all studies. The homogeneity analysis showed a lower Q (14.86) than the critical value of the χ^2 distribution ($\chi^2_8 = 17{,}49$; $p > .05$), indicating a homogeneous distribution of *d*s. The *d*s thus share a common underlying effect size and therefore seem to come from similar populations and experimental conditions. The analysis of effect sizes of results from studies of patients with PTSD thus does not indicate statistical differences in the effect of PTSD on PM between the studies.

Discussion

Based on the available literature, this review analyzed the effects of stress on PM in healthy adults and patients with PTSD depending on (a) the stressor characteristics, (b) whether PM is time- or event-based, and (c) which phase of PM processing is directly affected by the stressor. Results indicate that the effects of stress on PM are modulated by stressor characteristics and different types (i.e., time- and event-

Table 3
Summary of Descriptive Facets and Average Effect Sizes of PM Performance for the Included Clinical Studies

Study	Number of subjects (age in years = Mean)	Subjects	Type of PM	PM task	PM phase affected by stress	Stress effect on PM	n_d	Hedges *g*/*d*
Kleim et al. (2014)	PTSD 10 ♂; 20 ♀ (M = 42.30) Non-PTSD 10 ♂; 10 ♀ (M = 37.00)	PTSD vs. non-PTSD subjects	Imaged future events	Future-based autobiographical memory test	—	PTSD patients imagined future events fewer specific.	2	$d^{\text{PM performance}} = -.47$
Brown et al. (2013)	PTSD 12 ♂ (M = 30.25) Non-PTSD 16 ♂ (M = 31.38)	Combat veterans with PTSD vs. non-PTSD combat veterans	Imaged future events	Autobiographical memory test	—	PTSD patients overgenerate future events.	2	$d^{\text{PM performance}} = -1{,}99$
Brown et al. (2014)	PTSD 12 ♂ Non-PTSD 16 ♂ (M = 31.38; M = 30.25)	Combat veterans with PTSD vs. non-PTSD combat veterans	Imaged future events	Autobiographical interview (AI)	—	PTSD generated more external, than episodic internal details of imagined future. Greater symptom severity leads to fewer episodic details.	2	$d^{\text{PM performance}} = .14$
Scott et al. (2016)	PTSD 34 ♂; 6 ♀ (M = 35.2) Non-PTSD 35♂; 3 ♀ (M = 32.9)	Combat veterans with PTSD vs. non-PTSD combat veterans	Time-based PM and event-based PM	Memory for intention task (MIST)	—	PTSD patients show poorer PM, especially for time-based PM.	0	$d^{\text{PM performance}} = -.58$ $d^{\text{event-based PM}} = -.39$ $d^{\text{time-based PM}} = -.56$
McFarland et al. (2016)	PTSD 35 ♂, 5 ♀ (M = 39.6)	Combat veterans with PTSD	Event-based PM	Ongoing task paradigm	—	Elevated PTSD symptoms are associated with more difficulties in event-based PM.	3	$d^{\text{PM performance}} = -.96$

Note. N_d = total number of *d*s; Hedges *g*/*d* = (average) effect size of a particular study. PTSD = posttraumatic stress disorder; PM = Prospective Memory.

dependent) of PM in healthy adults. The calculation of effect size demonstrates that the experimental induction of short acute stress may improve PM. For studies that investigated basal stress neither an improving nor a deteriorating association between stress levels and PM performance could be detected. Effect sizes for the influence of stress on distinct phases of PM could not be calculated because too few studies are published on this issue. Effects sizes for studies of patients with PTSD demonstrate a deteriorating effect of the disorder on overall PM. Based on the results of our review we will now discuss our hypotheses on the modulating factors of PM in healthy adults and patients with PTSD.

Healthy Adults

Effects of stressor characteristics. We hypothesized that the reported diverging effects of stress on PM performance may be a result of different stress characteristics. Ihle et al. (2012) and Nakayama, Takahashi, and Radford (2005) both investigated the influence of basal stress levels on PM (i.e., stress levels in the absence of stress treatment). Nakayama et al. (2005) found no effect of different basal stress levels whereas Ihle and collegueas (2012) reported better PM performance under lower stress levels. However, only Nakayama et al. (2005) relied their results on the measured stress hormone cortisol. Ihle et al. (2012) differentiated their results on either subjective stress ratings. Supposedly, subjective stress measures and cortisol measurement describe different stress reactions. Subjective stress ratings usually contain the SAM-system. Bodily changes during the fast stress reaction are much better detectable for subjects and are reflected by physiological measure like blood pressure, than the HPA-axis that results in the release in cortisol. Therefore, different influence of stress may be a result of different stress reactions that were measured. Certainly Ihle et al. (2012) applied an everyday PM task, which aimed at assessing retrieval and execution of intentions over a time period of 5 days in a naturalistic study design. Participants formed and encoded their intentions during their everyday routine. Encoded intentions had to be retrieved and executed on the following day. Nakayama et al. (2005), in contrast, tested PM by an event-dependent PM paradigm. Participants were required to mark words that could be classified as clothing (neutral) or words that are classified as names of symptoms of physical disorders (negative emotional). Based on the study designs they are also different in their capabilities to standardize experimental conditions. Therefore, it is reasonable that the combination of different stressor characteristics and task demands may explain the different results for the influence of basal stress on PM. In another study, Ihle et al. (2014) investigated whether aging-related differences in time-based PM performance may be due to higher basal levels of stress in the elderly (relative to young adults) during a laboratory testing situation. Ihle et al. (2014) also measured basal stress niveau but even lowered the basal stress levels by conducting a relaxation intervention. In their experimental paradigm, participants performed a working memory task while pressing a computer button every minute to test time-dependent PM. Half of the older and half of the younger participants accomplished a relaxation intervention before the time-based PM task. Ihle et al. (2014) found that the relaxation intervention decreased subjective and physiological stress levels in both young and older participants. However, stress levels did not differ between the age groups such that the amount of stress could not explain aging-related differences in PM performance in this laboratory context. This study underpins the results of Nakayama et al. (2005) because both tested PM under controlled conditions and found no effect of basal stress on PM, although, again different forms of stress were measured.

Glienke and Piefke (2016); Nater et al. (2006) and Walser et al. (2013) induced systematically and standardized acute stress by social stress tests. While Glienke and Piefke (2016) applied the socially evaluated cold pressure test (SECPT), Nater et al. (2006) and Walser et al. (2013) conducted the trier social stress test (TSST). Both stress exposure methods resulted in a reliable cortisol increase (Kirschbaum, Pirke, & Hellhammer, 1993; Schwabe, Haddad, & Schachinger, 2008). Nater et al. (2006) reported selectively improved time-based PM performance after the induction of psychosocial stress, with no such effect on event-based PM (see Figure 2). Nater and colleagues (2006) implied an ongoing word-rating task, wherein the participant was asked to either press a target

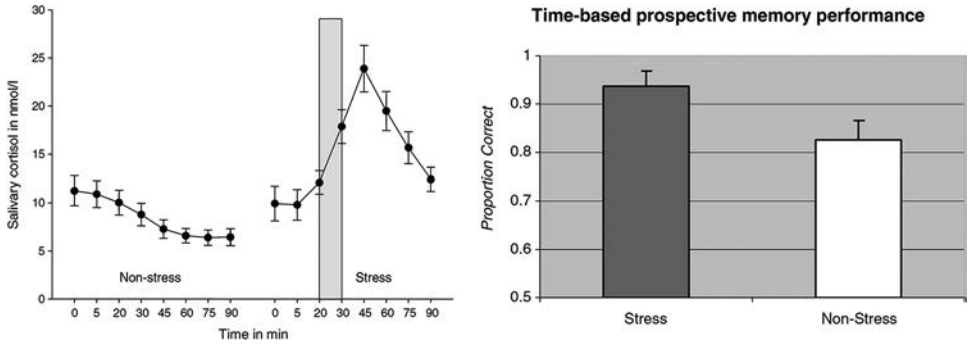


Figure 2. Differential effects of stress on time-based PM. In a study by Nater et al. (2006) it is shown that time-based PM was improved by the stressor, while there was no effect of stress on event-based PM. Nater et al. (2006) did not differentiate between the distinct phases of PM illustrated in Figure 1. It is most likely that the effects of stress on PM may also depend on the time point of stress induction (i.e., before the planning, the retention, the performance, or the evaluation phase).

key every 2 min (time-dependent PM) or whenever a target word appeared on the screen (event-dependent PM). Walser et al. (2013) intended to explore the missing effect of stress on event-based PM in the paradigm of Nater et al. (2006). In their study, the authors specified and expanded the event-depended PM condition by including (a) response times into their analyses and (b) implementing an additional deactivation phase into the paradigm. The latter experimental variation allowed them to test potential effects of stress on intention-deactivation after the completion of each PM trial. This approach is based on the view that after having completed a PM challenge intentions, which are no longer relevant, are deactivated to avoid interference with the demands of following PM tasks. Following this account, Walser and colleagues (2013) aimed at exploring whether stress may lead to changes of intention-deactivation by measuring if participants responded to PM cues that were required in the PM block before. Neither in event-dependent PM performance, nor in the response times, Walser et al. (2013) found differences between the stress and the control groups. Likewise, intention-deactivation after completion of each trial did not differ between the two groups. Given these results, the authors proposed that processes like intention retrieval and intention-deactivation may preserve even under acute stress conditions. Recently Glienke and Piefke (2016) separated PM in its different phases and applied a complex real life-like PM task that involved the planning, retention, and performance of intentions during a fictional holiday week. Half of the subjects were stressed with the SECPT before the planning of intentions, and the other half of the participants underwent a control procedure at the same time. Glienke and Piefke (2016) found that the performance of participants treated with the SECPT procedure before the planning phase remained constantly better time- and event-dependent PM retrieval over the performance phase, while performance of controls declined.

With respect to the hypothesis that the stressor characteristics may explain at least in part, we also have to take into account some important differences between the two most frequently applied experimental stressors in neuroendocrinological and neuropsychological research: the SECPT and the TSST. Both stressors have repeatedly been shown to induce robust effects on the physiological marker cortisol (Giles, Mahoney, Brunyé, Taylor, & Kanarek, 2014; Kirschbaum et al., 1993; Schwabe et al., 2008), on mood and emotion processing (e.g., Giles et al., 2014), and on cognitive functions (e.g., Het et al., 2005; Schwabe, Joëls, Roozendaal, Wolf, & Oitzl, 2012; Schwabe, Wolf, & Oitzl, 2010). Conceptually, the TSST represents a social stressor, whereas the SECPT includes both a social stressor component and—with the ice cold water condition—a physiological component. Additionally, the TSST proce-

dure lasts 15 min whereas the SECPT is much shorter with its 3-min stress procedure. It has repeatedly been shown that the TSST increases cortisol levels immediately after stress induction, while the SECPT increases cortisol within 20 min post stress treatment (Giles et al., 2014; Kirschbaum et al., 1993; Schwabe et al., 2008). The differences between the SECPT and the TSST in the magnitude of induced cortisol levels were only marginal (Giles et al., 2014). Note, however, that the SECPT induced across studies slightly higher levels of cortisol than the TSST, although the SECPT stress procedure lasts only one-fifth of the duration of the TSST stress procedure. Available data on the effects of the SECPT and TSST stress induction methods thus suggest that a combined social and physiological stressor (represented by the SECPT) is slightly more efficient in the induction of cortisol release than a mere social stressor (represented by the TSST). Since different experimental paradigms were applied in the studies on PM, which used either the SECPT or the TSST, one cannot decide whether the reported differences in cortisol release depended on differential stressor characteristics, distinct PM paradigms, or both (Glienke & Piefke, 2016; Nater et al., 2006; Walser et al., 2013). Moreover, issues related to stressor characteristics interfere with different time points of stress induction during PM processing in the reviewed studies (Glienke & Piefke, 2016; Nater et al., 2006; Walser et al., 2013). Based on the currently available data, we propose that stressor characteristics may at least in part explain the varying results of studies on the effects of stress on PM performance. Importantly, the calculation of effect sizes demonstrates that studies applying a standardized acute experimental stressor demonstrate comparably improving effects on PM (with effect sizes significantly greater than zero). In contrast, effect sizes for studies investigating the influence of basal stress levels on PM do not indicate a similarity between the measured effects. It is likely that in these studies the stressor characteristics are not sufficiently defined, such that they may not explain the varying reported effects of stress on PM performance.

Effects of PM type. We also hypothesized that time- and event-dependent PM may be differentially affected by stress. Cona, Arcara, Tarantino, and Bisiacchi (2012) found, besides a common frontal activation, different even-related potentials (ERPs) for event- and time-dependent PM. Especially in event-based PM the authors found ERPs that reflected increased recruitment of resources required for checking the occurrence PM cues. This is in line with the assumption that event-dependent PM is a self-initiated execution of intentions in response to an external PM cue, whereas time-dependent PM reflects a more internal self-initiated execution to a specific time and is therefore of higher cognitive demand (Nater et al., 2006). Strategic monitoring may be recruited in cases where PM cues possess a low salience and demand less cognitive resources (Cona et al., 2015; McDaniel & Einstein, 2000; Scullin et al., 2013). In consequence, it is reasonable that strategic monitoring is more likely the underlying strategy of event-dependent PM; however, the more demanding a PM task is, the fewer resources are available for strategic monitoring. Spontaneous retrieval has been explained by a reflexive association between the PM cue and the intention stored in PM. However, spontaneous retrieval may also be a result of the salience of the PM cue (Cona et al., 2015; McDaniel & Einstein, 2000; Scullin et al., 2013). PM tasks with high demands like time-dependent PM and salient PM cues would thus be most likely to provoke spontaneous retrieval (Cona et al., 2015; McDaniel & Einstein, 2000; Scullin et al., 2013). Time-dependent PM may thus be likely vulnerable for stress. The analysis of effect sizes revealed significantly increased time- and event-dependent PM in response to stress; however, there was a (nonsignificant) tendency toward a stronger improvement of time-dependent than event-dependent PM. Effect sizes of studies that did not distinguish between time- and event-dependent PM were heterogeneous (i.e., they did not differ from zero). In summary, the calculation of effect sizes corroborated our hypothesis that time- and event-dependent PM are differentially affected by stress in terms of a distinct degree of improvement.

The differential experimental paradigms used to measure time- and event-dependent PM, however, also need to be considered in the debate on putative differences between time- and event-dependent PM in the vulnerability to stress. For example, Nakayama et al. (2005) and

Ihle et al. (2014) applied experimental ongoing task paradigms, whereas Ihle and colleagues (2012) implemented a rather naturalistic PM paradigm. Nater et al. (2006) and Walser and colleagues (2013) applied an ongoing PM task, while Glienke and Piefke (2016) used a complex computerized real-life-related PM task. Ihle and colleagues (2012) did not differentiate between time-based or event-based PM. Nakayama et al. (2005) and Walser et al. (2013) investigated event-dependent PM, and Ihle et al. (2014) solely addressed time-depenendent PM. Nater et al. (2006) as well as Glienke and Piefke (2016) differentiated between time- and event-dependent PM and investigated the influence of stress on both types of PM. In paradigms where ongoing tasks are used, time-dependent PM is supposedly more cognitive demanding than event-dependent PM, and may therefore be more vulnerable to stress. Paradigms possessing a higher complexity may increase the cognitive demands of event-dependent PM. Task complexity may therefore explain at least in part why studies applying naturalistic paradigms found that event-dependent PM was also affected by stress. Following these assumptions, both PM types and study designs may explain the diverging patterns of results on the influence of stress on PM in healthy adults.

Effects of PM phase. As displayed in Table 1 only three studies are published that differentiated between the phases of PM (Glienke & Piefke, 2016; Ihle et al., 2012; Walser et al., 2013). Given the differences in stressor characteristics and experimental designs, results of the three studies cannot be compared with each other concerning the impact of stress on different phases of PM. For the same reasons, effect sizes could not be calculated for the influence of stress on different PM phases. However, following the assumption of the "multiprocess framework" (Cona et al., 2015; McDaniel & Einstein, 2000; Scullin et al., 2013; see also Introduction) for the retrieval and execution phases of PM, it is likely that the effects of stress on the retrieval phase is different from the influence of stress exerted on the planning and retention phases of PM. Since PM has a high relevance in everyday life, future research is needed to further explore during which phase of PM acute stress may have enhancing effects, and during which phase of PM it may deteriorate performance.

Patients With PTSD

Although PTSD represents a paradigmatic model in psychobiological research on the influence of stress on brain functions and behavior, results of studies on PM in PTSD patients cannot easily be analyzed and interpreted with reference to studies of the modulation of PM by stress in healthy participants. This is mainly due to the lack of psychobiological measures such as cortisol concentration and heart rate and the application of different PM paradigms in studies of healthy participants and PTSD patients. Nonetheless, the calculation of effect sizes for overall PM performance indicates that PTSD has a deteriorating effect on PM (average effect size of $d = -.58$).

Effects of stressor characteristics. With regard to our hypothesis that stressor characteristics may explain enhancing or disturbing effects on PM, it needs to be considered that stressor characteristics in PTSD patients need to be defined by the traumatic event that caused the disorder. The results of our review demonstrate that available studies focusing on PM in patients with PTSD recruited combat veterans for their investigations—except the study of Kleim et al. (2014; see Table 3). Characteristics of the stress situation causing the disorder were probably widely comparable in the studies of combat veterans, such that we can assume that the characteristics of combat-related traumatic stress may at least in part explain disturbing effects of PTSD on PM performance.

None of the studies addressed the role of the stress hormone cortisol. In prior research, it is discussed that low-dose cortisol treatment reduces cortisol concentrations in PTSD patients and improves symptoms (Aerni et al., 2004). Based on this assumption, a short and moderate stress exposure that actives the HPA-axis should have a similar effect in patients with PTSD. The putative relevance of this hypothesis for PM capacities in PTSD patients needs to be further investigated.

Effects of PM type. Only two studies on PM in combat veterans with PTSD are published, which differentiated between time- and event-dependent PM (Scott et al., 2016; McFarland et al., 2016). Scott and colleagues (2016)

consulted the Memory for Intention Test (MIST; Kamat et al., 2014; Raskin, 2009; Scott et al., 2016) and found significantly poorer PM performance in PTSD patients for time-dependent PM (but not for event-dependent PM) compared to combat veterans without PTSD. The MIST consists of eight PM trials over 30 min that are counterbalanced by PM type (i.e., time-based vs. event-based), response (i.e., physical vs. verbal), and delay (i.e., 2 min vs. 15 min; Kamat et al., 2014; Raskin, 2009; Scott et al., 2016). McFarland et al. (2016) applied an ongoing PM task similar to the one used by Nater et al. (2006), but only for event-dependent PM. They found a negative correlation between the severity of PTSD symptoms and event-based PM performance. Other authors used imaginative task procedures (Brown et al., 2013, 2014; Kleim et al., 2014) that did not distinguish between time- and event-dependent PM. Brown et al. (2013) presented neutral word cues to combat veterans who were instructed to generate autobiographical memories or imagine future autobiographical events. Data were analyzed for episodic specificity and content. Events of the future were reported more overgeneral by combat veterans with PTSD compared to combat veterans without PTSD. Brown et al. (2014) employed the autobiographical interview (Levine, Svoboda, Hay, Winocur, & Moscovitch, 2002) to investigate autobiographical memory and imagined future event narratives generated by combat veterans with and without PTSD. Responses were coded for the number of episodic and semantic details. Compared to combat veterans without PTSD, those with PTSD generated more semantic than episodic details when recalling past or imagining future events. Interestingly, fewer episodic details were associated with greater symptom severity. Kleim et al. (2014) were the only authors who did not investigate combat veterans in their study. In the PTSD patients included in their investigation, the disorder resulted from assault and motor vehicle accidents. The stressor characteristics therefore differed from those of studies of combat veterans with war-related PTSD. The PM task used by Kleim et al. (2014) addressed the effects of cues with differential emotional valence on the accuracy of autobiographical PM in PTSD patients and healthy controls. They reported that PTSD patients imagined future events less accurately and specifically in response to positive, but not to negative cues relative to the control group. Although the study design and the PM task used by Kleim et al. (2014) are not directly comparable to those applied in the PM studies in combat veterans, the results fit in the overall picture of data on the effect of war-related PTSD on PM.

In summary, different study paradigms make it difficult to interpret the effect of PTSD on time- and event-dependent PM performance. However, the pattern of results from studies of PTSD patients and the respective effect sizes clearly indicate that PTSD deteriorates overall PM performance.

Studies on the impact of stress on different phases of PM (see Hypothesis 3) in patients with PTSD have not been published yet. We know from RM in healthy adults and PTSD patients that stress may have different effects on memory phases (e.g., Het et al., 2005; Wingenfeld et al., 2012; Wingenfeld & Wolf, 2015; Wolf, 2009). Future research is needed to clarify whether similar effects can be found for PM. It may be of particular interest for the treatment of the disorder to examine putative differential positive or negative effects of distinct types of short acute stressors on each phase of PM in patients with PTSD (Wingenfeld et al., 2012). This would probably enable us to develop more precise and efficient rehabilitation interventions.

Conclusions

The present review demonstrates that a short acute stressor may have enhancing effects on PM in healthy humans, depending on the characteristics of the stressor and the type of PM. With regard to the stressor attributes, available data are confounded with the use of differential PM paradigms and the induction of stress at different time points during PM processing. In summary, however, the results of our review indicate that variations of stressor characteristics may in part be relevant for the effects of stress on PM (Giles et al., 2014). Moreover, time-dependent PM appears to be more vulnerable to stress. This may be due to the higher cognitive demands (e.g., memory load, executive capacity) in time-dependent relative to event-dependent PM. Interestingly, there is some evidence across studies that different basal cortisol

levels do not modulate PM performance. In contrast, acute short stress induction and the corresponding differential increases of cortisol may improve PM performance (Glienke & Piefke, 2016). Concerning a putative relationship between stress induction and the different phases of PM, only three studies are published that differentiated between the distinct phases of PM. Moreover, stressor characteristics and experimental designs highly differed between these studies such that one hardly may compare and interpret the study results with respect to the impact of stress on PM phases. Note, in everyday life stress may occur in all phases of PM. It would thus be interesting to explore in future research during which phases of PM acute stress may enhance or deteriorate PM. The knowledge about beneficial effects of stress on certain phases of PM may also be relevant for clinical samples. On the clinical side, data on influences of acute stress on PM in patients with PTSD are rare. These mainly relate PTSD symptoms to declined PM functions. The influence of a short acute stressor on the differential phases of PM has not been investigated yet. Interestingly, some recent studies suggest that the level of cortisol may be an important biomarker for diagnostic and therapeutic considerations in the treatment of patients with PTSD. Therefore, future research on the psychological and neuroendocrinological interactions underlying disturbances of PM in patients with PTSD possesses a central clinical and scientific relevance. It would be of particular interest to investigate how cortisol levels induced by a short acute stress exposure at different time points (i.e., before the planning, retention, or retrieval/execution phases) influences PM performance in patients with PTSD.

References

Addis, D. R., Wong, A. T., & Schacter, D. L. (2007). Remembering the past and imagining the future: Common and distinct neural substrates during event construction and elaboration. *Neuropsychologia, 45,* 1363–1377. http://dx.doi.org/10.1016/j.neuropsychologia.2006.10.016

Aerni, A., Traber, R., Hock, C., Roozendaal, B., Schelling, G., Papassotiropoulos, A., . . . de Quervain, D. J. (2004). Low-dose cortisol for symptoms of posttraumatic stress disorder. *The American Journal of Psychiatry, 161,* 1488–1490. http://dx.doi.org/10.1176/appi.ajp.161.8.1488

Beblo, T., Driessen, M., Mertens, M., Wingenfeld, K., Piefke, M., Rullkoetter, N., . . . Woermann, F. G. (2006). Functional MRI correlates of the recall of unresolved life events in borderline personality disorder. *Psychological Medicine, 36,* 845–856. http://dx.doi.org/10.1017/S0033291706007227

Begg, K. D. (1994). Publication bias. In H. Cooper & L. V. Hedges (Eds.), *The handbook of research synthesis* (pp. 399–409). New York, NY: Russell Sage Foundation.

Brandimonte, M. A., Einstein, G. O., & McDaniel, M. A. (1996). *Prospective memory: Theory and applications*. Mahwah, NJ: Lawrence Erlbaum.

Brandimonte, M. A., Ferrante, D., Feresin, C., & Delbello, R. (2001). Dissociating prospective memory from vigilance processes. *Psicológica, 22,* 97–113.

Bremner, J. D. (2006). Traumatic stress: Effects on the brain. *Dialogues in Clinical Neuroscience, 8,* 445–461.

Brown, A. D., Addis, D. R., Romano, T. A., Marmar, C. R., Bryant, R. A., Hirst, W., & Schacter, D. L. (2014). Episodic and semantic components of autobiographical memories and imagined future events in post-traumatic stress disorder. *Memory, 22,* 595–604. http://dx.doi.org/10.1080/09658211.2013.807842

Brown, A. D., Root, J. C., Romano, T. A., Chang, L. J., Bryant, R. A., & Hirst, W. (2013). Overgeneralized autobiographical memory and future thinking in combat veterans with posttraumatic stress disorder. *Journal of Behavior Therapy and Experimental Psychiatry, 44,* 129–134. http://dx.doi.org/10.1016/j.jbtep.2011.11.004

Buckley, T. C., Blanchard, E. B., & Neill, W. T. (2000). Information processing and PTSD: A review of the empirical literature. *Clinical Psychology Review, 20,* 1041–1065. http://dx.doi.org/10.1016/S0272-7358(99)00030-6

Cahill, L., & Alkire, M. T. (2003). Epinephrine enhancement of human memory consolidation: Interaction with arousal at encoding. *Neurobiology of Learning and Memory, 79,* 194–198. http://dx.doi.org/10.1016/S1074-7427(02)00036-9

Cannon, W. B. (1929). *Bodily changes in pain, hunger, fear and rage* (2nd ed.). New York, NY: Appleton.

Cheng, H.-D., Wang, K., Xi, C. H., Niu, C. S., & Fu, X.-M. (2008). Prefrontal cortex involvement in the event-based prospective memory: Evidence from patients with lesions in the prefrontal cortex. *Brain Injury, 22,* 697–704. http://dx.doi.org/10.1080/02699050802263035

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*. New York, NY: Lawrence Erlbaum.

Cona, G., Arcara, G., Tarantino, V., & Bisiacchi, P. S. (2012). Electrophysiological correlates of strategic monitoring in event-based and time-based prospective memory. *PLoS ONE, 7*(2), e31659. http://dx.doi.org/10.1371/journal.pone.0031659

Cona, G., Scarpazza, C., Sartori, G., Moscovitch, M., & Bisiacchi, P. S. (2015). Neural bases of prospective memory: A meta-analysis and the "Attention to Delayed Intention" (AtoDI) model. *Neuroscience and Biobehavioral Reviews, 52,* 21–37. http://dx.doi.org/10.1016/j.neubiorev.2015.02.007

Dedovic, K., Duchesne, A., Andrews, J., Engert, V., & Pruessner, J. C. (2009). The brain and the stress axis: The neural correlates of cortisol regulation in response to stress. *NeuroImage, 47,* 864–871. http://dx.doi.org/10.1016/j.neuroimage.2009.05.074

de Kloet, E. R., Joëls, M., & Holsboer, F. (2005). Stress and the brain: From adaptation to disease. *Nature Reviews Neuroscience, 6,* 463–475. http://dx.doi.org/10.1038/nrn1683

de Quervain, D. J.-F., Henke, K., Aerni, A., Treyer, V., McGaugh, J. L., Berthold, T., . . . Hock, C. (2003). Glucocorticoid-induced impairment of declarative memory retrieval is associated with reduced blood flow in the medial temporal lobe. *European Journal of Neuroscience, 17,* 1296–1302. http://dx.doi.org/10.1046/j.1460-9568.2003.02542.x

Einstein, G. O., & McDaniel, M. A. (1990). Normal aging and prospective memory. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 16,* 717–726. http://dx.doi.org/10.1037/0278-7393.16.4.717

Ellis, J. A. (1996). Prospective memory or the realization of delayed intentions: A conceptual framework for research. In M. Brandimonte, G. Einstein, & M. A. McDaniel (Eds.), *Prospective memory: Theory and applications* (pp. 1–22). Mahwah, NJ: Lawrence Erlbaum.

Elmlinger, M. W., Kühnel, W., & Ranke, M. B. (2002). Reference ranges for serum concentrations of lutropin (LH), follitropin (FSH), estradiol (E2), prolactin, progesterone, sex hormone-binding globulin (SHBG), dehydroepiandrosterone sulfate (DHEAS), cortisol and ferritin in neonates, children and young adults. *Clinical Chemistry and Laboratory Medicine, 40,* 1151–1160. http://dx.doi.org/10.1515/cclm.2002.202

Elzinga, B. M., Roelofs, K., Tollenaar, M. S., Bakvis, P., van Pelt, J., & Spinhoven, P. (2008). Diminished cortisol responses to psychosocial stress associated with lifetime adverse events: A study among healthy young subjects. *Psychoneuroendocrinology, 33,* 227–237. http://dx.doi.org/10.1016/j.psyneuen.2007.11.004

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*(12), e113618. http://dx.doi.org/10.1371/journal.pone.0113618

Glienke, K., & Piefke, M. (2016). Acute social stress before the planning phase improves memory performance in a complex real life-related prospective memory task. *Neurobiology of Learning and Memory, 133,* 171–181. http://dx.doi.org/10.1016/j.nlm.2016.06.025

Goldstein, D. S., & Kopin, I. J. (2007). Evolution of concepts of stress. *Stress, 10,* 109–120. http://dx.doi.org/10.1080/10253890701288935

Goldstein, D. S., & McEwen, B. (2002). Allostasis, homeostats, and the nature of stress. *Stress, 5,* 55–58. http://dx.doi.org/10.1080/10253890290012345

Graf, P., & Uttl, B. (2001). Prospective memory: A new focus for research. *Consciousness and Cognition, 10,* 437–450. http://dx.doi.org/10.1006/ccog.2001.0504

Guynn, M. J. (2003). A two-process model of strategic monitoring in event-based prospective memory: Activation/retrieval mode and checking. *International Journal of Psychology, 38,* 245–256. http://dx.doi.org/10.1080/00207590344000178

Hedges, L. V., & Olkin, I. (1985). *Statistical Methods for Meta-analysis.* Orlando, FL: Academic Press.

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

Holsboer, F., & Ising, M. (2010). Stress hormone regulation: Biological role and translation into therapy. *Annual Review of Psychology, 61,* 81–109, C1–C11. http://dx.doi.org/10.1146/annurev.psych.093008.100321

Ihle, A., Kliegel, M., Hering, A., Ballhausen, N., Lagner, P., Benusch, J., . . . Schnitzspahn, K. M. (2014). Adult age differences in prospective memory in the laboratory: Are they related to higher stress levels in the elderly? *Frontiers in Human Neuroscience, 8,* 1021. http://dx.doi.org/10.3389/fnhum.2014.01021

Ihle, A., Schnitzspahn, K., Rendell, P. G., Luong, C., & Kliegel, M. (2012). Age benefits in everyday prospective memory: The influence of personal task importance, use of reminders and everyday stress. *Aging, Neuropsychology, and Cognition, 19,* 84–101. http://dx.doi.org/10.1080/13825585.2011.629288

Kamat, R., Weinborn, M., Kellogg, E. J., Bucks, R. S., Velnoweth, A., & Woods, S. P. (2014). Construct validity of the Memory for Intentions Screening Test (MIST) in healthy older adults. *Assessment, 21,* 742–753. http://dx.doi.org/10.1177/1073191114530774

Kirschbaum, C., & Hellhammer, D. H. (1994). Salivary cortisol in psychoneuroendocrine research: Recent developments and applications. *Psychoneuroendocrinology, 19,* 313–333. http://dx.doi.org/10.1016/0306-4530(94)90013-2

Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test'—A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28,* 76–81. http://dx.doi.org/10.1159/000119004

Kleim, B., Graham, B., Fihosy, S., Stott, R., & Ehlers, A. (2014). Reduced specificity in episodic future thinking in posttraumatic stress disorder. *Clinical Psychological Science, 2,* 165–173. http://dx.doi.org/10.1177/2167702613495199

Kliegel, M., Martin, M., McDaniel, M. A., & Einstein, G. O. (2001). Varying the importance of a prospective memory task: Differential effects across time- and event-based prospective memory. *Memory, 9,* 1–11. http://dx.doi.org/10.1080/09658210042000003

Kremen, W. S., O'Brien, R. C., Panizzon, M. S., Prom-Wormley, E., Eaves, L. J., Eisen, S. A., . . . Franz, C. E. (2010). Salivary cortisol and prefrontal cortical thickness in middle-aged men: A twin study. *NeuroImage, 53,* 1093–1102. http://dx.doi.org/10.1016/j.neuroimage.2010.02.026

Kudielka, B. M., Buske-Kirschbaum, A., Hellhammer, D. H., & Kirschbaum, C. (2004). HPA axis responses to laboratory psychosocial stress in healthy elderly adults, younger adults, and children: Impact of age and gender. *Psychoneuroendocrinology, 29,* 83–98. http://dx.doi.org/10.1016/S0306-4530(02)00146-4

LaGarde, G., Doyon, J., & Brunet, A. (2010). Memory and executive dysfunctions associated with acute posttraumatic stress disorder. *Psychiatry Research, 177,* 144–149. http://dx.doi.org/10.1016/j.psychres.2009.02.002

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York, NY: Springer.

Levine, B., Svoboda, E., Hay, J. F., Winocur, G., & Moscovitch, M. (2002). Aging and autobiographical memory: Dissociating episodic from semantic retrieval. *Psychology and Aging, 17,* 677–689. http://dx.doi.org/10.1037/0882-7974.17.4.677

Lupien, S. J., Maheu, F., Tu, M., Fiocco, A., & Schramek, T. E. (2007). The effects of stress and stress hormones on human cognition: Implications for the field of brain and cognition. *Brain and Cognition, 65,* 209–237. http://dx.doi.org/10.1016/j.bandc.2007.02.007

Mason, J. W. (1968). A review of psychoendocrine research on the sympathetic-adrenal medullary system. *Psychosomatic Medicine, 30*(Suppl.), 631–653. http://dx.doi.org/10.1097/00006842-196809000-00022

McDaniel, M. A., & Einstein, G. O. (2000). Strategic and automatic processes in prospective memory retrieval: A multiprocess framework. *Applied Cognitive Psychology, 14,* S127–S144. http://dx.doi.org/10.1002/acp.775

McDaniel, M. A., & Einstein, G. O. (2007). *Prospective memory: An overview and synthesis of an emerging field*. Thousand Oaks, CA: SAGE.

McFarland, C. P., Clark, J. B., Lee, L. O., Grande, L. J., Marx, B. P., & Vasterling, J. J. (2016). Event-based prospective memory among veterans: The role of posttraumatic stress disorder symptom severity in executing intentions. *Journal of Clinical and Experimental Neuropsychology, 38,* 251–260. http://dx.doi.org/10.1080/13803395.2015.1102203

McNally, R. J. (2006). Cognitive abnormalities in posttraumatic stress disorder. *Trends in Cognitive Sciences, 10,* 271–277. http://dx.doi.org/10.1016/j.tics.2006.04.007

Meewisse, M.-L., Reitsma, J. B., de Vries, G.-J., Gersons, B. P. R., & Olff, M. (2007). Cortisol and posttraumatic stress disorder in adults: Systematic review and meta-analysis. *The British Journal of Psychiatry, 191,* 387–392. http://dx.doi.org/10.1192/bjp.bp.106.024877

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin, 133,* 25–45. http://dx.doi.org/10.1037/0033-2909.133.1.25

Nakayama, Y., Takahashi, T., & Radford, M. H. (2005). Cortisol levels and prospective and retrospective memory in humans. *Neuroendocrinology Letters, 26,* 599–602.

Nater, U. M., Okere, U., Stallkamp, R., Moor, C., Ehlert, U., & Kliegel, M. (2006). Psychosocial stress enhances time-based prospective memory in healthy young men. *Neurobiology of Learning and Memory, 86,* 344–348. http://dx.doi.org/10.1016/j.nlm.2006.04.006

Okuda, J., Fujii, T., Ohtake, H., Tsukiura, T., Yamadori, A., Frith, C. D., & Burgess, P. W. (2007). Differential involvement of regions of rostral prefrontal cortex (Brodmann area 10) in time- and event-based prospective memory. *International Journal of Psychophysiology, 64,* 233–246. http://dx.doi.org/10.1016/j.ijpsycho.2006.09.009

Peeters, F., Nicolson, N. A., & Berkhof, J. (2004). Levels and variability of daily life cortisol secretion in major depression. *Psychiatry Research, 126,* 1–13. http://dx.doi.org/10.1016/j.psychres.2003.12.010

Piefke, M., Pestinger, M., Arin, T., Kohl, B., Kastrau, F., Schnitker, R., . . . Flatten, G. (2008). The neurofunctional mechanisms of traumatic and non-traumatic memory in patients with acute PTSD following accident trauma. *Neurocase, 13,* 342–

357. http://dx.doi.org/10.1080/13554790701851494

Raskin, S. (2009). Memory for Intentions Screening Test: Psychometric properties and clinical evidence. *Brain Impairment, 10,* 23–33. http://dx.doi.org/10.1375/brim.10.1.23

Schacter, D. L., Addis, D. R., & Buckner, R. L. (2007). Remembering the past to imagine the future: The prospective brain. *Nature Reviews Neuroscience, 8,* 657–661. http://dx.doi.org/10.1038/nrn2213

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., 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

Scott, J. C., Woods, S. P., Wrocklage, K. M., Schweinsburg, B. C., Southwick, S. M., & Krystal, J. H. (2016). Prospective memory in posttraumatic stress disorder. *Journal of the International Neuropsychological Society, 22,* 724–734. http://dx.doi.org/10.1017/S1355617716000564

Scullin, M. K., McDaniel, M. A., & Shelton, J. T. (2013). The Dynamic Multiprocess Framework: Evidence from prospective memory with contextual variability. *Cognitive Psychology, 67,* 55–71. http://dx.doi.org/10.1016/j.cogpsych.2013.07.001

Simons, J. S., Schölvinck, M. L., Gilbert, S. J., Frith, C. D., & Burgess, P. W. (2006). Differential components of prospective memory? Evidence from fMRI. *Neuropsychologia, 44,* 1388–1397. http://dx.doi.org/10.1016/j.neuropsychologia.2006.01.005

Terrett, G., Rose, N. S., Henry, J. D., Bailey, P. E., Altgassen, M., Phillips, L. H., . . . Rendell, P. G. (2015). The relationship between prospective memory and episodic future thinking in younger and older adulthood. *The Quarterly Journal of Experimental Psychology, 69,* 310–323. http://dx.doi.org/10.1080/17470218.2015.1054294

Walser, M., Fischer, R., Goschke, T., Kirschbaum, C., & Plessow, F. (2013). Intention retrieval and deactivation following an acute psychosocial stressor. *PLoS ONE, 8*(12), e85685. http://dx.doi.org/10.1371/journal.pone.0085685

West, R., McNerney, M. W., & Krauss, I. (2007). Impaired strategic monitoring as the locus of a focal prospective memory deficit. *Neurocase, 13,* 115–126. http://dx.doi.org/10.1080/13554790701399247

Wingenfeld, K., Driessen, M., Terfehr, K., Schlosser, N., Fernando, S. C., Otte, C., . . . Wolf, O. T. (2012). Cortisol has enhancing, rather than impairing effects on memory retrieval in PTSD. *Psychoneuroendocrinology, 37,* 1048–1056. http://dx.doi.org/10.1016/j.psyneuen.2011.12.002

Wingenfeld, K., & Wolf, O. T. (2015). Effects of cortisol on cognition in major depressive disorder, posttraumatic stress disorder and borderline personality disorder—2014 Curt Richter Award winner. *Psychoneuroendocrinology, 51,* 282–295. http://dx.doi.org/10.1016/j.psyneuen.2014.10.009

Wolf, O. T. (2009). Stress and memory in humans: Twelve years of progress? *Brain Research, 1293,* 142–154. http://dx.doi.org/10.1016/j.brainres.2009.04.013

Yehuda, R. (2002). Posttraumatic stress disorder. *The New England Journal of Medicine, 346,* 108–114. http://dx.doi.org/10.1056/NEJMra012941

Yehuda, R. (2006). Advances in understanding neuroendocrine alterations in PTSD and their therapeutic implications. *Annals of the New York Academy of Sciences, 1071,* 137–166. http://dx.doi.org/10.1196/annals.1364.012

Yehuda, R., Teicher, M. H., Trestman, R. L., Levengood, R. A., & Siever, L. J. (1996). Cortisol regulation in posttraumatic stress disorder and major depression: A chronobiological analysis. *Biological Psychiatry, 40,* 79–88. http://dx.doi.org/10.1016/0006-3223(95)00451-3

Zubin, J., & Spring, B. (1977). Vulnerability—A new view of schizophrenia. *Journal of Abnormal Psychology, 86,* 103–126. http://dx.doi.org/10.1037/0021-843X.86.2.103

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