Memories of and influenced by the Trier Social Stress Test

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ABSTRACT

Psychosocial stress influences cognition, affect and behavior. This current review summarizes the impact of acute stress on human long-term memory taking a neuroendocrine perspective. In this respect the stress associated increase in activity of the sympathetic nervous system (SNS) and the hypothalamus-pituitary-adrenal (HPA) axis are key. A special focus will be placed on findings obtained with the Trier Social Stress Test (TSST). This paradigm can be used to induce stress before or after a memory task. It was shown repeatedly that stress enhances long-term consolidation but impairs long term memory retrieval. However the TSST can also be used to assess memories of this stressful episode itself. The latter requires a standardized presentation of relevant stimuli during the TSST as well as a carefully designed control condition. Moreover special care has to be taken to control potential influences on visual exploration and working memory in order to correctly interpret observed effects on memory. The results obtained so far fit the idea of enhanced encoding of salient information under stress. These findings are of relevance for educational, organizational and clinical applications.

1. Introduction

Stress has obtained a rather bad reputation nowadays. It is often related to impaired cognitive performance and when occurring chronically it is commonly associated with physical and mental health problems. However, research has illustrated that the impact of stress on cognitive functions such as learning and memory are far more complex than initially assumed. Stress may enhance or impair memory depending on several key modulators and mediators. Both quantitative and qualitative shifts take place (Schwabe and Wolf, 2013). Stress effects on memory will be discussed in the present selective review. It is part of the special issue devoted to work of Professor Hellihammer and thus will have a special focus on studies which have used the Trier Social Stress Test (TSST (Kirschbaum et al., 1993)).

A common definition is that stress occurs when a person perceives a challenge to their internal balance (homeostasis (De Kloet et al., 2005)). Thus, a discrepancy between what “should be” and “what is” induces stress (Ursin and Eriksen, 2010). A stressor can be physical (e.g. heat, thirst, pain) or psychological (e.g. deadlines at work, mobbing, relationship-problems). Moreover stressors can be acute or chronic (McEwen, 1998). For us humans a threat to the social self (social evaluative threat), in combination with uncontrollability of the situation, is especially potent in triggering a stress response (Dickerson and Kemeny, 2004). The initial subjective evaluation of the stressor (primary appraisal) and of existing coping resources (secondary appraisal) determines its impact on the individual (Lazarus, 1993). Something perceived as a major threat by one person might be perceived as an exciting opportunity by another. There is substantial inter-individual variability in the response to stress and its cognitive consequences, an issue discussed at the end of this review.

The stress response evolved as an adaptive reaction aimed at maintaining physiologic integrity (homeostasis) in the face of anticipated or actual threat to physiological or psychological well-being (De Kloet et al., 2005; McEwen, 1998). The effects of stress manifest themselves on multiple levels, including behaviour, subjective experience, cognitive function, and physiology. The same responses which are mostly adaptive under acute stress can, however, promote disease processes in vulnerable individuals (De Kloet et al., 2005; McEwen, 1998).

Stress leads to hormonal responses aimed at facilitating adaptation. The sympathetic nervous system (SNS) and the hypothalamus-pituitary-adrenal (HPA) axis are the two key players. SNS activity causes the rapid release of (nor)epinephrine from the adrenal medulla. This constitutes the first rapid response wave. Increased activity of the HPA axis induces the release of glucocorticoids (GCs; cortisol in humans) from the adrenal cortex. This response is slower and constitutes the second response wave (De Kloet et al., 2005). Cortisol levels start to rise approximately 10–15 min after stress onset and typically reach their peak
around 30 min after the beginning of the stressor (Dickerson and Kemeny, 2004).

GCs are lipophilic hormones and therefore can pass the blood brain barrier. There they affect regions involved in cognitive functions (e.g. prefrontal cortex, amygdala, hippocampus, striatum). These effects are mediated by two receptors: the mineralocorticoid receptor (MR) and the glucocorticoid receptor (GR). They differ in their affinity for the hormone and in their localization. While MR activation leads to enhanced neuronal excitability, GR activation causes a delayed suppression or normalization of the neuronal network (Joëls et al., 2008). In addition, GCs can exert rapid non-genomic effects which, in part, are mediated by recently described membrane-bound MRs (Joëls et al., 2008) and GRs (Roozendaal et al., 2010). Thus GCs have time dependent effects comprising of rapid non-genomic effects and later occurring slower genomic effects (Joëls et al., 2011; Wolf, 2017).

After acute stress, the HPA axis’ negative feedback cycle causes GC levels to return to baseline concentrations within hours (De Kloet et al., 2005; Dickerson and Kemeny, 2004). In periods of chronic stress persistent changes of the HPA axis can occur, leading to permanently elevated cortisol levels. However, high cortisol levels as often observed in major depressive disorder (MDD), are not always the consequence of chronic stress (Wolf, 2008). For example, lower cortisol levels occur in several stress-associated somatoform disorders (Fries et al., 2005) as well as in post-traumatic stress disorder (Yehuda, 2002; Wingenfeld and Wolf, 2015).

When researchers want to investigate the impact of stress on cognition experimentally they face the dilemma as to how to induce efficiently stress in the laboratory. The Trier Social Stress Test (TSST (Kirschbaum et al., 1993)) developed by Clemens Kirschbaum and Dirk Hellhammer combines a videotaped free speech (a job interview role play) in front of a neutral and reserved acting committee with a mental arithmetic task (also in front of a committee). It reliably induces negative affect and activates the SNS and the HPA. The combination of social evaluative threat, motivated performance and uncontrollability makes this stressor so powerful (Dickerson and Kemeny, 2004). A meta-analysis on the impact of variations in the TSST protocol on the HPA response to this paradigm has recently been published (Goodman et al., 2017). Alternative laboratory stressors which are not in the center of the present review are the socially evaluated cold pressor test (SECPCT (Schwabe et al., 2008)) and the Maastricht acute stress test (MAST (Smeets et al., 2012)). Findings obtained with these stressors which combine a physiological stressor (pain) with a psychosocial stressor (social evaluative threat) will be referred to especially in those occasions where they cover an area of relevance for the present review which has not been addressed with the TSST.

2. Stress and cognition

The hormonal stress response substantially influences cognitive and affective processes during stress and its aftermath. This has been named the afferent pathway. Indeed, stress has been shown to influence the entire information processing stream. It affects early sensory detection. For example stress lead to a lower sensory threshold for an unpleasant chemical odour (Pacharra et al., 2016). However more complex visual judgements appear to be impaired (Paul et al., 2016). Stress increases vigilance (Hermans et al., 2014) by activating the salience network in the brain. This appears to go along with impaired selective attention, mediated by the executive control network (Oei et al., 2012). Furthermore, stress prioritizes habitual stimulus-response behaviour (the “inner autopilot”), mediated by parts of the striatum, at the expense of cognitive/rational goal-directed behaviour, known to rely on regions within the prefrontal cortex (PFC) (Schwabe and Wolf, 2013). Thus under stress bottom up (stimulus driven) processes appear to dominate our behaviour. The ability to exert cognitive (top down) control in contrast is limited (Hermans et al., 2014; Shields et al., 2016). Working memory capacity is also typically impaired during and in the immediate aftermath of stress (Shields et al., 2016). Similarly stress affects our abilities to make good decisions. For example participants who had experienced a stressful laboratory situation displayed riskier and less successful behaviour in a gambling task (game of dice task (Starcke et al., 2008)). A recent meta-analyses concluded that stress impairs decision making especially under those conditions where reward seeking and risk taking is disadvantageous (Starcke et al., 2008; Starcke and Brand, 2016). The findings on decision making are in line with the stress induced deficits in executive functions mentioned above. Impaired decision making under risk is of relevance for safety behaviour of employees (Starcke et al., 2016) or for stock-market traders (Coates and Herbert, 2008; Cueva et al., 2015). Impaired decision making under stress can also promote addictive behaviour or relapse (Brand et al., 2016).

3. Stress and long-term memory

The effects of stress on long-term memory (LTM) have received considerable attention during the last decades. LTM can be subdivided into declarative or explicit and non-declarative or procedural (implicit) memory. Based on its content, declarative memory can be subdivided into episodic memory (recall of a specific event which can be located in space and time) and semantic memory (overall knowledge of the world) (Squire, 1992). The medial temporal lobe is critical for declarative memory, with the hippocampus being especially important for episodic memory (Nadel and Moscovitch, 1997).

Long-term memory consists of at least three memory phases, namely encoding (or acquisition), consolidation (or storage), and retrieval (or recall). The literature on the impact of stress on episodic memory was initially divergent and confusing, with groups reporting both enhancing as well as impairing effects of GCs on this form of memory. However, it has become apparent that this is largely due to the fact that the different memory phases outlined above are modulated by GCs in an opposite manner (Roozendaal, 2002; Shields et al., 2017).

Pre-learning stress studies have led to a somewhat inconsistent picture (see Fig. 1). The exact timing of the stressor (e.g. (Zoladz et al., 2011)), the emotionality of the learning material (e.g. (Payne et al., 2007)) and the relation of the learning material to the stressor (e.g. (Smeets et al., 2009)) appear to be important modulatory factors (Wolf, 2009, 2017). A recent meta-analysis (Shields et al., 2017) revealed that pre-encoding stress typically was associated with impaired long-term memories, especially when the stressor and the memory task were separated in space and time and the learning material was not related to the stressor (e.g. (Cadle and Zoladz, 2015)). In those studies the initial emotional (noradrenergic) arousal is already gone and the influence of cortisol dominates. This constellation apparently is typically linked to poorer encoding (Joëls et al., 2006). In line with this interpretation several studies reported negative correlations between the (pre-encoding) stress induced cortisol increase and memory (Kirschbaum et al., 1996; Wolf et al., 2001b). However this association is not always detected (Shields et al., 2017) probably reflecting inter-individual differences in glucocorticoid sensitivity (Rohleder et al., 2009) as well as parallel and interacting influences of SNS activity (Roozendaal et al., 2009) and affect (Abercrumbie et al., 2005; Wiemers et al., 2018).

Stress boosts memory consolidation, this process representing the adaptive and beneficial side of the action of stress hormones in the central nervous system (see Fig. 1). It has been characterized as the beneficial effects of ‘stress within the learning context’, or ‘intrinsic stress’ (Joëls et al., 2006). This terminology emphasizes the fact that the central aspects of a stressful episode are remembered better, an issue we investigated experimentally in humans with the TSST (see below).

Immediate post-learning stress specifically targeting memory consolidation has repeatedly been linked to enhanced LTM. Participants who were exposed to the cold pressor stressor directly after viewing a series of slides had better long-term memories of the slides compared to the stress free control group (Cahill et al., 2003). Supporting evidence
comes from pharmacological studies (e.g. [Buchanan and Lovallo, 2001]). Often these enhancing effects of stress on consolidation are stronger for emotional material (Cahill et al., 2003) even though they also have been found when neutral material was used. For example we observed that participants who had viewed emotional and neutral pictures before being exposed to the TSST showed enhanced memories for the neutral pictures when tested 24 h later (Preuß and Wolf, 2009). Several studies reported that the enhanced memory consolidation was associated with the stress induced increase in cortisol and/or noradrenergic arousal (Andreano and Cahill, 2006; Smeets et al., 2008; Zoladz et al., 2011). Moreover an interaction between cortisol and negative affect in predicting the stress induced memory enhancement has also been observed (Abercrombie et al., 2005).

Neuroimaging studies have provided evidence for a stress-induced modulation of amygdala and hippocampal activity (Henckens et al., 2009; van Stegeren, 2009). These effects are mediated by the action of stress-released GCs on the hippocampal formation. Studies in rodents have shown that an adrenergic activation in the basolateral amygdala (BLA) appears to be a pre-requisite for the modulating effects of GCs on other brain regions (e.g. the hippocampus). Lesions in the BLA as well as beta blockade abolish the enhancing effects of post-training stress (Roozendaal et al., 2009).

More recently the effects of stress on reconsolidation have been investigated. Reconsolidation refers to the re-stabilization of a memory trace which has become labile during retrieval/reactivation (Nader and Hardt, 2009). This process occurs when reactivation is accompanied by a prediction error (Sevenster et al., 2013). So far enhancing (Bus et al., 2014; Coccoz et al., 2011; Hupbach and Dorskind, 2014) as well as impairing (Larrosa et al., 2017; Schwabe and Wolf, 2010) effects of stress on reconsolidation have been reported. The exact timing of the stressor with respect to the reactivation might be one determining factor. The interested reader is referred to the following recent review (Meir Drexler and Wolf, 2018).

While an enhanced memory consolidation is typically adaptive, this effect appears to occur at the cost of impaired retrieval (see Fig. 1). Using a one day delay, de Quervain and colleagues were able to show that stress or treatment with GCs shortly before retrieval testing impairs memory retrieval in rats in the Morris Water Maze (de Quervain et al., 1998). Importantly this effect occurred 30 min after stress induction (at times of high corticosterone levels) but was absent immediately as well as four hours after stress induction. Moreover the impairing effects of stress could be blocked using glucocorticoid synthesis inhibitor Metyrapone and could be mimicked by administering corticosterone. Taken together these experiments provided strong evidence for a causal role of the stress induced corticosterone increase in impairing memory retrieval (de Quervain et al., 1998).

Again very similar findings were observed in humans. Participants exposed to the TSST showed poorer memory retrieval of words learned 24 h earlier (Kuhlmann et al., 2005). Similar retrieval impairing effects were observed for social stimuli like faces and autobiographical notes (Merz et al., 2010). Retrieval impairing effects have also been observed in several studies using the CPT (e.g. Buchanan, 2007; Smeets, 2011). In line with these examples the aforementioned meta-analysis (Shields et al., 2017) reported a significant impairing effect of stress on memory retrieval. Pharmacological studies administering glucocorticoids to human participants in a double blind placebo controlled design also repeatedly observed an impairment in memory retrieval after GC administration (de Quervain et al., 2000; Het et al., 2005). This effect appears to be primarily driven by the GR (Rimmle et al., 2013). Roozendaal has summarized these findings as indicative of stress putting the brain into a consolidation mode, accompanied by impaired retrieval (Roozendaal et al., 2009). A reduction in retrieval might support consolidation by reducing interference (Wolf, 2017).

Such an impairing effect of social stress on memory retrieval might explain retrieval deficits occurring during school or university exams. Especially oral exams, which exert a strong social evaluative threat, lead to a pronounced activation of stress hormones (Preuss et al., 2010). Another area where the impact of stress needs to be considered more is eyewitness memories (Christianson, 1992). Last but not least these findings have relevance for the development and treatment of anxiety disorders and post-traumatic stress disorder (de Quervain et al., 2017).

Initially the impairing effects of stress on memory retrieval were thought to be restricted to episodic memories. However research conducted during the last years revealed that stress also impairs stimulus response memory retrieval (Atsak et al., 2016) or extinction memory retrieval (Kinner et al., 2016). In addition the effect appears to be rather long lasting (up to several hours).

4. Memories of the TSST

In most previous stress and memory studies conducted in the laboratory the learning material and the stressor were unrelated (but see for an exception (Smeets et al., 2009)). Participants would study words or see pictures before or after being exposed to a standardized stressor (e.g. (Smeets et al., 2008; Zoladz et al., 2011)). While this work is important and has generated a sizeable amount of data it cannot answer the question what we remember from a stressful episode itself. This is, however, of major relevance for the understanding of eye-witness
memories or traumatic memories.

We therefore conducted a series of experiments where participants were tested for their memories of the TSST itself. As a first step we developed a version of the TSST which contained a number of objects which were presented on the TSST-table (Wiemers et al., 2013a). Moreover the committee interacted with half of these objects in a standardized fashion (e.g. using the stapler in order to staple paper). These items were called `central' in order to indicate that they are connected to the central aspect of the stressor (the committee). A picture of the TSST-committee with the used objects is presented in Fig. 2 (left side). On the next day a surprise recognition test took place where participants had to recognize the objects as well as the faces of the committee members out of a number of distractors. The memory of the participants was compared to the memory of a control group participating in a newly developed control condition termed the friendly TSST (Wiemers et al., 2013b). Here participants have to talk about some freely chosen aspects of their CV (e.g. hobbies, favorite movies etc.). In contrast to the Placebo-TSST where the participant conducts a speech and a simple calculation task alone in a room (Het et al., 2009), the friendly TSST takes place in front of a committee. However in sharp contrast to the TSST the committee in the friendly TSST responds in a very friendly and open manner and no video recordings take place (Wiemers et al., 2013b). A picture of the f-TSST-committee with the used objects is presented in Fig. 2 (right side). This control condition does not activate the HPA axis and also does not lead to an increase in negative affect. It does, however, increase sAA to a similar amount as the TSST does (Wiemers et al., 2013b).

When we compared the recognition memory performance of participants exposed to the stressful TSST with the performance of participants allocated to the control group we observed enhanced memories in the stress group for the central items of the TSST (see Fig. 3) as well as for the faces of the TSST committee members (Wiemers et al., 2013a). The finding of enhanced memories for central objects presented during the TSST was replicated in three follow up studies (Herten et al., 2017a, b; Wiemers et al., 2014).

In order to understand the mechanisms behind the stress induced enhancement of memories for central items we conducted additional experiments. In the first one we tested the potential causal role of the stress hormone cortisol (Wiemers and Wolf, 2015). In a pharmacological double blind study participants exposed to the friendly TSST received either cortisol or placebo. We observed that cortisol enhanced memory (in men) for peripheral items but not for central items. Thus the memory enhancement induced by cortisol within the context of a friendly episode differs from the memory enhancement induced by stress (Wiemers and Wolf, 2015).

In another experiment we wanted to test the potential impact of altered visual exploration during a stressful versus a non-stressful social situation (Herten et al., 2017a). Participants wore a mobile eye-tracker during the TSST and the fTSST. Again their memories for the office items were tested the next day. In line with the previous findings the stress group showed better memories for the central items. Interestingly they also spend more time fixating these items. However the fixation measures did not mediate the impact of stress on long-term memory suggesting that changes in visual exploration alone are not sufficient to explain the observed memory enhancement in the stress group (Herten et al., 2017a).

Last but not least we tested the memories of the office item shortly after the TSST (and not on the next day) (Herten et al., 2017b). This experiment could thus detect effects of the stressor on immediate recall, which could not be explained by stress-induced changes in consolidation. Participants exposed to the TSST again showed enhanced memories for the central items illustrating that this effect can be already detected shortly after stress cessation. This points towards a beneficial effect of stress on encoding of central items which are connected/re- lated to the emotional source of the situation.

These findings are in line with the Easterbrook hypothesis (Easterbrook, 1959) on the enhanced usage of central cues under emotional distress. They could also be explained by emotional binding accounts put forward by Mather (2007) or Yonelinas (Yonelinas and Ritchey, 2015). Last but not least they also fit to the ideas of the synaptic tagging hypothesis (McReynolds and McIntyre, 2012; Richter-Levin and Akirav, 2003). Taken together our studies on memories from the TSST indicate that items experienced during stress and which are related to the stressor are remembered especially well. The joint sequential activation of the SNS and the HPA axis apparently leads to enhanced memories for those items which are central to the current episode and which are contextually and/or conceptually related to the main aspect of the stressor. This conclusion is in line with the model from Joëls (Joëls et al., 2006). In fact a similar conclusion was recently put forward by a meta-analysis of experimental stress studies in humans (Shields et al., 2017).

5. Some moderators to be considered

5.1. Developmental changes

The studies reviewed above were conducted in healthy young adult participants. They can thus not answer the question about possible developmental changes. With respect to childhood evidence exists that...
the effects of stress on memory are relatively similar in school children compared to adults. For example Quas and Yim could show that memories for an age appropriate version of the TSST are related to their cortisol reactivity (Quas et al., 2010). Using the Trier Social Stress Test for Children (TSST-C (Buske-Kirschbaum et al., 1997)) we could show that elementary school children showed an impairing effect of stress on memory retrieval (Quesada et al., 2012). A finding highly similar to the observations made in adults (Kuhlmann et al., 2005). Even though data in children is still sparse the basic findings on stress and memory appear to be rather similar to those obtained in adults.

With respect to aging few studies exist which systematically compare young and older adults. Results are far from consistent with stronger but also weaker or even absent effects of stress on specific aspects of memory being reported (e.g. (Hidalgo et al., 2014; Pulopulos et al., 2013)). In a pharmacological study we observed that the impairing effects of stress on memory retrieval were highly similar for young and older participants (Wolf et al., 2001a). In contrast impairing effects on working memory were only detectable in young but not in old participants. Thus aging associated changes in stress sensitivity might depend on the involved brain regions and are potentially further modulated by sex (Hidalgo et al., 2015).

5.2. Sex differences

Men and women differ in their psychosocial stress response and apparently also in the impact of the stress response on cognition. Studies using the TSST repeatedly observed more pronounced responses in men when compared to women (see for a recent meta-analysis (Liu et al., 2017)). In order to complicate things it has to be mentioned that the menstrual cycle and especially the usage of oral contraceptives influences the (salivary) cortisol response to acute stress. Women using OCs typically display a blunted cortisol increase (Kirschbaum et al., 1999), which is caused by an OC induced increase in sex hormone binding globulin (Hellhammer et al., 2009). These sex and sex hormone effects need to be taken into account when designing experiments and when interpreting empirical findings (Merz and Wolf, 2017).

In addition to sex differences in neuroendocrine stress responsibility there is also evidence for sex differences in the sensitivity to stress hormones when it comes to cognition. Differential effects of stress or cortisol on a variety of cognitive processes have been reported even though the findings are currently far from being consistent. Often the impact of stress on memory appears to be stronger and more robust for men, when compared to women ((Andreano and Cahill, 2006) but see (Zoladz et al., 2014)). Moreover, again an impact of menstrual cycle phase and even more so of OC usage appears to exist (Merz and Wolf, 2017). For example the impairing effect of stress on memory retrieval could not be detected in women in the luteal phase (Schoofs and Wolf, 2009). Beneficial effects of post-encoding stress on memory were stronger in the follicular compared to the luteal phase (Zoladz et al., 2015). Cortisol, which impairs memory retrieval in men, did not do so in women using OCs (Kuhlmann and Wolf, 2005). Similarly studies using the TSST or the CPT observed blunted or absent effects of stress on memory encoding in women using oral contraceptives (Cornelisse et al., 2011; Zoladz et al., 2013). Neuroimaging studies in the domain of fear conditioning repeatedly observed almost opposing effects of stress or cortisol administration on the neural correlates of emotional learning (Merz et al., 2012, 2013). Recent reviews on this highly important topic are provided by (Merz and Wolf, 2017; Stockhorst and Antov, 2015).

Taken together empirical (Merz and Wolf, 2017) as well as conceptual (Taylor et al., 2000) evidence emphasizes the need to consider sex differences in psychoneuroendocrine stress research. The usage of small sample sizes with unbalanced participants from both sexes is not advisable since it may mask stress effects thereby hindering scientific progress in this important area. The exclusive focus on one sex (most often men in this sort of research) can be understood from a pragmatic point of view, but has led to a lack of information on stress effects in women. This state of affairs cannot be accepted anymore and is increasingly being recognized by journals and funding agencies (see (Cahill, 2017)).

5.3. Genetic influences

Genes involved in the regulation of the SNS and HPA response to stress can substantially influence the effects of stress on cognition. For example variations in a gene encoding the alpha 2 receptor influence the ability to form emotional memories (de Quervain et al., 2007b; Rasch et al., 2009). Moreover it also modulates the impact of acute stress on emotional memories and their neural correlates (Li et al., 2014, 2013). Thus genetic alterations within the stress system can influence the susceptibility to stressful events.

Other examples are alterations in the genes encoding the MR and GR. Under stress, the MR might be responsible for switching cognitive processing towards simpler, habitual/automatic response styles (Schwabe and Wolf, 2013). The GR appears to be especially important for memory consolidation (Roozendaal et al., 2009). Variability in the function and balance of these key stress mediators is likely to be related to interindividual differences in stress responsibility and sensitivity (Joëls et al., 2008). Studies have recently begun to characterize the impact of genetic polymorphisms in the MR and GR genes on HPA axis (re)activity and disease risk (De Kloet et al., 2016). The discovery of epigenetic modulation of these receptors by early life stress allows a new look at gene-environment interplay (Turecki and Meaney, 2016). Thus we are at the beginning of a new area in psychoneuroendocrinology which combines experimental laboratory studies with behavioural genetics.

6. Interventions

Some of the effects of stress on cognition might not be desirable in a specific situation. For example the impairing effects of stress on memory retrieval could lead to sub-optimal performance in an exam. Here stress reduction techniques or social support might be useful strategies to buffer the stress response (Ditzen and Heinrichs, 2014). In addition specific learning techniques such as retrieval practice might be able to reduce or prevent stress effects (Smith et al., 2016; Wolf and Kluge, 2017). Last but not least pharmacological interventions (e.g. the beta-blocker propranolol) can protect memory retrieval from the impairing effect of stress (de Quervain et al., 2007a). Future work translating these basic science studies into applied settings (e.g. schools, factories, mental health clinics) is needed.

7. Conclusion and outlook

In this review the impact of acute stress on memory has been reviewed with a focus on studies using the TSST. Opposing effects on memory consolidation versus memory retrieval are two fairly well established effects. Moreover stressed participants are better in remembering the central aspects of a stressful episode. Developmental changes, sex differences and genetic influences are moderators to be considered in future research. An enhanced understanding and a more differentiated view of the beneficial and detrimental effects of acute stress on human memory will in the long run help scientists to improve individual and societal well-being by promoting resilience.

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