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What Matters Most: Selective Memory Enhancement Surrounding Acute Stress

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Abstract

When stress occurs, this changes our memory for events around the stressor. These stress-driven changes in memory are not universal, but depend on various factors. Nevertheless, the factors driving this selectivity remain elusive. The behavioural tagging hypothesis offers a framework for understanding the etiology of such selectivity. Based on this framework, we proposed two mechanisms that may tag encoded information, allowing for selective memory enhancement if a stressful encounter occurs in the temporal periphery. Study I investigated whether a cue to a stressful context yields memory improvements for subsequently encoded information. While we did not find selective memory improvement for information that cued a stressful experience, we found a general memory improvement for material encoded before stress, specifically when the stressful experience was well represented in memory. In studies IIa and IIb, we explored whether stress selectively enhances the memory for information associated with a prediction error (PE). In Study IIa, we observed selective memory enhancement for high-PE information encoded both before and after the stressor. In Study IIb, the effect was specific to information encoded after the stressful encounter. These findings highlight that the influence of stress on memory is selective, and PE stands out as potential "gating" mechanism through which stress amplifies specific memories. Such selective memory enhancement can be considered adaptive based on the predictive brain hypothesis – when stress occurs, this may encourage the prioritization of specific information that helps to make better predictions in the future. These findings may be leveraged in educational settings and help us understand the development of strong post-traumatic memories.

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List of abbreviations

SNS	Sympathetic nervous system
HPA	Hypothalamic-pituitary-adrenal
LTP	Long-term potentiation
LC	Locus coeruleus
NE	Noradrenaline
TSST	Trier social stress test
eTSST	Enriched Trier social stress test
BTH	Behavioural tagging hypothesis
l-LTP	Late long-term potentiation
e-LTP	Early long-term potentiation
PRP	Plasticity related product
AMPA	α-amino-3-hydroxyl-5-methyl-4-isoxazole-propionate
PE	Prediction error
SLIMM	Schema-linked interactions between medial prefrontal and medial temporal regions
MDBF	Mehrdimensionale Befindlichkeitsfragebogen
STAI-S	State-Trait Anxiety Inventory - State
TICS	Trier Inventory of Chronic Stress
SCR	Skin conductance response
CDA	Continuous decomposition analysis
EEG	Electroencephalography
ERP	Event-related potential

FPL	Fractional peak latency
EST	Event segmentation theory
SPS	Sensory processing sensitivity

1. Introduction

What do you remember from your final exams at school? What about other events on that day, such as the funny looking dog you saw on your way to the exam hall, the weather in the morning, or the things you did afterwards? During a stressful situation, we often have the impression that our memory suffers. Indeed, under acute stress our ability to recall previously learned information can be significantly impaired (de Quervain et al., 1998; Klier & Buratto, 2020; Smeets et al., 2008). However, when it comes to learning new information during stress, the research shows a more dynamic picture. Specifically, experiencing stress can actually improve encoding of new information, especially for central details of the stressful situation (Bierbrauer et al., 2021; Kalbe et al., 2020; Lin et al., 2022; Stanek et al., 2024). Meanwhile, your memory for events that occurred before or after the experience might either improve or deteriorate depending on the context, how much time has passed, and relevance to the stressor (Shields et al., 2017). Remarkably, your memory for neutral events occurring before or after the stressful situation appears to not be affected in a uniform way. Some events seem to be remembered better, whereas others don't (see Shields et al., 2017 for a review). Thus, there are factors that selectively gate which events from around the time of the stressor will profit mnemonically from stress. The drivers of such selectivity and the cognitive mechanisms behind these factors remain poorly understood. Understanding what drives the effect of stress on the memory for surrounding information is crucial for clinical and educational settings, and carries important implications for eyewitness testimony.

1.1 The dynamic impact of stress on memory processes

Stress impacts memory through several cascades, hormones and neurotransmitters, occurring at varying time-scales (Fig 1.1). Two key players stand out in the stress response system – the autonomic nervous system, specifically via the activation of the sympathetic nervous system (SNS; Richter and Wright, 2020), and the hypothalamic-pituitary-adrenal (HPA) axis (Ulrich-Lai & Herman, 2009). The rapid, immediate stress response through the activation of the SNS activates the fight-or-flight response, which wanes quickly as the parasympathetic nervous system is reflexively activated, or as the stressful experience subsides (Joëls & Baram, 2009; Richter & Wright, 2020; Ulrich-Lai & Herman, 2009). The adrenal glands play a key role in the deployment of stress-related signalling molecules. The activation of the SNS results in the release of adrenaline and noradrenaline from the adrenal medulla, with projections to the amygala (Segal, 2020). SNS activation results in the increase of blood glucose levels, and this facilitates enhancement of learning and memory (Segal, 2020). The medial prefrontal cortex also sees elevated noradrenaline levels in response to acute stress (Ma & Morilak, 2005; Morilak et al., 2005). Other monoamines are also rapidly released from other sites as part of the stress response (Joëls & Baram, 2009). Serotonin levels increase in the hippocampus and prefrontal cortex (Beekman et al., 2005; Linthorst & Reul, 2008). Moreover, dopamine is increased in the prefrontal cortex via innervation from the ventral tegmental area (Goto et al., 2007). The nucleus accumbens also shows elevated dopamine levels in response to acute stress (Goto et al., 2007; Gresch et al., 1994). This dopaminergic increase has been found to enhance long-term potentiation (LTP; Goto et al., 2007).

This first line of response is followed closely by the synthesis and release of neuropeptides and steroid hormones (Joëls & Baram, 2009). The HPA axis is activated as stress stimulates the release of corticotropin-releasing-hormone from the hypothalamus (Ulrich-Lai & Herman, 2009). This in turn stimulates the pi-

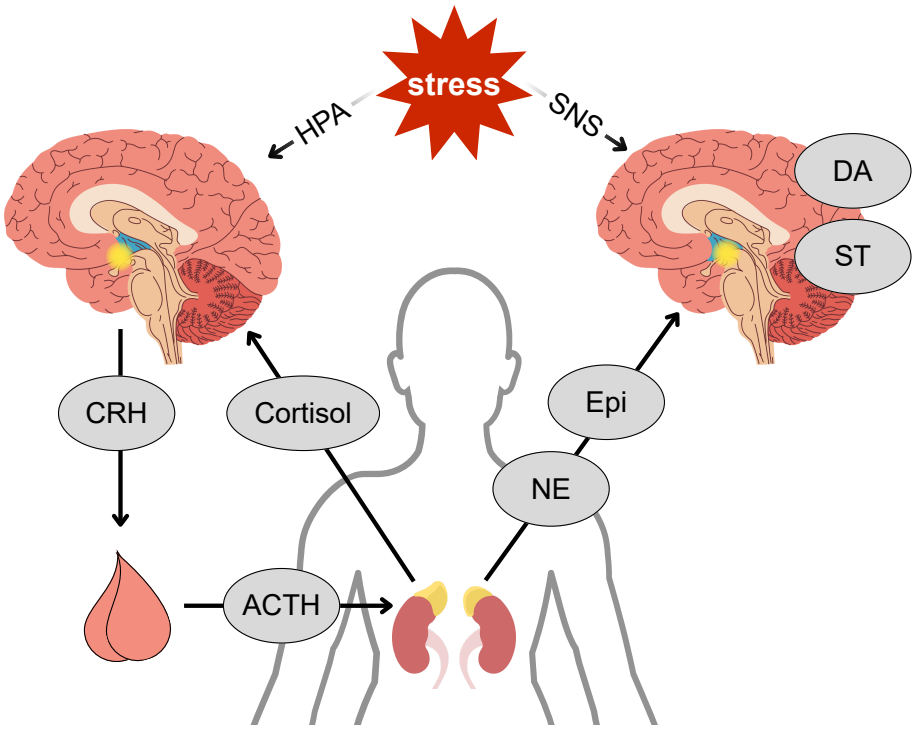

Figure 1.1 Simplified stress response. At the onset of a stressor, the sympathetic nervous system (SNS) is activated, resulting in the release of noradrenaline (NE) and adrenaline (Epi) from the adrenal glands and their projection to the amygdala. Meanwhile, dopamine (DA) and serotonin (ST) concentrations also increase in various brain areas. In addition to the SNS, the hypothalamic-pituitary-adrenal (HPA) axis results in the release of corticotropin-releasing hormone (CRH) from the hypothalamus, which results in the release of adrenocorticotropic hormone (ACTH) from the pituitary glands, which stimulates the adrenal glands to release cortisol. The fluorophore references the hypothalamus (left) and the amygdala (right).

tuitary glands to release adrenocorticotropic hormone, which finally triggers the release of glucocorticoids from the adrenal glands, specifically cortisol in humans (Ulrich-Lai & Herman, 2009). Cortisol binds to two kinds of receptors in the brain – mineralocorticoid and glucocorticoid receptors (De Kloet et al., 2005). Mineralocorticoid receptors are implicated at the onset of the stressful episode, and have a notably higher affinity than glucocorticoid receptors (De Kloet et al., 2005). Glucocorticoid receptor activity contributes to memory storage in preparation for coping with future events (De Kloet et al., 2005). Notably, these glucocorticoid receptors are concentrated in the hippocampus, alongside other areas that play a key role in memory making mechanisms (Joëls & Baram, 2009). The effects of cortisol can occur at two time-scales – slowly, through gene-mediated effects, but

also quickly, by acting on near-membrane mineralocorticoid receptors in the hippocampus to increase glutamate release (Karst et al., 2005). In this way, cortisol contributes not only to a slower gene-mediated stress response, but also allows the brain to adapt to the stressor within minutes of cortisol release (Karst et al., 2005). Thus, the early stress response consists of two waves – first, cortisol levels rise, with fast acting agents like monoamines and possibly cortisol enhancing attention and information processing (De Kloet et al., 2005). Gradually, gene mediated effects of cortisol start to exert their effect (De Kloet et al., 2005).

These stress mediators impact neural activity, specifically in prefrontal and medial-temporal areas, and thus experiencing stress can exert a profound impact on learning and memory processes (Diamond et al., 2007; Roozendaal et al., 2009; Schwabe et al., 2022). The amygdala and hippocampus stand out as key players in stress modulation of learning and memory. The hippocampus is a canonically important area for memory formation (R. J. McDonald & White, 1993; Scoville & Milner, 1957). Stress has been found to impair hippocampal LTP, as well as to disrupt hippocampus-dependent memory (Diamond & Rose, 1994). Furthermore, noradrenergic activation after encoding has been found to alter the involvement of the hippocampus in memory (Atucha et al., 2017; Krenz et al., 2021). Specifically, while the involvement of the hippocampus in memory normally reduces over time thorugh systems consolidation, whereby memories become increasingly more dependent on neocortical areas instead, noradrenergic activation after encoding has been shown to maintain hippocampal involvement and preserve detail memory of the learned content (Atucha et al., 2017; Krenz et al., 2021).

Meanwhile, the amygdala shares reciprocal projections with the hippocampus (A. J. McDonald & Mott, 2017). The amygdala has been found to be central to the processing of emotional memories and their details (Adolphs et al., 2005; Cahill et al., 1995; McGaugh, 2004). Specifically, the basolateral amygdala is a key driver of emotional memory enhancement through the joint actions of noradrenaline and cortisol (Roozendaal et al., 2006). The amygdala thus seems to

modulate the effect of stress on hippocampal learning (Kim et al., 2001). When rats underwent an acutely stressful experience in the form of restraint or a tail shock, hippocampal LTP was impaired, alongside memory of a water maze (Kim et al., 2001). This impairment was abolished in rats with a lesioned amygdala, highlighting the role of this region in mediating the effect of a stressful experience on memory formation. In humans, the hippocampus and amygdala have been found to interact with glucocorticoids in shaping memory (Ritchey et al., 2017). Participants encoded neutral and emotionally arousing images before being subjected to stress through a cold pressor test. The cortisol response to the stressor directly related to activity in medial temporal areas during encoding – in other words, if participants showed an elevated cortisol response to a stressful experience, subsequent successful memory recall depended on how much activation there was in the hippocampus and amygdala during encoding (Ritchey et al., 2017).

Stress further triggers network reorganization in the brain in order to support the stress response (Schwabe et al., 2022). Hermans et al. (2011) exposed people to aversive movies during fMRI scanning, and found increased responsiveness and interconnectivity in a widespread network encompassing multiple cortical areas as well as deeper structures, like the amygdala and hypothalamus. This increase was directly related to the strength of the measured stress response (Hermans et al., 2011). This shift in network activity and connectivity appeared to be modulated by noradrenaline, as administration of the noradrenaline antagonist propranolol diminished the increase in activity and connectivity of this network (Hermans et al., 2011). During the stress response, noradrenaline, together with cortisol, switches the brain to a memory formation mode (Schwabe et al., 2022). Part of this memory formation mode entails the shift from cognitive to more habit foucsed memory systems (Schwabe et al., 2022; Vogel et al., 2017). Stress has been found to drive an increase in amygdala activity, as well as an increased connectivity between the amygdala and the striatum (Vogel et al., 2017).

This increase was related to an enhancement of striatum dependent learning, and directly depended on mineralocorticoid receptor activity, which highlights the role of cortisol in modulating the memory systems active during stress (Vogel et al., 2017). Moreover, this stress driven memory formation mode involves an increase in salience network activity, encompassing the anterior cingulate cortex, amygdala, frontal insula, and inferior temporal cortex (Schwabe et al., 2022). Parallel to salience network activity is the locus coeruleus-noradrenaline (LC-NE) complex. At homeostasis, the LC operates at a high signal-to-noise ratio via high phasic and lower tonic activity (Joëls & Baram, 2009; Valentino & Van Bockstaele, 2008). This fosters focused attention (Valentino & Van Bockstaele, 2008). However, during stress, corticotropin-releasing-hormone activity shifts the LC to a mode of elevated tonic activity, thus promoting arousal, environment scanning, and decision making (Valentino & Van Bockstaele, 2008).

This way, the effect of stress on memory highlights the interacting effects of noradrenaline and cortisol. As discussed above, stress triggers noradrenaline release in the amygdala, which facilitates the activation of the HPA axis (Ma & Morilak, 2005). Noradrenaline further induces a hypervigilant state, which aids in memory formation, and is further enhanced by cortisol released into the amygdala (Joëls et al., 2011). Concurrent activity of noradrenaline and cortisol in the basolateral amygdala is necessary for emotional memory enhancement (Roozendaal et al., 2006). Rodents completed object recognition training in either a familiar or novel context, before the injection of corticosteroids (Roozendaal et al., 2006). Twenty-four hours later, context-naive rats showed the expected memory enhancement for the object recognition training. Meanwhile, blocking noradrenergic activity in the basolateral amygdala prevented this memory benefit, while stimulating it restored memory enhancement even in habituated rats (Roozendaal et al., 2006). Thus, rather than directly creating stronger memories for emotional material, stressful events trigger the activation of systems that modulate how strongly memories are consolidated (Roozendaal & McGaugh, 2011). Acute

stress activates sympathetic and adrenal systems, and the basolateral amygdala integrates these signals through noradrenergic mechanisms, ultimately influencing the consolidation of important events (McGaugh, 2004; Roozendaal & McGaugh, 2011).

Thus, stress exerts both transient and longer lasting effects on the brain. Certain modulators, like noradrenaline, exert their effects in a transient manner, while others, like cortisol, act over a longer time scale. These dynamics, together with shifts in neural network activity, indicate that temporal and contextual factors determine whether stress will have an enhancing or impairing effect on memory.

1.2 The effect of stress on memory is non-universal and time-dependent

Prior work has generally approached stress effects on memory through a global influence driven by the effects of monoamines and cortisol (Joëls et al., 2006; Roozendaal et al., 2006). However, literature has shown that the impact of stress on memory is selective, not universal (see e.g. Kensinger, 2009; Laney et al., 2004; Shields et al., 2017). As discussed above, episodic memory for emotional material follows a trajectory determined by the configuration of stress-related neuromodulators as well as activity and connectivity in large scale neural networks. Specifically, elevated arousal has been suggested to conditionally enhance memory if there is an increase in noradrenaline, combined with the release of corticosteroids and promoted connectivity between the basolateral amygdala and other brain areas (McGaugh, 2004). Thus, attention grabbing stimuli releasing noradrenaline may be preferentially encoded given concurrent activation of the stress system. Accordingly, this kind of selective memory enhancement has been suggested to occur due to a stress driven "attention magnet" whereby the gist, or central features, of a stressful situation are prioritized in memory (Kensinger, 2009; Laney

et al., 2004). The effects of stress on encoding during and around the time of the stressor are thus determined by several factors, which explains the multitude of effects seen in the literature.

One factor driving this selective effect of stress on memory is the relatedness of the stressor and the encoded information. For instance, stressor-relevant words encoded after stress were rememebered better, and this memory enhancement was directly related to the cortisol response and adrenergic activity triggered by stress (Smeets et al., 2009). The Trier Social Stress Test (TSST) is the gold standard for inducing acute, psychosocial stress in a laboratory setting (Kirschbaum et al., 1993). The TSST mimics a stressful job interview, and consists of a public speaking task as well as an arithmetic task, performed in front of a neutral, non-reinforcing committee of two actors. To study participants' memory for central and peripheral details of such stressful experiences, an enriched version of the TSST was developed (eTSST; Bierbrauer et al., 2021; Kalbe et al., 2020; Lin et al., 2022). In the eTSST, the room where the TSST takes place is decorated with objects that are either central or peripheral to the stressful experience. Central objects include items that are part of the stressful experience, for instance, a cup from which one of the panel members drinks. Peripheral objects, such as a poster on the wall, remain in the background and are thus not relevant for the stressful experience. Compared to a friendly control experience, stressed participants tend to remember more central objects (Bierbrauer et al., 2021; Kalbe et al., 2020; Lin et al., 2022; Stanek et al., 2024). In other words, the eTSST produces a centrality bias in the memory for the stressful situation. In Bierbrauer et al. (2021), experiencing stress altered the neural representation of stress-related objects, depending on their centrality to the experience. Specifically, the neural representation of central items in the amygdala became more similar, and the more similar they were, the better they were represented in memory (Bierbrauer et al., 2021). This suggests that the relatedness of some information to stress impacts the memory for this information through changes in amygdala representations of this memory.

Such a centrality bias is consistent with findings showing that the gist of emotional information is preferentially remembered over the details (Adolphs et al., 2005; Kensinger et al., 2007). In Kensinger et al. (2007), participants encoded neutral and negative objects superimposed on neutral scene images. They found a memory enhancement for central, negative information, but also a shift in contextual processing whereby these negative objects triggered a trade-off in the processing of gist and detail information. In other words, the gist memory of the scenes in the background was enhanced at the expense of detail memory (Kensinger et al., 2007). This gist-prioritization effect is not present in patients with damage to the medial temporal lobe or amygdala, highlighting the role of these brain areas in the encoding of stress-relevant information (Adolphs et al., 2005). Similarly, Simon-Kutscher et al. (2019) found that acute stress specifically impaired contextual fear learning, rather than cue-based fear learning. This relative shift from context to cue-based learning was directly related to the strength of the cortisol response to the stressor (Simon-Kutscher et al., 2019).

In addition, it has been suggested that negatively valenced information is preferentially encoded due to stress (Kensinger, 2009). This may be due to the preferential engagement of key brain regions during encoding, such as the amygdala. Specifically, engaging emotion processing regions during in the brain could increase the probability that those events will be remembered later (Kensinger, 2009). Ritchey et al. (2017) found that memories that engaged the medial temporal lobe more during encoding benefited more from post-encoding release of corticosteroids. Emotional words encoded around a stressful experience have been found to be remembered better than neutral words (Elzinga et al., 2005; Smeets et al., 2008). The strength of the stress response in form of salivary cortisol and alpha-amylase correlated positively with this selective enhancement for emotional words encoded before a stressful experience (Smeets et al., 2008). Meanwhile, some evidence suggests that a stronger cortisol response correlates negatively with memory performance for words encoded after a stressful situation,

suggesting differing effects of cortisol on the memory enhancement for negatively valenced information depending on the timing of encoding (Elzinga et al., 2005). However, the effect of emotionality on memory is not always consistent – while most of the findings presented here suggest a selective stress driven memory enhancement for negatively valenced information, Ritchey et al. (2017) found that corticosteroid driven enhancement for memories that engaged the medial temporal lobe more during encoding was specifically pronounced for neutral, rather than emotional information.

Timing dependent effects of stress on memory strongly depend on the temporal dynamics of monoamines and cortisol, often showing memory enhancement for events that are encoded within the same temporal and spatial context as the stressor, while impairing memory for non-stress related information (Diamond et al., 2005; Joëls et al., 2006; Schwabe et al., 2022; Smeets et al., 2007). Whether neutral item memory will be enhanced by subsequent arousal depends in part on whether it predicts the onset of emotional information (Knight & Mather, 2009). Neutral information encoded within seconds before emotional item encoding is enhanced in memory (Anderson et al., 2006; Knight & Mather, 2009). At longer time scales, when arousal is separated from the encoding by minutes to hours, these effects can look different. Further, a complex, psychosocial stressor like the TSST often involves a context shift, which brings into question the predictive utility of preceding neutral material.

Timing dependent effects have been found to interact with other variables that shape the effect of stress on memory. Smeets et al. (2008) found a memory enhancement for emotional words specifically if stress occurred after the encoding. While Smeets et al. (2008) found no memory effects for information encoded after a stressful encounter, several other studies have found enhancing effects (Domes et al., 2002; Goldfarb et al., 2019; Schwabe et al., 2008; Smeets et al., 2009; Vogel & Schwabe, 2016). Meanwhile, others have even shown impairing effects (Elzinga et al., 2005; Kirschbaum et al., 1996; Quaedflieg et al., 2013). A meta-analysis sug-

gested that post-stress encoding is generally impaired, except for when the encoded material is directly related to the stressful situation, or when the time-delay between stress and encoding is short (Shields et al., 2017). These inconsistencies are often attributed to differences in the timing of stress relative to encoding (Joëls et al., 2006; Shields et al., 2017; Zoladz et al., 2011). Heterogeneity between study paradigms likely also explains some of the divergent findings of stress on peri-stress encoding, suggesting that the effect of stress on memory for surrounding material is selective only for some kinds of content.

The findings discussed here suggest that stress and arousal have lasting impacts on episodic memory encoding. However, these effects do not manifest in a universal manner – rather they depend on several factors, such as the emotionality of the encoded information, the timing of the stressor, and the recruitment of key brain regions, such as the amygdala and medial temporal lobe, during encoding (Ritchey et al., 2017; Shields et al., 2017). Thus, the literature shows that the prospective and retrospective effect of stress on memory for surrounding non-stressful material can be selective. One theory which can shed light on the mechanisms behind this selectivity is the behavioural tagging hypothesis (Ballarini et al., 2009).

1.3 The behavioural tagging hypothesis

The behavioural tagging hypothesis (BTH) provides a framework for understanding the mechanisms that may be behind this selectivity. According to the BTH, a weak memory trace can be consolidated into long term memory, provided that a subsequent significant event occurs within a critical time window (Ballarini et al., 2009). Thus, a strong learning event, such as navigating a new environment, can have lingering prospective and retrospective effects on the memory for events that occurred around the time of this learning experience (Fig. 1.2). Importantly, the BTH states that the strong learning event may occur either before or after the weak learning experience (Ballarini et al., 2009; Viola et al., 2024). In

other words, the significant event can exert its memory enhancements effects for the weak learning trace both prospectively, when the weak learning experience occurs after the significant event, and retrospectively, when the weak learning experience occurs before the significant event. The mechanism that allows for this bidirectional effect can be understood through the molecular process that is hypothesised to underlie behavioural tagging – synaptic tag-and capture (Redondo & Morris, 2011).

Synaptic tag-and-capture explains how, in a continuous stream of incoming information, where events overlap and neurons are facing multiple encoding and consolidation cascades at different stages, the brain is able to distinguish which synapses need to be strengthened for future adaptation (Frey & Morris, 1997). Experiments showed that late long-term potentiation (l-LTP), a canonical cellular mechanism of long term memory formation, occurs via synaptic tag-and-capture (Frey & Morris, 1997). Synaptic tag-and-capture involves four stages – first, weak stimulation of a neuron produces early long-term potentiation (e-LTP) and places a tag at the stimulated synapse, which remains for up to three hours (Frey & Morris, 1997). Then, plasticity related products (PRPs) needed for LTP are synthesised and distributed throughout the brain (Frey & Morris, 1997; Redondo & Morris, 2011). Only the tagged synapses can capture these proteins, allowing stabilisation and strengthening of the synapse (Frey & Morris, 1997; Redondo & Morris, 2011).

The strong event that triggers the release of PRPs is often characterized by elevated arousal and novelty, activating ventral tegmental area and LC, and recruiting dopaminergic and adrenergic pathways (Dunsmoor et al., 2022; Moncada et al., 2015). Tags on the other hand are believed to involve the interplay of multiple molecules, and constitutes a state of the synapse involving structural changes to the dendrite (Moncada et al., 2015; Redondo & Morris, 2011).

A key feature of synaptic tag-and-capture is the idea that events occurring at different synapses can influence each other – in other words, the event that trig-

gers the tagging process does not have to be the same as the event triggering the synthesis and distribution of PRPs (Viola et al., 2024). Moreover, more recent revisions of synaptic tag-and-capture have described distinguishable roles of functional and structural plasticity (Redondo & Morris, 2011). Specifically, the weak stimulation that results in functional plasticity, in part via e-LTP and the insertion of α-amino-3-hydroxyl-5-methyl-4-isoxazole-propionate (AMPA) receptors, is separable from structural plasticity, which takes the form of changes to the cytoskeletal configuration of the dendrite (Redondo & Morris, 2011). It is precisely these transient structural changes to the dendrite that stand out as the state that constitutes a synaptic tag (Redondo & Morris, 2011).

The behavioural tagging hypothesis extends the idea of synaptic tag-and-capture by suggesting that a weak learning event can be consolidated into long-term memory if there is a second, stronger experience in the temporal periphery of the learning event (Ballarini et al., 2009). When spatial object recognition or contextual fear conditioning tasks were combined with exploration of an open field within a restricted time window, rodents formed a long-term memory for the object or fear context (Ballarini et al., 2009). In the same study, when the open field exploration was completed in a familiar environment, there was no long-term memory formation of the objects or fear context. In humans, a novel school lesson within one hour of a literary or visual learning experience enhanced the memory of the learned material, compared to when the lesson was not novel (Ballarini et al., 2013). This enhancement did not occur when the novel lesson took place four hours before or after the weak learning, suggesting that there is a critical time window in which the tag-and-capture must occur. With longer time delays between the strong event and weak learning, the tag deteriorates, and the action of PRPs fade (Ballarini et al., 2009; Redondo & Morris, 2011).

These findings underscore a crucial component of the BTH presented above – namely, the symmetry of the tag-and-capture process. The memory for the objects was improved regardless whether the significant learning experience occurred

before or after object encoding (Ballarini et al., 2009). This means that a significant experience influences the memory not only prospectively for episodic memories encoded after the experience, but also retrospectively, for episodic memories encoded before the experience. This has crucial implications for understanding how strong experiences – like experiencing a stressful event – can influence our memory for non-stress related information that was encoded before or after the stressful event.

Stress constitutes a strong learning event, and has also been explored as the driver of PRP synthesis in the context of behavioural tagging. When rodents were exposed to an acute stressor in the form of an elevated platform, a previously weakly learned spatial memory was strengthened, (Lopes da Cunha et al., 2019). This effect was also seen with an inhibitory avoidance task as the weak learning experience (Lopes da Cunha et al., 2022). In both studies, the memory enhancement was dependent on the activity of mineralocorticoid and glucocorticoid receptors, highlighting the role of corticosteroid hormones in the effect of stress on memory (Lopes da Cunha et al., 2019, 2022). Meanwhile, in humans, exam stress showed both improving and impairing effects on the memory for unrelated information in high school students (Lopes da Cunha et al., 2018).

Thus, strong learning events, such as stress, do not uniformly affect the memory for previously learned material. This selectivity has been previously explained via concurrent effects of noradrenaline and glutamate. According to this explanation, noradrenaline has dual effects - it suppresses activity at weakly stimulated sites, but amplifies strongly stimulated sites (Dunsmoor et al., 2022). Thus, dopamine and noradrenaline, when released by a strong learning event, mainly promote the memory for strong sensory input events (Dunsmoor et al., 2022). Such activity dependent memory enhancement is also demonstrated in behavioural findings. Images that engaged the medial temporal lobe more were more likely to be remembered later, if the participant had showed a strong cortisol response to a physical stressor that occurred after image encoding (Ritchey et

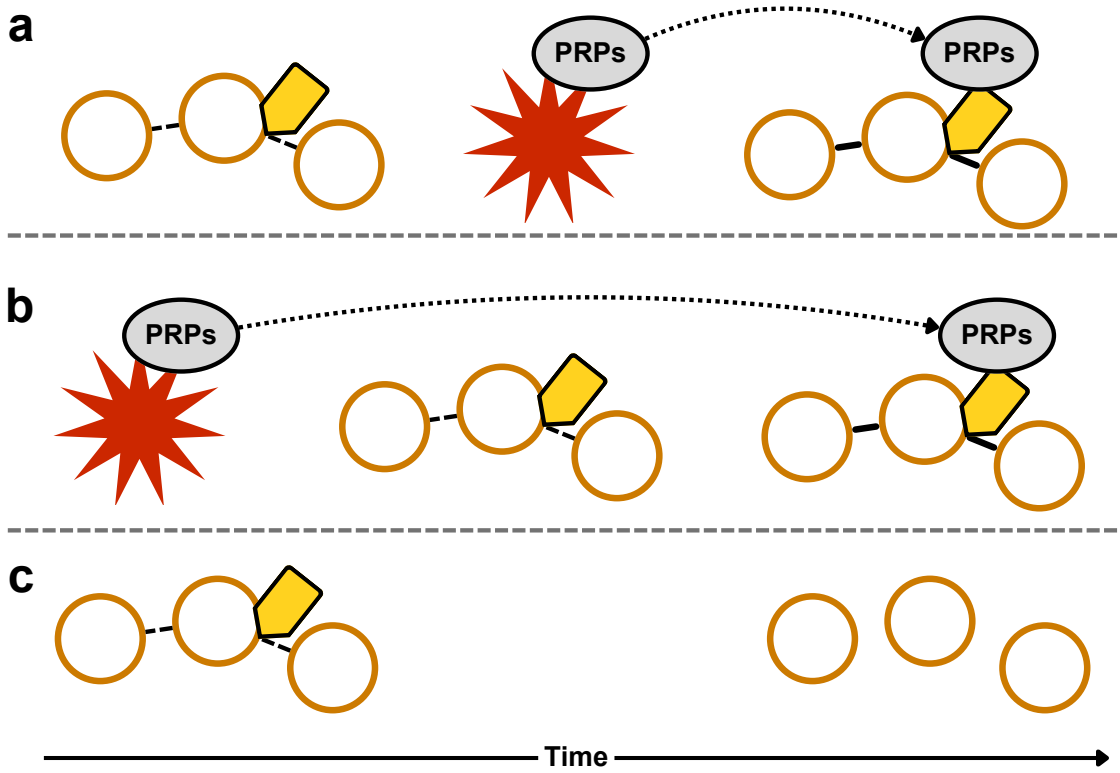

Figure 1.2 Schematic of the behavioural tagging hypothesis. Weak encoding of learning material initiates early long-term potentiation (e-LTP) processes, and results in the placement of a learning tag. If there is a subsequent strong learning experience, such as stress, plasticity related products (PRPs) are released. These PRPs can then be captured by the learning tag, which results in consolidation of the engram into long term memory. This process is symmetric, with the weak learning occurring either before (a) or after (b) the strong learning experience. If there is weak encoding with a learning tag, but no release of PRPs by a significant encounter within the temporal periphery, the tag fades and the memory is not consolidated via late long-term potentiation (l-LTP) (c).

al., 2017). Similarly, neutral information surrounding emotionally arousing material is remembered better if the neutral information receives sufficient attention at encoding (Knight & Mather, 2009). Some findings also indicate that conceptual overlap between an emotionally relevant event and neutral information results in memory enhancement for the neutral information (Dunsmoor et al., 2022; cf. Kalbe and Schwabe, 2022a). Animal research further suggests that the memory enhancing effects of stress depend on the strength of the learning experience – only experiences which are initially weakly encoded can be enhanced by the stressful experience, possibly due to interfering consolidation pathways (Lopes

da Cunha et al., 2019, 2022).

Thus, the behavioural tagging hypothesis provides a framework useful for understanding the carry-over effects of a significant event, such as an acute stressor, on the memory for experiences in the temporal periphery. Importantly, it provides a framework to understand how such selectivity may occur both prospectively, when stress occurs before the weak learning, and retrospectively, when stress occurs after the weak learning event. Furthermore, given that tag setting is a selective process, there should be distinguishable factors that determine whether it will take place or not. The following sections outline two possible mechanisms that may drive tagging.

1.4 Tag setting via cueing a significant experience

One potential driver of this kind of selective enhancement of memory is overlap in the neural representation of the memories of two experiences. The neural representation of a memory is generally believed to consist of an engram – a constellation of neurons that become interconnected in a memory-specific fashion (Tonegawa et al., 2015). Engrams representing two separate memories may become connected depending on when the events were encoded (Josselyn & Frankland, 2018). Encoding two memories close in time results in a higher degree of engram overlap in the mouse hippocampus and lateral amygdala (Cai et al., 2016; Rashid et al., 2016). Similarly, in humans, partially overlapping pieces of information were better integrated, and shared more similar neural representations, when they were learned closer together in time (Zeithamova & Preston, 2017).

Aside from time-dependent overlap, conceptual overlap might also contribute to linking memories together. Recalling a memory should reactivate the engram that represents it, returning it to a labile state (Josselyn & Frankland, 2018). A recently proposed cuing mechanism proposes that this reactivation allows for new encoding material to become connected to the already established memory trace (Josselyn & Frankland, 2018). This way, temporally separated pieces of informa-

tion can become linked through partial overlap of the engrams representing each memory. This linking has been shown via acquired equivalence tasks, whereby partially overlapping associations are learned (A-X, B-X, A-Y; Shohamy and Wagner, 2008). Here, the implicit association of two stimuli that were only connected indirectly (B-Y) indicated successful learning of the pairing that was not encoded explicitly. This kind of memory generalization is supported by the hippocampus and midbrain activity (Shohamy & Wagner, 2008). Applying an adjacent concept to emotional learning, Zhu et al. (2022) used a sensory preconditioning paradigm to show memory enhancement via overlapping neural representation and functional reorganization. Face-object associations were learned, followed by sensory conditioning whereby some faces were coupled with an aversive scream (Zhu et al., 2022). This emotional learning resulted in improved memory for the face-object pair, and this memory enhancement was directly related to the reinstatement of the face-object neural pattern during sensory conditioning. These findings suggest that reactivating a neutral memory during emotional learning enhances not only the memory for the emotional material, but also neutral material that was previously associated with it (Zhu et al., 2022). Thus, the question arises whether a cue to a stressful experience works as a selective tagging mechanism, resulting in engram overlap of the stress memory and the new experience, and prioritized consolidation of the new experience into long-term memory.

Cuing a stressful experience may also enhance the memory for subsequent neutral information, regardless whether there is an explicit tie to the stressor. Emotional word encoding has been shown to enhance the recall of subsequent words, regardless of semantic relevance to the emotional words (McClay et al., 2025). The same study also found a retrospective memory enhancement for neutral words seen before the emotional words, but only when the neutral words were semantically unrelated to the emotional word (McClay et al., 2025). Whether a similar effect would be seen at longer time scales, where the emotional relevance of some encoded material is only established in a separate episode, is completely

unknown.

Implicit to the question of whether the reactivation of a stressful memory places a transient learning tag on new, non-stress related information, is the assumption that the stressful episode should be well represented in memory. If the stressful experience is not distinctly remembered, then no memory benefit afforded to the stressful experience can transfer to the neutral information. Since stressful events result in memory enhancement through joint interactions of glucocorticoids and noradrenergic arousal (Roozendaal et al., 2006), the memory enhancement for surrounding neutral information that cues the stressful experience should also be directly linked to the cortisol response.

1.5 Tagging via prediction error

Another possible mechanism behind selective tag-setting is through prediction errors (PE). PEs occur when we experience something that deviates from what the brain predicted would occur. Such deviance between expectations and reality results in an updating of neural representations to better match reality in the future (Nolden et al., 2024). Indeed, learning models, such as the Rescorla-Wagner model, generally formalize error-based learning as the updating of associative strength in order to minimize mismatch between expectations and reality (Rescorla & Wagner, 1972). Error-based learning is considered adaptive because it ensures efficient resource allocation: predictions are generated continuously, and only deviations from those predictions are propagated to update higher-level representations, whereas accurate predictions require no model revision (Clark, 2013). PEs thus constitute a learning signal that can occur across neural hierarchies, to the extent that the brain has even been described as a prediction or inference machine (Clark, 2013; Friston, 2010).

The predictive brain hypothesis reversed the classical view of the brain as a passive, stimulus-driven system (Clark, 2014). Specifically, instead of bottom-up signals being continuously used to create a mental model of the world, the pre-

dictive brain hypothesis proposes that a top-down model is first projected onto the world, and sensory input is continuously compared against predictions made by this model (Clark, 2014). Across cortical hierarchies, there exist representation units and error units (Clark, 2014). At each layer of the hierarchy, an incoming sensory signal is compared to top-down predictions generated in the layer above, and only the mismatch between an incoming signal and top-down prediction is propagated to higher layers for further processing. The brain thus functions as a generative model, whereby each layer is able to create the data that may be conveyed from the layer below (Clark, 2013, 2014). Importantly, the model continuously improves based on error feedback from lower layers – successful predictions can increase the posterior probability of the model, and thus the system keeps improving its predictive models based on the continuous accumulation of evidence (Clark, 2013, 2014).

Aligned with the predictive brain hypothesis is the free-energy principle and the Bayesian brain hypothesis, according to which the brain constantly tries to optimize probabilistic interpretations of the underlying causes of sensory input (Friston, 2010). Aiming for a state of homeostasis, the brain constantly works to minimize the amount of free energy – information that is not aligned with whatever model is currently being applied (Friston, 2010). Formally, minimizing the amount of free energy amounts to minimizing PE across the neural hierarchies (Friston, 2010). Inherent to the Bayesian brain hypothesis and modern formulations of Hebbian neuroplasticity is the assumption that PEs arise when sensory input conflicts with top-down generated predictions, and this results in both updated beliefs about the current state of the world as well as updating the predictive model itself (Friston, 2010). Thus, PEs arise not only to facilitate perception and an understanding of the current state of the world – they also allow us to learn and improve the model we have of the world itself. In short, the brain keeps what helps it guess better.

On a neural level, unsigned (valence-independent) and signed (reward- and

punishment-related) PEs recruit distinct neural pathways, with unsigned PEs specifically recruiting the LC-NE system (Rouhani, Niv, et al., 2023). Alongside noradrenaline, the LC also co-releases dopamine, which may benefit memory making mechanisms via enhancement of hippocampal plasticity (Rouhani, Niv, et al., 2023). PE fosters stronger reinstatement of representational patterns of memory in the hippocampus, suggesting that memory traces become more stable when they are associated with PE (Boeltzig et al., 2025). The medial temporal lobe generally supports the creation of new, distinct memories when PE is high (Nolden et al., 2024). Neural state transitions also seem to be triggered by PEs, in both the medial prefrontal cortex, V1 and precuneus (Antony et al., 2021). PEs operate across multiple levels of the cortical hierarchy, extending from sensory deviations to more abstract violations encoded in higher-order areas such as the dorsolateral prefrontal cortex (Den Ouden et al., 2012).

PEs have frequently been found to enhance memory (Antony et al., 2021; Bein et al., 2021; Boeltzig et al., 2025; Kalbe & Schwabe, 2020; Rouhani, Niv, et al., 2023). However, some studies suggest a more complex relationship between these variables, with some findings failing to find a memory benefit for unexpected information, or conversely, finding a memory benefit for schema-congruent information (Huang et al., 2023; Turan et al., 2025). The Schema-Linked Interaction Between Medial Prefrontal and Medial Temporal Regions framework (SLIMM) proposes that PEs shape memory in a U-shaped manner, based on the degree of semantic overlap between the encountered stimuli (Van Kesteren et al., 2012). Schema congruency is detected by the medial prefrontal cortex, and this strengthens existing neocortical connections and fosters a strong schema-based memory. Schema incongruency on the other hand occurs when there are no established connections to support schema-based memory strengthening, resulting in disinhibition of the medial temporal lobe and the encoding of a new memory trace (Van Kesteren et al., 2012). The worst memory outcome occurs for moderate schema violations – here, the encountered information cannot be

integrated into existing memory representations, but it also does not allow for full disinhibition of the medial temporal lobe nor the creation of a new, separate memory trace (Van Kesteren et al., 2012). Whereas some studies have found evidence supporting this U-shaped relationship of PE and memory (Boeltzig et al., 2025; Greve et al., 2019), others report an inverted U-shaped pattern, suggesting that moderate—but not low or high—PE most strongly benefits item memory (Ortiz-Tudela et al., 2023).

To further complicate matters, the type of PE may play a key role in determining whether memory enhancement will take place. Specifically, only PEs which target the overall meaning of an encoded episode benefit memory, while surface level deviations from predictions are forgotten (Liedtke et al., 2025). Accordingly, while PE also correlates with elevated arousal in form of skin conductance and pupil size (Antony et al., 2021; Stemerding et al., 2022), PE driven memory enhancements seem to operate independently of the arousal response (Kalbe & Schwabe, 2020). These dynamic findings in the effect that PE has on memory may be due to the task-relevance of the surprising information – only task-relevant PEs should benefit memory (Nolden et al., 2024). Accordingly, only information which is useful for updating model predictions should be encoded successfully (Ortiz-Tudela et al., 2023).

The dynamic findings pertaining to the effect of PE on memory thus suggests that other variables may be at play. Specifically, how PEs may interact with arousal and affective learning remains an open question (Rouhani, Niv, et al., 2023). Given the behavioural tagging account, one perspective is that learning through PEs can be affected by the experience of an adjacent, unrelated event. Thus, events may be tagged by a moderate PE, and later captured by PRPs released by a significant encounter.

1.6 Rationale of the thesis

As discussed above, stress influences memory in a non-universal manner, with only some details being enhanced. For instance, studies have found enhanced memory for emotional material in the periphery of a stressor, as well as for the central details of stressful experiences (see e.g. Bierbrauer et al., 2021; Kensinger, 2009). Based on the free energy principle, such selectivity is useful as memory is prioritized specifically for elements that are adaptive for future success, leaving resources available for more accurate modeling of the ongoing situation (Clark, 2013; Friston, 2010). According to the sparse coding framework, a strategy of using a minimal amount of active neurons at a time to encode relevant information is adaptive as it conserves energy and makes for efficient encoding that can follow local, Hebbian learning rules (Olshausen & Field, 2004). From an energy conservation principle, under stress, such sparse coding that prioritises select information may be even more advantageous, as the stress response is activated and resources are directed towards mobilizing the autonomic and endocrine stress responses. Further, if the general purpose of the brain is to use a large chunk of its resources to generate top-down predictions that model the environment as accurately as possible (Clark, 2013; Friston, 2010), in coping with a stressor, model updating may benefit from efficient learning of select pieces of information that are of central relevance to the situation at hand. Specifically, cuing a stressful experience may send a signal that subsequent information is of central relevance, and thus should be prioritized. Further, PEs propagating across levels in the cortical hierarchy may be preferentially encoded as they are processed to minimize free energy (Friston, 2010). Stress may further encourage this process in order to optimize resource use when activating the stress response and encoding relevant information.

Research on the impact of stress on the memory for surrounding neutral material reveals a nuanced picture with several mechanism contributing to the push-

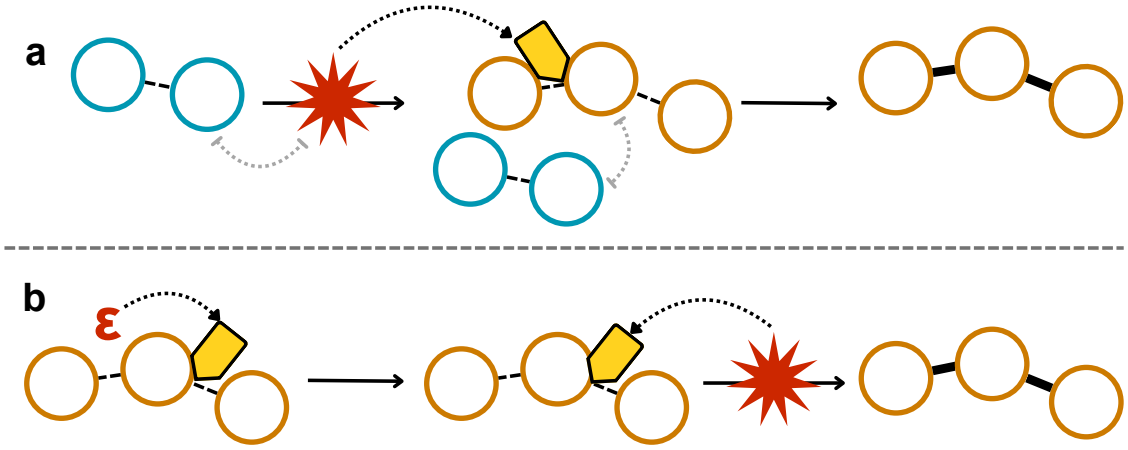

Figure 1.3 Schematic description of the hypothesis, proposing cuing a stressful experience and prediction errors (PEs) as tagging mechanisms. If newly encoded information involves a cue to a significant encounter, this places a learning tag at relevant synapses (a). This learning tag is able to capture plasticity related products (PRPs) released by a strong learning event in the temporal periphery. This results in the consolidation of the weak memory trace into long-term memory. Conversely, a tag may be placed at relevant synapses if a weakly encoded event is associated with a PE (ϵ) (b). Similar to the cuing process, this tag would then be able to capture PRPs released by the significant encounter, thus consolidating the surprising information into long-term memory.

and-pull of memory enhancement. Through the framework of the BTH, we identified two mechanisms that may drive this selectivity, specifically via setting the learning tag – cuing an emotionally relevant experience, and PEs (Fig. 1.3). Tag setting through one of these mechanisms would leave a transient learning trace in the form of a learning tag, that could then be captured by PRPs released during a significant experience, namely, a stressful encounter. Thus, we hypothesise that stress can selectively enhance the memory for non-stressor related material encoded around the time of the stressor.

Study I focused on the proposed cuing mechanism – here, we presented participants with cues to various contexts, one of which was the same context in which the participant experienced a psychosocial stressor. Here, we expected that cuing an emotionally relevant experience temporarily reactivates the memory trace for that experience, which allows for the memory enhancement afforded to the stressful experience to be transferred to newly encoded, neutral information. We thus expected that a stressful context cue triggers the placement of a tag

on subsequently encoded neutral information, which can be captured by PRPs released by the stressor.

Studies IIa and IIb in turn focused on tagging via PE. Participants were presented with images belonging to various categories and rated how expected each image was. The images were ordered in a manner designed to trigger varying levels of PE. We expected that experiencing a stressful encounter before or after encoding would selectively enhance the memory for images rated as more unexpected. In study IIb we further investigated the neural markers behind tag setting by PE.

2. Stress-driven memory enhancement of surrounding information via cuing

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2.1 Study I

Above, we discussed the the selective effect of stress on the memory for neutral, peri-stress information, and identified two possible mechanisms driving this selectivity. In this first study, we investigated one of these potential mechanisms. Specifically, we tested whether cueing a stressful event during the encoding of neutral information modulates the memory for this information. We reasoned that cueing the stressful context would result in reactivation of the stressful experience, thus extending the memory enhancement for the stressful experience to unrelated, neutral information. Therefore, images that were cued by a stressor context should be better remembered than images cued by a neutral context. We further hypothesized that a stressful episode would influence the memory for surrounding events only if this episode itself is distinctly represented in memory. We predicted that this effect would depend on the individual glucocorticoid response to the stressor. For information encoded before the stressor, we expected a stress-related memory enhancement based on the known beneficial effects of

stress and glucocorticoids on consolidation (Cahill et al., 2003; Roozendaal et al., 2006; Shields et al., 2017). For information encoded after the stressor, however, we predicted a stress-related memory impairment, due to the potential competition with the consolidation of the stressful event itself.

2.2 Methods

2.2.1 Participants and experimental design

One-hundred twenty-seven healthy volunteers participated in the experiment (68 female, mean age = 24.09 years, SD = 3.62 years). Participants were screened in a standardized interview for the following exclusion criteria: lifetime history or current neurological or psychological disorders, hormonal contraceptive use, or medication intake. Only nonsmokers were included, and women were not tested during their periods, because these factors are known to influence cortisol concentrations. The sample size was based on a power analysis using G-Power (version 3.1.9.7) assuming a small effect size of partial η^2 = 0.02, and a power of 0.85. We accounted for a dropout rate of $\sim$15%. Five participants were excluded due to being extreme outliers in the recognition memory task about material encoded before or after the treatment (> 3 SD from the mean, or false alarm-rate > hit-rate, n = 4) or due to experimenter error (n = 1), thus resulting in a final sample size of 122 participants (66 female). In the free recall task, one further outlier was identified and excluded. In the analysis on memory for the stressful episode, two further participants were identified as outliers in recognition memory performance (> 3 SD from mean) and were excluded from the subsequent analysis (stress: n = 59, control: n = 61). Based on the same cutoff, one participant was excluded from the free recall data analysis (stress: n = 60, control: n = 61). All participants provided written informed consent before the beginning of the experiment, and the study was approved by the local ethics committee of the University of Hamburg. Participants received a moderate monetary compensation for their participation.

In a between-subjects design, participants were pseudorandomly assigned to a stress (n = 61; 32 female, age: M = 24.00 years, SD = 4.21 years; 29 male, age: M = 23.90 years, SD = 3.27 years) or control group (n = 61; 34 female, age: M = 23.30 years, SD = 2.72 years; 27 male, age: M = 24.40 years, SD = 3.20 years) to ensure a comparable distribution of men and women in both conditions.

2.2.2 Experimental procedure and tasks

The experiment took place over 2 days, separated by approximately 24 h. Testing took place between 13.00 and 19.00 in order to control for the diurnal rhythm of cortisol. The experiment consisted of four parts: the context familiarization task, pretreatment encoding task, stress (or control) episode, and posttreatment encoding task. All tasks were programmed in Matlab (version R2019b, 9.7.0.1319299) with Psychtoolbox (Brainard, 1997; version 3.0.16).

Day 1: context familiarization task

At arrival on day 1, we measured salivary cortisol using a Salivette (Sarstedt), blood pressure (OMRON M500, OMRON Healthcare Europe), mood (Mehrdimensionale Befindlicheitsfragebogen [MDBF]; Steyer et al., 1997) and state anxiety (State-Trait Anxiety Inventory-State [STAI-S]; Laux, 1981) using standardized questionnaires. We also measured the number of hours that participants slept the night before the experiment.

The participants then completed the context familiarization task, which served to familiarize participants with all context images that were used as cues in the subsequent encoding phases in order to prevent that the memory cueing was confounded by context familiarity. On each trial of the context familiarization task, participants saw an image of a context (i.e., room) for 3 sec on a computer screen. There were ten different context images. Each was presented six times during the familiarization task, with the order of the context images being fully randomized. The ten context images belonged to one of three types of context

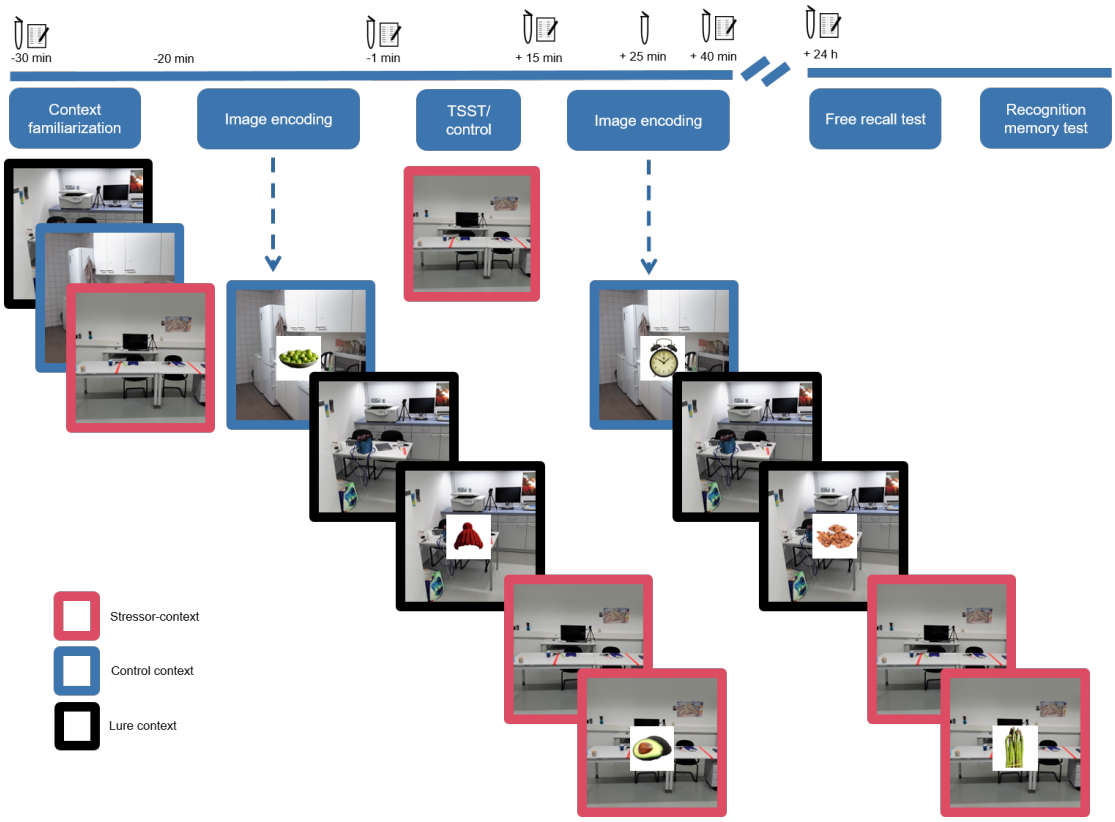

Figure 2.1 Experimental procedure. On day 1, participants first saw images of several rooms that would later be cued as contexts in the image encoding task. Participants then completed an image encoding task before and after a stressful encounter, or a control treatment. In the image encoding tasks, we first cued a context that was either the same room where the stressful encounter took place (stressor context), a lure context that looked similar to the stressor context, or an unrelated control room (e.g., a kitchen); indicated with colored frames for illustrative purposes. The participants were presented with 320 images in total (160 before the stressor and 160 thereafter). We also collected questionnaires and salivary cortisol throughout day 1 to track subjective and physiological stress responses. On day 2, 24 h later, participants completed free recall and recognition memory tasks for the encoded images as well as the stressful/control encounter.

cues: treatment context (n = 1), lure context (n = 1), and control contexts (n = 8). The treatment context cue was an image of the room where the stress (or control) manipulation would take place later. The lure context was included in order to control for the possibility that the treatment room was remembered better simply due to its distinctiveness. The lure room looked similar to the treatment room: the furniture set-up and objects in the room were similar, as well as the angle from which the picture was taken. Thus, this lure room would reference a context that was visually similar to the treatment room, but was not associated with the treat-

ment itself. Importantly, it was counterbalanced across participants and groups whether the stress (or control) manipulation would take place in one or the other context; that is, one room served as treatment context for some participants but as lure context for others (and vice versa). This allowed us to test memory cueing effects that were independent of the specific room cue but solely related to the fact that the experimental treatment took place in that room or not. The control rooms were eight photographs of rooms or places that were independent of the treatment or lure rooms. These were photographs of, for example, a supermarket aisle, a meeting room, or a gym. We presented control images in 80% of the trials and the stress context image in only 10% of the trials in order to prevent habituation to the stressor-context cue.

Day 1: Pretreatment encoding session

Immediately after the context familiarization task, participants completed the pretreatment encoding task. The stimuli in this task consisted of 160 trial-unique images of neutral items. The images were sourced from a variety of online databases (Brodeur et al., 2010; Hovhannisyan et al., 2020). At the start of each trial, one of the context cues was presented for 3 sec such that it covered the whole screen. Then, the stimulus image was presented on top of the context cue for 2 sec. In order to control for participants' attention and promote deeper encoding, participants were instructed to press a button if the item fits into a shoebox. The size of the stimulus image was 30% of the screen height. Thus, the context cue was still visible in the background during the image presentation. The trials were separated by a fixation cross presented for 2.5–3 sec. This trial structure allowed us to cue a specific context—which was linked to the subsequent treatment or not—at each trial, and then present unrelated information while continuing to cue the context.

The room cue presentation was pseudorandomized: the encoding session was broken into ten blocks of 16 trials, within which the order of the context cues was

randomized. Each room cue was presented once per block. The same context cue was never shown twice in a row.

Participants were instructed to memorize the stimulus images, as their memory for these images would be tested on the next day, alongside their memory for the room cue that was presented during that stimulus image. The pretreatment encoding task lasted ∼21 min. During encoding, we measured pupil dilation (SMI RED250, SensoMotoric Instruments), skin conductance response (SCR), and electrocardiography (BIOPAC MP150, BIOPAC Systems Inc.). These physiological measures are not analyzed here and thus will not be reported further.

Day 1: Stress and control manipulation

Immediately after the pretreatment encoding task, participants gave a second saliva sample, we measured blood pressure and pulse (OMRON M500) and the participants completed the mood questionnaire (MDBF) again. Participants were then escorted to a different room where they underwent either the stress or control treatment.

To induce stress in half of the participants, we used the TSST (Kirschbaum et al., 1993). In the TSST, the participant is brought into a room with a nonreinforcing panel of two experimenters wearing lab coats. The participant is told to imagine they are interviewing for their dream job, and that the panel will evaluate their performance. The participant is videotaped and can see themselves on a large screen. The TSST consists of three parts: (1) a 3 min preparation phase, during which the participant can make some notes about the upcoming speaking task, (2) a 5 min free speech, during which the participant has to argue why they are the ideal candidate for their dream job, and (3) a 5 min mental arithmetic task, during which the participant has to count as fast as possible backward from 2043 in steps of 17. We measured blood pressure and pulse twice during the mental arithmetic task (Dinamap, Critikon).

The remaining half of the participants took part in the control treatment. The

control treatment is structured similarly to the TSST, but the panel acts in a friendly manner and does not wear lab coats. The participant is not being videotaped and the speaking task is replaced with an informal discussion with the panel, during which the participant can freely choose a topic (e.g., a book they like). The mental arithmetic task is replaced with a counting game played together with the panel. Blood pressure and pulse were measured twice during the counting game.

As described above, the TSST or control treatment took place in one of two rooms (room A/room B), in order to rule out a confounding of the fact that the treatment took place in that room and a potentially different memorability of that room in the mnemonic context cueing.

The TSST and control manipulation were enriched by placing several objects in the room where the treatment took place. The panel members interacted with some of the objects (central objects), while others remained in the periphery (peripheral objects). Central objects included, for example, a cup that one of the panel members drank from, or a stapler that the panel member used to staple some notes together. The peripheral objects included, for example, a poster on the wall, a plant, and an umbrella. In total, there were twelve peripheral and ten central objects. The object interactions were exactly the same in the TSST and control manipulation.

Day 1: Posttreatment encoding session

After the TSST or control manipulation, participants were escorted back to the room where the preencoding task had taken place. Here, they gave a third saliva sample and completed a mood questionnaire (MDBF) as well as a questionnaire assessing the difficulty and stressfulness of the TSST or control manipulation. We also measured blood pressure and pulse (OMRON M500).

About 5 min after the end of the TSST (or control manipulation), participants completed the posttreatment encoding task. This task was structurally identical to the encoding task that took place before the treatment, but all stimulus im-

ages were new. During the posttreatment encoding session, we obtained a fourth saliva sample, because salivary cortisol was expected to peak ∼25 min after stressor onset.

After completing the posttreatment encoding task, about 45 min after treatment onset, participants gave a fifth saliva sample and completed a mood questionnaire, and we measured blood pressure and pulse (OMRON M500).

Day 2: Free recall test for images

Approximately 24 h later, participants returned for memory testing. Participants first completed mood (MDBF), sleep, and anxiety questionnaires (STAI-S), gave a final saliva sample, and we measured blood pressure and pulse (OMRON M500) to rule out that groups differed in their stress level before memory testing.

First, we assessed participants' memory for the encoded pictures in a free recall test. In this task, participants were given ten minutes to name all item images they remembered seeing during the encoding sessions on day 1. The participants were instructed to verbally list all images they remembered, while the responses were written down by the experimenter and audio-recorded. After completing data collection, the free recall data was analyzed by identifying all mentioned images in the pretreatment and posttreatment encoding tasks. If the mentioned image had not been presented, it was recorded as a false alarm. If the image was not clearly identifiable, it was recorded as a miss and was not further analyzed.

Day 2: Recognition test for images

Next, we assessed participants' recognition memory of the encoded images. Here, participants saw all 320 images that were presented on day 1 ("old") as well as 160 new ones in randomized order. Participants were asked to indicate with the computer mouse for each image whether the image was old or new. They could select between two levels of certainty (rather old/definitely old/rather new/definitely new). If the participant selected either "old" answer, they were shown three con-

text cues and asked to select which one had been presented while the image was shown. For old images (i.e., hits), two additional context cues were randomly shown. For new images (i.e., false alarms), three room cues were randomly shown. This task was self-paced.

Day 2: Free recall and recognition test for the stressful or control episode

We next assessed participants' memory for the TSST or control manipulation via free recall and recognition memory tasks. In the free recall task, participants were asked to verbally recall details about the TSST from the previous day. These details included questions asked by the panel, the rules of the mental arithmetic task and their own performance, and listing the objects that were present in the room. This task was self-paced.

In the recognition memory task, participants were shown images of all central and peripheral objects from the room where they completed the TSST or control treatment, pictures of the panel members' faces, as well as 22 lure objects and two lure panel members' faces. For each image, participants were asked whether it was old or new. They could select between three levels of certainty (maybe, sure, very sure). This task was self-paced. Finally, participants were debriefed about the purposes of the study and paid for their participation.

2.2.3 Salivary cortisol sampling and analysis

As outlined above, salivary cortisol was measured in total six times for each participant, using Salivette collection devices (Sarstedt), and were stored at -18°C until analysis at the end of data collection. The thawed and centrifuged samples were analyzed using chemiluminescence immunoassay (IBL, Tecan). Cortisol was assessed as the fraction of free cortisol (nmol/L). In total, cortisol samples from eight participants (five control) were not analyzed due to insufficient volume of saliva. Seven (six control) participants were missing only one sample. For these participants, we used regression imputation to estimate the salivary cortisol

concentration in the missing sample (Tabachnick & Fidell, 2019). In the remaining 114 complete data sets (58 stress), salivary cortisol was log-transformed and analyzed with a mixed-design ANOVA.

2.2.4 Control variables

We assessed potential group differences in chronic stress and depressive symptoms using standardized questionnaires (TICS, Schulz and Schlotz, 1999; IDAS-II, Wester et al., 2021) at the beginning of the experiment.

2.2.5 Statistical analysis of behavioral data

To verify successful stress induction, mixed-design ANOVAs and Welch's t-tests were used to assess mood, blood pressure, pulse, and cortisol (nmol/L) throughout testing, with condition (stress vs. control), and time point of measurement as predictors. We used Holm–Bonferroni to correct for multiple testing in regressions and Tukey's HSD for pairwise comparisons where more than two groups were compared. When sphericity was violated, the Greenhouse–Geisser correction was applied. We compared stress and control participants in autonomic nervous system activity (blood pressure and pulse) at baseline on both days as well as during the treatment. The blood pressure and pulse measurements from before the stressor, after the stressor, as well as at the end of day 1 were not analyzed, as we were mainly interested in comparing stress vs. control participants' pulse and blood pressure during the treatment, as well as controlling for baseline differences between groups. Due to technical failure, three blood pressure measurements were missing during the stress treatment (n = 2) and at the start of day 2 (n = 1).

Memory performance was analyzed using ANOVAs and generalized linear mixed regression models in RStudio (version 4.2.2), using the afex (Singmann et al., 2024), emmeans (Lenth et al., 2024), rstatix (Kassambara, 2023), lme4 (Bates et al., 2014), and lmerTest (Kuznetsova et al., 2017) packages. To assess memory

for the stressful episode, the participant indicated for each image whether it was old or new and how certain they were (maybe, sure, very sure). We analyzed the data with mixed-design ANOVAs with group (stress vs. control) and object type (central vs. peripheral) as predictors. If memory differed depending on the room (room A/room B), it was included as a covariate. We considered only high confidence hits (sure old/very sure old). We opted to analyze hit-rate rather than d' as we included two kinds of object types (central vs. peripheral) and two encoding phases (pre- vs. posttreatment) in the experiment, and lures could not be separated into one of the two categories. We thus ran a control analysis checking for a difference between stress and control participants' false alarm rate. As response bias still may obscure the memory effects, we further ran control analyses with d' in accordance with the signal detection framework, here subtracting the z-scored false alarm rate for the participant from the z-scored hit-rates for central and peripheral objects (see Supplemental Material). We next computed a hit-rate separately for pretreatment and posttreatment encoding for stress and control participants, and ran a mixed-design ANOVA to analyze the effect of stress on the memory for information encoded before or after the stressor. We again acknowledge the possibility of response bias confounding a possible memory effect here, and thus computed d' with the z-scored false alarm rate for the participant, subsequently subtracting it from the z-scored hit-rate for material encoded before or after the stressor (see Supplemental Material). We also ran linear mixed models to assess the effect of stressor memory and cortisol response on the hit-rate for information encoded before or after the stressor. Finally, we predicted the probability of a hit with cue type (treatment vs. lure vs. control), condition (stress vs. control), phase (pre- vs. posttreatment) and distance to treatment (number of trials from treatment onset and offset, respectively). Room (room A/room B) was included as a covariate to control for potential differences between the treatment rooms. A random intercept was included for each participant. Effect sizes are reported as generalized eta squared (η^2, ANOVAs) and as Cohen's d (d, t-tests).

2.3 Results

2.3.1 Successful stress induction

Subjective, autonomic, and endocrine measures confirmed the successful stress induction by the TSST (Fig. 2.2).

Mixed-design ANOVAs revealed a significant time × condition (stress vs. control) interaction for all mood ratings (all $F > 6.64$, all $p < 0.001$, all $\eta^2 > 0.019$). Follow-up tests using pairwise comparisons showed that immediately after the treatment, participants of the stress group rated their mood as less awake (Supplemental Fig. S1A), calm (Supplemental Fig. S1B), and good (Fig. 2.2A) compared to control participants (all $t(120) > 3.914$, all $p < 0.001$). Furthermore, stressed participants rated their mood as less calm at the end of day 1 ($t(120) = 2.490$, $p = 0.014$). There was no difference in these parameters between the groups at the start of day 1 or before the treatment (all $|t| < 0.965$, all $p > 0.336$; Supplemental Table S1). Moreover, participants in the stress group rated the treatment as significantly more difficult, stressful, and unpleasant than those in the control group (all $F > 91.12$, all $p < 0.001$, all $\eta^2 > 0.435$; Fig. 2.2B).

At the autonomic arousal level, mixed-design ANOVAs revealed significant time × condition interactions for pulse, systolic, and diastolic blood pressure (all $F > 17.56$, all $p < 0.001$, all $\eta^2 > 0.031$; Fig. 2.2C–E). Follow-up pairwise comparisons showed that during the treatment, participants in the stress group had a significantly higher pulse, systolic, and diastolic blood pressure relative to control participants (all $|t| > 2.842$, all $p < 0.006$), while there was no difference between the stress and control groups at the start of day 1 (all $|t| < 1.557$, all $p > 0.122$).

Finally, the exposure to the TSST led also to a significant increase in salivary cortisol. A mixed-design ANOVA showed significant main effects of time point ($F(1.83, 205.09) = 8.75$, $p < 0.001$, $\eta^2 = 0.023$) and condition ($F(1, 112) = 12.52$, $p < 0.001$, $\eta^2 = 0.072$), as well as a significant interaction between these factors ($F(1.83, 205.09) = 16.03$, $p < 0.001$, $\eta^2 = 0.042$; Fig. 2.2F). Follow-up analyses showed

that cortisol was significantly higher in stressed participants, relative to controls, at the offset of the stressful episode, 25 min after the onset of the stressful episode, and at the end of the experiment on day 1, ~40 min after the onset of the stressor (all $|t| > 2.492$, all $p < 0.015$). There were no differences between the conditions at baseline on day 1 or right before the stress/control treatment (both $|t| < 0.695$, both $p > 0.489$).

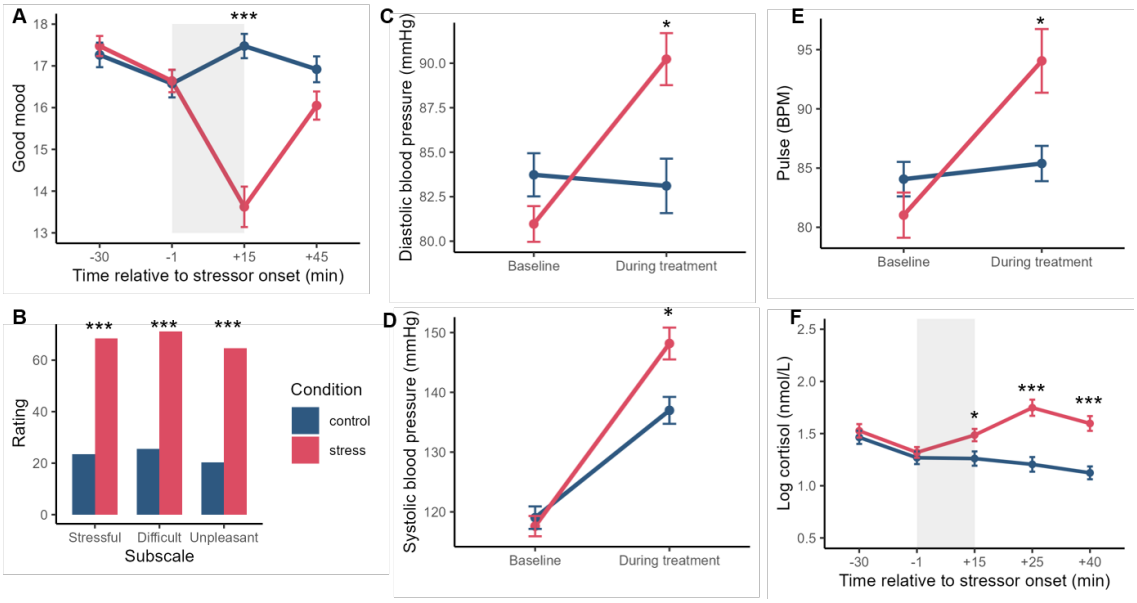

Figure 2.2 Successful stress induction. After the stress versus control treatment, participants in the stress condition rated their mood as significantly worse (A), found the treatment to be more difficult, unpleasant, and stressful (B), and had elevated blood pressure (C,D) and pulse (E). Stressed participants also showed elevated salivary cortisol immediately after, 25 min, and 40 min after treatment onset (F). Error bars represent standard error of the mean (SEM). (*) $p < 0.05$ and (***) $p < 0.001$, for stress compared to control group.

2.3.2 Enhanced memory for the stressful episode

We tested participants' memory for the stressful (vs. control) episode 24 h later. At the time of memory testing, groups did not differ in subjective stress levels (all $|t| < 1.591$, all $p > 0.113$), autonomic arousal (all $|t| < 1.190$, all $p > 0.239$), or salivary cortisol ($t(112) = 0.982$, $p = 0.328$; Supplemental Table S1). In a recognition test, we presented images of central and peripheral objects that were present during the treatment as well as of novel objects. For each image, participants

selected whether the object was present in the treatment room (old) or not (new).

Using a mixed-design ANOVA with condition and object type (central vs. peripheral) as predictors, we found a significant main effect of object type ($F(1, 118) = 401.12$, $p < 0.001$, $\eta^2 = 0.543$), as well as a significant interaction of condition and object type ($F(1, 118) = 8.04$, $p = 0.005$, $\eta^2 = 0.023$, Fig. 2.3A). Pairwise comparisons, however, revealed no significant differences between the stress and control group for central ($t(118) = -1.715$, $p = .089$) or peripheral items ($t(118) = 1.643$, $p = 0.103$). We next computed a centrality bias index for each participant by subtracting the hit-rate for peripheral objects from the hit-rate for central objects and compared stress and control participants using a t-test. The centrality bias reflects the prioritization of memory and was significantly higher in the stress group than in the control group ($t(118) = -2.838$, $p = 0.005$, $d = -0.518$, Fig. 2.3C). The false alarm rate did not differ between groups (stress: $M = 0.106$, $SD = 0.083$; control: $M = 0.120$, $SD = 0.112$; $t(110.890) = 0.756$, $p = 0.451$, $d = 0.138$).

In the next step, we tested for a potential role of the stress-induced cortisol response in stress effects on memory of the treatment episode. To this end, we divided stressed participants into two groups based on their cortisol response to the stressor and compared each to the control group. Participants with an increase in cortisol above 1.5 nmol/L were considered to be high responders ($n = 29$), the rest were considered low responders ($n = 29$) (Miller et al., 2013). A mixed-design ANOVA revealed a significant interaction of responder group and object type ($F(2, 109) = 3.99$, $p = 0.021$, $\eta^2 = 0.026$, Fig. 2.3E), but no follow-up tests were significant (all $|t| < 1.899$, all $p > 0.143$, Tukey-corrected). We then used a one-way ANOVA to test for differences in centrality bias depending on cortisol response (high vs. low vs. control). There was a significant difference in centrality bias depending on the group ($F(2, 109) = 3.99$, $p = 0.021$, $\eta^2 = 0.068$, Fig. 2.3G), and pairwise comparisons indicated that high cortisol responders had a significantly higher centrality bias than control participants ($t(109) = -2.711$, $p = 0.021$, Tukey-corrected), while the differences between high responders and low responders

and between low responders and controls remained nonsignificant (both $|t| < 1.654$, both $p > 0.228$, Tukey-corrected). The lack of a difference between the low responders and controls may however be due to the effect being too small to detect in this comparison (Fig. 2.3G). To test whether autonomic nervous system activity during the stressor influenced the extent of this centrality bias, we computed separate linear models predicting the centrality bias with blood pressure, pulse, and group, as well as the interaction between them. No interactions reach significance (all $|\beta| < 0.003$, all $p > 0.434$), suggesting that the autonomic response was not directly associated with the prioritization of stressor memory.

In addition to the recognition test, participants completed also a free recall test for the stressful (vs. control) episode. In this free recall test, participants were asked to verbally list all objects they remembered seeing in the treatment room. A mixed ANOVA revealed a significant main effect of object type ($F(1, 118) = 176.54$, $p < 0.001$, $\eta^2 = 0.371$) and a significant interaction of condition and object type ($F(1, 118) = 7.36$, $p = 0.008$, $\eta^2 = 0.024$) on free recall performance (Fig. 2.3B). Pairwise comparisons revealed that stressed participants recalled significantly fewer peripheral objects than the control participants ($t(118) = 2.121$, $p = 0.036$). There was no difference between stressed and control participants in free recall memory for central objects ($t(118) = -1.441$, $p = 0.152$). Same as for the recognition test data, we further calculated a centrality bias by subtracting the proportion of recalled peripheral objects from the proportion of recalled central objects. This centrality bias was significantly higher in the stress compared to the control group ($t(117.83) = -2.713$, $p = 0.008$, $d = -0.493$, Fig. 2.3D).

We next tested whether free recall memory depended on the cortisol response (high vs. low vs. control). We found a significant effect of object type ($F(1, 110) = 161.58$, $p < .001$, $\eta^2 = 0.361$) as well as an interaction effect of responder group and object type ($F(2, 110) = 3.62$, $p = 0.030$, $\eta^2 = 0.025$, Fig. 2.3F). Pairwise comparisons, however, showed no significant differences (all $|t| < 1.881$, all $p > 0.149$, Tukey-corrected). A one-way ANOVA comparing centrality bias

depending on cortisol response (high vs. low vs. control) showed an effect of group on centrality bias (F (2, 110) = 3.62, p = 0.030, η^2 = 0.062, Fig. 2.3H). However, none of the follow-up tests approached significance (all $|t| < 2.214$, all $p > 0.073$, Tukey-corrected). To test the impact of ANS activity on centrality bias, we again ran separate linear models for pulse and blood pressure, which did, however, not yield any significant interactions between group and autonomic arousal parameters (all $|\beta| < 0.003$, all $p > 0.153$).

Notably, although groups did not differ in the overall false alarm rate, we re-ran the recognition memory mixed ANOVAs with d' instead of the hit-rate, which left the pattern of results mostly unchanged, yet even showed a significant difference between stressed and control participants for central objects (see Supplemental Material).

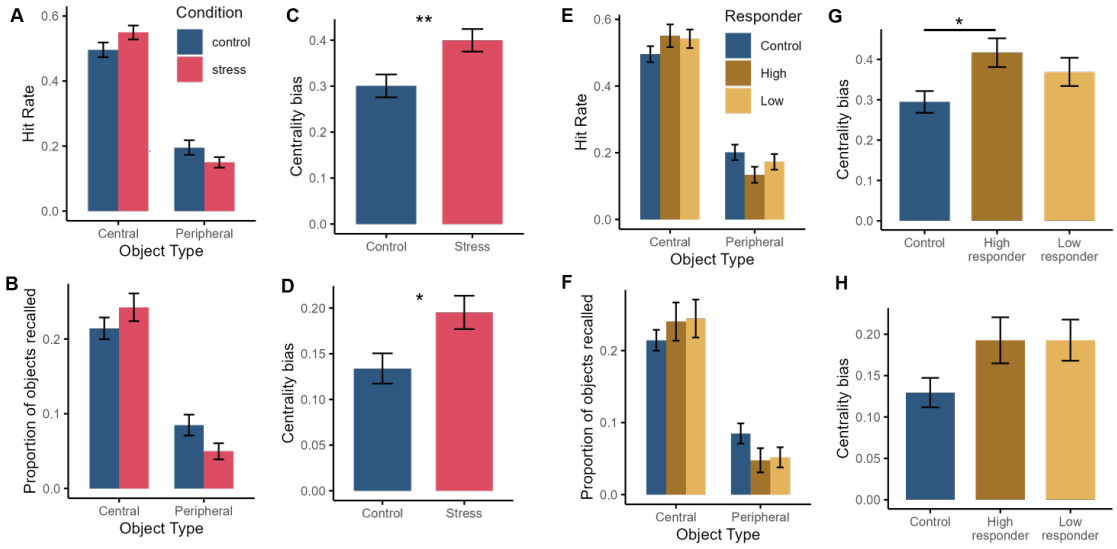

Figure 2.3 Memory for the stressful episode. Participants in the stress condition tended to remember more central and less peripheral objects in a recognition task (A,E) and free recall task (B,F) than those in the control group. There was a significantly higher centrality bias (difference between recognized or recalled central and peripheral objects) in the stress group than in the control group, both in the recognition test (C) and in the free recall test (D). Specifically, stressed participants with a high salivary cortisol response had a significantly higher centrality bias compared to participants in the control group (G). A similar, but nonsignificant, pattern was also shown in the free recall task (H). Error bars represent SEM. (*) $p < 0.05$, (**) $p < 0.01$.

2.3.3 Memory for events surrounding the stressful episode

We next assessed the probability of remembering each individual stimulus image from the pretreatment and posttreatment encoding phases with mixed-design ANOVAs and GLMMs. We first ran a mixed-design ANOVA to analyze the effect of condition and phase (pretreatment vs. posttreatment) on the hit-rate.

This model revealed a significant effect of phase ($F(1, 120) = 87.51$, $p < 0.001$, $\eta^2 = 0.033$) as well as a significant interaction of phase and condition ($F(1, 120) = 15.32$, $p < 0.001$, $\eta^2 = 0.006$). The significant interaction between phase and condition was driven by a memory enhancement for stimuli encoded before the treatment in the stress group. As shown in Fig. 2.4A, participants remembered overall more of the items from the pretreatment than from the posttreatment encoding session, but this difference was more pronounced in the stress group than in the control group (control: $t(120) = 3.847$, $p < 0.001$, stress: $t(120) = 9.383$, $p < 0.001$). However, pairwise comparisons between the stress and control groups for the pretreatment ($t(120) = -1.568$, $p = 0.120$) and posttreatment encoding ($t(120) = 0.093$, $p = 0.926$) sessions separately remained nonsignificant.

We next tested for a potential role of the stress-induced cortisol response in stress effects on the memory for surrounding neutral events. A mixed-design ANOVA revealed a significant effect of phase ($F(1, 111) = 91.55$, $p < 0.001$, $\eta^2 = 0.036$) and a significant interaction of phase and responder group (high vs. low vs. control; $F(2, 111) = 7.99$, $p < 0.001$, $\eta^2 = 0.007$, Fig. 2.4B). Tukey-corrected pairwise comparisons showed that stressed participants with a high cortisol response recognized significantly more stimuli encoded prestress, than the control group ($t(111) = -2.588$, $p = 0.029$). No other pairwise comparisons reached significance (all $|t| < 1.890$, all $p > 0.146$). To test whether autonomic nervous system activity (blood pressure and pulse) during the stressor predicted memory for events surrounding the stressor, we ran separate mixed linear models with condition (stress vs. control), phase (pretreatment vs. posttreatment), and autonomic nervous system activity, as well as an interaction term, as predictors. Only the inter-

action term of group × phase × systolic blood pressure approached significance (β = -0.027, p = 0.059; both other $|\beta| < 0.023$, both other p > 0.086). Follow-up simple slopes analyses assessing the effect of stress at high, moderate, and low systolic blood pressure in the pretreatment or posttreatment revealed no significant differences between the stress and control groups (all $|\beta| < 0.031$, all p > 0.397, Holm–Bonferroni-corrected). The false alarm rate did not differ between conditions (t (111.060) =-0.494, p = 0.622, d = –0.089) or depending on the cortisol response (F (2, 111) = 0.89, p = 0.411, η^2 = 0.016). The pattern of results remained largely unchanged when using d' instead of hit-rate as outcome measure in the recognition memory mixed ANOVAs (see Supplemental Material).

With respect to the potential cueing of memory by the stressor context, we ran a GLMM with phase, condition, and cue type (treatment vs. lure vs. control), as well as distance to treatment as predictors. We found no interaction of condition, cue type, and phase or distance (all $|\beta| < 0.192$, all p > 0.196). The reduced model with two-way interactions of the variables again showed no interaction of cue type and condition (both $|\beta| < 0.022$, both p > 0.771). Moreover, there was no effect of cue type on hit probability (both $|\beta| < 0.100$, both p > 0.118), indicating that images were equally likely to be recognized irrespective of whether they were cued by a stressful context or not. Thus, cueing a stressful context did not affect the memory for subsequent neutral information. We next added the cortisol response (high vs. low vs. control) to the model and repeated the analyses. There was no interaction of cue type, responder group, and phase or distance (all $|\beta| < 0.286$, all p > 0.132). The reduced model with two-way interactions of the variables again showed no interaction of cue type and responder group (all $|\beta| < 0.078$, all p > 0.401). Predicted probabilities are presented in the Supplemental Material (stress vs. control, Supplemental Table S2; high cortisol vs. low cortisol vs. control, Supplemental Table S3).

In addition to the recognition memory task, participants were asked in a free recall task to verbally list all objects they recalled seeing in the pretreatment and

posttreatment encoding tasks. We used GLMMs to analyze the probability of an item being recalled, with the same predictors as for the recognition task. We found no interaction of condition, cue type, and phase or distance (all $|\beta| <$ 0.110, all p > 0.665). The reduced model included two-way interactions and again showed no interaction of cue type and condition (both $|\beta| < 0.197$, both p > 0.148). We next repeated the analyses with the responder group (high vs. low vs. control) as a predictor. Similar to the analyses above, there was no interaction of cue type, responder group, and phase or distance (all $|\beta| < 0.360$, all p > 0.227). The reduced model with two-way interactions of the variables showed a significant interaction of low responders and treatment cue (β = 0.346, p = 0.049). However, follow-up simple slopes analyses showed no significant effects of the response group for treatment cues (all $|\beta| < 0.374$, all p > 0.118). Predicted probabilities are presented in the Supplemental Material (stress vs. control, Supplemental Table S4; high cortisol vs. low cortisol vs. control, Supplemental Table S5).

2.3.4 Memory for events surrounding a stressful episode depends on the memory prioritization for the stressful episode itself

We next analyzed whether memory for the stressful episode itself would moderate the influence of stress on the memory for surrounding events. We reasoned that stress would affect the memory for surrounding events only if there is also a memory prioritization (i.e., centrality bias) for the stressful episode itself. To this end, we ran a linear mixed model with condition (stress vs. control), phase (pretreatment vs. posttreatment), and centrality bias as predictors. We also included state anxiety score as a covariate in order to control for baseline differences in state anxiety on day 2 (see below). We found a significant phase × condition × centrality bias interaction (β = -0.035, p = 0.025). We followed up this significant interaction with simple slopes analyses at 1 SD below and above mean centrality bias. Simple slopes analyses revealed that stress significantly enhanced memory

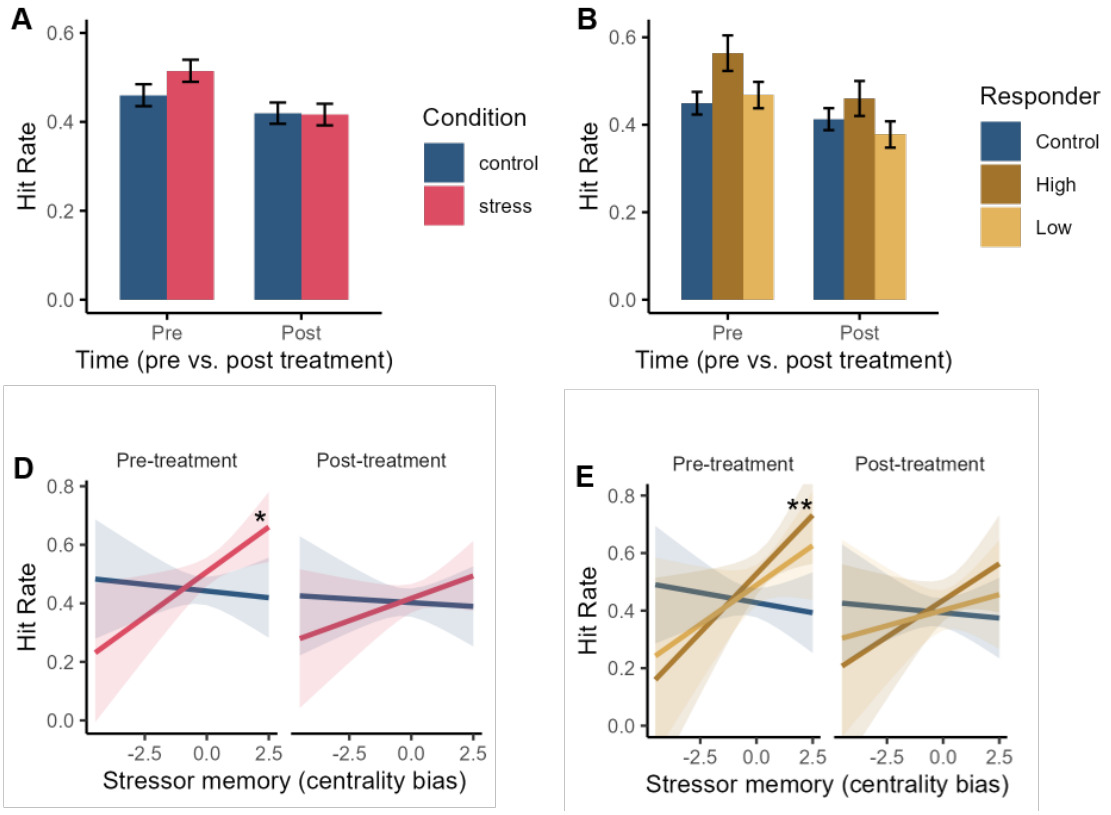

Figure 2.4 Memory for events occurring before or after the stressor or control treatment. Stressed participants tended to remember more objects encoded before the stressor (A), yet this pattern did not reach significance. This was driven by high cortisol responders recognizing more images that were encoded before the stressor (B). When stressed participants showed memory prioritization for the stressful episode, they recognized significantly more images than control participants (C). This was especially the case for stressed participants showing a pronounced cortisol response to the stressor (D). Error bars show SEM. (*) $p < 0.05$, (**) $p < 0.01$.

for items encoded before the treatment, if participants had a high centrality bias, that is, more pronounced memory for central aspects of the stressful episode (β = 0.136, p = 0.036, Fig. 2.4C). The effect of condition was nonsignificant in all other simple slopes analyses (at moderate and low centrality bias levels, in the posttreatment encoding phase, all $|\beta| < 0.066$, all $p > 0.306$; Holm–Bonferroni-corrected).

To zoom into the effect of stress further, we next divided stressed participants into high and low cortisol responders and reran the linear mixed model presented above. Simple slopes analyses revealed that high cortisol responders who also

had a high centrality bias showed a significant memory enhancement for items encoded before the stressor (β = 0.196, p = 0.006). Low cortisol responders also showed this effect at a descriptive level, but did not differ from the control group after Holm–Bonferroni-correction (β = 0.130, p = 0.248, Fig. 2.4D). None of the other simple slopes analyses showed a significant effect of responder group (all $|\beta|$ < 0.102, all p > 0.135). We next ran an exploratory analysis comparing low cortisol responders to high responders, and found no significant difference in their memory performance (all $|\beta|$ < 0.047, all p > 0.340). These findings suggest a memory enhancement for information encoded before the stressor that is dependent on memory prioritization of the stressful episode, but largely independent of the stress-related cortisol response. The pattern of results remained mostly unchanged when using d' instead of hit-rate as outcome measure (see Supplemental Material). In particular, the significant interaction effect between condition, phase, and centrality bias as well as the interaction between cortisol response and phase remained. With d' as the outcome measure, however, the post hoc analysis contrasting stressed and control participants with a high centrality bias for material encoded before the stressor was not significant anymore. However, the difference between high cortisol responders and control participants with a high centrality bias remained.

Lastly, we assessed whether stressor cueing would enhance memory for participants with an elevated centrality bias. In this model, we found no significant interaction of cue type, condition, centrality bias, and encoding phase (both $|\beta|$ < 0.164, both p > 0.295).

2.3.5 Control variables

To control for baseline differences between stressed and control participants, we measured chronic stress, anxiety or depressive symptoms, state anxiety, sleep duration, and quality. Groups did not differ in any of these variables (all |t| < 1.001, all p > 0.301, see Supplemental Table S1) except for state anxiety at the

beginning of experimental day 2 (t (118.51) = -2.370, p = 0.019, d = -0.431). Participants in the stress group had higher state anxiety at the beginning of day 2 than controls, which may be due to different expectations based on the stressful event experienced on day 1. In order to control for this difference between groups, we included the state anxiety score on day 2 as an additional predictor in our final linear mixed models.

2.4 Discussion

Acute stress is known to be a powerful modulator of memory (Diamond et al., 2007; Joëls et al., 2006; Schwabe et al., 2022). Although it is well established that stressful events are typically well remembered (Bierbrauer et al., 2021; Kalbe et al., 2020; Sandi & Pinelo-Nava, 2007; Vogel & Schwabe, 2016) and that they can interfere with the retrieval of unrelated information (de Quervain et al., 1998; Shields et al., 2017), the impact of these events on the memory for surrounding neutral events and the cognitive mechanisms involved remain elusive. In this experiment, we probed a cueing mechanism whereby stress-induced memory alterations would selectively affect surrounding neutral information cued by the stressful encounter. In addition, we tested how the effect of stress on the memory for surrounding events relates to the memory of the stressor itself. While our data did not support the proposed cueing mechanism, they demonstrated a memory enhancement for events encoded before the stressor, which was directly linked to the memory enhancement for the stressful episode itself.

Significant increases in subjective stress responses, autonomic arousal, and salivary cortisol validated the successful stress induction through the eTSST. Consistent with previous studies, the stressful episode was more distinctly represented in memory, compared with the nonstressful control episode (Bierbrauer et al., 2021; Kalbe et al., 2020; Lin et al., 2022; Stanek et al., 2024). Specifically, we obtained a memory prioritization or centrality bias for the stressful episode, reflected in a more pronounced difference in the memory for central and pe-

ripheral features of the episode. Previous research has suggested a trade-off of memory during stress, whereby arousal enhances the memory for high-priority information but impairs the memory for low-priority information (Mather, 2007; Sakaki et al., 2014). A potential mechanism behind this trade-off effect is the proposed arousal-driven reconfiguration of large-scale neural networks. In particular, arousal has been suggested to induce a shift from default- or executive control networks toward the salience network, resulting in the prioritized encoding of highly salient information at the expense of less relevant, peripheral information (Hermans et al., 2011, 2014; Schwabe et al., 2022). However, it is to be noted that this large-scale network reconfiguration was shown to be driven by noradrenergic arousal (Hermans et al., 2011) and that we did not find associations between the memory prioritization (i.e., centrality bias) for the stressful episode and parameters of autonomic arousal in the present study. Moreover, several studies that assessed this trade-off in the past did not show an improvement for central feature memory at the expense of peripheral features (Bierbrauer et al., 2021; Kalbe et al., 2020; Lin et al., 2022), suggesting that the process might be more dynamic and depend on additional factors (Kensinger et al., 2007).

In addition to the memory prioritization for the stressful episode, stress affected the memory for neutral information encoded before the stressor. Exposure to the stressful episode enhanced the subsequent memory of information encoded before the stressor. Importantly, however, this memory boost occurred only if the stressful episode itself was distinctly represented in memory, as reflected in the centrality bias. Interestingly, the memory for information encoded before the stressor did not differ between stressed participants with a low versus high cortisol response when these participants showed a pronounced centrality bias for the stressful episode, suggesting that if the stressful event itself is distinctly represented in memory, it may impact the subsequent memory of previously encoded events, even in the absence of a pronounced cortisol response. The centrality bias in memory of a stressor has recently been linked to distinct repre-

sentational changes in the basolateral amygdala (Bierbrauer et al., 2021), the same region shown to integrate glucocorticoid and noradrenergic actions to modulate memory processes in other brain areas (McGaugh, 2015; Roozendaal et al., 2009). Based on these data, an interesting question for future research is whether the representational changes in the basolateral amygdala are necessary for potential stress (hormone) effects on memory, as has been suggested in the case of stress-induced effects on gist-memory (Adolphs et al., 2005), or whether these changes result from the interaction of glucocorticoids and noradrenergic arousal.

While memory for stimuli encoded before the stressor was enhanced in stressed participants, memory for stimuli encoded after stressor exposure remained unaffected. This result is generally in line with a meta-analysis suggesting that the effect of preencoding stress on memory is rather inconsistent (Shields et al., 2017). It has been proposed that stress effects on subsequent encoding depend on the temporal distance between stressor and encoding (Joëls et al., 2006; Zoladz et al., 2011). Thus, it could be argued that there may have been differential, potentially opposite, effects of stress on subsequent encoding of stimuli presented shortly after the stressor exposure or after a longer time delay. However, our results showed no modulation of the stress effect by the distance of a stimulus to the stressor. Our data did show, however, that memory performance was lower in the posttreatment phase for all participants, regardless whether they experienced a stressful or neutral encounter, which may have been due to fatigue or an already high memory load. This overall low memory performance for stimuli encoded after the treatment may have obscured any effects of stress on subsequent encoding.

The memory enhancement for stimuli encoded before the stressor when there is also a memory prioritization for the stressor aligns well with the BTH, which predicts that emotionally arousing events can boost memory for previously weakly encoded events (Ballarini et al., 2009). In the present study, we probed an additional cueing mechanism, assuming that neutral stimuli would be linked to the

stressor and therefore preferentially stored in memory if the stressor was cued at the time of stimulus encoding. Our results did not confirm this proposed cueing mechanism: cueing a stressful encounter did not modulate subsequent memory for stimuli that were encoded before or after the stressor. This finding suggests that the proposed cueing mechanism may not be relevant for stressor effects on surrounding neutral events. There is, however, an alternative explanation for the absence of a cueing effect in our experiment, related to the used contextual stressor cues. Accumulating evidence indicates that contextual information is less well processed under stress (Kaouane et al., 2012; Schwabe et al., 2009; Simon-Kutscher et al., 2019) and that stress induces a shift from a hippocampus-based system, which is relevant for contextual memory, toward a dorsal striatal system, which focuses on single cues (Kim et al., 2001; Vogel et al., 2016; Wirz et al., 2017). Thus, if spatial and contextual cues are less well processed under stress, these cues might not be well-suited to effectively cue the stressor. Future studies are required to test the proposed cueing mechanism using central aspects of the stressor, such as faces of the panel members in the TSST. Moreover, the effectiveness of the stressor context cueing may have been reduced in the present study by the relatively frequent presentation of the stressor context cue (16 times in total), which might have resulted in habituation to this cue. Finally, it is important to note that we used exclusively emotionally neutral images in the encoding sessions before and after the stressor. Stressor-cueing effects on the memory for surrounding information might only occur if the surrounding information involves some degree of emotional arousal, as suggested by studies showing that stress or glucocorticoid effects on memory require concurrent noradrenergic arousal (Joëls et al., 2011; Roozendaal et al., 2006).

In sum, we demonstrate that the impact of stress on memory for stimuli preceding the stressor is contingent on the recall of the stressor itself. Our results show a centrality bias in the memory of the stressful episode, indicated by a pronounced differentiation between the storage of central and peripheral features of

the event. While cueing the stressful encounter did not affect the later recall of surrounding information, memory for information encountered before the stressor was enhanced. This enhancement, however, was observed only in stressed individuals who exhibited a pronounced centrality bias in their memory of the stressful encounter. Collectively, these findings provide novel insights into the relationship between memory for the stressor itself and its impact on memory for surrounding (neutral) events. Specifically, these findings suggest that stress-related consolidation enhancements require a distinct memory representation of the stressful event itself.

3. Prediction errors gate stress-induced memory enhancements

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3.1 Study IIa

Next, we sought to investigate a second possible mechanism driving the selective effects of stress on the memory for surrounding, neutral material. Namely, our objective was to test whether PEs would place a transient behavioural tag on surprising information, which could be captured by stressful encounter occurring in the temporal periphery. Strong learning experiences marked by novelty trigger both the setting of a tag as well as the synthesis of PRPs (Viola et al., 2024). In contrast, moderate PEs arising from environmental changes, while causing insufficient neural activation for successful long-term memory formation independently, may leave a learning tag that can be captured by stress-related consolidation resources. Returning to the exam scenario, the stress response should preferentially strengthen memory for moderately unusual moments – such as the funny looking dog – rather than mundane aspects of the journey to the exam.

In the following two studies, participants encoded images presented in structured category sequences designed to elicit varying degrees of PE. Switches between main categories (e.g., animals to vehicles) were expected to produce larger

PEs than switches between subcategories (e.g., birds to fish), which in turn were expected to exceed repetitions within a subcategory. Participants provided trial-by-trial PE ratings. Between two encoding sessions, participants underwent either a psychosocial stressor or a control procedure. Memory was assessed the following day. The behavioural findings of Study IIa motivated us to repeat the experiment in a preregistered replication, combining the same encoding task structure with Electroencephalography (EEG) recordings in Study IIb. Any deviations from the preregistration can be seen in Supplemental table S6.

First, in Study IIa, we predicted that stress would selectively enhance memory for surrounding high-PE images, which should be associated with increased arousal, indexed by pupil size and SCR. In accordance with the symmetry inherent to the BTH, we expected this enhancement to take place both for images encoded before, as well as after the stressful encounter.

3.2 Methods

3.2.1 Participants and experimental design

One-hundred-eighteen healthy volunteers participated in this experiment (mean age = 24.99 years, SD = 3.93 years, 81 female). This sample size was based on a power calculation in G-Power (version 3.1.9.7) (effect size f = 0.2, power = 0.90), as well as on previous studies with similar research designs from our group (Kalbe et al., 2020; Lin et al., 2022). Participants were screened in a standardized phone interview for the following exclusion criteria: lifetime history of any mental or neurological disorder, use of medication or illicit drugs, use of hormonal contraceptives, and smoking.

Six participants were excluded due to excessive missing data (see below), resulting in a final sample size of 112 participants. Participants were pseudorandomly assigned to a stress (n = 53; 35 female; age: M = 24.23 years, SD = 3.25 years) or control group (n = 59; 42 female; age: M = 25.53 years, SD = 4.44 years)

to ensure a comparable number of stressed and control participants. One participant was excluded from mood data analysis due to missing data on Day 1, which left 111 participants in the analysis for mood (stress: n = 52, control: n = 59). Due to technical failure of the equipment, six participants' blood pressure and pulse measurements were missing from at least one time point, leaving 106 participants in the analysis for autonomic activity (stress: n = 50; control: n = 56). All participants provided written informed consent before the start of the experiment, which was approved by the local ethics committee.

3.2.2 Experimental procedure and tasks

All testing was conducted in the afternoon, from 13:00-19:00, to control for the diurnal rhythm of cortisol concentrations. The experiment spanned two experimental days, separated by a 24-hour interval. Day 1 consisted of three parts: a pre-treatment encoding phase, a standardized stress or control manipulation, and a post-treatment encoding phase (Fig. 3.1). On Day 2, participants underwent free recall and recognition memory testing. All experimental tasks were programmed in Matlab (version R2019b, 9.7.0.1319299) with Psychtoolbox (Brainard, 1997; version 3.0.16).

Day 1: pre-treatment encoding

After their arrival on Day 1, participants provided informed consent, followed by the collection of a salivary cortisol sample using a Salivette collection device (Sarstedt, Numbrecht, Germany). They then completed a multidimensional mood questionnaire (MDBF, Steyer et al., 1997) and rated their sleep quality on a slider, ranging from 0 to 100.

Participants then encoded 160 emotionally neutral images belonging to four different categories. For each image, they rated how expected it was by clicking on a slider ranging from 0 ("not at all expected") to 100 ("very expected"). This scale was transformed such that it ranged from 0 (low PE; very expected) to 100

(high PE; very unexpected). Images were presented sequentially, organized by category. Overall, there were eight main categories of images: animals, scenes, buildings, furniture, foods, kitchen equipment, clothing, and vehicles. Four of these categories were randomly selected for the pre-treatment encoding phase. Each main category was subdivided into four subcategories. For example, animals were divided into birds, insects, exotic, and marine animals; vehicles were divided into cars, planes, rail vehicles, and boats. Participants viewed ten images from each subcategory presented consecutively, before moving to the next subcategory. Once all subcategories within a category were shown, images from a new main category were presented in the same sequential format. This presentation method allowed participants to form expectations about subsequent images as the task progressed. For example, after viewing 40 images of animals, encountering a car would likely elicit a high PE, while viewing a plane after ten cars would result in a moderate PE. Repeated exposure to similar images within a subcategory (e.g., a seventh plane) would likely generate a low PE.

Each image was shown for three seconds on the screen, followed by a three-second window for participants to indicate their expectedness (i.e. PE) rating. There was an inter-trial-interval with a fixation cross presented for at least one second, with any remaining time from the three-second response time being added to the ITI, after which the next trial began. If participants did not respond within the three-second window, the trial was recorded as missing. The number of missing trials per participant was tracked across three trial types: main category switches, subcategory switches, and within-category switches. Participants were excluded if more than 16.7% of responses were missing for any trial type (more than one main category trial; more than four subcategory trials; more than 48 within category trials). Based on this criterion, six participants were excluded.

Day 1: stress and control treatment

After completing the pre-treatment encoding phase, participants provided a second saliva sample and completed the mood questionnaire (MDBF) again. Thereafter, approximately five minutes after the end of the pre-treatment encoding session, participants were randomly assigned to either the stress or control group. Similar to Study I, in the stress group, participants underwent the enriched version of the TSST (Kirschbaum et al., 1993). Furthermore, as in Study I, in the control group, participants took part in the non-stressful version of the eTSST. Blood pressure and pulse were measured during the arithmetic task in the stress group, and during the counting game in the control group (Dinamap, Critikon).

As in Study I, the room where the experimental manipulation took place was enriched with several objects scattered throughout the room. These objects were categorized as either central to the treatment (e.g. a mug that one of the panel members drank from) or peripheral (e.g. a plant on a shelf). In total, there were twelve peripheral and ten central objects, identical across the stress and control procedures.

Day 1: post-treatment encoding

After completing the TSST or control manipulation, participants returned to the room where the pre-treatment encoding task had taken place and gave a third saliva sample. They also completed the MDBF again and rated how stressful, difficult and unpleasant they had experienced the treatment.

Participants then performed the post-treatment encoding task, during which they viewed 160 new, neutral images and rated how expected each image was on a slider from 0 to 100. The images were drawn from the four remaining main categories, each again subdivided into four subcategories. The presentation order mirrored the structure of the pre-treatment encoding task: ten images from a subcategory were shown consecutively, followed by the remaining three subcategories from the same main category in sequence. The order of the (sub)categories

and which categories were presented during the pre- and post-treatment encoding task was fully randomized across participants.

A fourth saliva sample was collected during the post-treatment encoding phase, 25-30 minutes after stressor onset, as cortisol responses to the TSST typically peak within this time frame (Kirschbaum et al., 1993; Vogel & Schwabe, 2016). After completing the post-treatment encoding task, participants gave a fifth saliva sample and completed the MDBF again.

Day 1: pupillometry and skin conductance recordings

During both encoding sessions, we measured pupil size and skin conductance. Pupil size was tracked using the EyeLink 1000 Plus in remote mode (SR Research, Ottawa, ON, Canada). We recorded the left eye at 1000 Hz in a dimly lit room. All image stimuli were black and white and were made equiluminant.

Skin conductance responses were measured using the BIOPAC MP160 with the wireless BioNomadix EDA transmitter (BIOPAC Systems Inc., Goleta, CA, USA). Disposable electrodes were placed atop the thenar and hypothenar muscles of the left hand.

We also recorded continuous blood pressure and pulse (CNAP, CNSystems Medizintechnik AG, Graz, Austria). We averaged the blood pressure and pulse across each encoding session to obtain estimates for pre- and post-treatment autonomic nervous system activation.

Day 2: recognition test for encoded images

Twenty-four hours after Day 1, participants returned for a memory test for the encoded images and the treatment episode. We measured mood (MDBF), salivary cortisol, blood pressure and pulse to control for potential group differences in these stress measures before memory testing, and asked participants to rate the quality of their sleep during the previous night on a slider. In a recognition task, participants were presented with all encoded images from Day 1, as well

as an equal number of lure items from the same categories and subcategories as the target images. For each image, participants were asked to indicate whether the image was old or new. They could select between two levels of certainty (maybe/sure). The order of the images was randomized and the task was self-paced.

Day 2: free recall and recognition test for the stressful or control episode

Next, we tested participants' memory for the stressful or control episode in a free recall task. Participants were asked to recall details about the experience, such as the rules of the counting game and questions asked by the panel members. They were also asked to list all objects they remembered seeing in the room where the stress or control manipulation had taken place, while the experimenter wrote the participants' answers down. The task was self-paced. We later rated free recall performance by counting how many old objects the participant mentioned. Only mentions of pre-defined central and peripheral objects were considered as hits.

We also tested recognition memory for the stressful or control episode. Images of all central and peripheral objects, as well as the faces of the two panel members, were presented together with an equal number of lure items, including two lure faces. Participants indicated whether they had seen the specific item during the Day 1 treatment episode or not. They could select between three levels of certainty (maybe/sure/very sure).

3.2.3 Salivary cortisol sampling and analysis

A total of six saliva samples were collected using Salivette collection devices (Sarstedt, Numbrecht, Germany) and stored at -18°C until analysis. After data collection, the thawed and centrifuged samples were analysed using a chemiluminescence immunoassay (IBL, Tecan). Salivary cortisol level was assessed as the fraction of free cortisol (nmol/L).

Five participants were excluded from cortisol analysis due to insufficient saliva

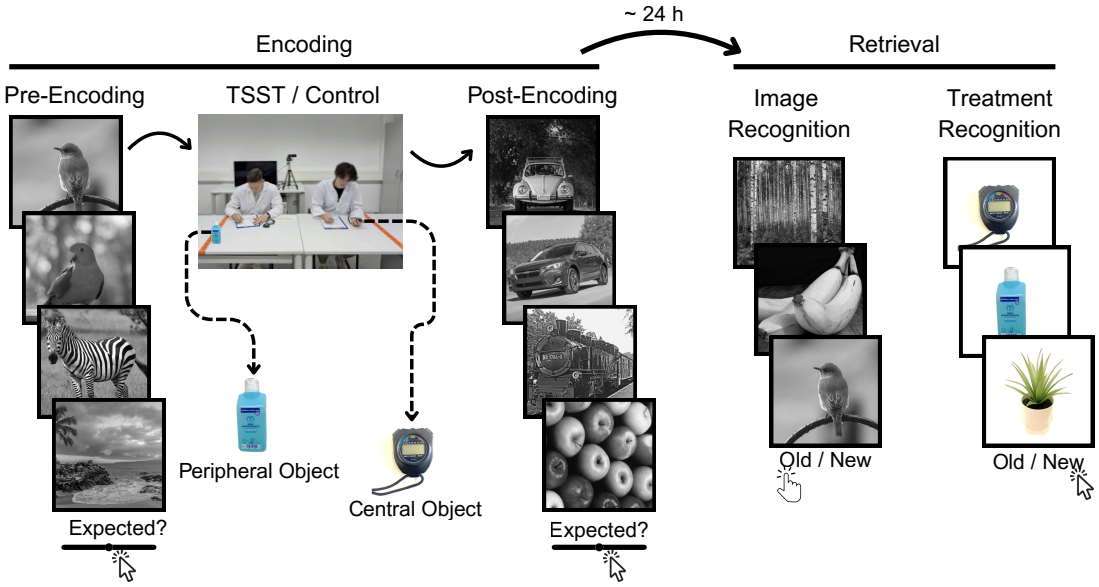

Figure 3.1 Experimental procedure. On Day 1, participants first encoded 160 neutral images and rated how expected each one was on a slider. The responses were reversed for analysis such that the scale ranged from 0 (very expected, ie. low PE) to 100 (very unexpected, ie. high PE). The images belonged to different subcategories, and were presented sequentially such that 10 images (Study 1) or 8-12 images (Study 2) of the same subcategory followed one another before switching to the next (sub)category. Next, participants participated in either an enriched version of the TSST or a control treatment before encoding and rating a new set of images. We collected mood ratings and salivary cortisol samples throughout Day 1 to track the stress response. One day later, participants first completed a recognition task, followed by a free recall and recognition task for the treatment episode.

volume in more than one sample. For ten participants (6 stress) with only one sample showing insufficient volume, regression imputation was used to estimate the cortisol concentration of the missing sample based on the existing data of the participant (Tabachnick & Fidell, 2019). One participant was excluded from the cortisol analysis due to a negative value after regression imputation, resulting in 106 complete datasets (57 control) for the cortisol analysis. The data were log transformed and analysed using a mixed-design ANOVA.

3.2.4 Control variables

To assess baseline differences in anxiety, depression, and chronic stress levels, participants completed two standardized questionnaires at home: the Trier In-

ventory of Chronic Stress (TICS, Schultz, 1998) and the IDAS-II (Wester et al., 2021). For 22 participants, one item in the chronic stress questionnaire was missing. The results did not change when those participants were excluded from the analysis, thus the reported results reflect the whole sample.

3.2.5 Statistical analysis of behavioral data

To verify that the stress manipulation was effective, we used mixed-design ANOVAs and t-tests to compare stress and control participants' blood pressure, pulse, salivary cortisol, mood and stress ratings. We used adjusted FDR to correct for multiple comparisons. Effect sizes are reported as generalized eta squared (η^2, ANOVAs) or Cohen's d (d, t-tests).

Memory performance was analysed in RStudio (version 4.2.2) using mixed-design ANOVAs and logistic regression models for trial-wise analyses, using the afex (Singmann et al., 2024), emmeans (Lenth et al., 2024), lme4 (Bates et al., 2014) and lmerTest (Kuznetsova et al., 2017) packages. We ran a mixed-design ANOVA to analyse the memory for the stressful vs. control episode. We only considered confident hits (sure/very sure), as confidence has been found to relate to a stronger memory trace (Heinbockel et al., 2024). We included treatment (stress vs. control) and object type (central vs. peripheral) as independent variables. We analysed hit rates as opposed to d', as false alarm rates could not be obtained separately for the central and peripheral objects. One participant was excluded from the analysis due to being an outlier in the recognition task (hit rate > 3 SD from the mean).

We used a logistic multilevel regression to analyse the effect of PE and stress on the probability of recognizing the old image. We only considered confident hits where the response was given within the mean response time (approximately 2.5 seconds), as faster responses have been shown to reflect higher confidence in one's memory (Weidemann & Kahana, 2016). Faster reaction times also relate to hippocampal activity during retrieval (Heinbockel et al., 2024). We included

treatment (stress vs. control), PE rating, phase (pre vs. post-treatment), and distance to treatment as predictors. To avoid overparameterization, we included a random intercept for participant as well as a random slope for PE, but no other parameters (Bates et al., 2018). GLMMs were computed in a step-wise fashion, starting with a simple model and gradually adding complexity by including interaction terms. Follow-up simple slopes analyses were corrected using adjusted FDR.

3.2.6 Statistical analysis of physiological data

SCR was analysed using continuous decomposition analysis (CDA) in Ledalab (version 3.4.9; Benedek and Kaernbach, 2010). We downsampled the data to 50 Hz and extracted the average phasic driver within 1-4 seconds from stimulus onset. The minimum amplitude was set to 0.01 uS. To account for interindividual differences in thickness of corneum, we cluster-mean centered the extracted phasic SCR and divided the centered value by the SD of the respective participant.

We obtained a measure of mean pupil size for each trial using the Dataviewer trial report, downsampling the data to 20 Hz (version 5.2, SR Research, Ottawa, ON, Canada). GazeR was used for pupil preprocessing (Geller et al., 2020). We extended blinks by 100 ms and used linear interpolation to before smoothing the data with a 2-point moving average. Next, we removed spurious pupil values based on visual inspection of the data distribution. We excluded datasets from 14 participants due to more than 20 percent missing data after spurious values were removed. Finally, we baseline corrected pupil dilation by subtracting the median pupil size from 100 ms prior to trial onset before averaging trial-wise pupil dilation for 1-3 seconds post stimulus appearance. Pupil dilation and SCR measurements were analysed in relation to behavioural variables using RStudio.

3.3 Results

3.3.1 Successful stress induction

To verify the successful stress induction, we used mixed-design ANOVAs to compare stressed vs. control participants' mood ratings across the experiment. We found significant time × group interactions for all mood ratings (mixed-design ANOVAs; all F $\geq$ 3.56, all p $\leq$ .016, all $\eta^2 \geq$ .01). Follow-up pairwise comparisons showed that participants in the stress group rated their mood as less good (Fig. 3.2a) and calm (Supplemental Table S7) immediately after the stressor exposure as well as at the end of testing on Day 1 (all |t| $\geq$ 2.97, all pFDR $\leq$ .007). For wakefulness ratings, pairwise comparisons showed no difference between stressed and control participants at any timepoint (all |t| $\leq$ 1.79, all pFDR $\geq$ .304). Moreover, there was no difference between stressed and control participants' mood ratings at baseline on Day 1 (all |t| $\leq$ 1.39, all pFDR $\geq$ .166). Stressed participants rated the treatment as more stressful, difficult and unpleasant than control participants (Fig. 3.2.c; all |t| $\geq$ 6.88, all p < .001, all |d| $\geq$ 1.30).

In addition to subjective stress levels, the exposure to the TSST led to increased autonomic activity. Mixed-designed ANOVAs comparing stressed and control participants' pulse, systolic and diastolic blood pressure in the pre-encoding phase, during the treatment, and in the post-encoding phase showed time × group interactions for all autonomic measures (all F $\geq$ 3.69, all p $\leq$ .028, all $\eta^2 \geq$ .01). Pairwise comparisons showed elevated pulse, systolic and diastolic blood pressure during the treatment episode in the stress (vs. control) group (Fig. 3.2g; all |t| $\geq$ 2.53, all pFDR $\leq$ .039). Stressed participants also showed elevated diastolic blood pressure during the post-encoding phase (t (104) = -2.47, pFDR = .023).

Finally, the TSST also resulted in elevated salivary cortisol concentrations. A time × group interaction (mixed-design ANOVA; F (1.84, 191.28) = 10.21, p < .001, η^2 = .03) followed by pairwise comparisons indicated that participants in

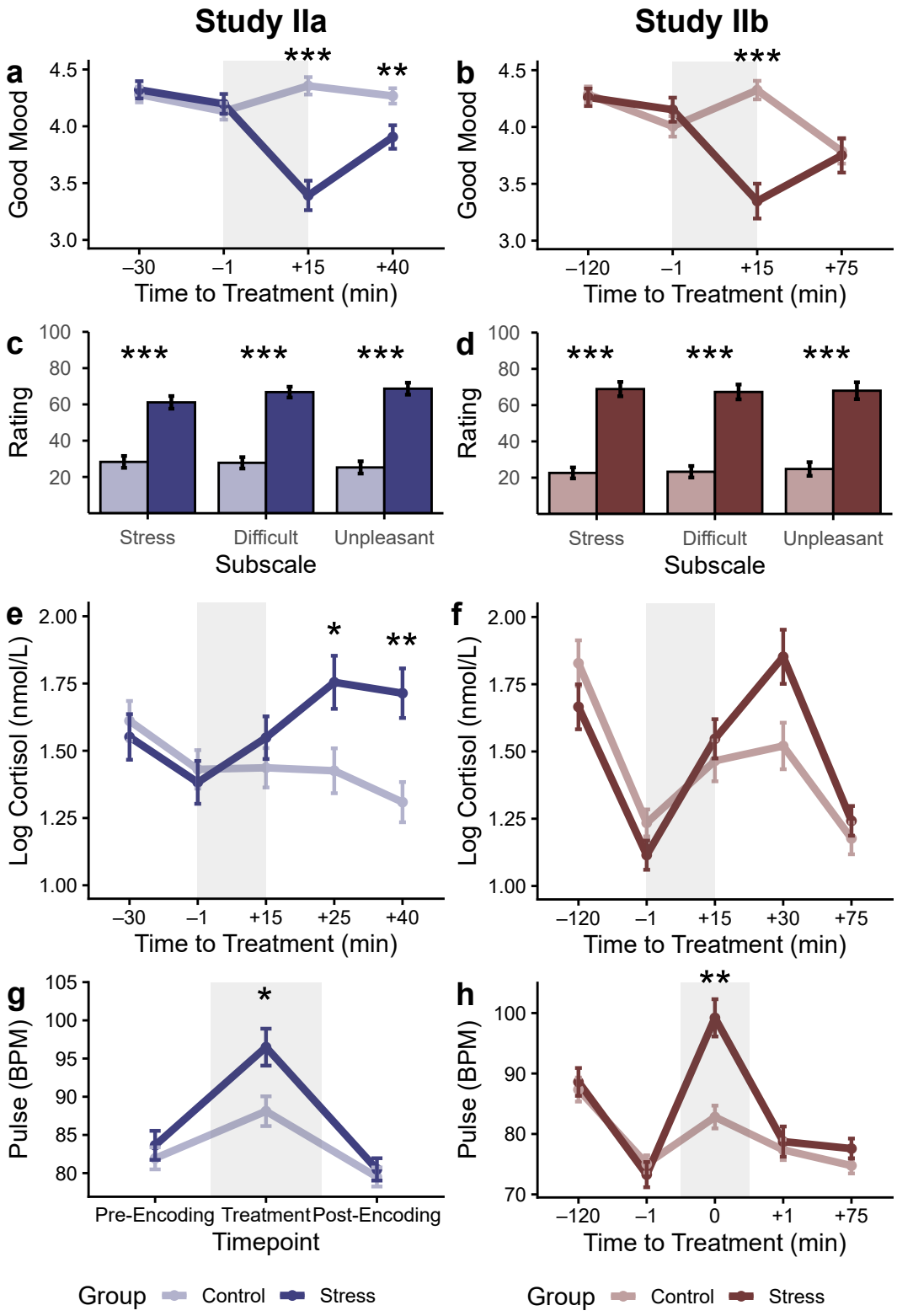

Figure 3.2 Successful stress induction in both (a, c, e, g) Study IIa and (b, d, f, h) Study IIb. Participants in the stress group rated their mood as significantly worse after the treatment (a, b) and found the treatment to be more stressful, difficult and unpleasant (c, d). Stressed participants also showed an elevated cortisol response 25-30 minutes after the stressor (e, f) as well as a higher pulse during the stressor (g, h). The gray bar illustrates the timing and duration of the treatment. Error bars represent standard error of the mean (SEM). (***) pFDR < .001, (**) pFDR < .01 and (*) pFDR < .05 for stress compared to control group.

the stress group had higher salivary cortisol levels than control participants at 25 minutes after stressor onset, as well as at the end of Day 1, around 40 minutes after stressor onset (Fig. 3.2e; both $|t| \geq 2.56$, both pFDR $\leq .030$). There was no difference between stressed and control participants at baseline, immediately before or immediately after the treatment (all $|t| \leq 1.04$, all pFDR $\geq .504$).

3.3.2 Prioritized memory for the stressful episode

At the time of memory testing on Day 2, there was no difference between stressed and control participants' mood ratings (all $|t| \leq 0.54$, all $p \geq .589$, all $d \leq .10$), autonomic activity (all $|t| \leq 1.25$, all $p \geq .215$, all $|d| \leq 0.24$), or salivary cortisol concentration (stress: M = 3.68, SD = 3.08, control: M = 4.00, SD = 3.21; t (97.4) = 0.90, p = .368, d = 0.18), suggesting that the stress level did not differ between groups before memory testing.

We used a mixed-design ANOVA to compare stressed and control participants' recognition memory for the stressful episode. We calculated separate hit rates for central and peripheral items. We found a group × item type interaction (mixed-design ANOVA; F (1, 109) = 6.62, p = .011, η^2 = .02). Pairwise comparisons, however, showed no difference between stressed and control participants for central or peripheral items (Fig. 3.3a; both $|t| \leq 1.82$, both pFDR $\geq .139$). Similar to Study I, we next computed a centrality bias index by subtracting the proportion of recognized peripheral objects from the proportion of recognized central objects. Stressed participants had a higher centrality bias in their memory of the treatment episode compared to control participants (Fig. 3.3b; t (106.42) = -2.57, p = .012, d = -0.49).

We also compared stressed vs. control participants' free recall memory for the central and peripheral objects. A mixed-design ANOVA revealed a main effect of object type whereby more central than peripheral objects were recalled, regardless of treatment (F (1, 106) = 354.18, p < .001, η^2 = .66). No other effects were significant (both $F \leq 0.56$, both $p \geq .458$). We also computed the centrality bias for

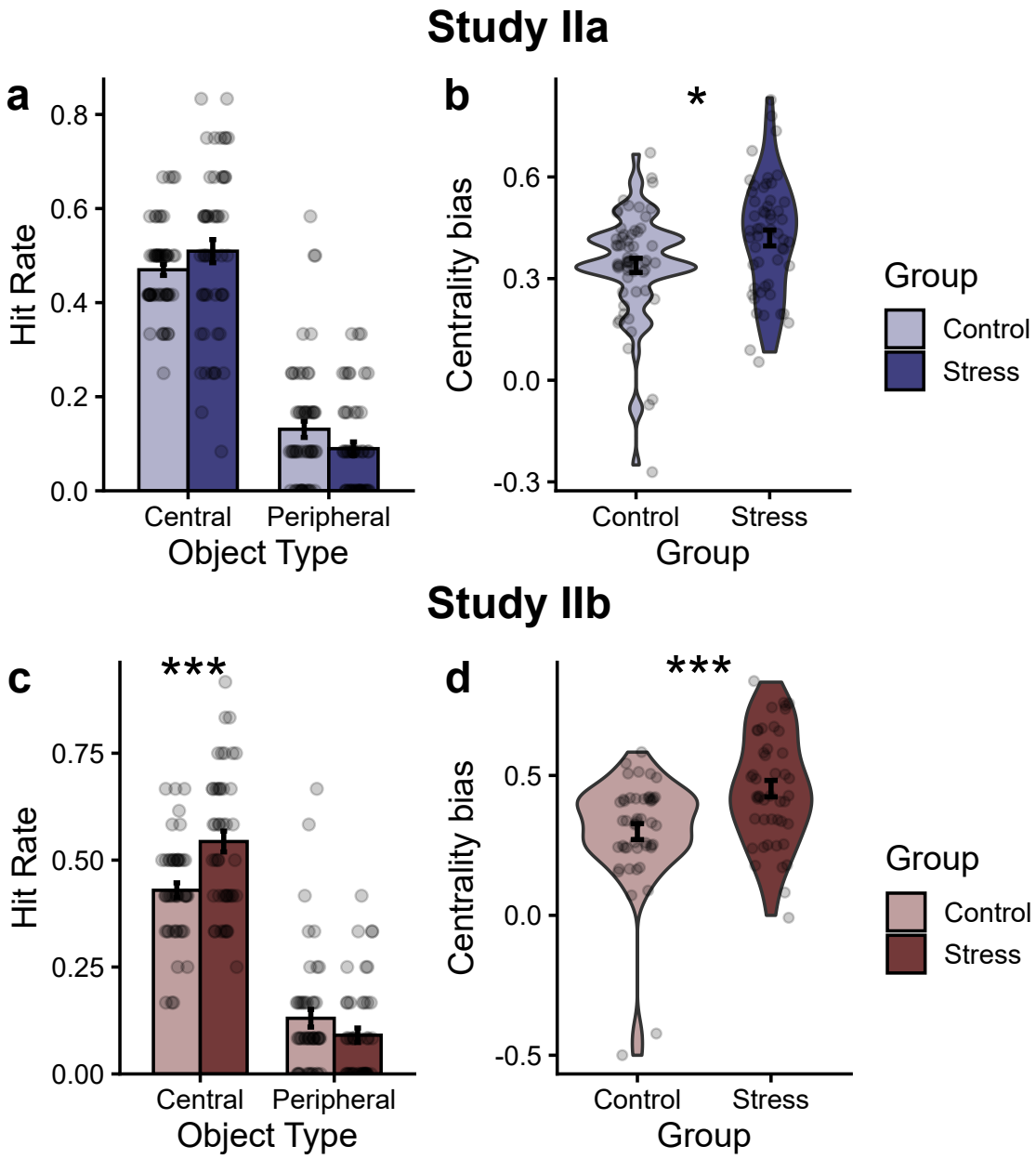

Figure 3.3 Memory for the stressful episode in (a-b) Study IIa and (c-d) Study IIb. Participants in the stress group recognized more central objects than the control group, at trend level in (a) Study IIa and significantly more in (c) Study IIb. The centrality bias (difference in proportion of recognized central and peripheral items) was significantly higher in the stress than the control group in both studies (b, d). Error bars represent SEM. (***) pFDR < .001 and (*) pFDR < .05 for stress compared to control group.

free recall, similarly to recognition data, but found no difference between stressed and control participants (t (91.32) = -1.08, p = .285, d = -0.21). Notably, free recall memory performance was overall very low (mean N objects recalled per participant = 2.61, SD = 1.45), resulting in a floor effect that may have prevented the detection of any group differences in free recall performance.

3.3.3 Stress effect on the memory for surrounding events depends on their elicited prediction error

To test whether the impact of stress on the memory for surrounding neutral events depends on the PE evoked by these events, we presented images sequentially, both in the pre-treatment and in the post-treatment encoding session. Images were organized by subcategory and main category, assuming that in particular subcategory and main category shifts would result in varying degrees of PEs. To test this assumption, we first grouped trials based on whether the preceding stimulus image belonged to the same subcategory (within subcategory switch), the same main category but a different subcategory (subcategory switch), or a different main category (main category switch). As expected, PE ratings differed significantly for the different types of trials (Fig. 3.4a; F (1.43, 158.40) = 776.41, p < .001, η^2 = .67). We also found an effect of encoding phase (F (1, 111) = 30.83, p < .001, η^2 = .02) as well as an interaction of trial type and encoding phase (F (1.78, 198.47) = 5.48, p = .007, η^2 = .00), suggesting that PE-ratings were overall lower in the post- compared to the pre-encoding phase. The significant interaction was driven by a subtle drift towards lower PE ratings in the main and subcategory switch trials, but not in the within subcategory switch trials (Fig. 3.4a).

Importantly however, regardless of the encoding phase, main category switches resulted in higher PE-ratings than the subcategory and within category switch trials, and subcategory switch trials resulted in higher PE-ratings than within category switch trials (all |t| > 16.43, all pFDR < .001). Moreover, there was no difference in PE ratings between stressed and control participants (β = -0.00, ±

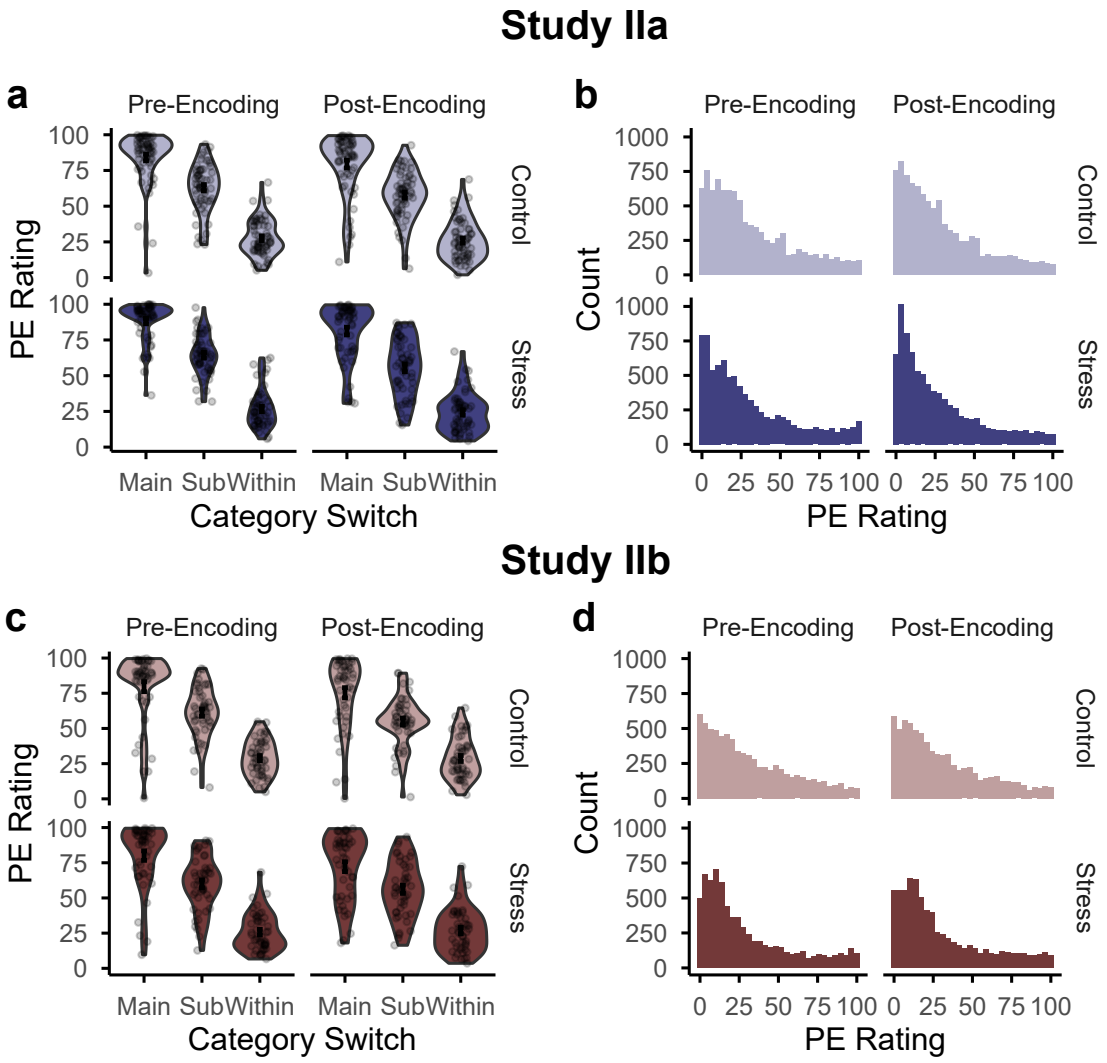

Figure 3.4 Prediction error (PE) ratings in Study (a-b) IIa and (c-d) Study IIb. Images immediately following a main category switch (e.g. animals to vehicles) were rated with a higher PE (i.e. less expected) than images following a subcategory switch (e.g. birds to insects), and images following another picture within the same subcategory were rated with the lowest PE (i.e. very expected) (a, c). The PE-ratings were, as expected, very skewed towards low PE ratings, with relatively few high PE ratings (b, d). Error bars represent SEM.

0.01, t (35537.00) = -0.00, p = .999). We further found correlations of PE ratings with both SCR (Supplemental Fig. S3a.; ρ = .03, p < .001) and pupil size (Supplemental Fig. S3b.; ρ = .05, p < .001).

As expected, the distribution of PE-ratings was overall skewed towards more low than high PE-ratings, as most trials were within the same subcategory of the previous trial (Fig. 3.4b). This trial structure was necessary in order to facilitate the creation of predictions about the upcoming images in the participants.

Importantly, however, the full range of the rating scale was used in both the pre-treatment encoding phase (range = 0.03-100.00) and post-treatment encoding phase (range = 0.03-100). Moreover, while PE-ratings decreased over the course of the encoding tasks (pre-treatment: β = -0.06, ± 0.00, t (17635.27) = -14.25, p < .001; post-treatment: β = -0.03, ± 0.00, t (17680.02) = -7.35, p < .001), the drift towards lower PE-ratings was relatively small, given the full scale of possible PE-ratings (Supplemental Fig. S2a.).

To analyse whether stress selectively affected the memory for surrounding events that were associated with elevated PE, we next computed mixed logistic regression models and compared them to identify the model with the optimal fit using likelihood ratio tests. We gradually increased the complexity of the models by adding higher order interaction terms. Model comparison showed that the model including two-way interaction terms of treatment (stress vs. control), PE-rating, phase (pre vs. post-treatment), and distance (trials from treatment onset/offset) best described the probability of a hit, when compared to the simpler model with direct effects only (Fig. 3.5b; χ^2 (6) = 66.65, p < .001). Moreover, the model fit did not improve when comparing the deviance to a more complex model with three-way interactions (χ^2 (4) = 2.31, p = .679). The most complex possible model also did not have a better fit to the observed data (χ^2 (1) = 1.49, p = .223).

This best fitting model showed a significant interaction effect of treatment and PE rating (β = -0.09, ± 0.03, z = -2.67, p = .008), indicating that the effect of stress on memory depends on the PE level associated with the specific event at encoding. Stress and control groups differed in particular when the PE associated with the encoded stimulus was high (Fig. 3.5a). Accordingly, follow-up simple slopes analyses showed that elevated PE resulted in a higher probability of a hit in stressed (β = 0.12, ± 0.03, z = 4.69, pFDR < .001), but not control participants (β = 0.04, ± 0.03, z = 1.37, pFDR = .171).

As evidenced by the lack of improved fit when including the three-way in-

teractions in the model, the effect of stress and PE on memory did not depend on whether the encoded material was encountered before or after the treatment. There was no interaction between treatment, PE, and encoding phase (Fig. 3.5c; β = -0.03, ± 0.05, z = -0.56, p = .566). This suggests that stress influences the memory for surprising material in a similar way regardless of whether the material was encoded shortly before or shortly after the stressful episode. Further, the interaction of treatment and PE also did not depend on the distance to the treatment episode (PE × group × distance interaction: β = -0.03, ± 0.03, z = -1.23, p = .220). In other words, the memory enhancement afforded to high PE stimuli in stressed participants was the same, irrespective of whether stimuli were encoded in close temporal proximity of the treatment or further away in time. The full regression table of the best fitting model is provided in Supplemental Table S8.

Finally, we also computed GLMMs including trial-wise arousal measures in the form of pupil size and SCR as additional predictors in the winning model. Neither pupil size (β = 0.02, ± 0.03, z = 0.89, p = .372) nor SCR (β = -0.40, ± 1.93, z = -0.21, p = .835) modulated the stress driven memory enhancement for high-PE information.

3.3.4 Control variables

To control for baseline differences between the stressed and control participants in sleep, we asked participants to rate their sleep quality on a numeric slider at the start of both days. The sleep ratings were missing from one participant. There was no difference between stressed and control participants sleep quality on Day 1 (t (103.56) = 0.60, p = .548, d = 0.11), nor on Day 2 (t (103.45) = 1.66, p = .246, d = 0.22). Stressed participants also did not differ from control participants in chronic stress levels (t (109.84) = 1.23, p = .223, d = 0.23), depressive or anxiety symptoms (t (84.58) = 0.56, p = .576, d = 0.12).

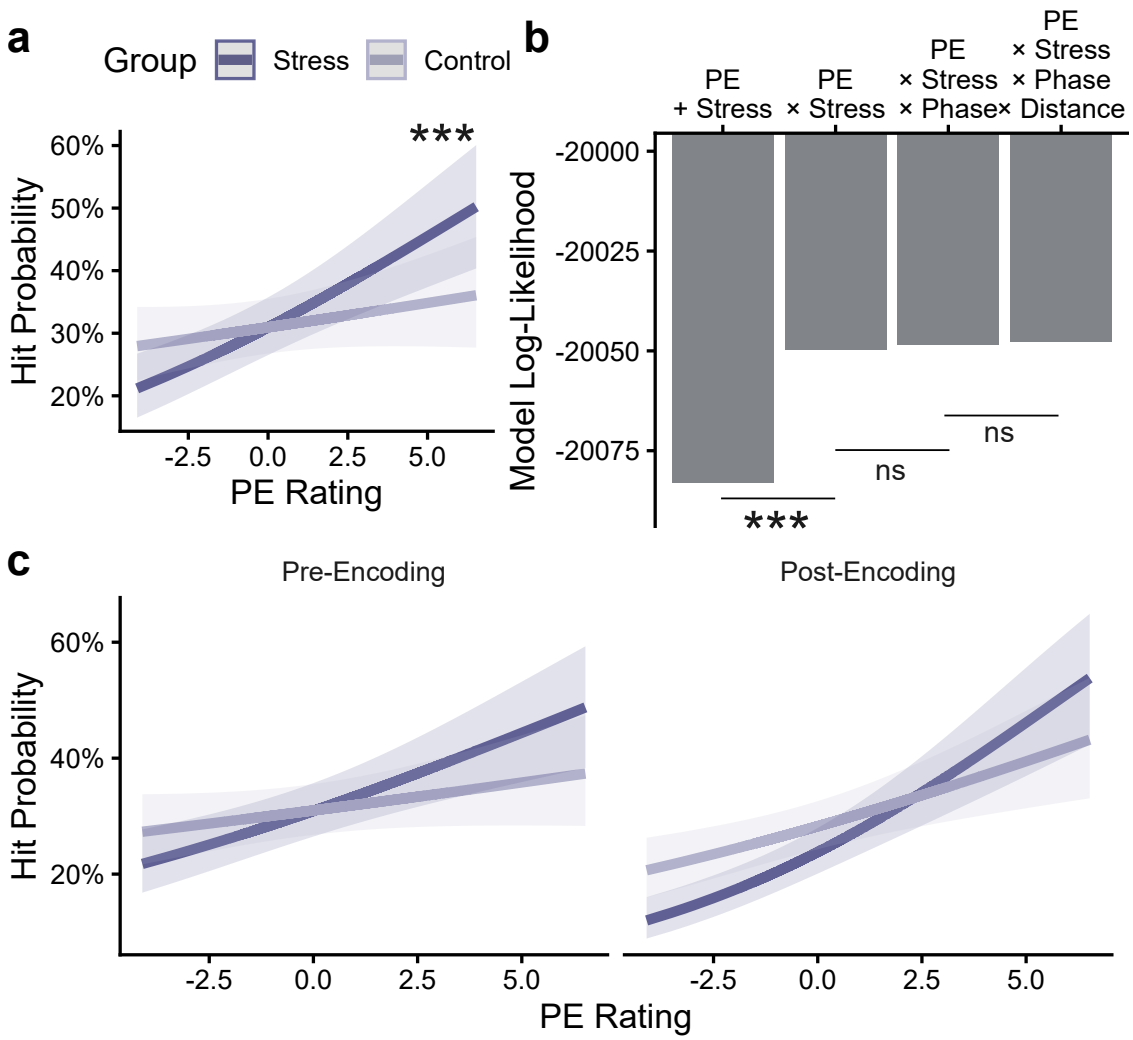

Figure 3.5 Stress effect on memory depends on the prediction error (PE) associated with the encoded image in Study IIa. The stress effect on Day 2 recognition memory depended on the PE associated with the image during Day 1 encoding (a). Model comparison suggested that this effect did not differ between the pre- and post-treatment encoding phases (b, c). Shaded areas indicate 95% confidence intervals. (***) $p < .001$ for (a) regression slopes and (b) model comparisons.

3.4 Study IIb

To test the robustness of these effects, we next conducted a preregistered (https://osf.io/ywbrc) replication study that used an analogous experimental design as Study IIa. We further wished to examine the neural basis of PE and stress driven memory enhancements using electroencephalography (EEG). We focused on the P300 and N400 event-related potentials because N400 has been shown to be sensitive to semantic processing, while P300 amplitude and latency

have been shown to be neural markers of attentional processing, model updating, and to be inversely related to expectancy (Duncan-Johnson & Donchin, 1981; Kutas & Federmeier, 2011; Linden, 2005). Furthermore, we examined whether post-stimulus reactivation processes contribute to the PE-dependent modulation of memory by stress.

3.5 Methods

3.5.1 Participants and experimental design

One-hundred-and-three participants (mean age = 24.92 years, SD = 4.42 years; 52 female) were pseudorandomly assigned to either a stress (n = 53; 27 female; age: M = 24.42 years, SD = 4.31 years) or a control group (n = 50; 25 female; age: M = 24.44 years, SD = 4.52 years). Participants had to meet the same inclusion criteria as in Study IIa, in addition to having a BMI between 18-27 (Björntorp & Rosmond, 2000). The sample size was based on a summary-statistics based power calculation (Murayama et al., 2022). Aiming for a power of 0.80 and an alpha level of 0.05, the power analysis indicated 94 participants would be needed. To account for a 5% drop-out rate, we increased this target sample size to 100. We chose to aim for a power level of 0.80 instead of 0.90 as in Study IIa due to the inclusion of EEG measurement and laboratory availability. Three participants were excluded due to technical error during testing. Similar to Study IIb, we excluded six participants due to excessive missing data in the PE task, leaving us with a sample of 94 participants (46 stress). Due to missing data, three participants were excluded from mood or autonomic nervous system activity analysis (one stress), and one control participant from cortisol analysis. Thirteen participants were excluded from pupillometry analysis due to more than 20% missing data, and one participant was missing free recall data for the treatment episode. Testing took place in the afternoon between 13:00-19:00.

3.5.2 Experimental procedure and tasks

The experimental procedure largely mirrored that of Study IIa, with the primary modifications being the addition of EEG measurements during the encoding sessions and the inclusion of a classifier task.

Participants completed two days of testing. On Day 1, the protocol involved the following steps: baseline assessments of mood, cortisol levels, blood pressure, and pulse; administration of an EEG classifier task; pre-treatment encoding phase; re-assessment of mood, cortisol, blood pressure, and pulse; application of either stress or control treatment; post-treatment assessments of mood, cortisol, blood pressure, and pulse; a second encoding phase (during which another cortisol sample was obtained, 30 minutes after the treatment onset); and finally, a second classifier task. On Day 2, the procedure was the same as in Study IIa.

We aimed to identify EEG activity patterns specific to the different image categories and their potential reactivation. To this end, participants completed a classifier task, which was divided into two sessions to control for potential temporal drifts in the EEG signal and to mitigate fatigue effects – one at the beginning of Day 1 and the other at the end of the same day. During each session, ten images from each subcategory (images not used during the subsequent encoding or test sessions) were presented on the screen for 500 ms, followed by a five-second ITI. Each subcategory appeared four times per session, with the order of subcategory presentation fully randomized.

The encoding task closely resembled the one of Study IIa, with three minor adjustments. First, participants had only 2 seconds to respond to the unexpectedness question. Second, the number of images per subcategory block was jittered between 8 and 12 stimuli to reduce habituation effects. Third, non-informative visual oddballs were embedded in 20% of the trials. At each trial, a fixation cross remained superimposed on the stimulus image for one second. In the oddball trials, the fixation cross was replaced by a neutral symbol (circle, diamond, pentagon, square, or triangle) at stimulus onset. These oddball trials did not relate

to the category switches and were fully task-irrelevant. They served to assess whether purely perceptual PEs would also interact with stress effects on memory formation.

3.5.3 EEG measurement and analysis

For EEG recording, a Biosemi ActiveThree system (BioSemi, Amsterdam, The Netherlands) with 64 electrodes was arranged according to the 10-20 system. Additional electrodes were placed on the mastoids, approximately 1 cm above and below the right eye's orbital ridge, and at the outer canthi of both eyes. Impedances were kept below 30 kΩ. Offline preprocessing was performed with Fieldtrip (Oostenveld et al., 2011) and custom MATLAB scripts (version R2025b, 25.2.0.3042426).

The data was epoched into trials (from 1 s before to 3 s after stimulus), filtered (low pass at 100 Hz, high pass at 0.3 Hz), referenced to the average of all EEG channels, baseline corrected via demeaning, and notch-filtered at 50 Hz. Up to one noisy channel per participant was rejected (M = 0.92, SD = 0.26). We opted to not reject more than one channel in order to avoid using interpolated channels for the correction of other noisy channels. Due to the overall low number of high PE trials, no trials were rejected during preprocessing. The data was downsampled to 256 Hz. ICA was used to identify eye-blink components based on visual inspection and connectivity analysis (coherence > 0.9). If more than two components were identified in the connectivity analysis, only the manually identified eye blink components were rejected. Finally, the rejected channel was interpolated based on the average of neighbouring electrodes.

A pre-registered aim of the EEG analysis was to decode stimulus category relevant activity patterns from the longer ITI in the classifier task, and apply this decoder to the encoding task. We expected that higher PE ratings would be reflected in reactivation of category specific neural activation patterns (Loock et al., 2025). To this end, a support vector machine was trained on the ITI of classifier

trials. However, decoding accuracy did not exceed chance level for any category, thus rendering the transfer to the encoding task not meaningful. Consequently, the classifier data was not used further in the analyses.

Beyond the decoding analysis, we pre-registered three event-related potential (ERP) components of interest which were computed relative to a pre-stimulus baseline (-0.2 to 0 s). The N400 amplitude was averaged across 300–500 ms post-stimulus at electrodes F3, F4, Fz, C3, C4, Cz (Šoškić et al., 2022), and the P300 amplitude across 250–500 ms at Cz, Pz, and Fz (Polich, 2007). Furthermore, the P300 peak latency was defined as the time when amplitude reached 50% of its local maximum (Fractional Peak Latency, FPL). These ERPs were calculated both by averaging across category types (within-category switch, subcategory switch, main category switch) and on a trial-by-trial basis.

3.5.4 Statistical analysis

Statistical analyses were performed as in Study IIa. Significant interactions were followed up with simple slopes (GLMMs) and pairwise comparisons (GLMMs, ANOVAs) and corrected using FDR. We further computed GLMMs incorporating trial-wise P300 and N400 amplitude as well as P300 FPL as additional predictors of memory performance.

3.6 Results

3.6.1 Successful stress induction

Same as in Study IIa, subjective, autonomic, and endocrine data showed a successful stress induction through the TSST. We found a drift toward overall lower mood ratings across the experiment (mixed-design ANOVAs; all $F \geq 13.05$, all $p < .001$). For good mood and calmness, we observed group × time interactions (both $F \geq 9.94$, both $p < .001$, $\eta^2 \geq .05$). Pairwise comparisons revealed that stressed participants scored lower than controls immediately after the treatment (Fig. 3.2b;

both t (89) $\geq$ 4.33, both pFDR < .001), with no group differences at baseline (both t $\leq$ 0.31, both pFDR > .774). There was no group × mood interaction for wakefulness (F (2.87, 255.41) = 0.63, p = .591, η^2 < .01). Stressed participants rated the treatment episode as more stressful, difficult, and unpleasant than those in the control group (Fig. 3.2d; all |t| $\geq$ 7.20, all p < .001).

Mixed ANOVAs for pulse, diastolic, and systolic blood pressure showed significant effects of time (all F $\geq$ 36.94, all p < .001) and group × time interactions for pulse and systolic blood pressure (both F $\geq$ 3.62, both p < .019). Follow-up analyses indicated higher pulse in the stress (vs. control) group during the stressor (Fig. 3.2h; t (89) = -4.83, pFDR < .001). Descriptively, systolic blood pressure tended to be higher in the stress group during the treatment (stress: M = 153.28, SD = 19.36; control: M = 145.22, SD = 20.06), which was however not significant after correction for multiple testing (all |t| $\leq$ 1.92, all pFDR $\geq$ .291). There was no group × time interaction for diastolic blood pressure (F (3.33, 296.67) = 1.14, p = .336, η^2 = .006). No group differences were observed at the start of Day 1 (all |t| $\leq$ 0.49, all pFDR $\geq$ .683).

For salivary cortisol, we found an effect of time (mixed-design ANOVA; F (2.29, 208.50) = 47.08, p < .001, η^2 = .18) and a group × time interaction (F (2.29, 208.50) = 6.47, p = .001, η^2 = .03). Cortisol levels decreased in both groups from the beginning of the experiment to before the treatment, due to the diurnal rhythm of cortisol (Fig. 3.2f). As expected, cortisol levels increased after the treatment in the stress (t (91) = 6.93, pFDR < .001), but not in the control group (t (91) = 1.27, pFDR = .208).

3.6.2 Prioritized memory for the stressful episode

Before the memory test on Day 2, stress and control groups did not differ in good mood or calmness (Supplemental Table S9; both t $\leq$ 0.60, both p > .553), systolic and diastolic blood pressure (both $\leq$ 1.83, both p > .070), or salivary cortisol (stress: M = 6.31, SD = 6.44, control: M = 5.18, SD = 4.97; t (87.51) = .434, p =

.666). Participants in the control group had higher awake ratings than stressed participants (t (88.98) = -2.05, p = .044) and a higher pulse (t (90.86) = -2.07, p = .042). However, including these measures into the subsequent analyses left the results largely unaffected.

We computed mixed ANOVAs to assess memory for the treatment episode. This analysis revealed an effect of item type (F (1, 92) = 336.65, p < .001, η^2 = .66) as well as a group × item type interaction (F (1, 92) = 14.04, p < .001, η^2 = .08), suggesting that groups differed in memory for the treatment episode depending on whether an item was central or peripheral. Pairwise comparisons revealed that stressed participants remembered more central objects than those of the control group (Fig. 3.3c; t (92) = -3.85, pFDR < .001). For peripheral objects, while there was no statistically significant difference between the groups (t (92) = 1.48, pFDR = .141), descriptively, participants in the stress group recognized fewer objects than those in the control group (stress: M = 0.09, SD = 0.11; control: M= .13, SD = 0.14). Similar to Studies I and IIa, we next computed a centrality bias for each participant by subtracting the number of peripheral items recognized from the number of central items recognized. We found that participants in the stress group had a higher centrality bias, suggesting mnemonic prioritization for the central details of the stressful episode (Fig. 3.3d; t (91.78) = -3.75, p < .001, d = -0.77).

We also compared stressed and control participants' free recall memory for central and peripheral details of the treatment episode. We found effects of group (mixed-design ANOVA; F (1, 91) = 11.21, p = .001, η^2 = .069), item type (F (1, 91) = 422.54, p < .001, η^2 = .652) as well as an interaction of the two (F (1, 91) = 16.70, p < .001, η^2 = .069). Follow-up tests showed that stressed participants recalled more central (t (91) = -4.61, p < .001), but not peripheral objects (t (91) = 0.00, p = .993). We also computed the centrality bias for each participant, subtracting the proportion of recalled peripheral objects from the proportion of central objects recalled. Supporting the recognition memory findings above, we found a stronger

centrality bias for free recall in the stress group (t (78.92) = -4.62, p < .001, d = -0.97).

3.6.3 Stress effect on memory for subsequent events depends on their elicited prediction error

Participants' PE ratings largely mirrored the pattern observed in Study IIa. PE ratings differed by trial type (Fig. 3.4c; F (1.34, 124.37) = 247.37, p < .001, η^2 = .53) and by encoding session (F (1, 93) = 12.32, p < .001, η^2 = .01), and were skewed towards mostly low PE ratings (Fig. 3.4d). We again found an interaction of trial type and encoding session (F (1.68, 156.21) = 6.84, p = .003, η^2 = .01), driven by an overall subtler drift in PE ratings in the post-treatment phase (Fig. 3.4c). Regardless of the encoding phase, main category switches resulted in higher PE-ratings than the subcategory and within subcategory switch trials, and subcategory switch trials resulted in higher PE-ratings than within subcategory switch trials (all $|t| \geq 7.98$, all pFDR < .001). Importantly, PE ratings did not differ between the stress and control group (β = -2.30, ± 2.59, t (92) = -0.89, p = .376). PE ratings correlated negatively with pupil size (Supplemental Fig. S4b.; ρ = 0.06, p < .001) but not with SCR (Supplemental Fig. S4a.; ρ = 0.01, p = .140). Further, pupil size was larger in the post- than in the pre-treatment encoding phase (β = 21.30, ± 1.41, t (92) = 15.15, p < .001).

PE-ratings were overall skewed towards more low than high PE-ratings, as expected based on the experimental design, but the full range was used in both encoding phases (pre-treatment: 0.03-100.00; post-treatment: 0.03-100). PE-ratings decreased over the course of the encoding task (pre-treatment: β = -0.03, ± 0.00, t (14515.25) = -6.87, p < .001; post-treatment: β = -0.02, ± 0.00, t (14521.22) = -4.61, p < .001), but the drift towards lower PE-ratings was relatively small (Supplemental Fig. S2b.).

We next computed mixed logistic regression models and compared them to identify the model with the optimal fit using likelihood ratio tests. Model com-

parisons showed that the model including three-way interaction terms of treatment (stress vs. control), PE-rating, phase (pre- vs. post-treatment) and distance (trials from treatment onset or offset) best described the probability of a hit when compared to the simpler model including two-way interactions only (Fig. 3.6b; χ^2 (4) = 13.96, p = .007). The model fit did not improve when comparing this model to a model including a four-way interaction (χ^2 (1) = 1.30, p = .254).

This best fitting model showed an interaction effect of treatment, PE rating, and encoding phase (Fig. 3.6a; β = 0.16, ± 0.05, z = 3.04, p = .002), indicating that the interactive effect of stress and PE rating depended on the encoding phase. Follow-up simple slopes analyses showed that high PEs increased the hit probability in stressed participants in the post-treatment encoding phase (β = 0.09, ± 0.03, z = 2.94, pFDR = .012) but not in the pre-treatment encoding phase (β = -0.02, ± 0.03, z = -0.71, pFDR = .475). No other simple slopes were significant (all $|\beta| \leq 0.04$, $|z| \leq 1.21$, pFDR $\geq$ .225). Moreover, the memory enhancing effect of stress for high-PE events did not depend on the number of trials from the treatment episode (β = -0.01, ± 0.03, z = -0.50, p = .615). The full regression table of the winning model can be seen in Supplemental Table S10.

We next computed a GLMM predicting memory based on whether the trial included a perceptual oddball or not, as well as encoding phase, distance to treatment, and group. In this model, there was no difference between oddballs and standards in terms of how stress and encoding phase impacted memory (β = 0.08, ± 0.13, z = 0.57, p = .566). In other words, the lack of an effect for oddballs suggests that the PE-related memory enhancement we found above is most likely not a perceptual effect, but rather requires semantic processing.

We also computed GLMMs with pupil size or SCR as additional predictors in the winning model. Neither SCR (β = 1.05, ± 2.80, z = 0.37, p = .708) nor pupil size (β = -0.02, ± 0.06, z = -0.31, p = .756) modulated the memory effect seen for high-PE stimuli in the post-stress encoding phase.

3.6.4 EEG correlates of prediction error driven memory enhancement

We predicted that the PE-dependent effect of stress on memory would be linked to the P300 and N400 amplitude, as well as P300 latency, neural signatures of attentional and semantic processing as well as model updating (Duncan-Johnson & Donchin, 1981; Kutas & Federmeier, 2011; Linden, 2005). We further expected that these neural markers would directly relate to PE-ratings. To address these predictions, we first ran separate GLMMs predicting the ERP based on PE rating, group and encoding phase. None of the neural markers was modulated by PE magnitude (all $|\beta| \leq 0.04$, $|t| \leq 1.75$, $p \geq .084$).

Next, we analysed whether the interactive effect of stress and PE would be associated with these neural measures. Separate GLMMs showed that higher N400 (β = 0.01, ± 0.00, z = 2.56, p = .010) or P300 amplitude (β = 0.01, ± 0.00, z = 3.58, p < .001) as well as a later P300 peak (β = 0.04, ± 0.01, z = 2.81, p = .005) in general predicted better memory performance. None of these neural measures differed between groups (all $|\beta| \leq 0.15$, $|t| \leq 0.56$, $p \geq .577$). We further tested whether the effect of PE and stress on memory depended on the N400 or P300 amplitude, both in models with the neural measures in four-way interactions with PE, group, and encoding phase, and in separate models for each encoding phase. Neither N400 nor P300 amplitude modulated the effect of PE and stress on memory (all $|\beta| \leq 0.02$, $|z| \leq 1.50$, $p \geq .134$).

For P300 FPL, however, we found a trending four-way interaction of PE × group × phase × FPL (β = 0.10, ± 0.05, z = 1.87, p = .061). As we found a behavioural effect of PE specifically in the post-treatment encoding phase, we next ran separate GLMMs for the pre- and post-treatment encoding phases. Here we obtained a significant PE × group × P300 FPL interaction in the post-treatment phase (β = 0.08, ± 0.04, z = 2.11, p = .035), but not in the pre-treatment phase (β = -0.02, ± 0.03, z = -0.72, p = .474). Notably, this interaction was mainly driven

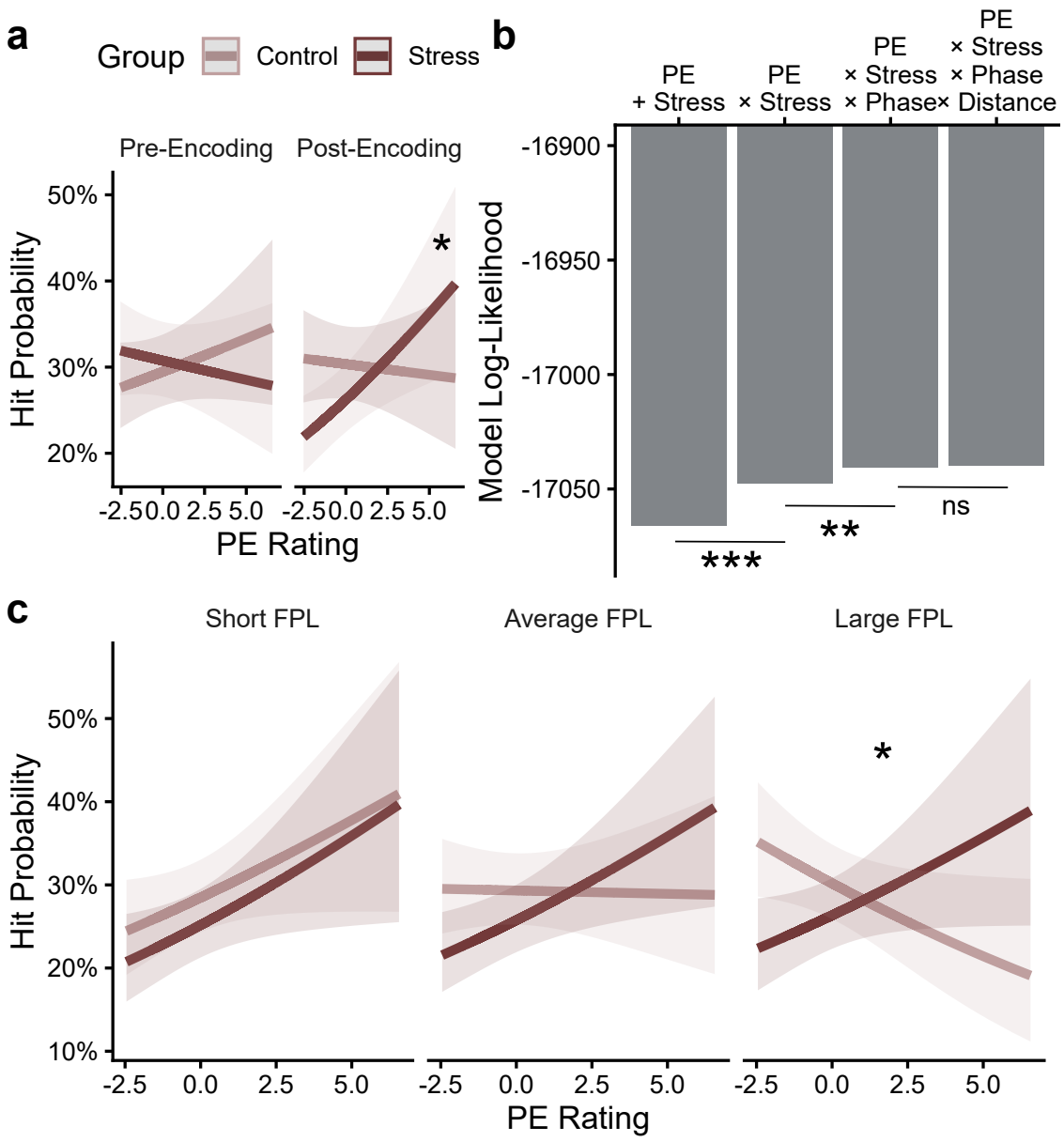

Figure 3.6 Stress effect on memory depends on the prediction error (PE) associated with the encoded image in Study IIb. Stress effect on memory depends on PE associated with the encoded material as well as timepoint of encoding, such that only high PE images encoded post-stress were more likely to be remembered (a). Model comparison showed that including a term of encoding phase (pre vs. post-treatment) significantly improved model fit (b). An earlier P300 peak (FPL) modulated PE-driven memory enhancement in the control group specifically, while the PE-effect on memory did not rely on the FPL in the stress group (c). Shaded areas indicate 95% confidence intervals. (***) $p < .001$, (**) $p < .01$ and (*) $p < .05$ for (a) regression slopes, (b) model comparisons, and (c) slope differences between stress and control group.

by a change in the effect of PE on memory performance in the control group (Fig. 3.6c). Specifically, follow-up analyses indicated that the effect of PE on memory differed significantly between stressed and control participants, depending on the P300 FPL latency. In other words, the effect of PE on memory differed between participants in the stress vs. control group when there was a later P300 FPL ($z = 2.75$, $pFDR = .018$), but not at average ($z = -1.86$, $pFDR = .094$) or early FPL ($z = -0.28$, $pFDR = .782$). The predicted probabilities revealed that, at higher (later) P300 FPL, PE enhanced memory in the stress group. In contrast, in the control group, higher P300 FPL was associated with a reduction in PE-driven memory enhancement (Fig. 3.6c).

3.7 Discussion

Across Studies IIa and IIb, we demonstrate that stress enhances memory for surrounding neutral events depending on the PE associated with these events. Study IIa indicated that this PE-dependent effect of stress on memory occurs for material encoded both before and after the stressful event, whereas Study IIb replicated this effect specifically for images encoded after the stressor. Additionally, Study IIb revealed that, while the P300 latency, a neural correlate linked to cognitive efficiency, was associated with the PE-dependent memory enhancement in the control group, this relationship was absent in stressed participants.

Our findings align with earlier studies showing that stressful experiences are not only more distinctly represented in memory themselves (Bierbrauer et al., 2021; Lin et al., 2022), resulting in a prioritization of central features, but also enhances memory for events occurring immediately before or after the stressor (Joëls et al., 2006; Shields et al., 2017). However, previous research suggested that these effects are mainly driven by the activity of major physiological stress response systems (Joëls et al., 2006; Schwabe et al., 2022), predicting comparable, uniform memory performance for items encoded close in time and under similar physiological conditions. In contrast, our results demonstrate that memory

for items encoded at similar temporal distances from a stressor can differ significantly depending on the PE associated with each event. Specifically, stress selectively enhanced memory for surrounding neutral events when these were associated with higher PEs. This pattern is in line with theories proposing that memory is future-oriented and that we tend to preferentially encode memories that aid in predicting future outcomes (Bar, 2009; Shohamy & Adcock, 2010). The preferential storage of surprising events surrounding a stressor may facilitate forming an accurate model of the stressful encounter and its potential consequences, thereby supporting adaptive coping.

Previous research has suggested that stress effects on memory are primarily evident for emotionally arousing events, requiring an interaction of glucocorticoids and noradrenergic arousal (Roozendaal et al., 2006). To avoid confounding stimulus emotionality with PE, we deliberately used emotionally neutral materials and aimed to induce mainly moderate PEs. While our pupil and SCR data in Study IIa suggest that larger PEs evoked stronger arousal responses, this correlation was rather modest. This response may reflect the moderate nature of the PEs produced during encoding, making them insufficient to trigger all cascades that are required for long-term memory formation, but still leaving an activity trace that can benefit from the release of PRPs by the subsequent stressor. Such PE-driven behavioral tagging could explain why memory enhancement under stress selectively affects some, but not all, encoded events. Very novel events are likely to be memorized regardless of context, as the BTH posits that a strong learning event is sufficient for transfer into long term memory through the simultaneous setting of a learning tag and the synthesis of plasticity-related proteins (Viola et al., 2024).

According to the behavioral tagging framework, the release of PRPs and their interaction with memory tags is symmetric – meaning that tags can be captured both if set before or after protein synthesis (Viola et al., 2024). While Study IIa showed that stress enhanced memory for high-PE stimuli encoded both before

and after the stressor, Study IIb indicated this effect specifically for stimuli encoded after the stressor. Notably, the relationship between PE and arousal measures in Study IIb was inconsistent, and, in contrast to Study IIa, pupil size was overall lower in the pre-treatment encoding phase. Thus, this discrepancy could suggest that the pre-treatment encoding phase in Study IIb failed to trigger tag setting equivalent to Study IIa. Methodological differences – such as the inclusion of an EEG classifier task before pre- but not post-treatment encoding in Study IIb – may have induced fatigue, impacting tag setting in the pre-treatment encoding phase only. Moreover, tagging effects following stress might be generally more robust than those before a stressor because stress responses, especially elevated cortisol, can influence not only consolidation but also initial encoding processes.

Importantly, stress did not alter PE ratings themselves, indicating that stress influences memory encoding processes rather than novelty detection. Our EEG analyses provided initial insights into the neural substrates of the PE-dependent effects of stress on memory formation. While we were not able to test whether PE and stress interactively influence the post-encoding reactivation of image categories – because decoding the image categories from the EEG data was not feasible, likely due to the large number of different (sub)categories and the limited spatial resolution of EEG – we observed that an earlier P300 was associated with PE-driven memory enhancement in the control group, but not in the stress group, during post-treatment encoding. Our findings suggest that an earlier P300 may facilitate effective 'tagging' of surprising stimuli when stress is absent, whereas stress may induce alternative encoding pathways. The latency of the P300 is associated with attention and information processing speed (Duncan-Johnson & Donchin, 1981; Linden, 2005), implying that efficient processing reflected by an earlier P300 supports learning in non-stressed conditions. In contrast, our findings indicate that stress may diminish reliance on P300 latency. Importantly, the latency of P300 per se was not affected by stress. These findings suggest that, in a stressed state, stress response system activation may be sufficient to boost the

processing of surprising information, potentially through the recruitment of other processing pathways than under non-stressful conditions. Future studies should use neuroimaging tools with higher spatial resolution such as fMRI to elucidate the brain mechanisms underlying the PE-dependent impact of stress on memory formation for surrounding events.

To conclude, we show that stress prioritizes the storage of unexpected events surrounding a stressful encounter. Across two experiments, we found strong support for prospective, selective memory enhancement by stress for subsequent surprising events. These findings underscore that stress does not uniformly impact surrounding memories but may specifically target information critical for understanding stressful situations. Beyond its relevance for forming adaptive models of stressful events, this PE-dependent stress effect on memory formation may be relevant in the context of eyewitness testimony and for memory distortions in stress-related mental disorders.

4. General discussion

Historically, the impact of stress on the memory of surrounding events has been studied from a perspective assuming a global influence driven by interactions of noradrenaline and corticosteroids (Joëls et al., 2006; Roozendaal et al., 2006). Research has however indicated that this influence is selective (see e.g. Kensinger, 2009; Shields et al., 2017). Across three studies, we investigated two mechanisms that may drive selective prospective and retrospective memory enhancement induced by stress. Based on the framework of the BTH, we hypothesised that a stressful encounter acts as a significant event that is encoded effectively and results in the release of PRPs. We suggested that stress can thus influence the learning of unrelated, non-stressful information that is encountered within a short time period either before or after the stressor.

Most literature on the BTH has assumed that the requirements that need to be met for the tagging and capture to take place are (1) a restricted time delay between the weak learning event and significant encounter (Ballarini et al., 2009), (2) a weak, rather than strong, learning trace (Lopes da Cunha et al., 2018), (3) emotional relevance of the weakly encoded information (Dunsmoor et al., 2022). Based on the literature, we identified two further possible mechanisms that may determine whether the tagging and capturing will take place – PEs, and a cue to a context that reactivates the memory trace of the stressful encounter. Across the experiments presented in this thesis, we demonstrate a selective stress-driven enhancement of surprising information. The selective memory enhancement by PEs was consistently demonstrated when the information was encoded after the stressor – for surprising material encoded before the stressor, the results were

inconsistent. In contrast, we found no evidence for cueing a stressful encounter as a tagging mechanism.

4.1 Selective retrospective and prospective memory enhancement by stress

Many studies have shown results that suggest the influence of stress on memory to be selective, rather than general (see e.g. Bierbrauer et al., 2021; Kensinger, 2009; Kensinger et al., 2007; Simon-Kutscher et al., 2019). The mechanisms behind this selectivity remain elusive, but the BTH offers a framework through which we can understand how a significant event can influence the consolidation of unrelated information encoded in the temporal periphery. A key feature of the BTH is the assumption of symmetry, meaning that the tag can be placed both before (retrospective effect of the significant event) or after (prospective effect of the significant event) the occurrence of a significant encounter (Ballarini et al., 2009; Viola et al., 2024). Thus, a strong learning event that releases PRPs could enhance the consolidation of both memories that are encoded before as well as after the placement of the learning tag.

Indeed, the findings in Study IIa suggested an equivalent memory enhancement of surprising information by stress in the pre- and post-stress encoding phase. Meanwhile, the findings in Study IIb show this selective memory enhancement only for surprising information encoded after the stressful encounter. These findings raise the question of whether the mechanisms behind these effects are the same, regardless of whether the surprising information was encountered before or after the stressor. According to the framework proposed by the BTH, the mechanisms should be equivalent (Ballarini et al., 2009; Viola et al., 2024). Per synaptic tag-and-capture, the neural mechanism that constitutes tag setting is a structural, temporary change in state of the dendrite enabling the capture of PRPs, ultimately resulting in more slots available for AMPA receptors (Redondo

& Morris, 2011; Viola et al., 2024). This results in the expression of l-LTP, ultimately stabilizing the memory trace (Redondo & Morris, 2011). Whether the tag is placed before or after the production of PRPs should not matter, rather, the key temporal determinant of successful tag-and-capture is the gradual synthesis of PRPs as well as their half lives (Ballarini et al., 2009). Thus, from the pure perspective of the BTH, the lack of a memory benefit for high PE information encoded before the stressor in Study IIb is puzzling, especially given that the prospecive, selective effect of stress remained intact.

Aside from the explanations derived through the BTH, an alternative view on these findings can be extracted from the role of the amygdala on encoding in a state of elevated arousal. The amygdala, specifically the basolateral nuclei, play a key role in consolidation mechanisms under stress, and has been proposed to be the key site where interactions between chemical messengers mobilised during the stress response take place (McGaugh, 2004). Through these stress-driven interactions, the amygdala modulates memory formation in cortical and subcortical brain areas (McGaugh, 2004). Amygdala activity has been found to play a key role in encoding when arousal is high, regardless of the valence of the encoded information (Mather & Sutherland, 2011). Moreover, stress may induce a persistent neural state that may carry over and influence the encoding of new information (Tambini et al., 2017). Thus, there is a possibility that the tag-and-capture of weakly encoded events occurs in tandem with amygdala mediated encoding and the carry-over effects of emotional brain states. Such interactions of emotional brain states and behavioural tagging may shed some light on the inconsistent findings in Studies IIa and IIb.

The effects of stress on subsequent encoding have shown variable outcomes, with both enhancing (Domes et al., 2002; Goldfarb et al., 2019; Schwabe et al., 2008; Smeets et al., 2009; Vogel & Schwabe, 2016), impairing (Elzinga et al., 2005; Kirschbaum et al., 1996; Quaedflieg et al., 2013), or even null effects (Smeets et al., 2008). One possible explanation for these variable findings is fine differences in

the temporal distance between stress and encoding. The gradual rise and fall of cortisol may exert drastically different effects on memory depending on the exact moment of encoding relative to stressor onset, eventually shifting the brain to a consolidation mode whereby the storage of recently learned information is prioritized above new encoding (de Quervain et al., 2017; Schwabe et al., 2012, 2022; Shields et al., 2017). Given that we consistently found selective improvement of surprising information encoded after a stressful episode, this suggests two possible mechanistic insights.

The first possibility is that, while stress and elevated cortisol normally prioritizes consolidation above new encoding, select mechanisms, such as the occurrence of a PE, may bypass this consolidation mode, resulting in selective memory enhancement of surprising information. In mobilizing the stress response, the organism redirects resources across bodily functions in order to optimize chances of survival (Charmandari et al., 2005). Under such conditions of constrained cognitive and metabolic resources, the processing of incoming information may become increasingly selective, prioritizing information that is most relevant for adaptive behavior. Within predictive processing frameworks, the brain is thought to minimize free energy by reducing PEs through updating internal models or acting on the environment (Clark, 2014; Friston, 2010). Stress-related states of uncertainty may therefore enhance the prioritization of high-PE information, as PEs carry information relevant for improving predictions and guiding behavior.

Such "gating" by PEs may be partially driven by noradrenergic firing. PEs have been shown to result in activation of the LC-NE system and dopamine firing, as well as elevated arousal, measured via pupil size and SCR (Antony et al., 2021; Rouhani, Niv, et al., 2023; Stemerding et al., 2022). Importantly, noradrenaline, indexed by pupil size, has been proposed to track absolute uncertainty, similarly to how dopamine tracks reward prediction errors (Preuschoff et al., 2011). Stress related memory enhancement has been proposed to require both cortisol and noradrenaline signalling in the amygdala (McGaugh, 2004). Thus,

PEs may trigger noradrenaline signalling, which together with stress-related increases in cortisol could result in memory enhancements for unexpected information.

Moreover, learning from a stressful environment may be adaptive as it allows for more accurate event models and predictions in the future (Clark, 2013; Friston, 2010). Stressful situations are inherently unpredictable and may act as teaching signals, predisposing the organism to process ongoing events (Trapp et al., 2018). Elevations in noradrenaline have been suggested to trigger a heightened "epistemic appetite" whereby learning rate is increased (Proietti & Pezzulo, n.d.). Thus, stress-related mechanisms may give rise to a state in which errors are more efficiently propagated in order to optimize predictions made for the future. This perspective offers an explanation as to why post-stress encoding in Studies IIa and IIb resulted in more consistent memory enhancement than pre-stress encoding – experiencing stress may have enabled a state in which specifically surprising information is pushed through. However, it is important to note that Studies IIa and IIb did not show a direct dependency of the PE-driven memory benefit on noradrenaline activation as measured by pupil size and SCR. Previous research has shown that PE-driven memory enhancement occurs independently of parallel physiological arousal (Kalbe & Schwabe, 2020). In Studies IIa and IIb we found that PE was not consistently related to such physiological measures of surprise, but still enhanced memory together with stress. This suggests that there may be an underlying cognitive effect of PE which does not depend on arousal.

Aside from the "gating-by-PE" explanation, another possibility is that successful encoding after the occurrence of a stressful encounter occurs due to compounding memory enhancements of PE and stress. Memory enhancement of non-stress related information may require two things – the carry over of emotional brain states and sustained arousal or amygdala activity, as well as the selective tagging of some information. The neural signature of the learning tag has been proposed to constitute a temporal state of an activated synapse, consisting

of structural changes to the affected dendrite, rather than the functional plasticity processes initiated during e-LTP (Redondo & Morris, 2011). This raises the intriguing possibility that the occurrence of a stressor promotes subsequent structural dendritic changes. In other words – while the mechanisms of tagging may be similar for information encoded before and after a significant encounter, further amplification or silencing of this tagging process may be introduced through specific qualities in the significant event – such as the presence of stress and stress-related neurochemicals like cortisol.

4.1.1 Cortisol as a candidate plasticity related product

Stress is a complex actor in memory making – aside from the release of quickly acting neurotransmitters that impact moment to moment encoding, there are also slower effects of cortisol (Joëls & Baram, 2009; Schwabe et al., 2022). Cortisol in itself also has complex effects on memory – it affects both consolidation and subsequent encoding in dynamic ways. As it slowly increases, the consolidation of information learned at a certain distance in the past is prioritized at the expense of the encoding of new, unrelated information (de Quervain et al., 2017; Schwabe et al., 2012, 2022). Additionally, stress drives changes in brain states and reconfigures neural network activity, further adding nuance to the processes behind selective memory enhancement of stress (Hermans et al., 2011; Schwabe et al., 2022). These factors make it complicated to disentangle the tagging and capturing effects from more general, wide-spread stress and cortisol mediated changes on encoding and consolidation.

Furthermore, the question arises whether cortisol might qualify as a PRP within the framework of the BTH. Cortisol has previously been identified as a key driver of selective memory enhancement for emotional information in rats (Roozendaal et al., 2006). Noradrenaline, released either pharmacologically or due to novelty in the learning environment, acts in the basolateral amygdala together with cortisol to drive memory enhancement of emotional information (McGaugh, 2004;

Roozendaal et al., 2006). Thus, noradrenaline released due to PE may result in a temporary learning tag which can later be captured due to stress driven synthesis of PRPs, such as cortisol. Such framing of cortisol as a PRP is supported by some studies (Lopes da Cunha et al., 2018; Lopes da Cunha et al., 2019, 2022). While novelty is most commonly used as the significant event that triggers the synthesis of PRPs (Ballarini et al., 2009, 2013; Vishnoi et al., 2016), other studies have used stress instead (Lopes da Cunha et al., 2018; Lopes da Cunha et al., 2019, 2022). Importantly, one study identified cortisol as a necessary component of the tag-and-capture process (alongside Brain-derived neurotrophic factor and protein synthesis in the hippocampus) (Lopes da Cunha et al., 2019). These findings suggest that stress-driven cortisol changes can be interpreted as an integral part of behavioural tagging, rather than as a separate, independent contributor to memory enhancement via stress.

Nevertheless, even in the behavioural tagging framework, the literature has revealed inconsistencies in how pre- and post-stress encoding is affected. Some studies showed that stress enhanced memory for weakly encoded information learned before the stressor (Lopes da Cunha et al., 2018), and this depended on glucocorticoid activity (Lopes da Cunha et al., 2019). However, stress impaired memory for strong learning traces placed before the stressor (Lopes da Cunha et al., 2018; Lopes da Cunha et al., 2019). Meanwhile, when learning followed stress, memory was impaired (Lopes da Cunha et al., 2018; Lopes da Cunha et al., 2019). Further findings showed this to be the case only when the learning itself was already strong, whereas weak learning taking place after the stressor was enhanced, and this enhancement depended on glucocorticoids (Lopes da Cunha et al., 2022). These conflicting findings suggest that other mediators exist that further complicate how the BTH manifests with stress as the significant experience, such as the strength of original encoding. Taken together, stress can be considered a significant event as described by the BTH, possibly with cortisol acting as a PRP, but the stress-related cascades of various neurotransmitters and hormones

may complicate the memory enhancement generally produced by a significant experience, leading to unexpected memory outcomes for weak learning events encoded in the temporal periphery.

4.2 Disentangling tagging by PE from tagging by reward or emotional relevance

Disentangling the effects of PE from the effects of emotional arousal can be complex. Studies often used electrical shocks to condition a given image category with an aversive outcome (Kalbe & Schwabe, 2022b; Loock et al., 2025; Rouhani, Niv, et al., 2023). Measuring participants' explicit predictions of shock occurrence allows for finetuned measures of either positive or negative deviations from the predicted outcome. However, such shock studies necessarily make stimuli emotionally relevant, either in a rewarding way via unexpected shock omission, or in a penalizing way via unexpected shock occurrence. Similarly, gambling or game-based designs include both aspects of unexpectedness and reward or punishment at each trial, and study designs vary in the ability to detangle these processes (Antony et al., 2021; Huang et al., 2023; Preuschoff et al., 2011). Thus, this kind of task structure may inherently conflate the occurrence of a PE with emotionality, combining PE with either reward or punishment. In Studies IIa and IIb, we aimed to avoid this structure by exclusively using neutral stimulus material. Such separation is relevant, because emotional experiences may exert their memory shaping effects precisely due to their unpredictable nature (Kalbe et al., 2020; Trapp et al., 2018). However, even with exclusively neutral material, meta-learning of the encoding task may inherently introduce experiences of reward and punishment at each trial, based on moment-to-moment expectations about stimulus occurrence and task enjoyment. This opens the question whether PEs can truly be separated from emotional relevance, as well as how this emotional relevance would interact with an adjacent stressful experience.

4.2.1 Emotional relevance of the learning material and strength of encoding

Across Studies IIa and IIb, we found a selective memory enhancement of neutrally valenced, surprising information when a stressful encounter occurred in the temporal periphery. Whether this finding would be replicated for emotional learning material, such as emotionally arousing images, is unclear. Studies have shown that behavioural tagging and capture by an adjacent stressor only occurs when the non-significant learning experience leaves a weak memory trace (Lopes da Cunha et al., 2018; Lopes da Cunha et al., 2019, 2022). Stress may even impair the memory of previously encoded information, if that information left a strong memory trace (Lopes da Cunha et al., 2022). The strength of initial encoding thus seems to be a key determinant of the success of behavioural tagging. While the tasks used in Studies IIa and IIb were designed to induce varying levels of surprise, the PEs remained rather moderate. This was reflected in the lack of a general effect of PE on memory – we only found a memory enhancing effect of PE in the stress group, while there was no effect in the control group. This contrasts with findings from previous studies showing that PE typically enhances memory (Bein et al., 2021; Loock et al., 2025; Rouhani, Niv, et al., 2023), but can be interpreted as evidence that the PEs induced by our task resulted in a relatively weak learning trace, allowing it to be consolidated by the PRPs released by the stressor. Stress driven memory impairment of strongly encoded information in the periphery has been explained through competition of PRPs, resulting in suboptimal conditions for consolidation for both events (Lopes da Cunha et al., 2019).

Stress-induced impairment of memory for strongly encoded information is at odds with findings that stress can selectively enhance memory for emotional material, or for information that receives enhanced attentional processing at encoding (Cahill et al., 2003; Knight & Mather, 2009; Mather & Sutherland, 2011).

Specifically, it has been shown that stress may induce an attentional bias towards negatively valenced stimuli, which prioritizes the encoding of emotional material (Elzinga et al., 2005; Kensinger, 2009; Smeets et al., 2008). Such findings suggest that stress should specifically enhance the memory for information that left a strong or emotionally relevant learning trace. The time-dependent effects of stress on memory offer a lens through which to reconcile these findings with the predictions made by the BTH.

When encoding emotional material, the amygdala is being modulated top-down by the medial prefrontal cortex through cognitive control mechanisms, as well as bottom-up by noradrenergic activity in the locus coeruleus (Joëls et al., 2011). These two pathways may work together to create optimal conditions for peri-stress encoding. Noradrenaline and corticotropin-releasing-hormone, both key actors of the stress response, have been shown to exert LTP-like mechanisms in the hippocampus, and facilitate electrical LTP independently (Joëls et al., 2006). As cortisol levels begin to rise, learning is supported, but as time passes and cortisol reaches peak concentration levels, cortisol may suppress further encoding in order to prevent new, irrelevant information from interfering with the existing memory trace (Joëls et al., 2006; Schwabe et al., 2012). This suggests that, in this time-window, other mechanisms must take over for successful encoding to take place. Moderate PEs may offer this support. Importantly, we found no modulation of the PE-dependent memory enhancement by the distance to the stressor. In other words, PE enhanced memory in stressed participants even when cortisol levels were at their peak, as well as afterwards. Thus, in the short term, stress may support the encoding of emotionally relevant information with a strong learning trace, but as time passes, co-occurring signals of lingering emotional arousal and new emotional or strong stimulus material may result in a "system overload" that prevents the new information from being successfully encoded. Indeed, it has been suggested that stress prioritizes non-emotional encoding in order to minimize the potential of maladaptive memories (Ritchey et al., 2017). Information

that leaves a weaker memory trace, such as moderately surprising information, may land at a sweet spot of encoding strength, allowing it to benefit from ongoing PRP synthesis and release.

PE size has been found to influence memory in a graded manner, and has been suggested to follow a U-shaped curve whereby high and low PEs both may enhance memory, while moderate PEs generally result in the worst memory outcomes (Boeltzig et al., 2025; Van Kesteren et al., 2012; cf. Ortiz-Tudela et al., 2023). Our findings, together with the literature, indicate that there exists an optimal point of convergence at which PEs and emotional relevance co-occur to enhance memory. Specifically, moderately surprising information may generally not generate a LTM, but a stressful event resulting in PRP release can rescue these relatively weak memory traces. However, if the learning material would be emotionally salient, it would likely leave a stronger memory trace, and thus memory for the learning material may even be impaired by the occurrence of a subsequent significant event (Lopes da Cunha et al., 2019). An intriguing parallel to this pattern of results can be found in the shock literature, whereby unexpected shock omissions generally result in memory enhancement, while unexpected shock occurrence may result in memory impairment (Kalbe & Schwabe, 2022b; Loock et al., 2025). These findings support the notion that there is an optimal meeting point of PE size and emotionality at which memory enhancements can take place. However, this remains speculative, and further research is needed to determine whether stress enhances, impairs, or does not affect memory for strongly encoded information in the periphery.

4.2.2 A change is as good as a break – the inherent reward of PEs

Within the framework of reinforcement learning, PEs work as teaching signals that convey to the brain that the outcome deviates from what was expected. When a reward is larger than anticipated, this results in a larger spike in dopamine, while when the reward is lower than expected, this lowers dopamine signalling

(Schultz, 2016). Whether our design can be interpreted through a lens of reinforcement learning is not clear – as we technically worked with unsigned PEs which were inherently non-rewarding, the dopaminergic midbrain release that updates expectations within reinforcement learning may not be triggered. However, it is also possible that even neutrally valenced PEs are, at least within this kind of study design, inherently rewarding. This is because the encoding task still engaged goal-directed behaviour, and the PEs were directly informative to the task structure. Specifically, whenever the category switched, there was a change in behaviour and context, which may have produced a reward signal due to being confronted with an aha-experience (Becker & Cabeza, 2025). This perspective may offer an explanation to some divergent findings within the PE literature. Some research has shown that accurate rather than inaccurate predictions support memory formation when playing a board game (Huang et al., 2023). Similarly, probable moves were remembered better due to their recruitment of schema knowledge (Huang et al., 2023). Different study designs may thus encourage PE-driven memory enhancement or schema-driven memory enhancement, depending on whether the PE is inherently rewarding through being goal-relevant and informative for the task at hand (Nolden et al., 2024).

Taken together, disentangling the effects of PE on memory from emotional relevance and reward processing is challenging, and future studies should aim to distinguish whether these processes and their memory driving effects can be truly separated.

4.3 The role of processing speed and P300 latency in memory enhancement of surprising information

Based on the findings from Study IIb, the neural correlates underlying the selective memory enhancement of stress for surprising information remain unclear. However, we found that latency of the P300 component directed PE related mem-

ory outcomes differently depending on whether stress had recently been experienced or not. P300 latency has previously been linked to processing speed of incoming information (Donchin, 1981; Linden, 2005). In Study IIb we found that, in the non-stressed participants, a shorter P300 FPL, or faster processing of the incoming information, allowed for PE related memory benefits to occur to the same extent as if a stressor had occurred shortly before. Importantly, this difference in the role of P300 in the effect of PE on memory was completely driven by the control group – for stressed participants, the effect of PE on memory was equivalent, regardless of the latency of P300. Thus, under normal conditions, in the absence of stress, faster processing of high PE stimuli may support memory formation. Slower processing speed on the other hand may result in failed memory formation. Indeed, some research suggests that when learning material elicited a shorter P300, it was more likely to be recognized later on (Johnson et al., 1985). Given that P300 latency has been suggested to relate to stimulus organization, this suggests that successful categorization of incoming information may support successful memory formation (Donchin, 1981). How stress may impact stimulus categorization and its utility in memory formation is unknown. The findings of Study IIb suggest that, under stressed conditions, PE-driven memory enhancement occurs independently of processing speed as indexed by P300 latency. Thus, a shorter P300 latency may benefit encoding under normal circumstances, but becomes less relevant when under stress. These findings are however preliminary, and should be investigated in future studies.

4.4 Interpretation of the findings through event segmentation theory

The findings from the presented studies could also be interpreted through the perspective of event segmentation theory (EST; Zacks et al., 2007). Based on previous experiences, we construct event models that guide us through incoming

sensory input. As we navigate the world, we are faced with a continuous stream of incoming information. Dividing this continuous stream into distinct episodes in memory requires the perception of event boundaries – moments in time when we perceive that the context, goal, or perceptual features have changed (Zacks et al., 2007). These event boundaries mark the shift of one event model to another being active. Crucially, event boundary perception has been suggested to arise due to gradual increases in PEs (Zacks et al., 2007). Perceptual input is constantly compared against the predictions made by the ongoing event model, and when this sensory input is inconsistent with the prediction, a PE arises, resulting in the perception of an event boundary (Zacks et al., 2007). As PEs accumulate, our event model is updated to better match environmental demands. EST thus serves as a behavioural parallel of the predictive brain hypothesis (Clark, 2014).

Event boundaries have consistently been found to structure memory through guiding time perception, and enhancing memory for contextual information and experiences around the event boundary (Clewett & McClay, 2025; Clewett et al., 2020; Cowan et al., 2021). Event boundaries can be decoded from cortical activity patterns, and hippocampal activity at event boundaries predicts cortical reactivation during recall, suggesting an active role of the hippocampus in event segmentation (Baldassano et al., 2017). Dynamic changes in pupil size align with event segmentation, suggesting that transient elevation in arousal in the form of LC-NE signalling is a mechanism through which event segmentation occurs (Clewett et al., 2020). Through this lens, and considering the key role that PEs play in event boundary perception, the findings in Studies IIa and IIb can be interpreted as reflecting the detection and encoding of event boundaries. Based on findings showing that emotional arousal may linger and serve as a context that supports mnemonic integration (Clewett & McClay, 2025), the experience of a stressor may generate a context in which surprising information is more likely to result in event boundary perception, resulting in memory enhancement of information associated with PE. This notion is further supported by the idea that no-

radrenaline may trigger an elevated "epistemic appetite", whereby learning rate increases (Proietti & Pezzulo, n.d.).

Additionally, the task structure we applied in Studies IIa and IIb aligns with event segmentation. As we presented images belonging to the different subcategories sequentially, we created events that consisted of different image categories. Thus, the first image in each given category may be seen as a shift from one event to the next, causing an event boundary and driving the memory enhancement for boundary items. Importantly, the task used in Studies IIa and IIb allowed for internally generated PEs to be captured as well. Internally generated shifts in goals also allow for event segmentation to take place (Wang et al., 2023). Thus, although no transient spike in PE was necessarily expected within any given image category, interindividual variability in interests and personal history may have triggered PEs even within these categories. Indeed, interindividual differences in personality variables have been found to influence the memory benefit of surprising information (Kafkas et al., 2025). Whether such PEs would constitute event boundaries in the same way as the systematic shifts from one category to another is unclear.

As discussed above, some findings have shown a U-shaped relationship of PEs and memory whereby moderate PEs see the worst memory outcomes (Boeltzig et al., 2025; Van Kesteren et al., 2012). In the SLIMM model, highly unexpected information supports the creation of a new, detailed memory, whereas highly expected information supports the integration of the incoming information into existing knowledge structures (Van Kesteren et al., 2012). Moderately surprising information that neither fits into existing schema nor causes the creation of a separate memory trace is thus the most prone to fade from memory (Van Kesteren et al., 2012). However, if an acute stressor occurs within a critical time window, this may shift processing of such moderately surprising information towards either schema-based encoding or the creation of a separate memory trace, as learning from even this intermediate level of schema-mismatching information may

be adaptive for updating event models. Thus, moderate surprise or lower level event boundaries may be retrospectively identified as significant.

While EST originally proposed PEs as the key mechanism resulting in event segmentation (Zacks et al., 2007), later accounts have demonstrated that PEs are neither necessary nor sufficient for boundary detection (Güler et al., 2023). For instance, predictable events still cause event boundaries to be perceived (Pettijohn & Radvansky, 2016). Accordingly, we found that participants showed signs of meta-learning throughout the tasks in Studies IIa and IIb, with PE ratings gradually drifting towards lower values. Thus, as the experiment progressed, participants likely learned to expect a category switch after a certain number of images. Nevertheless, the joint effects of PE and stress on memory did not depend on the number of trials that passed between stress and encoding, suggesting that while there was a drift in subjective PE ratings, this did not reduce the mnemonic advantage for images rated as more surprising. Further, we found that task irrelevant oddballs did not result in any memory improvement, suggesting that higher level processing of the stimulus is necessary for the combined memory benefit of PE and stress. It is thus possible that two mechanisms contributed to the memory enhancement for PEs in Studies IIa and IIb – on the one hand, memory enhancements occurred due to event segmentation based on category switches in the task, while on the other, due to internally generated, task-relevant PEs and event boundaries.

Emotional arousal, induced by strong emotional sounds, lingers to mnemonically tie discrete episodes together (Clewett & McClay, 2025). Aligned with this, in Study I, we expected that a cue to an emotional encounter would result in memory improvement for subsequently encoded, non-stress related material. We did not find evidence for this hypothesis. These findings can also be interpreted through an EST lens – the cues that were used to reactivate the memory for the stressful experience in Study I may not have induced emotional arousal to the necessary extent. The transient nature of an image cue, rather than a continuous

incoming stream of emotional music, may not provide a salient enough context to tie ongoing events together. The cue to a stressful situation may thus not result in a strong contextual switch. Event segmentation occurs at several levels and granularities, and thus, even if a lower level event boundary is perceived due to transient reactivation of a stressful memory, this reactivation may not be sufficient to create a shift in mental state that lingers for several seconds, allowing for enhanced encoding and binding together of subsequent information.

4.5 Cueing as a tagging mechanism

In Study I, while we did not find evidence for the proposed cueing mechanism, we nevertheless found that stress retrospectively enhanced memory for neutral information encoded before the stressor, specifically when the stressor was also well represented in memory. This finding aligns with the BTH – weakly encoded information was retrospectively consolidated into LTM due to experiencing a significant encounter. The fact that this effect depended on how vivid the memory for the stressful experience was, suggests that the significant experience has to be clearly represented in memory in order for the tag-and-capture mechanism to take place. In this way, the memory for the significant experience can be gauged as an indicator for whether the capturing will take place or not. Interestingly, while cortisol has previously been identified as a potential driver of tag-and-capture (Lopes da Cunha et al., 2019), we did not find strong evidence for the role of cortisol in the mnemonic enhancement of information encoded before a stressful event. While stressed participants with a stronger cortisol response showed the highest overall pre-stress memory enhancement on average, this effect remained nonsignificant. This suggests that several cellular mechanisms lie behind the capturing of tagged events (for instance, brain-derived neurotrophic factor and hippocampal synaptogenesis; Lopes da Cunha et al., 2019).

One of the key postulates of the BTH is that the tag and PRPs need to occur at the same neural substrate, or at the very least, in overlapping neural networks

(Moncada et al., 2015). For instance, open field exploration in mice engages hippocampal learning, and thus retrospectively enhances the memory for hippocampus dependent weak object learning and fear conditioning tasks (Ballarini et al., 2009). In contrast, non-hippocampal learning, such as conditioned taste aversion, was not enhanced by open field exploration, but was instead enhanced by experiencing a novel taste (Ballarini et al., 2009). Thus, the weak learning experience must engage similar learning pathways as the significant event. Such overlap implies that the significant event that results in the release of PRPs may become directly associated with the weak learning experience.

This raises the question of whether the weak learning events and significant experiences used in the present studies recruited enough of the same neural networks in order for the requirement of spatial overlap to be met. During stress, salience network activity is upregulated in order to aid in the encoding of stress-relevant information (Schwabe et al., 2022). Similarly, PEs have been associated with increased activity and connectivity in parts of the salience network (Kalbe & Schwabe, 2022b). Thus, it appears that stress and PEs may recruit parts of the same neural networks during encoding, possibly resulting in sufficient neural overlap for the PRPs released by stress to reach tags placed due to the occurrence of PEs. Future studies should investigate whether such overlap in neural network activity is paralleled by increased representational similarity of surprising material and stress-relevant information.

Meanwhile, in Study I, there was no specific feature of the encoded information that would mark it for possible relevance. Rather, its relevance was directly dependent on the context cue that preceded the stimulus. Aligned with the requirement of shared neural substrates, the proposed cueing mechanism requires engram overlap of the two memories (Josselyn & Frankland, 2018). In order for this overlap to occur, the memory trace for the stressor has to be successfully reactivated. As it is unclear whether the cue successfully reactivated the memory trace of the stressful episode in Study I, this may be another explanation for why

we did not find evidence for the cueing mechanism. Previous research has found that stressors result in increased neural similarity of central features of the stressful episode, and more distinct representations for peripheral features (Bierbrauer et al., 2021). Thus, general contextual cueing may be inefficient for reactivating the complex neural trace left by a stressful episode. Future studies could take advantage of this centrality bias to investigate whether the neural representational similarity of central features supports the memory for surrounding neutral information.

4.6 Multiple drivers of peri-stress memory enhancement

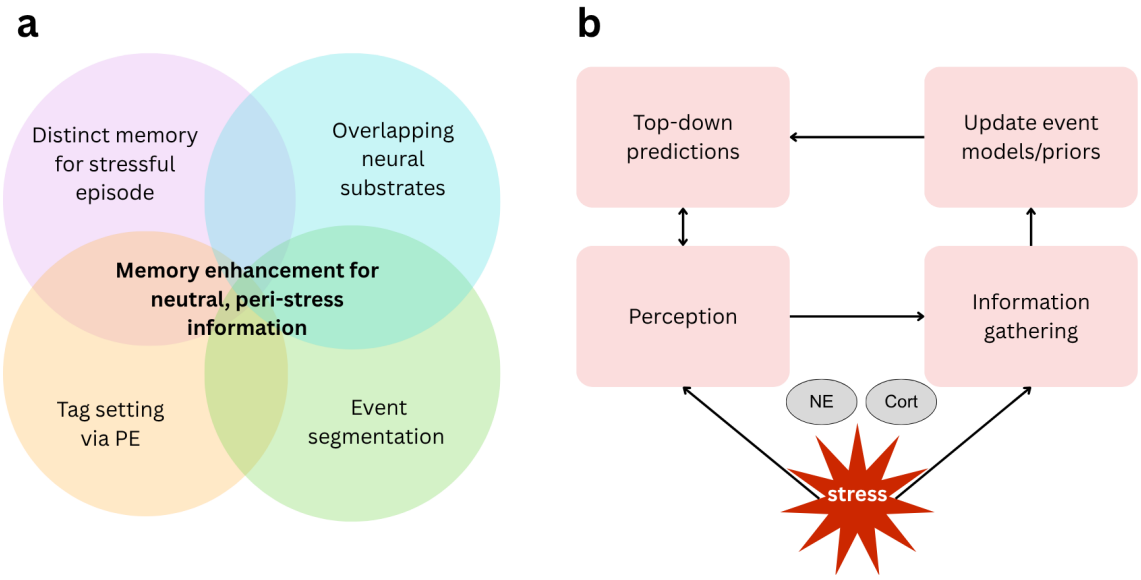

Figure 4.1 Stress can selectively enhance the memory for neutral information encoded in the periphery (a). This enhancement depends in part on the distinctiveness of the memory for the stressful experience, the prediction error (PE) associated with the encoded material, internally or externally generated event boundaries, and whether there is overlap in neural substrates for the stressor and encoded material. The general purpose of selective memory enhancement of stress may be to optimize the updating of event models and priors to facilitate more successful future predictions (b). The release of noradrenaline (NE) and cortisol (Cort) together contribute to a shift towards information gathering.

Allow us to return to your final examinations at school. You may remember what the room looked like; a clock on the wall, soft hum of the air conditioner, your friend sitting in front of you. Perhaps you can also remember other details from that day, even neutral, mundane things – the funny looking dog you saw in your neighbourhood on your way to the exam, the heavy rain that surprised you on your way home, or running into a friend you had not seen for a while outside the exam hall. Why do these details stick out in your memory? The studies presented here demonstrate various key factors that contribute to this kind of memory enhancement.

If the exam was more difficult than you anticipated, you might have felt very stressed, rather than just nervous. All three studies presented here suggest that, during such a stressful experience, central aspects of the situation become well represented in your memory. Central details are likely to be remembered well, while peripheral details may fade. Study I further demonstrated that when we form this kind of specific memory for the stressful event, there is a significant improvement for information encoded shortly before the onset of the stressor. A strong memory trace for the stressor may exert a more generalized, retrospective memory enhancement on previously encoded information, aligning with previous accounts of behavioural tagging (Ballarini et al., 2009, 2013; Lopes da Cunha et al., 2019).

Previous research has suggested that this memory enhancement may be selective for certain kinds of information. For instance, the emotionality of the encoded content (Elzinga et al., 2005; Smeets et al., 2008), or relatedness to the stressor (Bierbrauer et al., 2021) have both been found to receive selective memory enhancement due to stress. Study IIa and IIb demonstrated that experiencing a stressful encounter can selectively enhance your memory for information that induces a PE. In Study IIa we further found that surprising information encoded before the stressor was also more likely to be remembered. Thus, PE arises as a new factor allowing selective memory enhancement of stress on surrounding in-

formation. On the day of your exams, experiencing a PE due to taking a different bus route to the exam hall, or unexpectedly running into a friend, may form a stronger memory than on some other day when there was no exam. Meanwhile, mundane and expected parts of the exam day may not see this memory enhancement. The BTH assumes the co-occurrence of the learning tag and the significant encounter in overlapping neural substrates. The involvement of regions of the salience network in both PE and stress related encoding (Kalbe & Schwabe, 2022b; Schwabe et al., 2022) suggests that PEs could meet this requirement of the tag-and-capture process.

Finally, event segmentation may contribute to the memory enhancement for neutral information encountered before or after a stressor because transient shifts in emotional arousal can create a context in which events are effectively tied together into coherent episodes (Clewett & McClay, 2025). Moreover, a stressful event results in a lingering emotional neural state, biasing future encoding (Tambini et al., 2017). Another possibility is that event boundaries are, at times, retrospectively identified. When we are exposed to an acute stressor, this may trigger the automatic reprocessing of events that were experienced in the recent past. Specifically, events that were marked for possible future relevance may here be re-evaluated to assess whether they can be adaptively built into a new event model. Thus, event segmentation may contribute to the memory for peri-stress encoded neutral information through both tying events together and by re-assessing former experiences for distinct event boundaries (Fig 4.1.a).

4.6.1 Why so specific – information gathering as a general purpose behind selective memory enhancement of stress

A common feature of EST and the predictive brain hypothesis is that they both imply that a general purpose of cognition and the brain is to be a sense-maker. In EST, event models are used to process incoming information based on pre-existing knowledge of the world, and an unexpected shift in the environment

sends a signal that the event model should be updated or switched (Zacks et al., 2007). Meanwhile, the predictive brain hypothesis conceptualizes the brain as a generative model, whereby, at each layer of processing, top-down predictions are generated and only information that contradicts those predictions is propagated up in the hierarchy for further processing (Clark, 2013). The purpose of selective memory enhancement during a stressful situation could be seen to be driven by this kind of sense-making process inherent to EST and the predictive brain hypothesis.

When stress occurs, it is adaptive to find the underlying causes of the current state (Peters et al., 2017). Stress related neurochemical cascades may facilitate this sense-making process through various effects. Elevated noradrenaline may trigger an elevated epistemic appetite (Proietti & Pezzulo, n.d.) alongside facilitating efficient encoding through interactions with cortisol in the amygdala (McGaugh, 2004). HPA axis activity, specifically through corticotropin-releasing factor, also triggers tonic mode of LC-NE firing that amplifies scanning in environment (Valentino & Van Bockstaele, 2008). Moreover, increased activity of the salience network allows for prioritized processing of stress-relevant information (Schwabe et al., 2022). The brain can thus be understood as being in an information gathering state during stress (Fig 4.1.b). In order to efficiently cope and adapt for the future, the brain may seek to efficiently update the active event models or predictions generated at higher layers.

Different factors associated with selective memory enhancement around a stressor may therefore provide useful insight into the latent causes of a stressful environment. Research has identified several such factors, including the emotional salience of the encoded information, its central relevance to the stressor, and a prioritization of cue-based learning (Bierbrauer et al., 2021; Kensinger, 2009; Simon-Kutscher et al., 2019). These factors may receive stress-driven selective memory enhancement due to their value in facilitating precise future predictions, while preventing overgeneralization. Based on Studies IIa and IIb, selec-

tive memory enhancement may additionally be afforded to high-PE information, possibly due to the epistemic value of surprising content when updating event models and adjusting priors.

Further nuance to the processing of PEs is brought through their assigned precision – the "volume" or weighting of the PEs, regardless of size (Clark, 2013; Friston, 2010). Brains cannot simply revise top-down hypotheses whenever a PE occurs because sensory signals are noisy. Instead, an additional precision parameter judging the informativeness of the PE has to be computed (Friston, 2010). This weighting essentially governs the influence of bottom-up influence on top-down expectations (Friston, 2010). This precision parameter has been proposed to underlie the personality trait that shapes sensory processing sensitivity (SPS) (Greven et al., 2024). SPS is characterized by greater sensitivity to stimuli, emotional reactivity, and deep processing (Greven et al., 2024). SPS has been linked to both positive and negative outcomes, including higher levels of depression, anxiety and burn-out, but also higher creativity and resilience (Greven et al., 2024). Recently it has been proposed that the mechanistic explanation for SPS involves generally greater precision for sensory input (Greven et al., 2024). In other words, more incoming information is flagged as important and stored for the future. This also involves deeper model updating in response to errors, as well as a stronger response to PEs (Greven et al., 2024). In short, brains of high-SPS individuals assign high precision to incoming signals (Greven et al., 2024). This profile parallels the information gathering state proposed here to be triggered by stress. This raises the question whether a stressed state may drive a general shift toward a temporary state of high SPS. Further, future studies should address whether elevated baseline stress plays a role in the development of SPS.

Thus, an overarching theory of selective memory enhancement by stress may be summarized in alignment with the predictive brain hypothesis – the brain stores what helps it guess better in the future, and stress may amplify this process by adjusting the precision assigned to incoming information.

4.7 Conclusion

These three studies have demonstrated that your memory for the day of your final exams is shaped by several factors, but we can point out reasons for why you may remember some things better than others. As mentioned above, events that were slightly out of the ordinary, such as a different bus route or running into your friend, may be remembered better because of the PE attached to these events. Let us say your friend who sat in front of you afterwards has an even more specific memory for the exam situation – he also remembers the look of the pages, the pen in his hand, the water bottle he chose that morning. He may remember even more events from his morning due to the general retrospective memory enhancement that stress induced for him.

The findings presented here can help us understand why stress has such variable effects on the memory of surrounding events. People often feel that stress impairs their memory, but the body of literature as well as the three studies presented here reveal a more complicated picture. Through understanding the circumstances under which stress may in fact improve memory, we may be able to harness the stress-driven memory enhancements for improving learning outcomes in educational settings. Furthermore, we can better understand the distorted memories reported by eyewitnesses in courtroom settings, and identify drivers of maladaptive memory for peri-traumatic events in patients with post-traumatic stress disorder. Importantly, a deeper appreciation and sympathy for ourselves can be fostered by understanding why some details stand out and why others fade. Exposing ourselves to novel situations that are likely to stimulate the release of PRPs, even if we are nervous, will help us remember other events from that day as well. During the Covid-19 pandemic, the lockdown resulted in a compressed sense of remembered time (Rouhani, Stanley, et al., 2023). People frequently report subjective dissatisfaction with their memory, and this number seems to be on the rise (Begum et al., 2014; González García et al., 2024; Luck et

al., 2018). Subjective memory complaints are often related to worry and anxiety, even in the absence of objective metrics of memory problems (Pearman, 2021). A better understanding of how memories are formed around significant experiences or daily stressors can help foster more empathy and self-compassion, and promote agency and a feeling of control over one's own memory problems. While you might not have exactly enjoyed yourself during your final exams, you are left with memories of running into your friend after, having a nice chat, and seeing a funny looking dog on the way to the exam hall. Perhaps this made it all worth it.

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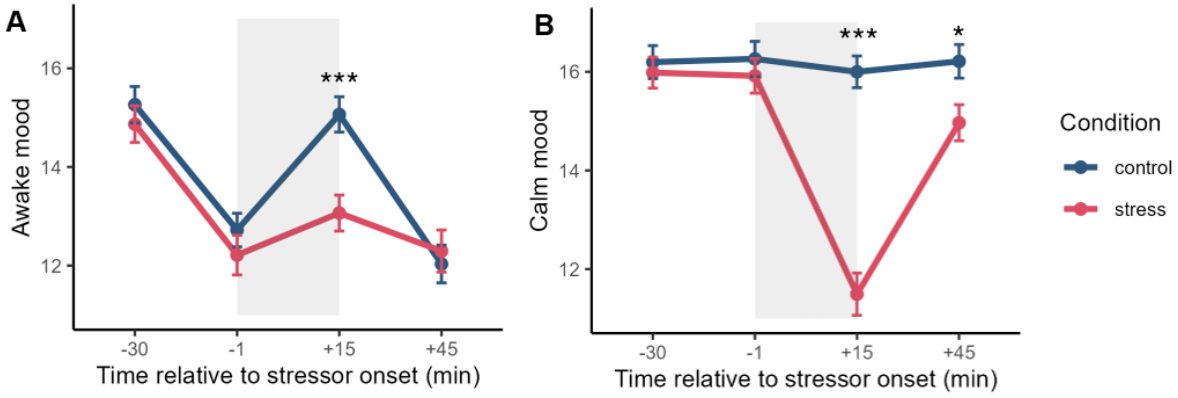
Supplementary materials

Supplemental Figure S1. Mood ratings in Study I.



Note. After the treatment, stressed participants rated their mood as less awake (A) and less calm (B) than control participants. Error bars show SEM. (***) P < .001.

Supplemental Table S1. Baseline measurements of chronic stress, depressive and anxiety symptoms, sleep, state anxiety, autonomic activity, mood and salivary cortisol in Study I.

	Day 1		Day 2	
	Control	**Stress**	**Control**	**Stress**
Trait chronic stress (TICS)	77.2 (29.1)	76.9 (27.9)		
Trait depression and anxiety (IDAS II)	181.0 (39.6)	175.0 (36.3)		
Sleep quality	75.8 (17.4)	73.7 (15.6)	70.2 (20.0)	73.8 (17.6)
Sleep duration (h)	7.5 (1.3)	7.5 (1.1)	7.3 (1.3)	7.5 (1.3)
State anxiety (STAI-S)	40.5 (6.3)	40.8 (7.2)	38.8 (6.5)	41.8 (7.0)
Systolic blood pressure (mmHg)	119.0 (14.7)	118.0 (13.3)	116.0 (13.5)	118.0 (11.1)
Diastolic blood pressure (mmHg)	83.7 (9.5)	81.0 (7.8)	79.7 (7.7)	79.9 (7.2)
Pulse (BPM)	84.1 (11.4)	81.0 (14.9)	87.7 (15.1)	84.6 (14.9)
Mood (good)	17.3 (2.3)	17.5 (1.9)	17.2 (2.4)	17.0 (2.3)
Mood (awake)	15.3 (2.9)	14.9 (2.9)	15.1 (2.9)	14.7 (3.2)
Mood (calm)	16.2 (2.6)	16.0 (2.5)	16.5 (2.4)	15.7 (3.0)
Log cortisol (nmol/L)	1.5 (0.5)	1.5 (0.5)	1.5 (0.5)	1.4 (0.5)

Means and (SD) are shown.

Supplemental Table S2. Predicted probabilities of recognizing material that cues stressor relevant vs. irrelevant information in Study I.

	Pre-treatment encoding		Post-treatment encoding	
	Control	**Stress**	**Control**	**Stress**
Stressor context	0.439 (0.186)	0.516 (0.191)	0.426 (0.181)	0.409 (0.178)
Lure context	0.443 (0.185)	0.500 (0.190)	0.405 (0.179)	0.405 (0.177)
Control context	0.465 (0.188)	0.517 (0.194)	0.421 (0.180)	0.419 (0.179)

Means and (SD) are shown.

Supplemental Table S3. Predicted probabilities of recognizing material that cues stressor relevant vs. irrelevant information depending on cortisol response in Study I.

	Pre-treatment encoding			Post-treatment encoding		
	Control	High cortisol response (stress)	Low cortisol response (stress)	Control	High cortisol response (stress)	Low cortisol response (stress)
Stressor context	0.427 (0.187)	0.567 (0.218)	0.472 (0.156)	0.422 (0.183)	0.439 (0.198)	0.390 (0.148)
Lure context	0.434 (0.187)	0.545 (0.214)	0.448 (0.159)	0.397 (0.180)	0.452 (0.200)	0.368 (0.145)
Control context	0.454 (0.190)	0.566 (0.218)	0.470 (0.162)	0.414 (0.182)	0.463 (0.203)	0.377 (0.146)

Means and (SD) are shown.

Supplemental Table S4. Predicted probabilities of recalling material that cues stressor relevant vs. irrelevant information in Study I.

	Pre-treatment encoding		Post-treatment encoding	
	Control	**Stress**	**Control**	**Stress**
Stressor context	0.069 (0.034)	0.086 (0.041)	0.047 (0.019)	0.058 (0.022)
Lure context	0.069 (0.037)	0.068 (0.038)	0.057 (0.021)	0.046 (0.017)
Control context	0.083 (0.038)	0.089 (0.043)	0.060 (0.023)	0.059 (0.023)

Means and (SD) are shown.

Supplemental Table S5. Predicted probabilities of recalling material that cues stressor relevant vs. irrelevant information depending on cortisol response in Study I.

	Pre-treatment encoding			Post-treatment encoding		
	Control	High cortisol response (stress)	Low cortisol response (stress)	Control	High cortisol response (stress)	Low cortisol response (stress)
Stressor context	0.065 (0.031)	0.088 (0.051)	0.086 (0.032)	0.045 (0.017)	0.060 (0.023)	0.056 (0.021)
Lure context	0.067 (0.036)	0.077 (0.038)	0.060 (0.031)	0.061 (0.026)	0.045 (0.016)	0.047 (0.019)
Control context	0.081 (0.037)	0.103 (0.053)	0.076 (0.034)	0.058 (0.023)	0.064 (0.024)	0.056 (0.022)

Means and (SD) are shown.

Supplemental results (Study I)

Response bias in memory for stressful episode. In order to control for response bias in the memory for the stressful episode, we computed d' scores by subtracting the participant's z-transformed false alarm rate from the z-transformed hit-rates for central and peripheral objects. We subsequently ran a mixed ANOVA with condition (stress vs. control) and object type (central vs. peripheral) as predictors. We found a near-significant condition x object type interaction ($F(1, 118) = 3.63$, $p = .059$, $\eta^2 = .010$) as well as a significant effect of object type ($F(1, 118) = 224.97$, $p < .001$, $\eta^2 = .388$). Pairwise comparisons showed improved memory for central objects in the stressed compared to control participants ($t(118) = -2.122$, $p = .036$). For peripheral objects there was no difference ($t(118) = 0.231$, $p = .818$). These findings show a slightly different pattern than what we saw for hit-rates. Specifically, for hit-rates, we did not find a significant difference between stressed and control participants for central objects.

We next ran a mixed ANOVA with salivary cortisol response (high vs. low vs. control) and object type (central vs. peripheral) as predictors. We found a main effect of object type ($F(1, 109) = 200.94$, $p < .001$, $\eta^2 = .380$) and an interaction of object type and responder ($F(2, 109) = 3.54$, $p = .032$, $\eta^2 = .021$). Tukey corrected post-hoc test showed no significant difference for central (all $|t| < 2.262$, all $p > .065$) or peripheral objects (all $|t| < 0.891$, all $p > .647$) depending on the cortisol response. These results reflect the same pattern as the hit-rates revealed.

Response bias in memory for events surrounding the stressful episode. Similarly, we computed d' scores by subtracting the z-transformed false alarm rate from the hit-rates for images encoded before or after the treatment. We ran a mixed ANOVA with condition (stress vs. control) and phase (pre vs. post treatment) as predictors. We found a significant main effect of phase ($F(1, 120) = 98.63$, $p < .001$, $\eta^2 = .029$) and an interaction of condition and phase ($F(1, 120) = 19.03$, $p < .001$, $\eta^2 = .006$). However, Tukey corrected post-hoc tests showed no significant differences between stressed and control participants in the pre-treatment encoding ($t(120) = -1.677$, $p = .096$) nor the post-treatment encoding phase ($t(120) = -0.025$, $p = .980$). These findings are in accordance with what we found with respect to analysis of hit-rates.

We next ran a mixed ANOVA with salivary cortisol response (high vs. low vs. control) and phase as predictors, and again found a significant main effect of phase ($F(1, 111) = 105.85$, $p < .001$, $\eta^2 = .033$) as well as an interaction of cortisol response and phase ($F(2, 111) = 10.46$, $p < .001$, $\eta^2 = .007$). The Tukey corrected pairwise comparisons showed a significantly higher d' in stressed participants with a high cortisol response, compared to control participants, for information encoded before the stressor ($t(111) = -2.568$, $p = .031$). No other follow-up tests were significant (all $|t| < 1.659$, all $p > .226$). Also these findings agree with what we found for hit-rates.

We also ran linear regression models with pulse, systolic and diastolic blood pressure as predictors in order to assess how autonomic nervous system activity might contribute to response bias corrected recognition memory performance of material encoded before or after a stressor. The interactions of condition, phase and pulse or diastolic blood pressure were nonsignificant (both $|\beta| < 0.058$, both $p > .113$). The interaction of condition, phase and

systolic blood pressure was significant ($\beta = -0.082$, $p = .045$). However, follow-up analyses showed no significant effects (all $|\beta| < 0.151$, all $p > .999$, Holm-Bonferroni corrected).

Next, we ran a linear regression model with condition, phase and centrality bias as predictors. The pattern of results was again the same, with a significant condition x phase x centrality bias interaction ($\beta = -0.084$, $p = .049$). However, follow-up simple slopes analyses showed no significant effects (all $|\beta| < 0.374$, all $p > .131$, Holm-Bonferroni corrected).

Finally, we ran a linear model with salivary cortisol response, phase, and centrality bias as predictors. Here, we found a significant interaction of low responder group, phase and centrality bias ($\beta = -0.113$, $p = .040$). Simple slopes analyses showed that high cortisol responders with a high centrality bias recalled more objects from the pre-encoding phase compared to controls ($\beta = 0.548$, $p = .030$, Holm-Bonferroni corrected). All other effects were nonsignificant (all $|\beta| < 0.353$, all $p > .100$, Holm-Bonferroni corrected). This mirrors the pattern of results we saw when analysing hit-rates.

Supplemental Table S6 Preregistration deviations.

#	Details		Original wording	Deviation description	Extent of deviation	Judgement of impact
1	Study	#2	"The difference between the memory for central vs. peripheral details will be directly related to the effect of the treatment or control episode on PE memory."	We found an unexpected 3-way interaction in our main analysis (PE × group × encoding phase). We opted not to follow up with a 4-way interaction including stressor memory due to lack of power for 4-way interactions.	Minor	As this analysis was not at the heart of the project, for brevity we opted to focus the manuscript on the core hypotheses pertaining to the interacting effect of stress and prediction error instead. The risk of this change is unknown. Evaluating the risk of bias with further analyses would require gathering more data.
	Type	Research questions / hypotheses				
	Reason	Other (Please Explain)				
	Timing	After data access				
2	Study	#2	"Visual oddballs will result in elevated mismatch negativity during stimulus presentation, but this will be unrelated to memory performance"	During data analysis, we did not find any memory enhancement for visual oddballs. We opted to focus our manuscript on the memory findings instead of adding extensive additional analyses focusing on perceptual rather than semantic prediction errors.	Minor	The risk of this change is minimal. Inclusion of visual oddball analyses would not substantially change any of the conclusions of the manuscript as is.
	Type	Research questions / hypotheses				
	Reason	Other (Please Explain)				
	Timing	After data access				
3	Study	#2	"For the physiological measures obtained during encoding (skin conductance response, pupil dilation), we will first assess their correlation with PE-rating (Pearson's R)."	During analysis, we found that the skin conductance and pupil dilation did not fulfil assumptions for Pearson's R calculation. We thus opted for Spearman's rho instead, as it is robust to those assumptions.	Minor	The risk for bias is low. Spearman's rho has been established to be preferable over Pearson's r with non-normal distributions and in the presence of outliers (de Winter et al., 2016).
	Type	Analyses				
	Reason	New knowledge				
	Timing	After data access				

4	Study	#2	"Where correction for multiple corrections are needed (e.g. for simple-slopes follow up analyses), we will apply the Holm-Bonferroni correction."	We opted to use FDR instead of Holm-Bonferroni correction as it was more flexible to use across all analyses where post-hoc testing was done.	Minor	The risk for bias is low. FDR yields similar outcomes in analyses with relatively few comparisons (Benjahmini & Hochberg, 1995).
	Type	Analyses				
	Reason	New knowledge				
	Timing	After data access				
5	Study	#1 & 2	"If there are significant interactions, we will complete post-hoc tests using simple slopes analyses."	Instead of simple slopes analysis, we conducted post-hoc tests for regression analyses using pairwise comparisons of slopes. This change was motivated by the discovery of significant 3-way interactions, where our main interest was in the difference between slopes for the stress and control group rather than the absolute magnitude of the regression slopes.	Minor	As the main focus of our analyses was specifically the difference between stressed and control participants with regards to the effect of PE on memory, the risk of this change is low.
	Type	Analyses				
	Reason	New knowledge				
	Timing	After data access				

Supplemental Table S7. Mood ratings in Study IIa.

	Start of Day 1		Before treatment		After treatment		End of Day 1		Start of Day 2	
	Control	**Stress**	**Control**	**Stress**	**Control**	**Stress**	**Control**	**Stress**	**Control**	**Stress**
Mood (calm)	4.0 (0.6)	3.8 (0.6)	4.1 (0.7)	3.9 (0.8)	4.1 (0.8)	3.0 (0.9) ***	4.1 (0.6)	3.6 (0.9) **	4.06 (0.8)	3.99 (0.6)
Mood (awake)	3.8 (0.7)	3.8 (0.7)	3.2 (0.8)	3.3 (0.9)	3.7 (0.8)	3.4 (0.9)	3.2 (0.8)	3.0 (0.9)	3.75 (0.8)	3.72 (0.7)

Note. Mean and SD mood ratings shown. (***) $p < .001$ and (**) $p < .01$ for stress compared to control group.

Supplemental Table S8. Regression table of winning model in Study IIa.

Predictors	*Odds Ratios*	*CI*	*p*
(Intercept)	0.45	0.36 – 0.56	**<0.001**
PE	1.13	1.08 – 1.19	**<0.001**
condition	1.00	0.74 – 1.35	0.995
phase	0.70	0.65 – 0.75	**<0.001**
distance scaled	1.24	1.19 – 1.29	**<0.001**
PE × condition	0.91	0.86 – 0.98	**0.008**
PE ×phase	1.07	1.02 – 1.13	**0.005**
PE × distance	1.01	0.99 – 1.03	0.439
condition × phase	1.26	1.15 – 1.39	**<0.001**
condition × distance	1.00	0.95 – 1.05	0.997
phase × distance	0.87	0.83 – 0.92	**<0.001**
Random Effects			
σ^2	3.29		
$\tau_{00\ participantID}$	0.61		
$\tau_{11\ participantID.PE}$	0.01		
ICC	0.16		
$N_{\ participantID}$	112		
Observations	35541		
Marginal R^2 / Conditional R^2	0.016 / 0.171		

Note. Odds ratios, confidence intervals (CI) and significance of predictors in winning regression model; PE = prediction error.

Supplemental Table S9. Mood ratings in Study IIb.

	Start of Day 1		Before treatment		After treatment		End of Day 1		Start of Day 2	
	Control	**Stress**	**Control**	**Stress**	**Control**	**Stress**	**Control**	**Stress**	**Control**	**Stress**
Mood (calm)	4.06 (0.7)	4.00 (0.7)	3.80 (0.8)	4.01 (0.8)	3.95 (0.7)	3.21 (1.0) ***	3.49 (0.8)	3.56 (1.0)	4.18 (0.7)	4.10 (0.7)
Mood (awake)	3.74 (0.7)	3.82 (0.7)	2.86 (0.8)	3.07 (1.1)	3.46 (0.8)	3.47 (0.9)	2.46 (0.8)	2.75 (1.0)	3.82 (0.7)	4.13 * (0.7)

Note. Mean and SD mood ratings shown. (***) $p < .001$ and (*) $p < .05$ for stress compared to control group.

Supplemental Table S10. Regression table of winning model, Study IIb.

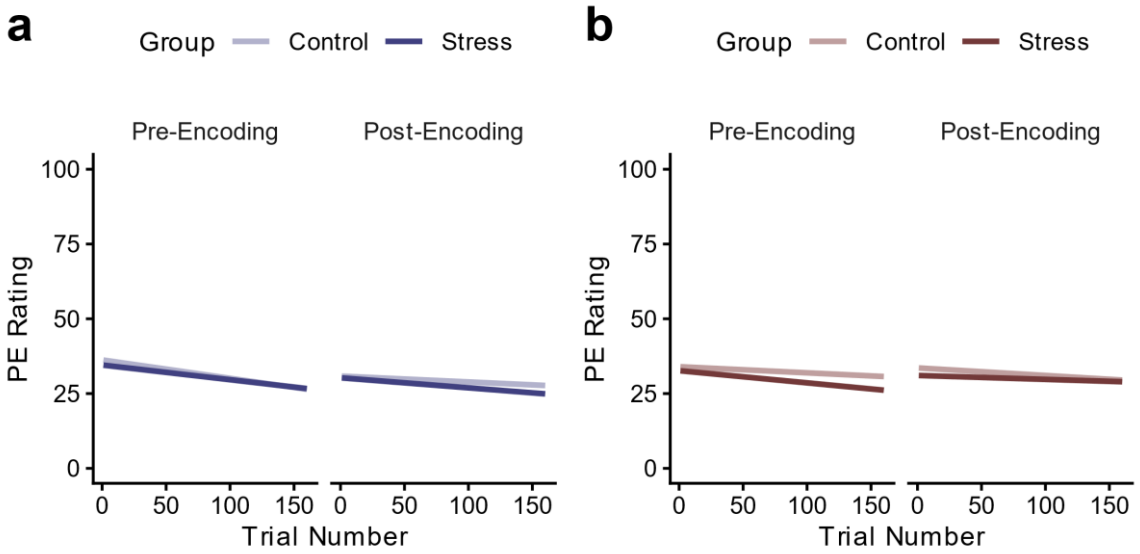
Predictors	*Odds Ratios*	*CI*	*p*
(Intercept)	0.42	0.34 – 0.51	**<0.001**
PE	1.04	0.98 – 1.10	0.225
condition	1.06	0.80 – 1.41	0.674
phase	1.04	0.97 – 1.12	0.254
distance	1.03	0.98 – 1.08	0.309
PE × condition	0.94	0.87 – 1.03	0.174
PE × phase	0.95	0.89 – 1.03	0.202
PE × distance	0.99	0.94 – 1.03	0.567
condition × phase	0.77	0.69 – 0.85	**<0.001**
condition × distance	0.92	0.85 – 0.99	**0.020**
phase × distance	1.01	0.94 – 1.09	0.792
PE × condition × phase	1.18	1.06 – 1.31	**0.002**
PE× condition × distance	0.99	0.94 – 1.04	0.615
PE × phase × distance	1.00	0.95 – 1.05	0.914
condition × phase × distance	1.12	1.01 – 1.24	**0.031**

Random Effects

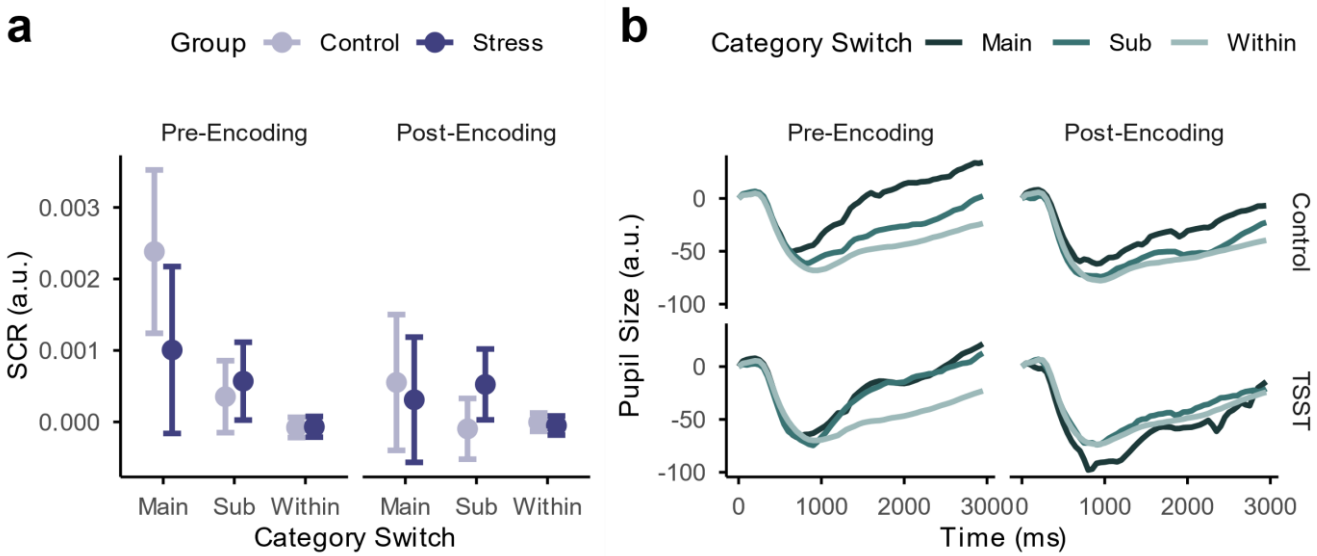
σ^2	3.29
$\tau_{00\ participantID}$	0.47
$\tau_{11\ participantID.PE}$	0.01
ICC	0.12
$N_{\ participantID}$	94
Observations	29226
Marginal R^2 / Conditional R^2	0.003 / 0.127

Note. Odds ratios, confidence intervals (CI) and significance of predictors in winning regression model; PE = prediction error.

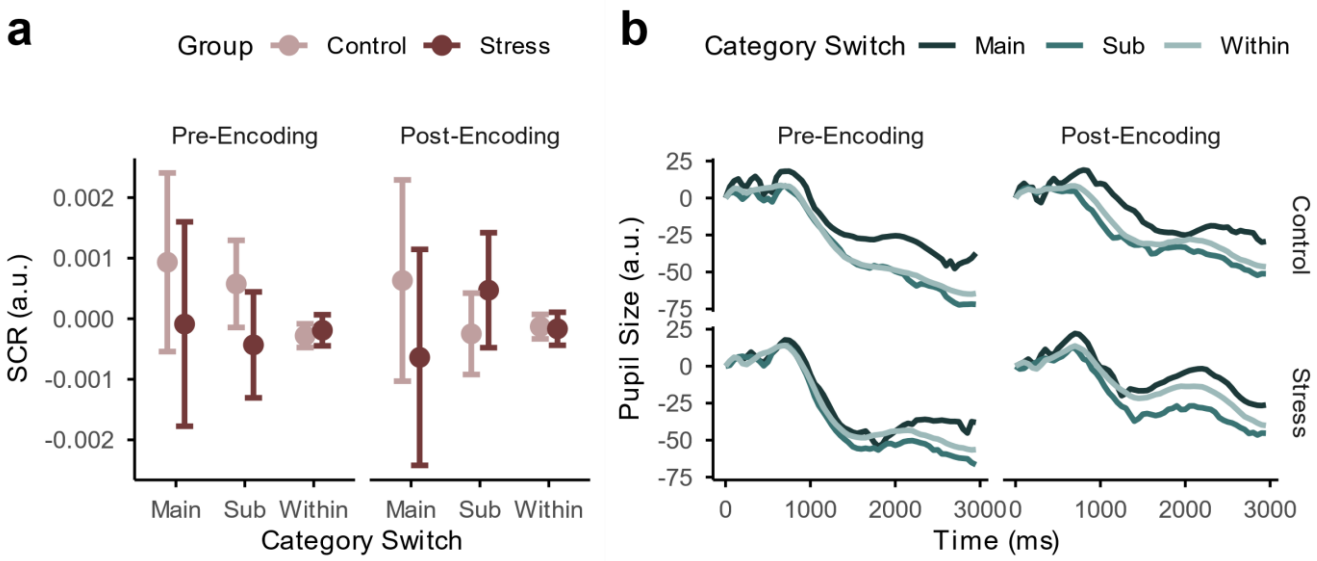
Supplemental Figure S2. Drift of prediction error (PE) ratings throughout the encoding task in (a) Study IIa and (b) Study IIb.



Supplemental Figure S3. Physiological correlates of category switches in encoding task (Study IIa). (a) SCR at category switches and (b) timecourse of pupil size at category switches.



Supplemental Figure S4. Physiological correlates of category switches in encoding task (Study IIb). (a) SCR at category switches and (b) timecourse of pupil size at category switches.



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FAKULTÄT
FÜR PSYCHOLOGIE UND
BEWEGUNGSWISSENSCHAFT
Institut für Bewegungswissenschaft
Institut für Psychologie

Declaration according to *(please mark with a cross where applicable)*

☐ **§ 4 (1c) of the Doctoral Degree Regulations of the Institute of Human Movement Science at the University of Hamburg dated 11.06.2025**

☒ **§ 5 (4d) of the Doctoral Degree Regulations of the Institute of Psychology at the University of Hamburg dated 20.08.2003**

I herby declare,

LILJA, ANTONIA (surname, name),

that I have not yet taken a doctoral examination at another university or faculty or applied for admission to a doctoral examination.

~~Hamburg~~ Prague, 18.5.2026
place, date

signature

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FAKULTÄT
FÜR PSYCHOLOGIE UND
BEWEGUNGSWISSENSCHAFT
Institut für Bewegungswissenschaft
Institut für Psychologie

Statutory declaration according to *(please mark with a cross where applicable)*

☐ **§ 7 (4) of the Doctoral Degree Regulations of the Institute of Human Movement Science at the University of Hamburg dated 18.08.2010**

☒ **§ 9 (1c and 1d) of the Doctoral Degree Regulations of the Institute of Psychology at the University of Hamburg dated 20.08.2003**

I hereby declare on oath,

1. that the dissertation I have submitted has not been the subject of another examination procedure or has not been assessed as unsatisfactory in such a procedure.
2. that I have written the dissertation I have submitted myself, have not used any sources or aids other than those specified and have not taken advantage of any commercial doctoral advice. I have marked the passages taken verbatim or in terms of content as such.

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