

The Effects of Stress on Prospective Memory

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ABSTRACT

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*

INTRODUCTION

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 hypothesize 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.

II. THE BIOLOGICAL STRESS REACTION

The stress reaction can be conceptualized as the adaptive response of an organism aiming at the reinstatement 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)

III. 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.

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, & 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 spontaneously 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 & Uttil, 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).

IV. 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.

V. METHOD

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

VI. 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 psychoendocrinological 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; Wingefeld & 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, & Olf, 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.

V. 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.

VIII. 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, post-traumatic 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.

IX. STRESS INFLUENCE 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.

X. 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 eventdependent) 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.

XI. HEALTHY ADULTS

Effects of stressor characteristics. 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 colleagues (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 every day 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.

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-dependent 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.

XII.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.

XIII.CONCLUSION

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 (Glienne & 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 every-day 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 neuro endocrinological 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.

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