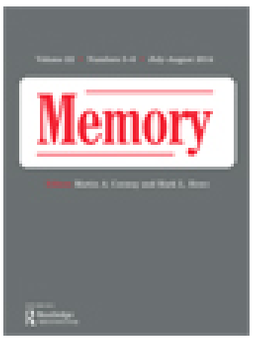

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REVIEW ARTICLE

Memory dynamics under stress

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ABSTRACT

Stressful events have a major impact on memory. They modulate memory formation in a time-dependent manner, closely linked to the temporal profile of action of major stress mediators, in particular catecholamines and glucocorticoids. Shortly after stressor onset, rapidly acting catecholamines and fast, non-genomic glucocorticoid actions direct cognitive resources to the processing and consolidation of the ongoing threat. In parallel, control of memory is biased towards rather rigid systems, promoting habitual forms of memory allowing efficient processing under stress, at the expense of "cognitive" systems supporting memory flexibility and specificity. In this review, we discuss the implications of this shift in the balance of multiple memory systems for the dynamics of the memory trace. Specifically, stress appears to hinder the incorporation of contextual details into the memory trace, to impede the integration of new information into existing knowledge structures, to impair the flexible generalisation across past experiences, and to hamper the modification of memories in light of new information. Delayed, genomic glucocorticoid actions might reverse the control of memory, thus restoring homeostasis and "cognitive" control of memory again.

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Stress; glucocorticoids; noradrenaline; hippocampus; multiple memory systems; memory generalisation; reconsolidation

Memories are highly dynamic entities. After initial encoding, memories remain fragile and susceptible to numerous amnesic agents or modifications by new information (Gordon & Spear, 1973; McGaugh, 1966). Over time, the new memory trace becomes more and more resistant to disruptions until it is consolidated (McGaugh, 2000; McGaugh, McIntyre, & Power, 2002). However, even consolidated memories are not stable and fixed (Dudai & Eisenberg, 2004; Hardt, Einarsson, & Nader, 2010; Misanin, Miller, & Lewis, 1968; Schneider & Sherman, 1968). In fact, several studies have shown that once reactivated during retrieval, memories may become transiently labile again requiring another period of stabilisation called "reconsolidation" (Nader & Hardt, 2009; Sara, 2000). During reconsolidation, seemingly stable memories can be weakened, strengthened or updated (Nader, 2015; Schwabe, Nader, & Pruessner, 2014). As long as memories remain relevant to the individual and the environment of the individual changes, the reactivation and modification rarely stops (Dudai, 2012; McKenzie & Eichenbaum, 2011). As time after learning proceeds, memories tend to be transformed from highly specific, episodic memories to more gist-like, semantic memories contributing to knowledge structures called schemas (Nadel, Hupbach, Gomez, & Newman-Smith, 2012). These schemas have a substantial impact on encoding, consolidation and retrieval; they enable learning against the background of prior experiences (Wang & Morris, 2010). The proposed circle of (re)consolidation and (schema) modification is well in line with a dynamic view of memory that dominates cognitive psychology and conceptualises memory as a highly constructive process (Schacter & Addis, 2007). Memory construction often involves a reorganisation of the stored information, making memories prone to distortions but allowing also updating of memories in the light of new information (Schacter, 1999; Schacter, Guerin & St Jacques, 2011; St Jacques & Schacter, 2013).

The formation, modification and re-modification of the dynamic engram are subject to many influences. One of the most powerful modulators of memory is emotional arousal and stress (Christianson & Mjorndal, 1985; Diamond, Campbell, Park, Halonen, & Zoladz, 2007; Joels, Fernandez, & Roozendaal, 2011; Schwabe, Joels, Roozendaal, Wolf, & Oitzl, 2012). Stressful events provoke an orientation of attentional and memory processes towards threat-related stimuli (de Kloet, Joels, & Holsboer, 2005; Hermans, Henckens, Joels, & Fernandez, 2014). This cognitive response to stressors is mediated through the many physiological changes that occur in response to stress. In particular, within seconds after stressor onset, the sympathetic branch of the autonomic nervous system (ANS) is activated, triggering the release of adrenaline and noradrenaline from the adrenal medulla. These catecholamines lead to well-known stress symptoms such

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as increased heart rate, sweating or accelerated breathing but at the same time enhance alertness, arousal and attention (Clark, Geffen, & Geffen, 1987; Robbins, 1984). This ANS-driven enhanced arousal state normalises soon after stressor offset, typically within a few minutes (Hermans et al., 2014; Joels & Baram, 2009; Ulrich-Lai & Herman, 2009). In parallel to the activation of the ANS, the hypothalamus activates an endocrine response system, the hypothalamic–pituitary–adrenal (HPA) axis. Activation of the HPA axis leads via intermediate steps and with a delay of about 15 minutes to the release of glucocorticoids (mainly cortisol in humans) from the adrenal cortex (Joels & Baram, 2009; Ulrich-Lai & Herman, 2009). Glucocorticoids can readily enter the brain where they bind to two types of receptors: The widely distributed glucocorticoid receptor (GR) and the mineralocorticoid receptor (MR) that is mainly located in limbic and prefrontal areas (Reul & de Kloet, 1985). Traditionally, GR and MR were thought to be mainly intra-cellular receptors enabling gene-mediated glucocorticoid effects that take several hours to develop (Derijk & de Kloet, 2008; Joels, Sarabdjitsingh, & Karst, 2012). More recent research, however, points also to membrane-associated MR and GR that allow rapid, non-genomic actions of glucocorticoids shortly after their release (see Figure 1(A)). Thus, the major physiological stress mediators operate on different time scales, ranging from milliseconds to days or even months (Joels & Baram, 2009).

Past research has mainly focused on the action of these stress mediators on different memory stages. It is well-known that stress has an opposite effect on memory consolidation and retrieval. Whereas consolidation is often enhanced by stress and stress hormones (Cahill, Gorski, & Le, 2003; Roozendaal & McGaugh, 1996; Roozendaal, Okuda, de Quervain, & McGaugh, 2006; Smeets, Otgaar, Candel, & Wolf, 2008), memory retrieval is typically impaired (Buchanan, Tranel, & Adolphs, 2006; de Quervain, Roozendaal, Nitsch, McGaugh, & Hock, 2000; Kuhlmann, Piel, & Wolf, 2005; Roozendaal, Griffith, Buranday, De Quervain, & McGaugh, 2003; Schwabe & Wolf, 2009a). Recent evidence, however, indicates that the impact of stress and stress mediators on memory formation and retrieval depends critically on the fine-tuned orchestration of the major stress response systems. Both the enhancement of memory formation and the impairment of memory retrieval require precisely timed interactions of glucocorticoids and arousal-related noradrenergic activity (as outlined below; Joels et al., 2011; Roozendaal, Okuda, Van der Zee, & McGaugh, 2006; Roozendaal, Quirarte, & McGaugh, 2002). De-synchronised glucocorticoid and noradrenergic activity may have different effects. In the first part of this review, we will give an overview of these temporal dynamics of stress effects on memory processes.

Beyond the modulation of memory consolidation and retrieval, the fine-tuned activation of stress response systems is also thought to result in large-scale network changes that promote processing of salient, threat-related events, at the expense of deliberate executive control processes during the acute stress phase (Hermans et al., 2014, 2011). This shift from executive control towards salience processing networks may set the stage for a shift in the brain systems that guide learning and memory (Schwabe, 2016). In fact, there is accumulating evidence showing that stressful events favour rather rigid "habitual" memory processes that involve learning automatised responses by associating behaviours with single stimuli over more flexible "cognitive" memory processes that built associative structures by using the relationships between multiple stimuli (Braun & Hauber, 2013; Kim, Lee, Han, & Packard, 2001; Packard & Wingard, 2004; Schwabe et al., 2007; Schwabe, Tegenthoff, Hoffken, & Wolf, 2010, 2013; Schwabe & Wolf, 2009b; Vogel, Fernandez, Joels, & Schwabe, 2016). Only recently, studies have started to look into the cognitive consequences of this stress-induced bias towards habitual memory (e.g., Dandolo & Schwabe, 2016; Hoscheidt, LaBar, Ryan, Jacobs, & Nadel, 2014; Kluen, Agorastos, Wiedemann, & Schwabe, 2017; Kluen, Nixon, Agorastos, Wiedemann, & Schwabe, 2016; Schmidt, Rosga, Schatto, Breidenstein, & Schwabe, 2014; van Ast, Cornelisse, Meeter, Joels, & Kindt, 2013). In the second part of this article, the implications of the stress effects on the flexibility with which memories are updated, transformed or integrated with existing knowledge structures, i.e., the memory dynamics, will be addressed. In the third and final part of our review, we will discuss possible implications of the impact of stress on the dynamics of memory and how these stress-related changes might contribute to cognitive adaptation to stressful events.

Temporal dynamics of stress effects on memory

As outlined above, stress results in an orchestrated physiological response involving several endocrine and neurotransmitter systems characterised by different temporal profiles (Joels & Baram, 2009). Within seconds following stressor onset, the release of catecholamines, including noradrenaline, is triggered. With a delay of several minutes, the HPA axis results in an increased secretion of glucocorticoids. Glucocorticoids exert rapid, non-genomic effects via membrane-associated receptors, followed by delayed genomic effects that are mediated through intracellular receptors, develop within 60–90 minutes, and may last for several days to weeks.

Time-dependent effects of stress mediators on the brain

The different temporal profiles of action of the major stress response systems result in highly dynamic stress effects on brain areas that are critically involved in memory processes. Fast noradrenergic activation is thought to enhance the connectivity within the salience network including the amygdala, hypothalamus, the dorsal anterior cingulate cortex and inferior temporal regions (Hermans

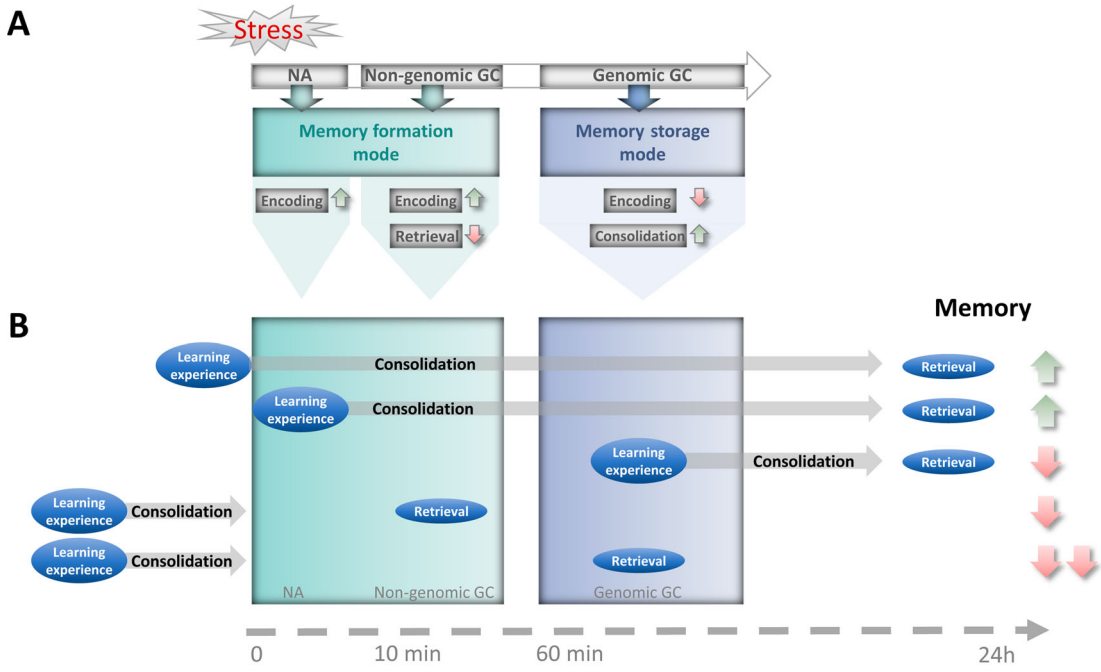

Figure 1. Temporal dynamics of stress effects on memory. (A) Rapid catecholaminergic and glucocorticoid actions set the brain in a memory formation mode that facilitates the encoding of stressor-related material but impairs retrieval of stressor-unrelated material. With time, as genomic glucocorticoid actions develop, during the memory storage mode, the threshold for encoding stressor-unrelated material is elevated whereas the consolidation of stressor-related material is facilitated. (B) Stress within the spatiotemporal context of a learning situation facilitates the ongoing encoding process and thus enhances subsequent memory (upper two). If stress is experienced some time before the learning experience and the learning experience takes place during the memory storage mode, subsequent memory is impaired (third). Lastly, if stress is experienced after the learning experience, during both the memory formation and storage mode, the capacity to retrieve stressor-irrelevant information is reduced with a more pronounced impairing effect on retrieval during the memory storage mode (lower two). NA – noradrenaline and GC – glucocorticoids.

et al., 2014; Schwabe, Wolf, et al., 2010; Seeley et al., 2007). This effect is specifically driven by noradrenergic activity. Pharmacological blockade of β-adrenergic receptors prevents this stress-induced network reconfiguration, whereas a blockade of glucocorticoid synthesis did not result in changes in network configuration (Hermans et al., 2011). Later on, when catecholamines coincide with rapid non-genomic glucocorticoid effects, glucocorticoids boost the effect of noradrenaline (Krugers, Karst, & Joels, 2012). Specifically, the fast noradrenergic response and non-genomic membrane MR activity result in a rapid increase of excitability in hippocampus and amygdala neurons (Joels, Krugers, & Karst, 2008; Karst, Berger, Erdmann, Schutz, & Joels, 2010; Roozendaal, McEwen, & Chattarji, 2009). Administration of hydrocortisone without simultaneous noradrenergic stimulation, however, resulted in a reduced activity in the hippocampus and amygdala as revealed by fMRI (Lovallo, Robinson, Glahn, & Fox, 2010). Thus, precisely timed interactions between corticosteroids and noradrenergic activity appear to have different effects on activity in memory-related brain areas.

The amygdala is a key part of the salience network and one of the fastest brain areas to react to a stressor. It excites the ANS and HPA responses, thus mediating the initial surge in vigilance and optimising detection of threats for homeostasis (de Kloet et al., 2005; Phillips, Drevets, Rauch, & Lane, 2003). The optimised threat-processing under stress may further be due to stress hormone-induced changes in the connectivity of the amygdala with a number of salience network areas (Quaedflieg et al., 2015; van Marle, Hermans, Qin, & Fernandez, 2010). Furthermore, glucocorticoid actions in the amygdala seem to modulate activity in areas that are reciprocally connected such as the hippocampus but also the prefrontal cortex (PFC; Kim et al., 2011; Roozendaal et al., 2009), albeit in a region-dependent manner. Specifically, it has been shown that during the immediate stress response PFC activity is reduced (van Stegeren, Roozendaal, Kindt, Wolf, & Joels, 2010), resulting in an impairment of cognitive processes supported by the executive control network, like working memory (Qin, Hermans, van Marle, Luo, & Fernandez, 2009; Roozendaal, McReynolds, & McGaugh, 2004) and goal-directed behaviour (Schwabe, Tegenthoff, Hoffken, & Wolf, 2012). This detrimental effect of the rapid stress response is thought to be reversed during the slow genomic glucocorticoid phase. Administration of hydrocortisone 4 hours before performing a memory-encoding task resulted in decreased activity in the hippocampus (Henckens et al., 2012). Moreover, targeting the slow genomic glucocorticoid mode, an opposite effect was found on the activity in the amygdala and dorsolateral PFC. Hydrocortisone administration 4 hours before fMRI scanning reduced amygdala activity and enhanced prefrontal activity during a working memory task (Henckens, van

Wingen, Joels, & Fernandez, 2011) or dynamic facial expression task (Henckens, van Wingen, Joels, & Fernandez, 2010). These studies support the idea that delayed genomic actions of glucocorticoids are thought to lead to a reinstatement of the executive control network activity and concurrently a suppression of salience network activity (Hermans et al., 2014).

In sum, stress effects on the brain are time-dependent and associated with the temporal profile of the ANS and HPA axis stress hormones. Moreover, the changes in brain systems are region-specific, with the amygdala, hippocampus and PFC as main target areas. These time- and region-specific effects of stress hormones may translate into differential effects of stress on distinct learning and memory processes (see Figure 1). Specifically, stress may have different effects on learning and memory depending on whether stress occurs at the same time and within the context of the learning (i.e., learning of *stressor-related* information) or not (i.e., learning of *stressor-unrelated* information).

Temporal dynamics of stress effects on memory formation

Neurophysiological studies targeting hippocampal plasticity mechanisms provided the first evidence that the different waves of the physiological stress response may translate into time-dependent effects on learning and memory. These studies have shown that hippocampal synaptic plasticity is only facilitated when corticosterone (the major rodent glucocorticoid) was applied during tetanic stimulation but not when corticosterone was administered 30 minutes before or directly after stimulation (Wiegert, Joels, & Krugers, 2006). When the slow, genomic glucocorticoid actions prevailed, however, hippocampal long-term potentiation was found to be suppressed (Joels, Pu, Wiegert, Oitzl, & Krugers, 2006; Kim & Diamond, 2002). These animal data led to the development of models on the temporal dynamics of stress effects on memory formation (Diamond et al., 2007; Joels et al., 2006). These models postulate a dual action mode of glucocorticoids, with rapid non-genomic glucocorticoid actions setting the brain in a memory formation mode that favours the encoding and early consolidation of threat-related material. Specifically, when acute stress is experienced within the spatiotemporal context of the learning episode, the fast noradrenergic activity and non-genomic glucocorticoid actions, mediated most likely via the membrane-associated MR, are assumed to result in a rapid increase of glutamate-mediated excitability in the hippocampus and the amygdala, thus facilitating memory formation (Joels, Karst, DeRijk, & de Kloet, 2008; Karst et al., 2010). The delayed genomic glucocorticoid actions, however, would induce a memory storage mode that suppresses the encoding of new information that does not converge in time or learning context and thus is not related to the stressor (i.e., stressor-unrelated). These delayed genomic actions protect the consolidation of the stressful event from interference (Diamond et al., 2007; Joels et al., 2011; Schwabe, Joels, et al., 2012).

The time-dependent non-genomic role of glucocorticoids in the memory formation mode has been tested in humans by varying the time interval between stressor exposure and encoding (see Figure 1(B)). For instance, one study exposed individuals to stress either immediately or 30 minutes before learning and tested memory retention 24 hours later. Acute stress immediately before encoding enhanced subsequent recognition of positive stimuli, whereas stress 30 minutes before encoding impaired recall of negative material (Zoladz et al., 2011). The role of emotional valence in this time-dependent effect remains unclear. However, very similar to these time-dependent effects of stress before learning, the timing of the stress exposure influenced the direction of the association between the increase in cortisol response and the number of remembered pictures 24 hours later (Quaedflieg, Schwabe, Meyer, & Smeets, 2013). Correctly recognised neutral pictures were positively associated with the cortisol response, but only if participants were stressed shortly before learning. In contrast, there was a negative association for participants who were stressed 30 minutes before learning (i.e., fewer pictures recognised with higher cortisol response). This behavioural association in the 30 minutes pre-learning stress condition was supplemented by a negative association between stress-induced cortisol and the amplitude of the late positive potential (LPP), an electrophysiological index of attention and visual processing (Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000). Stress 30 minutes before learning decreases attentional allocation to new stimuli, indicative of impaired processing of new information. In addition, a selective enhancement of the LPP for unpleasant pictures was found in participants stressed shortly before learning (Weymar, Schwabe, Low, & Hamm, 2012). Together these studies suggest that some of the effects of acute stress are due to a modulation of attention and indicate the need for a finer delineation of the effects of the two glucocorticoid modes on sub-processes like attention and early consolidation versus late consolidation.

The above discussed studies tested the proposed time-dependent effects of stress when peak cortisol concentrations are expected (i.e., around 30 minutes after stressor onset), indicating the need for a finer delineation of the precise time windows during which stress and glucocorticoids can affect memory-encoding processes. A recent study assessed the development of stress effects on memory formation in a natural environment over a 2-hour period after a stressful event (Vogel & Schwabe, 2016b). This study found a time-dependent enhancement of memory formation that was closely linked to the temporal profile of the rapid physiological stress responses. More specifically, the enhanced memory for the stressor itself was associated with the noradrenergic response, while the delayed cortisol response was associated with enhanced memory for events encoded between 41 to 65

minutes after stressor onset. Acute stress experienced within the spatiotemporal context of the learning episode induced a memory formation mode mediated by the fast, non-genomic stress hormone actions that last for approximately about 1 hour after stressor onset. Though, there was no effect of stress on memory formation of events encoded after 60 minutes, when genomic cortisol actions are thought to set in. The development of genomic glucocorticoid actions may depend on the species tested as well as on the specific brain area. Unravelling the brain site-specific temporal development of non-genomic and genomic glucocorticoid actions in humans is a major challenge on the way to understand how stress modulates human memory formation.

Temporal dynamics of stress effects on memory retrieval

Time-dependent effects of stress due to the temporal profiles of noradrenaline and glucocorticoid action were mainly discussed for memory encoding. For memory retrieval, however, such differences were rarely considered. In fact, the vast majority of studies tested the impact of stress on memory retrieval about 20 to 30 minutes after stressor exposure, when non-genomic glucocorticoid actions prevail, and these studies reported mainly detrimental effects of stress and glucocorticoids on retrieval performance (Buchanan et al., 2006; de Quervain, Roozendaal, & McGaugh, 1998; Kuhlmann et al., 2005; Roozendaal, Hahn, Nathan, de Quervain, & McGaugh, 2004; but see also Schilling et al., 2013; Schwabe et al., 2009; Smeets et al., 2008). This retrieval deficit has been explained by non-genomic glucocorticoid actions shifting the brain, in interaction with noradrenergic arousal (de Quervain, Aerni, & Roozendaal, 2007; Roozendaal, Hahn, et al., 2004), to a memory formation mode, resulting in a reduced capacity to retrieve stressor-irrelevant new information (see Figure 1(A)). The subsequent memory storage mode, induced by genomic glucocorticoid actions, was supposed to have an even more pronounced impairing effect on retrieval (Joels et al., 2006; Schwabe, Joels, et al., 2012). Support for this latter idea comes from a recent study that exposed individuals to stress about 90 minutes before retention testing of previously learned words and showed that, at this time, when genomic glucocorticoid actions should have developed, the retrieval impairment was stronger than at the time of the cortisol peak after stress (Schwabe & Wolf, 2014). Notably, no such impairment was obtained when retrieval was tested immediately after the stress, before cortisol levels were elevated, which is in line with earlier rodent data (de Quervain et al., 1998). Interestingly, a related study indicated that when stress is part of the retrieval situation and the performance level (free recall of learned words) is directly linked to the stressfulness of the situation (e.g., oral examination), the retrieval might even be enhanced by the noradrenergic stress response (Schonfeld, Ackermann, & Schwabe, 2014; see also Murchison et al., 2004 for related findings in animals). These findings point to potential time-dependent effects of stress on memory retrieval, with (moderate) noradrenergic arousal facilitating and rapid and delayed glucocorticoids impairing memory retrieval (see Figure 1(B)).

Stress and the dynamics of memory

Most of the research on the effects of stress on memory focused on hippocampal memory processes. Although the hippocampus is probably the most prominent memory system in the brain, intimately linked to episodic memory and the flexible integration of memories for discrete events (Backus, Schoffelen, Szebenyi, Hanslmayr, & Doeller, 2016; Burgess, Maguire, & O'Keefe, 2002; Eichenbaum, Schoenbaum, Young, & Bunsey, 1996; Squire, 2009), learning and memory can be supported by other systems as well (Eichenbaum, & Cohen, 2004; Squire, 2004). For long, it has been assumed that non-hippocampal memory is less sensitive to stress (Lupien & Lepage, 2001). However, more recent research demonstrates that stress may also alter non-hippocampal (e.g., dorsal striatal or insular) memory and that these stress effects on non-hippocampal memory processes resemble those on hippocampal memory (Atsak et al., 2016; Guenzel, Wolf, & Schwabe, 2013, 2014; Medina et al., 2007; Quirarte et al., 2009).

Beyond quantitative changes within hippocampal and non-hippocampal memory systems, stress may also have a profound effect on which memory system guides behaviour (Packard & Goodman, 2012; Packard & Wingard, 2004; Schwabe, 2016; Schwabe & Wolf, 2013). More specifically, a number of studies shows that stress before learning a task that can be solved both by a flexible but cognitively demanding hippocampus-dependent or by a simple but rigid dorsal striatal memory system, favours dorsal striatal over hippocampal learning (Kim et al., 2001; Schwabe & Wolf, 2010b; Schwabe et al., 2007, 2013; Schwabe, Tegenthoff, et al., 2012). Similarly, stress or the concurrent activation of glucocorticoids and noradrenaline has been shown to promote dorsal striatum-based habitual learning at the expense of PFC-based goal-directed learning (Braun & Hauber, 2013; Gourley et al., 2012; Schwabe, Tegenthoff, et al., 2010; Schwabe, Tegenthoff, et al., 2012; Schwabe & Wolf, 2009b, 2011; Seehagen, Schneider, Rudolph, Ernst, & Zmyj, 2015). Together, these data suggest that stress shifts memory from flexible "cognitive" control by the hippocampus and PFC, to rather rigid habit-based control of memory by the dorsal striatum (Schwabe & Wolf, 2013; Vogel, Klumpers, et al., 2017). This shift towards more habitual memory may be due to an enhancement of the habitual system or to an impairment of the cognitive system. There is empirical evidence for both of these alternatives, which are not mutually exclusive (Schwabe & Wolf, 2012; Schwabe, Tegenthoff, et al., 2012; Wirz, Wacker, Felten, Reuter, & Schwabe, 2017). Moreover, the amygdala appears to orchestrate the engagement of these systems as there are opposite effects of stress on amygdala

connectivity with the hippocampus and dorsal striatum, respectively (Schwabe et al., 2013; Vogel et al., 2017; Wirz et al., 2017; for review see Schwabe 2013).

The change in the control of memory may have considerable implications for the dynamic nature of memory. The altered nature of memory after stress may be reflected in altered memory contextualisation, changes in memory flexibility, and altered memory updating processes (Figure 2).

Stress and the specificity of memories

Acute stress changes the current environmental demands requiring the individual to rapidly extract the information that is critical for survival. For instance, acute stress reduces selective processing for items later remembered vs. forgotten in the hippocampus and midbrain regions during memory formation (Qin, Hermans, van Marle, & Fernandez, 2012). Specifically, stress and glucocorticoids may dynamically shift neural resources from areas involved in detailed episodic encoding, such as the hippocampus, to other areas that are implicated in more abstract, semantic representations (Schwabe, 2016; Schwabe & Wolf, 2013). Such a stress-induced impairment of memory systems supporting detailed encoding has become evident in a higher reliance on *gist-based memory formation* (Nadel & Payne, 2002). Gist-based information processing fosters the encoding of categories instead of discrete memory representations resulting in reduced memory specificity and a more liberal response bias when retrieving these memories (Payne et al., 2002). In line with these findings, ANS activation was associated with more liberal responding after stress (Qin et al., 2012). Using the Deese–Roediger–McDermott (DRM) paradigm, it has been demonstrated that stress before or after encoding neutral word lists increased the number of semantically related lure words recalled, indicative of gist-based memory formation (Pardilla-Delgado, Alger, Cunningham, Kinealy, & Payne, 2016; Payne, Nadel, Allen, Thomas, & Jacobs, 2002; but see Smeets, Jelicic, & Merckelbach, 2006 and Smeets et al., 2008 for studies that did not find a stress effect on false memory).

The stress-induced shift from detailed, episodic processing to processing of the essential parts of a stressful event is assumed to be due to attentional narrowing (Kensinger, 2004) and also changes the *contextualisation* of the memory trace. Integrating contextual information during encoding leads to a richer memory trace, the retrieval of which may subsequently be boosted by the presentation of such contextual cues (Smith & Vela, 2001). Stress 30 minutes before encoding abolished this so-called context-dependent memory enhancement (Schwabe & Wolf, 2009a), suggesting that stress reduced the incorporation of contextual cues into the memory trace during the memory formation mode. Stress may alter the way memories are integrated in their original internal (task-related) encoding context as well. This idea is also supported by findings showing that rapid non-genomic glucocorticoid actions impaired the contextualisation of negative memories. Interestingly, delayed, genomic glucocorticoid actions had the opposite effect (van Ast et al., 2013), suggesting that the effect of stress/glucocorticoids on memory contextualisation depends on the mode of glucocorticoid action. However, in contrast to the idea that non-genomic glucocorticoid actions disrupt memory contextualisation, stress-induced cortisol levels have been shown to enhance the contextualisation of negative and neutral items against background pictures in another study (van Ast, Cornelisse, Meeter, & Kindt, 2014). Differences in the experimental paradigm as well as general differences between stress and pharmacological manipulations may explain at least part of these divergent findings. Interestingly, the underlying mechanism of the non-genomic glucocorticoid effects on contextualisation has been suggested to be an enhanced feeling of familiarity, a cognitive less demanding and more automatic memory process (van Ast et al., 2014). This view is generally in line with the idea of a stress-induced shift towards cognitively less demanding processing that may foster cognitive adaptation under stress (Schwabe & Wolf, 2013; Vogel & Schwabe, 2016a).

Stress and the flexibility of memory

A core feature of the hippocampal memory system is that it encodes separate representations of events and thus enables building flexible memory representations that can be linked into mnemonics (Shohamy & Wagner, 2008). If stress promotes a shift from hippocampal towards dorsal striatal memory, which lacks this flexibility of memory representations (Myers et al., 2003), stress should also reduce the flexibility with which memories can be used. This flexibility is reflected in our ability to make inferences and to transfer acquired information to novel contexts (Eichenbaum, Stewart, & Morris, 1990; Shohamy & Wagner, 2008). Evidence for reduced memory flexibility after stress comes from a recent study indicating that stress reduced participants' ability to *generalise across overlapping past experiences* (Dandolo & Schwabe 2016), which may require integrative encoding processes that rely on the hippocampus (Shohamy & Wagner, 2008). This stress-induced generalisation deficit was linked to the interaction of noradrenaline and non-genomic glucocorticoid action (Dandolo & Schwabe, 2016). Additionally, a pharmacological study targeting the role of the major stress hormone systems in this effect showed that increased noradrenergic arousal is sufficient to impair individuals' capacity to generalise across past experiences, whereas increased glucocorticoid activity alone had no such effect (Kluen et al., 2017). Notably, this effect was only observed in women, not in men, pointing to relevant sex differences in the influence of stress hormones on memory generalisation.

Whereas the hippocampus is required for the specific encoding of novel events and the generalisation across past events, it is less involved in processing material related

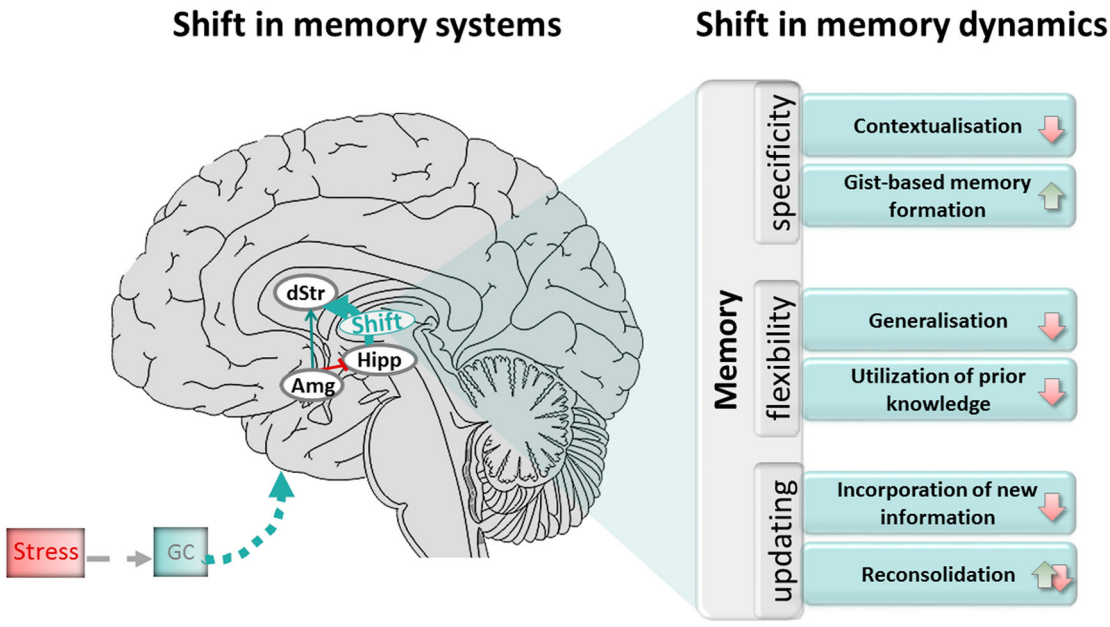

Figure 2. Stress-induced shift in memory systems alters the dynamics of the memory trace. (Left) Stress dynamically shifts memory control from "cognitive", flexible control by the hippocampus and PFC to rather rigid, habit-based control by the dorsal striatum. (Right) This change in the control of memory alters the nature of the memory trace after stress. Specifically, stress appears to hinder the incorporation of contextual details into the memory trace, to impede the integration of new information into existing knowledge structures, to impair the flexible generalisation across past experiences, and to hamper the modification of memories in light of new information. Abbreviations: dStr – dorsal striatum; Hipp – hippocampus; Amg – amygdala and GC – glucocorticoids.

to *prior knowledge*, that is, schema-based memory processes. When new information is congruent with existing knowledge, memory control is shifted from the hippocampus to the medial PFC (mPFC; van Kesteren et al., 2013). The mPFC then tends to inhibit the hippocampus and promotes the integration of the information into existing neocortical networks (van Kesteren et al., 2013). Interestingly, stress has been found to reduce mPFC activity and result in a shift of brain networks relevant for the processing of prior knowledge-related information to novel information (Vogel, Kluen, Fernandez, & Schwabe, 2017a, 2017b). Moreover, a recent study showed that stress impairs the utilisation of prior knowledge during learning, suggesting that stress not only disrupts the accessibility of stored memory traces but also their integration with new information (Kluen et al., 2016). This effect of stress was observed when learning took place under increased cortisol levels and it could be mimicked by the pharmacological elevation of glucocorticoid levels (but not by noradrenergic stimulation), indicating that rapid glucocorticoid effects may drive the stress-induced deficit in the utilisation of prior knowledge during learning. In sum, the putative stress-induced shift in the memory system guiding learning and behaviour appears to result in less flexible and less specific memories that are difficult to integrate with existing memory representations (Figure 2).

Stress and the modification of memories

An essential feature of adaptive memory is that it can be updated in the face of new information. The incorporation of new information into existing memories underlines the dynamic nature of memory. Several lines of research suggest that stress may interfere also with this updating of existing memory traces (Figure 2). Indications for the malleability of memory come, for instance, from research on the misinformation effect. Three decades of research have shown that presenting misleading information after encoding often biases subsequent memory in the direction of the misinformation (for review see Loftus, 2005), implicating that the memory was modified in light of the misleading information. Stress appears to "protect" memories against this updating. If participants were stressed before the presentation of misinformation, they were less likely to incorporate it into existing memories (Schmidt et al., 2014). This form of updating requires the reactivation of the existing trace as well as the encoding of the novel (incorrect) information and both might have been affected by stress. On the other hand, stress after encoding of the original information may result in stronger memories (Cahill et al., 2003; Roozendaal, Okuda, de Quervain, et al., 2006) that are in turn less sensitive to modifications by misleading information (Hoscheidt et al., 2014). Though, without acute stress, memories for stressful events seem to be more vulnerable to modification by exposure to misinformation (Morgan, Southwick, Steffian, Hazlett, & Loftus, 2013).

The mechanism underlying the misinformation effect might be reconsolidation (Schacter & Loftus, 2013; but see Hupbach, Gomez, Hardt & Nadel, 2007 and Johnson & Seifert, 1994 for alternative interpretations). Memory reconsolidation refers to a process of re-stabilisation after memory reactivation during which seemingly stable memories can

be weakened, strengthened or updated based on prior experience and anticipated outcomes, respectively (Hardt et al., 2010; Nader & Hardt, 2009; Sara, 2000). Reconsolidation is supported by the basolateral amygdala and areas of the cognitive memory system, in particular the hippocampus and PFC (Akirav & Maroun, 2013; Sandrini, Censor, Mishoe, & Cohen, 2013; Schwabe et al., 2014). Blockade of the β-adrenergic receptor during memory reactivation reduced the well-known emotional memory enhancement in a subsequent test and this effect was associated with altered activity in the amygdala and hippocampus (Schwabe, Nader, Wolf, Beaudry, & Pruessner, 2012). While these findings indicated a role of noradrenergic arousal in emotional memory reconsolidation, other studies reported also direct evidence for acute stress-induced changes in reconsolidation, although the direction of the effect is mixed (see Figure 2). On the one hand, acute stress following reactivation of autobiographical memories impaired the memory for the neutral episodes 1 week later, whereas the subsequent memory for the emotional episodes was not affected (Schwabe & Wolf, 2010a). In heroin addicts, acute stress following the reactivation of neutral, positive and negative addiction related words impaired reconsolidation of both positive and negative words whereas the reconsolidation of neutral words was not affected (Zhao, Zhang, Shi, Epstein, & Lu, 2009). On the other hand, stress following reactivation enhanced memory performance for emotional slideshows (Marin, Pilgrim, & Lupien, 2010) and for neutral and emotional words (Bos, Schuijer, Lodestijn, Beckers, & Kindt, 2014). Similarly, memory for neutral cue-syllables association was enhanced when the cold pressor stress administration was concurrent with the retrieved labile memory state (Coccoz, Maldonado, & Delorenzi, 2011). In addition, decreasing cortisol levels with a synthesis inhibitor before reactivation of an emotional slideshow impaired memory performance tested 4 days later (Marin, Hupbach, Maheu, Nader, & Lupien, 2011). Further studies are needed to test whether the effects of glucocorticoids on reconsolidation may depend on the memory type tested, for example neutral versus emotional or autobiographical versus slideshow, as well as on the specific brain area controlling learning and the temporal profile of action of major stress mediators. Examining these and related issues would be highly interesting as glucocorticoid-based modulation of reconsolidation processes might provide a useful tool for altering dysfunctional memories, with crucial implications for the treatment of post-traumatic stress disorder (PTSD) and other fear-related disorders (de Quervain, Schwabe, & Roozendaal, 2017).

Summary

Stressful events result in a fine-tuned physiological response, including numerous hormones, neurotransmitters and peptides (Joels & Baram, 2009). The orchestrated action of these stress mediators dynamically changes the processing in memory-related brain areas, enabling the prioritisation of cognitive resources towards the adaptation to the stressful event (Hermans et al., 2014; Joels et al., 2011; Schwabe, Joels, et al., 2012; Vogel et al., 2016). Specifically, synchronised catecholamine and rapid glucocorticoid actions set the brain in a memory formation mode that promotes building lasting memories of the stressful encounter. The subsequent memory storage mode further promotes the consolidation of the memory for the stressful episode. Beyond the modulation of memory formation and retrieval, stress induces a shift from flexible, "cognitive" towards more rigid, habitual control of memory (Packard & Goodman, 2012; Schwabe, 2013; Schwabe, & Wolf, 2013), which relies on rapid glucocorticoid action mediated via the MR (Schwabe, Tegenthoff, et al., 2010, 2013; Vogel et al., 2017). Delayed genomic actions of glucocorticoids are thought to boost a prefrontal executive control network, thus reversing the balance of multiple memory systems again and restoring homeostasis in the aftermath of stress (Diamond et al., 2007; Hermans et al., 2014; Joels et al., 2006, 2011; Robbins & Meyer, 1970; Schwabe, Joels, et al., 2012; Vogel et al., 2017).

The stress-induced shift towards habitual memory allows highly efficient processing in the face of increased environmental demands. It avoids hesitation, distraction and lets well-established routines guide behaviour. Together with a transient decrease in the retrieval of stressor-unrelated information and the enhanced consolidation of the stressful event, this bias towards habitual memory may be an integral part of the individuals' cognitive adaptation to stressful encounters (Vogel, Fernandez, et al., 2016). This form of adaption, however, comes at the cost of the specificity and flexibility of memory. Stress appears to reduce the integration of contextual details into the memory trace, to hinder the transfer or generalisation of acquired information to novel situations, to hamper the integration of new information and existing knowledge structures (schemata), and to impede the updating of memory in the light of new information. In other words, stress appears to transiently reduce the dynamic nature of memory.

Although this transient shift towards more rigid memory is thought to be generally adaptive under stress, it is important to shift back to the cognitive control of memory once the stressful event is over. The long-lasting recruitment of habitual forms of memory may result in inflexible and less well-integrated knowledge, with tremendous implications for educational settings (Vogel & Schwabe, 2016a). Moreover, an aberrant recruitment of inflexible habit memory may contribute to stress-related psychopathology (de Quervain et al., 2017). For instance, the extreme stress experienced during a traumatic event might promote habitual forms of memory reflected in strong associations between trauma-related cues and emotional responses, a hallmark feature of PTSD (Goodman, Leong, & Packard, 2012; Schwabe, Wolf, & Oitzl, 2010). Other fear-related disorders as well as drug addiction have been discussed as further disorders in which stress-related changes in memory processes are crucial (de Quervain et al., 2017; Everitt & Robbins, 2005; Sinha, 2007).

Based on the basic research on the impact of stress on learning and memory processes, several treatment approaches for stress-related mental disorders have been proposed. One potential approach involves the pharmacological manipulation of major stress mediators, in particular catecholamines and glucocorticoids. A blockade of noradrenergic arousal has been suggested to prevent emotional memory formation (Cahill, Gorski & Le, 2003; Roozendaal, Okuda, de Quervain, et al., 2006) as well as the impact of stress and glucocorticoids on memory retrieval (de Quervain et al., 2007; Roozendaal et al., 2003). First clinical trials suggested that pharmacological blockade of noradrenergic activity may indeed provide a way to prevent the overly strong consolidation of trauma memory (Pitman et al., 2002); subsequent studies, however, yielded mixed findings (Lonergan, Olivera-Figueroa, Pitman & Brunet, 2013). In addition to noradrenergic manipulations, pharmacological changes of glucocorticoid activity might be a promising avenue in the treatment of memory distortions in stress-related psychopathologies (for a review see de Quervain et al., 2017). For instance, oral cortisol treatment reduced fear responses in spider phobics and social phobics (Soravia et al., 2006). However, treatment approaches are not limited to pharmacological manipulations. Understanding the brain networks relevant for the stress-induced changes in memory, might be a first step to directly modulate these networks. A very recent study suggested that transcranial direct current stimulation over the dorsolateral PFC might prevent stress-induced working memory deficits (Bogdanov & Schwabe, 2016). Neurofeedback might be another tool to modulate the brain's response to stress (Quaedflieg et al., 2016).

The regular clinical use of such treatments is, however, a long way off and many open questions related to how exactly stress shapes our memory remain. One important issue concerns individual differences in the impact of stress on memory. There are in fact substantial individual differences in the cognitive response to stress that are at least partly genetically determined. A variant of the gene encoding the α2-adrenegic receptor (ADRA2B), for instance, has been linked to increased emotional memory formation (de Quervain et al., 2007; Rasch et al., 2009), increased amygdala responses to stress (Cousijn et al., 2010), and to a reduced ability to engage the appropriate memory system under stress (Wirz et al., 2017). Identifying individuals whose memory is particularly sensitive to the impact of stress may pave the way to personalised treatment approaches. Further questions relate to the actual mechanisms underlying the stress-induced changes in memory. How and when does the transition from rapid, non-genomic to delayed, genomic glucocorticoid actions on memory take place? How do stress-induced changes in the engagement of different memory systems develop in the aftermath of a stressful event? May the slow, genomic glucocorticoid actions boost memory flexibility and specificity? Answering these and related questions will significantly aid our understanding of the far-reaching impact of stressful events on the dynamics of the memory trace.

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