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The effects of stress hormones on cognitive emotion regulation: A systematic review and integrative model

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ABSTRACT

The experience of stress and the need to regulate emotions are pervasive in everyday life. Emotion regulation (ER) is particularly required under stress to facilitate successful adaptation and recovery. Importantly, a growing body of work has identified stress and ER deficits as transdiagnostic risk factors for psychopathology. This highlights the relevance of understanding how stress impacts ER to elucidate individual vulnerability to mental disorders. Stress alters cognitive and emotional functioning via stress hormones secreted by the two major stress systems: sympathetic nervous system and hypothalamus-pituitary adrenocortical axis. This review aims to compile and synthesize empirical studies in humans investigating the effects of acute stress and stress hormones on ER. A systematic literature search yielded 14 relevant studies, 11 investigating acute stress effects and 3 examining the influence of pharmacological cortisol elevations on ER. The results of the stress studies are mixed revealing either impairing, beneficial or no effects at all. Cortisol administration mostly facilitated ER attempts. Notably, we detected timing differences in measuring ER performance relative to stress exposure that potentially reconcile divergent findings. Here, we propose the **PRESSURE** model (***Pre**dominant **S**tress **S**ystem **U**nderpins **R**egulation of **E**motions*) postulating that the direction and magnitude of stress effects on ER depends on the relative predominance of one stress system over the other. Additionally, sex-stress hormone interactions, stimulus intensity and ER strategy are discussed as possible moderators. Finally, we highlight limitations in current research and provide recommendations for future studies that will further advance our understanding of the intricate relationship between stress and ER.

1. Introduction

Emotions can be considered as an "inner compass" that informs us about our internal needs, motivational goals and potential threats from the outer world in order to guide our behavior (Greenberg, 2008). However, emotions can also become maladaptive when occurring too intense, long-lasting, or misleading, challenging adequate psychological functioning in daily life (Sheppes et al., 2015). In stressful situations, ***emotion regulation*** (ER; cf. glossary) skills are particularly needed for adaptation and rapid recovery protecting an individual from developing chronic stress (Ragen et al., 2016). Importantly, people highly differ in their ability to deal with stressful situations making some of us resilient and others vulnerable to stress-related (psycho-)pathology. Accordingly, ER deficits have been identified as a transdiagnostic risk factor for the development and maintenance of mental disorders such as depression, anxiety and post-traumatic stress disorder (Eftekhari et al., 2009; Gross and Jazaieri, 2014; Sheppes et al., 2015). Detecting factors that may influence the ability to regulate emotions is thus essential to pave the way for advanced preventive and therapeutic interventions.

Acute stress and its physiological mediators (cf. 1.2) have frequently been shown to modulate cognitive functioning by acting on prefrontal and limbic structures (McEwen et al., 2016; cf. 1.3). Interestingly, these brain regions strongly overlap with the ***ER network*** (cf. glossary; Etkin et al., 2015) suggesting an interactive relationship. In fact, there is accumulating evidence showing acute stress effects on cognitive ER, however the literature is still scarce and somewhat inconsistent, revealing either impairing (Raio et al., 2013), beneficial (Jentsch et al., 2019; Langer et al., 2020) or null findings (Shermohammed et al., 2017). Given these inconsistencies, several moderators have been discussed, including the role of sex hormones, ER strategy use, or the timing-dependency of distinct stress mediators. Yet, despite its crucial clinical relevance, the influence of stress and its endocrine mediators on

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cognitive ER is by far not fully understood.

Accordingly, this systematic review (1) encompasses a comprehensive compilation of the available empirical work in humans investigating the influence of stress hormones on cognitive ER employing acute stress induction methods as well as pharmacological approaches. To integrate and streamline these findings, we (2) propose a theoretical model on the critical role of the relative predominance of the two major stress systems (sympathetic nervous system & hypothalamus-pituitary-adrenocortical axis; cf. 1.2) contributing to either beneficial or impairing effects of stress on cognitive ER. We further (3) discuss potential moderating factors that might interact with stress in altering ER processes. Finally, we (4) highlight methodological challenges and limitations of prior work, outline important open questions and provide suggestions for future research that could advance our understanding of the interplay between stress and ER.

1.1. Emotion regulation

In daily life, we are constantly required to regulate our emotions whether on a conscious, explicit or unconscious, implicit level (Koole, 2009). By definition, ER encompasses all deliberate and automatic attempts to change the type, duration or intensity of an emotional experience (Braunstein et al., 2017; Koole, 2009; Webb et al., 2012). A broad range of ER research to date has focused on the downregulation of negative emotions. However, ER relates to any emotional modification including both, up- and downregulation of positive or negative emotions depending on the current regulatory goal.

Different ER strategies can be employed varying in when and how they influence the emotional response, affecting subjective experiences, behavior and biological responses in the central as well as peripheral nervous system (McRae et al., 2009; Schönfelder et al., 2014; Zaehringer et al., 2020). To date, the so-called *process model of emotion regulation* (Fig. 1) is the most common and widely used taxonomical system, which classifies regulatory strategies according to the major nodes of the emotion generation process at which they intervene (Gross, 2015, 1998). *Cognitive **reappraisal*** and ***distraction*** (cf. glossary) have been identified as two of the most powerful cognitive strategies to deal with negative emotions (Webb et al., 2012). Cognitive reappraisal refers to the reframing of a given situation to change the meaning or relevance of emotional cues (cognitive change). Of note, different tactics are available to generate reappraisals of an emotional trigger: reinterpretation, distancing (Powers and LaBar, 2019) and ***acceptance*** (cf. glossary; Troy et al., 2018). Reinterpretation requires engagement with the emotional content by imagining it to either happen in another context or with another outcome to alter the valence of the emotional meaning. Distancing involves the generation of a new perspective by changing either spatial distance, temporal distance, or objectivity (e.g., taking the perspective of a neutral observer). Besides reappraisal of the triggering event, one may also reappraise the cognitive and emotional response to the stimulus as being "normal" which is known under the term *acceptance.* This strategy involves changing how one relates to his or her thoughts and feelings by becoming actively aware of them without any evaluation. In addition, distraction acts via shifting the attention away from the emotional stimulus (attentional deployment) either towards non-emotional aspects of the situation or a completely unrelated situation (Gross, 2015). Attention can be altered actively by thinking about positive or neutral situations or passively by working on a task (e.g., math tasks; Webb et al., 2012). Even though all of these regulatory tactics are potent to modify emotional activation, distraction intervenes earlier in the emotion generation process and requires less cognitive control resources than cognitive reappraisal (Silvers et al., 2015; Thiruchselvam et al., 2011).

Over the last decade, a growing body of empirical work has demonstrated that ER strategies cannot be categorized as inherently adaptive or maladaptive. Rather, the effectiveness of certain strategies critically depends on contextual factors such as emotional intensity (Shafir et al., 2015), situational demands (Kobylińska and Kusev, 2019) or availability of time (Sheppes and Meiran, 2007) as well as on individual characteristics including sex, age (McRae, 2016; Nolen-Hoeksema and Aldao, 2011) and cognitive resources (Adamczyk et al., 2022; Zaehringer et al., 2018). For instance, distraction appears to be superior in the short-term when a person deals with high intensity emotions while having only limited cognitive resources. Cognitive reappraisal, however, is more effective in the long run when being exposed to low intensity emotional stimuli that are expected to occur multiple times (Sheppes, 2020). Taken together, recent findings imply that successful coping with emotional challenges is less dependent on any regulatory process per se, but rather on the ability to flexibly choose and switch between various ER strategies according to contextual factors and individual resources (Aldao et al., 2015).

The ER process relies on a cognitive control network composed of the dorsolateral and ventrolateral prefrontal cortex (***dlPFC / vlPFC;*** cf. glossary) as well as parietal and cingulate cortex regions that exert top-down control on emotion-related limbic structures such as the amygdala (Dörfel et al., 2014; Etkin et al., 2015; Morawetz et al., 2020; Ochsner et al., 2004). Importantly, these brain structures are key target sites of stress hormones and thus particularly sensitive to their influence (Arnsten, 2009).

1.2. Acute stress and its physiological mediators

Stress has been conceptually defined as a real or implied threat to homeostasis (De Kloet et al., 2005). Although it is typically regarded as a negative phenomenon, the acute physiological stress response is highly

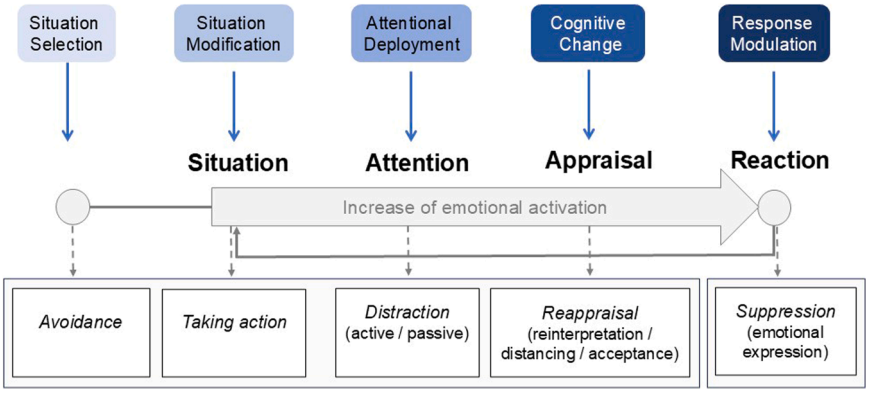

Fig. 1. The process model of emotion regulation. A situation triggers an emotional response via attentional and subsequent appraisal processes ("modal model" of emotion generation; middle row; Gross, 1998). The process model of emotion regulation classifies regulatory strategies by means of five categories located at different stages across the emotion generation process (top row). For each category, example strategies are displayed (bottom row; figure modified from Gross, 2015).

adaptive as it enables the organism to adequately cope with potential threats and to restore homeostasis afterwards (McEwen, 2004). Neurobiologically, stress is conveyed by the activation of two major stress axes: The sympathetic nervous system (***SNS;*** cf. glossary) represents the first, fast-acting pathway leading to a rapid release of ***catecholamines*** (cf. glossary) like adrenaline and noradrenaline from the adrenal medulla. Catecholamines bind to membrane-bound α- and β-adrenergic receptors within the peripheral and central nervous system, causing rapid increases in heart rate, blood pressure, pupil diameter or glucose availability to prepare the organism for immediate ***fight-or-flight*** behaviors (cf. glossary; Ulrich-Lai and Herman, 2009). Catecholamines cannot directly cross the blood-brain barrier but act indirectly through the activation of the vagus nerve prompting the nucleus of the solitary tract to stimulate adrenergic neurons in the ***locus coeruleus*** (LC; cf. glossary) from where noradrenergic projections reach multiple cortical and subcortical brain regions (Kvetnansky et al., 2009). The hypothalamus-pituitary-adrenocortical axis (***HPA axis***; cf. glossary) represents the second, somewhat slower-acting pathway resulting in a delayed secretion of ***glucocorticoids*** (GCs, in humans mainly cortisol; cf. glossary; Joëls and Baram, 2009) from the adrenal cortex. GCs can readily enter the brain and exert their effects upon binding to membrane-bound and intracellular **mineralocorticoid** (MR) and ***glucocorticoid receptors*** (*GR*; cf. glossary) located predominantly in the hypothalamus, amygdala, hippocampus, and prefrontal cortex (PFC). In addition to adrenaline, noradrenaline and GCs, various other hormones, neuropeptides, and neurotransmitters are released in response to an acute stressor with each mediator having its own functional, spatial, and temporal characteristics of release and action allowing for fine-tuned, timing-dependent changes in specific brain areas (Joëls and Baram, 2009). They yet also work in concert to orchestrate the most optimal response to diverse challenges, while suppressing functions that are not of immediate necessity.

In humans, novel, unpredictable or uncontrollable situations containing a threat to the social self are especially potent in prompting a stress response (Dickerson and Kemeny, 2004). In the laboratory, this can be realized by using standardized psychosocial stressors, such as public speaking tasks (e.g., the Trier Social Stress Test, ***TSST***; cf. glossary; Kirschbaum et al., 1993), physical stressors (e.g., Cold Pressor-Test, ***CPT***; cf. glossary; Hines et al., 1936) or hybrid formats combining physical and social-evaluative components (e.g., the Socially Evaluated Cold-Pressor Test, ***SECPT***; cf. glossary; Schwabe et al., 2008). In addition, there are several psychosocial stressors available which can be applied in the functional Magnetic Resonance Imaging (***fMRI;*** cf. glossary) environment (e.g., ScanStress; Streit et al., 2014). The TSST and the SECPT are amongst the most often used stress induction protocols. These stressors have been shown to reliably activate both, the SNS and the HPA axis (Allen et al., 2014; Schwabe and Schächinger, 2018). In addition, pharmacological manipulations blocking or activating specific receptor types (MR, GR or adrenergic receptors) can serve to isolate the effects of single mediators, as for instance the administration of hydrocortisone, a synthetic analogue to cortisol. Hydrocortisone administration leads to cortisol concentrations in the upper physiological to supraphysiological range (depending on the exact dosage) which are substantially higher than those induced by laboratory stressors (Jentsch et al., 2022). However, such a pharmacological intervention does not evoke an activation of other stress-responsive physiological systems (e.g., the SNS) or any experience of subjective stress. Additionally, hydrocortisone administration suppresses secretion of other hormones along the HPA axis (e.g., corticotropin-releasing hormone and adrenocorticotropic hormone).

1.3. Timing-dependent stress effects on brain activity

Given that physiological stress reactions differ in their temporal characteristics, there are three critical time windows of acute stress effects on brain activity and related cognitive and affective functions (Figs. 3a and 3c; Hermans et al., 2014).

In response to stress, levels of adrenaline and noradrenaline increase promptly and return to baseline rapidly after stress offset. Catecholaminergic effects thus emerge during or instantly after the encounter with a stressor shaping the first wave of stress effects in the brain (first time window). By contrast, GC levels rise more slowly (peaking approximately 25 min after stress onset; Dickerson and Kemeny, 2004) and remain elevated for a longer period of time (about 60–90 min) even though the stressor has already vanished. GCs act rapidly via ***non-genomic actions*** (cf. glossary) as soon as GCs reach target tissues and as long as GC levels are elevated (non-genomic pathway: second time window). In addition, GCs may shape neural activity in a delayed fashion via ***genomic actions*** (cf. glossary), which take at least 60 min to initiate and then continue for several hours (genomic pathway: third time window; Joëls et al., 2013).

Critically, timing-dependent stress effects on neural activity and cognitive-affective functioning may serve to quickly provide coping resources to the organism during acute stress states and foster a return to homeostasis later on (Hermans et al., 2014). For instance, imaging data revealed increased amygdala and thalamus activation (Oei et al., 2012; van Marle et al., 2009) but reduced dlPFC activation during cognitive control testing (Qin et al., 2009) either during or immediately after acute stress exposure. Crucially, these effects seem to be mediated via noradrenergic activation of β-adrenoceptors (Hermans et al., 2011), suggesting that rapidly increasing catecholamines may instantly provoke a state of excitation and hypervigilance to potential threats while dampening executive control functioning. In contrast, with longer temporal delays (i.e., 75–240 min after stress exposure or pharmacological treatment) GCs have been shown to reduce amygdala activation (Henckens et al., 2010) indicating dampened emotional responsivity while enhancing PFC signals and cognitive control performances (Henckens et al., 2012, 2011; Yuen et al., 2009). Accordingly, fast neural stress actions may promote salience network activation (i.e., increased amygdala and thalamus activation) at the cost of executive control functioning (i.e., reduced PFC activation) during the acute stress phase. By contrast, delayed GC actions may reverse these initial neural effects to facilitate higher-order cognitive processes and to normalize the systems when stress has subsided.

Given that these structures are also critically involved in ER processes, one may expect timing-dependent differences in stress effects on ER outcomes. To compile the current state of knowledge, we systematically searched for all available research articles that examined the effects of acute stress and stress hormones on ER. After summarizing study findings, we will discuss the role of timing of the ER task relative to stress manipulation as well as other potentially moderating factors that might contribute to divergent findings.

2. Systematic literature search

A systematic literature search was conducted to extract all published articles reporting on experimental studies which investigated the effects of acute stress or pharmacological challenges of the two major stress pathways on ER performances in healthy, adult human samples. The literature synthesis was carried out in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Moher et al., 2009).

2.1. Sources and search strategy

To identify all relevant studies, we performed an exhaustive search of the databases Scopus, PubMed, and Web of Science to obtain any original research article from the earliest publication date until May 2024. We used the following search string to reveal any hits in the title, abstract, and/or keywords:

(("acute stress" OR "cortisol" OR "glucocorticoids" OR "hydrocortisone" OR "noradrenaline" OR "norepinephrine" OR "catecholamines" OR

"yohimbine") AND ("emotion regulation" OR "emotion control"))

The databases offer different filter options. Whenever possible (Scopus, PubMed), we filtered for English-written articles reporting on healthy, human, adult samples. In this search, Scopus returned 404 results, PubMed returned 185 results, and Web of Science returned 502 results (1.091 hits in total; Fig. 2). Additionally, the snowball search method was used to detect further potentially relevant studies by reviewing references from relevant articles received from the databases (15 hits). After removing duplicates, 703 articles were screened for eligibility.

2.2. Screening procedure and eligibility criteria

Two investigators independently screened the titles, abstracts, and full-text articles. In case of disagreement, they reached a consensus through discussion with a third investigator. After removing duplicates, we extracted all studies that examined the effects of acute stress and/or pharmacological challenges of the GC and/or noradrenergic systems on ER. In the subsequent screening phase, studies were excluded if they met any of the following criteria: (a) non-human subjects, non-healthy, or non-exclusively adult samples (i.e., not $\geq$ 18 years old), (b) non-original research articles, (c) non-peer-reviewed journals, (d) non-English language articles. Next, the remaining studies were thoroughly reviewed and assessed for eligibility according to the following exclusion criteria: (a) absence of experimental stress manipulation (i.e., no acute laboratory stressor) or absence of pharmacological manipulation of the GC or noradrenergic systems (i.e., no administration of pharmacological agents that block or activate GC and/or noradrenergic receptors), (b) absence of a control group, (c) absence of an explicit ER task (i.e., emotional stimuli should be used to induce emotional responses, and participants should be asked to regulate their emotions using a specific ER strategy such as reappraisal, distraction or suppression), (d) absence of a measure of ER performance (e.g., self-report, physiological responses, neural activity), (e) absence of a stress measure.

3. Current state of research

3.1. Study characteristics

The systematic literature search resulted in 14 original research articles (Fig. 2) that report on experimental studies testing the effects of acute stress and / or pharmacological manipulation of the GC and / or noradrenergic system on cognitive ER outcomes in healthy adult samples, meeting the other search criteria mentioned above. Table 1 lists all

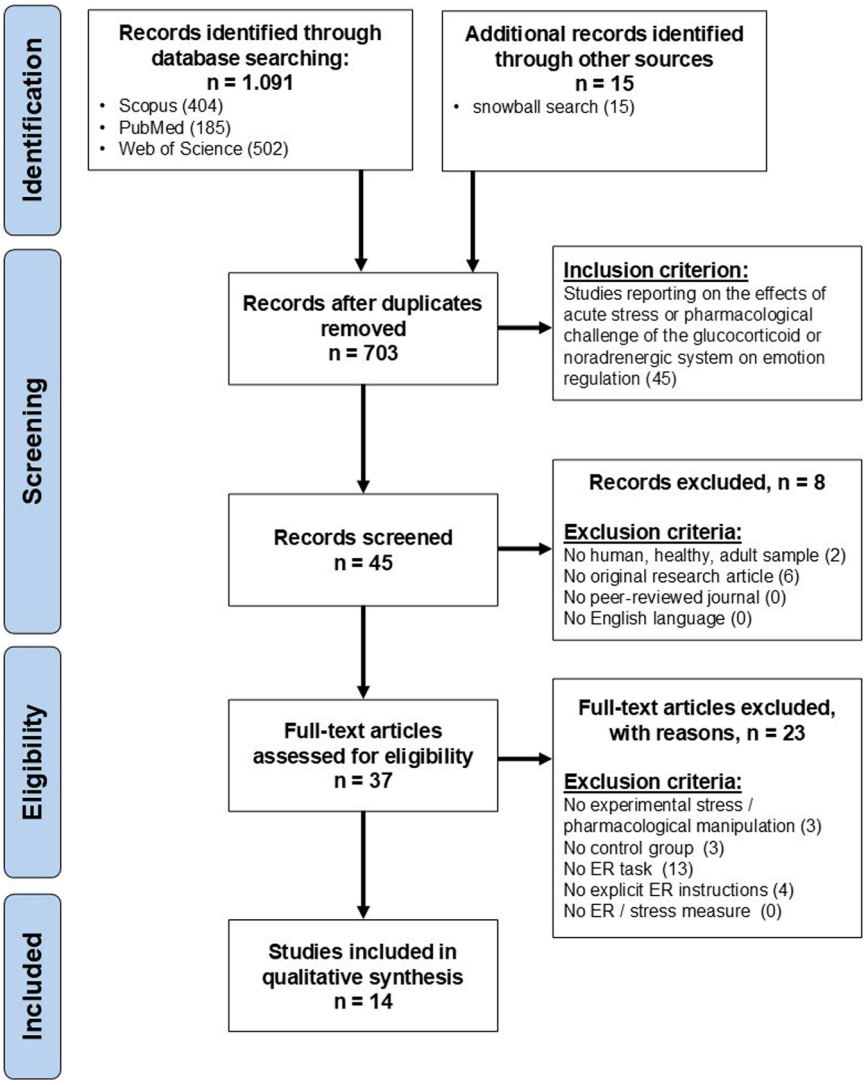

Fig. 2. Flowchart of article selection. Illustration of the systematic literature search process, from initial article identification at the different databases (Scopus, PubMed, and Web of Science) to the final study selection based on specific inclusion and exclusion criteria at the screening and eligibility stages. ER = emotion regulation.

Table 1
Study list. All selected studies ordered alphabetically and characterized by its methodological approach and the main direction of stress/cortisol effects on ER.

Publication (first author, year)	**Sample size** (number of females)	**Stress system manipulation**	**ER task delay** (after stress onset)	**Stimuli** (emotion induction)	**Intensity of used stimuli** (mean normative ratings)	**ER strategies**	**ER measures**	**Effect on ER**	**Correlated stress biomarkers** (with ER)
ACUTE STRESS									
Hamza, 2024	98 (f=73)	SECPT	18 min	pictures (IAPS)		reappraisal (down), acceptance	intensity ratings, HR, SCR, pupil dilation	**null**	
Kinner, 2014	72 (f=36)	SECPT	28 min	pictures (IAPS)		reappraisal (up & down), distraction	arousal, valence ratings	**negative & positive**	
Langer, 2023	80 (f=40)	SECPT	15 min	pictures (NAPS)	arousal: 7.36, valence: 2.27	reappraisal (down), distraction	arousal, valence, success ratings, pupil dilation	**negative & positive**	BP & HR cortisol
Langer, 2020	118 (f=78)	TSST	25 min	pictures (NAPS)	arousal: 4.35, valence: 3.55	reappraisal (up & down) distraction	arousal, valence, success ratings, pupil dilation	**positive**	cortisol
Langer, 2021b	81 (f=40)	TSST	90 min	pictures (NAPS)	arousal: 4.35, valence: 3.55	reappraisal (up & down) distraction	arousal, valence, success ratings, pupil dilation	**positive**	cortisol
Langer, 2022	80	TSST	25 min	pictures (NAPS)	*low intensity* arousal: 5.18, valence: 3.68 *high intensity* arousal: 7.26, valence: 2.34	reappraisal (down), distraction	arousal, valence, success ratings, pupil dilation	**positive**	cortisol
Raio, 2013	78 (f=39)	CPT	15 min	pictures (snakes & spiders) + shocks		reappraisal (down)	selection of fear-related words, SCR	**negative**	sAA
Sandner, 2021	81 (f=40)	ScanSTRESS-C	20 min	pictures (EmoPicS)		reappraisal (down), distraction	valence ratings, fMRI	**null**	
Shermohammed, 2017	54 (f=27)	free speech	no delay	pictures (IAPS)		reappraisal (down)	valence ratings, HR, SCR, fMRI	**null**	
Wessa, 2024	50 (f=26)	TSST	40 min	pictures (IAPS)	arousal: 6.59, valence: 2.09	reappraisal (down)	valence ratings, EEG, EMG activity	**negative**	
Zhan, 2017	180 (f=119)	CPT	15 min	anger provoking exchange		reappraisal (down)	selection of anger-related adjectives	**negative**	
PHARMACOLOGY									
Jentsch, 2019	64 (f=32)	30 mg hydrocortisone	90 min	pictures (IAPS)	arousal: 5.58, valence: 2.41	reappraisal (down), distraction	intensity ratings, fMRI	**positive**	
Langer, 2021a	85	10 mg hydrocortisone	30 min & 90 min	pictures (NAPS)	*low intensity* arousal: 6.5, valence: 3.21 *high intensity* arousal: 7.40, valence: 2.09	reappraisal (down), distraction	arousal, valence, success ratings, pupil dilation	**positive**	
Pan, 2023	105 (f=41)	20 mg hydrocortisone	30 min & 90 min	pictures (NAPS)	arousal: 6.58, valence: 2.99	reappraisal (up & down)	intensity ratings, fMRI	**negative & positive**	

Note: CPT = Cold-Pressor Test; SECPT = Socially Evaluated Cold-Pressor Test; TSST = Trier Social Stress Test; sAA = salivary alpha-amylase; BP = blood pressure; HR = heart rate; ER = emotion regulation; NAPS = Nencki Affective Picture System; IAPS = International Affective Picture System; fMRI = functional Magnetic Resonance Imaging, EEG = Electroencephalography; EMG = Electromyography; SCR= Skin Conductance Response; f=female (if no portion of female participants is specified, the whole sample consisted of male participants)

available stress and pharmacological studies in alphabetical order according to the first author and presents the methodological approach, the main direction of stress / pharmacological effects on ER as well as significant correlations between stress biomarkers and ER outcomes.

3.1.1. Emotion induction

In twelve studies (86 %), pictures either taken from the Nencki Affective Picture System (NAPS; Marchewka et al., 2014), the International Affective Picture System (IAPS; Lang et al., 2005) or the EmoPicS database (Wessa et al., 2010) were used to induce negative emotions via

computer-based ER paradigms. One study induced anger by asking participants to exchange views on a popular societal topic with a fictional person (prepared by the experimenter) via e-mail, which involved a confrontation with an extremely negative evaluation of the participant's viewpoint (Zhan et al., 2017). Another study used a fear conditioning paradigm, in which images of snakes and spiders were paired with mild electric shocks to induce conditioned emotional responses (Raio et al., 2013). Of note, the emotional stimuli varied in intensity between studies which was also experimentally manipulated within two studies (Langer et al., 2022, 2021a) to examine the role of stimulus intensity for stress/cortisol effects on ER.

3.1.2. Emotion regulation strategies and outcome measures

Existing studies showed slight variations in the type and implementation of ER strategies. In all studies, reappraisal was applied to downregulate negative emotional responses. Importantly, there are different tactics to generate reappraisals of an emotional stimulus (Powers and LaBar, 2019). Most participants were instructed to think of a positive reinterpretation of the presented stimuli (change of context), while two studies either allowed (Shermohammed et al., 2017) or specifically instructed (Kinner et al., 2014) the use of distancing tactics. Additionally, four studies (29 %) included an upregulation condition, asking participants to intensify their emotional responses by worsening negative interpretations (Kinner et al., 2014; Langer et al., 2021a, 2020; Pan et al., 2023). Distraction was applied in eight studies (57 %), either in its active form (Jentsch et al., 2019; Langer et al., 2023, 2022; Langer et al., 2021a; Langer et al., 2021b; Langer et al., 2020) or its passive form (Kinner et al., 2014; Sandner et al., 2021). In addition, one study included acceptance, asking participants to accept the emotions they feel as a natural response without seeking to change them (Hamza et al., 2024).

Besides differences in ER strategy implementation, different outcome measures were used to quantify ER performance. All studies included at least one self-report instrument to evaluate the emotional state, varying between direct measures of emotional intensity, arousal, valence, or regulatory success, and indirect measures of emotional activation through the selection of emotion-related words. In addition, skin conductance responses (SCRs), pupil dilation, and electromyographic (EMG) activity of the corrugator supercilii muscle served as physiological outcome measures. Some studies provided data on neural activity during ER, recorded via electroencephalography (EEG) or fMRI signals.

3.1.3. Manipulation of the stress systems

All studies included either an acute stressor (eleven studies: 79 %) or a pharmacological challenge of the GC system (three studies: 21 %), but none applied an isolated manipulation of the noradrenergic system. Stress induction methods varied between the SECPT, the CPT, the TSST, the free speech part of the original TSST, and the ScanStress-C (for detailed information on the stressors, see Section 1.2). In all pharmacological studies, participants were administered either hydrocortisone (a GC receptor ***agonist***; cf. glossary) with varying dosages, 10 mg (Langer et al., 2021a), 20 mg (Pan et al., 2023) and 30 mg (Jentsch et al., 2019), or a placebo prior to the ER task.

To verify successful manipulation of the stress systems, all studies included repeated saliva sampling to assess free cortisol concentrations. In six studies (43 %), sAA levels were additionally measured as an indirect marker of noradrenergic activity (Nater and Rohleder, 2009), while others alternatively recorded cardiovascular reactivity (blood pressure: two studies: 15 %; heart rate: three studies: 23 %). In most studies, participants repeatedly rated their affective state (eight studies: 57 %) and/or subjective feelings of stress (five studies: 36 %) throughout the experimental procedure.

3.1.4. Timing-related variations of the ER task

Given that physiological stress mediators differ in their temporal characteristics of release and action (Joëls and Baram, 2009), variations in the timing of the ER task relative to stress exposure or pharmacological administration might be critical for the interpretation of the findings. In the present studies, the ER task was either performed during stress in alternating blocks of stress and ER (Shermohammed et al., 2017) or started immediately up to 15 min after stress onset or pill intake (Langer et al., 2023; Raio et al., 2013; Zhan et al., 2017), 18–40 min (Hamza et al., 2024; Kinner et al., 2014; Langer et al., 2022, 2020; Sandner et al., 2021; Wessa et al., 2024), or 90 min afterwards (Jentsch et al., 2019; Langer et al., 2021b). In three studies, the delay was experimentally varied between 30 and 90 min (Langer et al., 2021a; Pan et al., 2023) to capture time windows of rapid, non-genomic and slow, genomic cortisol effects, or between 20 and 40 min (Wessa et al., 2024) to directly compare catecholaminergic and non-genomic cortisol effects on ER within one study design. Besides variations in timing of the ER task, the studies also differ in the number of trials and presentation time of the stimuli resulting in distinct durations of regulatory efforts. For instance, there are two studies that used rather short regulation periods of 11 min (Raio et al., 2013) and 5 min (Zhan et al., 2017), whereas ER tasks of other studies last about 30–40 min (Kinner et al., 2014; Langer et al., 2022; Wessa et al., 2024). Given timing-dependent molecular actions in the brain after stress (cf. 1.3), these methodological differences might affect stress effects on ER. In addition, differences in cognitive fatigue as a confounding factor cannot be excluded.

3.2. Summary of findings

In sum, three of the stress studies reported impairing effects, three showed beneficial effects, two revealed both beneficial and impairing effects on ER outcomes depending on sex (Langer et al., 2023) and ER strategy (Kinner et al., 2014) and three resulted in null findings (cf. Table 1). Two of the pharmacological studies showed that cortisol facilitates ER attempts, whereas one study provides mixed findings. In the following, these results are summarized and described in more detail.

3.2.1. Detrimental effects of stress hormones on ER

In line with research showing stress to compromise executive control functions (Shields et al., 2016), there is evidence for detrimental stress effects on the cognitive control of emotions. In detail, Raio et al. (2013) revealed that stress impairs ER performances when participants applied reappraisal to downregulate conditioned fear. In contrast to the control group, stressed participants were not able to successfully downregulate fear arousal 15 min after stress onset, as indicated by stronger SCRs and a higher propensity to select fear-related words. While no associations between cortisol and ER performance were found, sAA levels were positively related to fear arousal during the regulation task, suggesting noradrenergic reactivity to mediate the ER impairment after stress. Consistently, a second experiment showed that participants did not effectively downregulate subjective feelings of anger via reappraisal when having been stressed 15 min before (Zhan et al., 2017). Further evidence for detrimental stress effects comes from a recent study (Wessa et al., 2024) demonstrating stress to reduce the effectiveness of reappraisal to downregulate negative emotional activity 40 min, but not 20 min, after stress onset. In this study, all participants underwent a 40 min ER task divided in an early and late post-stress phase. Given the resulting relatively long duration of constant cognitive effort needed to concentrate on the regulatory task, group differences in the late phase might be attributed to higher mental fatigue after stress. In favor of this argumentation, the effect was at least partially driven by increases in ER performance over time in the control group only. Further in line with the findings showing rapidly impairing effects on ER, pharmacological cortisol elevations increased amygdala, but also dlPFC activation, 30 min after hydrocortisone administration (Pan et al., 2023) when comparing the down- and upregulation condition of reappraisal. These findings suggest that cortisol rapidly reduced the effectiveness of reappraisal to change the emotional activation in the intended direction (either impaired down- or upregulation or both). More specifically, the

increased dlPFC activation in combination with enhanced amygdala activation after cortisol intake may indicate an effortful (i.e. enhanced cognitive regulatory engagement) but still ineffective regulation of negative emotions. Of note, however, cortisol effects were neither found when directly comparing down- or upregulating negative emotions with the viewing only condition nor reflected in emotional intensity ratings leading to some degree of ambiguity in data interpretation. Another study revealed that stress impaired downregulation of negative emotions via distraction but not reappraisal 28 min after stress onset (Kinner et al., 2014), suggesting that stress effects on ER performance may critically depend on the particular strategy used. This finding aligns with data showing that stress is indirectly related to higher arousal ratings mediated via cardiovascular reactivity (blood pressure) when women tried to actively distract themselves from negative stimuli 15 min after stress exposure (Langer et al., 2023). Interestingly however, no such impairing effects on ER were found in male participants.

Together, these findings provide evidence that acute stress may impair one's ability to downregulate negative emotions. Some studies linked the ER impairments to noradrenergic reactivity (sAA, cardiovascular activity) but not cortisol levels, suggesting the SNS to guide detrimental stress effects on ER. In addition, participant sex as well as ER strategy emerge as two potential moderating factors. It should be noted, however, that to the best of our knowledge the role of noradrenergic activity in modulating ER processes has never been tested in isolation, e.g. by a pharmacological activation or blockade of noradrenergic receptors.

3.2.2. Beneficial effects of stress hormones on ER

In contrast to the findings described before, there is a similar number of studies providing evidence for beneficial stress effects on ER. Kinner et al. (2014) were the first to show that acute stress may improve the effectiveness of reappraisal to downregulate negative emotions. More specifically, stressed women rated negative pictures as more positive when applying reappraisal 28 min after stress onset compared to control women, whereas no such effect was found in men. Notably, in this study, ER strategy was realized as a between-subjects factor resulting in a relatively small cell size. The stress x sex interaction should thus be treated with caution. This power issue was resolved in a subsequent study (Langer et al., 2020) that included ER strategy as a within-subject factor asking participants (men, naturally cycling women, and women taking oral contraceptives) to apply reappraisal (up & down) and distraction 25 min after stress exposure. Although the stress-induced ER improvement in women could not be replicated, in this study, stress increased the effectiveness of reappraisal to downregulate negative emotions in men, as evidenced by reduced arousal, enhanced valence and success ratings. Stressed men also displayed larger pupil size increases during reappraisal. Pupil dilation is positively related to emotional arousal but also to the cognitive effort needed for ER attempts (Kinner et al., 2017; Maier and Grueschow, 2021; van der Wel and van Steenbergen, 2018). It is therefore reasonable to assume that the increases in pupil diameter found in the study by Langer et al. (2020) might reflect greater cognitive regulatory engagement which in turn may have improved ER performance. Critically, subjective reappraisal success was related to increases in cortisol, but not sAA, indicating that beneficial stress effects on ER might be mediated by GC actions. Supporting this hypothesis, another study replicated the beneficial stress effects on ER, showing increased subjective regulatory success of reappraisal when men reappraised negative emotions of high-, but not low-intensity, 25 min after stress (Langer et al., 2022). These effects were again positively correlated with cortisol, but not sAA increases. Stressed participants also reported to be more successful in distracting themselves from high-intensity negative stimuli than controls, suggesting that stress may improve both, reappraisal and distraction. However, these effects were not corroborated by other (physiological) ER outcome measures, raising the possibility that stressed participants were simply more convinced of their regulatory performance than controls. Contrary to this view, other studies also showed that stress exposure either indirectly (Langer et al., 2021b) or directly (Langer et al., 2023) led to reduced emotional arousal when participants distract themselves from negative stimuli, which thus further supports the idea that stress improves distraction outcomes. In both studies, regulatory improvements were mediated by cortisol, but not sAA increases. Given that stress not only triggers cortisol secretion but also other neurophysiological reactions (Joëls and Baram, 2009), no causal conclusions regarding cortisol effects on ER can be drawn from this research. To this end, Jentsch et al. (2019) used a pharmacological approach examining the influence of hydrocortisone administration on ER in the scanner. In this study, cortisol led to increased vlPFC activation when participants distracted themselves from negative stimuli and reduced activation in the right amygdala when applying reappraisal 90 min after pill intake suggesting cortisol to facilitate ER attempts. Consistently, a recent study again showed that cortisol dampened amygdala activation when applying reappraisal 90 min after pharmacological administration, but resulted also in reduced dlPFC activation and connectivity between these two structures (Pan et al., 2023). The authors discussed these findings to reflect cortisol-induced ER improvements without the need to activate additional prefrontal control resources. Consistent with the buffering effects of cortisol on amygdala activation, cortisol intake lowered subjective emotional arousal when downregulating high-, but not low-intensity emotions via reappraisal and distraction 30 and 90 min afterwards (Langer et al., 2021a).

In sum, there is accumulating evidence that stress may also benefit ER attempts. These regulatory improvements have been repeatedly related to cortisol increases. Together with the data from pharmacological studies using hydrocortisone administration, one may assume favorable GC actions on the ER network as a driving mechanism for ER improvements in response to stress. These effects appear to be moderated by interindividual and contextual factors such as participant sex, emotional stimuli intensity and the applied ER strategy.

3.2.3. Null findings

The literature review resulted in three studies in which no significant stress effects on ER were found (Hamza et al., 2024; Sandner et al., 2021; Shermohammed et al., 2017). Even though stress manipulation successfully induced psychophysiological stress responses, there were no significant group differences between stressed and control participants in self-report measures of emotional intensity and valence, physiological indices of emotional arousal and cognitive regulatory effort as well as neural activation in key structures of the ER network. In addition to reappraisal, the ER tasks required participants to downregulate emotional responses via acceptance (Hamza et al., 2024) or distraction (Sandner et al., 2021). They either took place in alternating blocks of stress and ER (Shermohammed et al., 2017) or lasted from 18 min until 31 min (Hamza et al., 2024) or 40 min (Sandner et al., 2021) after stress onset. Together, some studies have found no evidence of changes in ER performance following acute stress, highlighting the need to integrate these divergent findings.

4. Integration and discussion

4.1. The PRESSURE model: Pre dominant stress system underpins regulation of emotions

As described in the introduction, the two primary stress systems - the SNS and HPA axis - differ in the timing of peak secretion of their physiological end-products. The delay between stress exposure and cognitive testing might thus play a crucial role in determining the impact of any neurophysiological action on cognitive and emotional functioning. Hermans et al. (2014) postulated opposing effects of rapid and rather slow actions of stress hormones on brain networks including structures that are also critically engaged in ER (e.g., PFC and amygdala). In line with this idea, our systematic literature search revealed a

substantial variety in the time interval between stress onset and ER measurement in the studies reviewed. Some of them assessed ER during time windows of primary HPA axis dominance, while rather quick but short-lived SNS-driven actions should already be vanished (i.e., ≥25 min after stress onset). Most of these studies revealed beneficial stress effects on ER, which has been attributed to excitatory effects of cortisol on the dlPFC and dampening effects on the amygdala (Jentsch et al., 2019). In line with this notion, cortisol responses to stress were repeatedly linked to better ER outcomes (Langer et al., 2023, 2022; Langer et al., 2021b; Langer et al., 2020). Congruently, cortisol administration increased ER performances 30 and 90 min after pharmacological manipulation (Langer et al., 2021a) indicating that both, rapid, non-genomic as well as slow, genomic cortisol actions may contribute to ER improvements in the aftermath of stress.

By contrast, another set of studies pointed at stress to reduce the regulatory efficacy of reappraisal on anger (Zhan et al., 2017) and conditioned fear (Raio et al., 2013) that has been related to noradrenergic reactivity. Moreover, stress has indirectly been linked to decrements of distraction outcomes via cardiovascular responses (Langer et al., 2023). Importantly, in these studies two methodological characteristics should be considered. The time interval between stress onset and the ER task (15 min) and the duration of the ER tasks was shorter (5–11 min) than in studies reporting beneficial effects (interval ≥25 min; duration ~30 min). Given that neural actions of the SNS predominate in this early time window after stress, it is reasonable to assume that fast-acting catecholaminergic actions impede ER attempts.

To reconcile the obtained divergent findings, we propose the *Predominant Stress System Underpins Regulation of Emotions* (PRESSURE) model postulating that the direction of stress effects on ER performance is determined by the relative predominance of one stress system over the other (SNS vs. HPA axis). According to this hypothesis, catecholaminergic actions rapidly impairing cognitive ER are somewhat later counteracted by beneficial (non-genomic and genomic) GC actions. This biphasic response may initially serve to maximize resources for coping with the imminent stressor at the cost of cognitive regulatory flexibility (first phase), while somewhat later these initial effects are reversed to support the return to homeostasis once the stressor is gone (second phase). Fig. 3 illustrates this biphasic process, predicting ER performance based on the dominant stress pathway and the underlying molecular actions of stress hormones.

In favor of this model, it has been shown that administration of hydrocortisone (GC receptor agonist) but not yohimbine (adrenoceptor agonist) enhances attentional shifts from negative to neutral emotional stimuli (Metz et al., 2021) suggesting GCs, but not catecholamines, to facilitate ER processes. Conversely, pharmacological blockade of *β*-adrenergic receptors (via propranolol) but not cortisol synthesis (via metyrapone) reduced functional connectivity between the amygdala and other regions of the salience network (Hermans et al., 2011). Interconnectivity between these network structures sensitized the organism for emotional stimuli and increased emotional processing indicating reduced inhibitory control of emotions. However, in these pharmacological studies, single nodes of the stress pathways were manipulated disregarding effects of and interactions to other physiological outputs. Importantly, (Finke et al., 2018) provided first evidence for the opposing effects of SNS and HPA activation after stress exposure on emotional arousal. Here, increases in sympathetic activation were linked to heightened emotional reactivity, whereas subsequent cortisol increases predicted the reversed outcome. Consistent with the latter, cortisol was frequently shown to dampen negative affect (Het et al., 2012; Reuter, 2002) and to augment exposure therapy outcomes by reducing phobic fear (De Quervain et al., 2019). Furthermore, there is evidence for cortisol administration to dampen amygdala reactivity (Henckens et al., 2010) and increase dlPFC activation which probably enables better cognitive control performances (Henckens et al., 2011). Collectively, SNS dominance may excite emotional responding and impair cognitive control capacities that in turn hampers ER attempts,

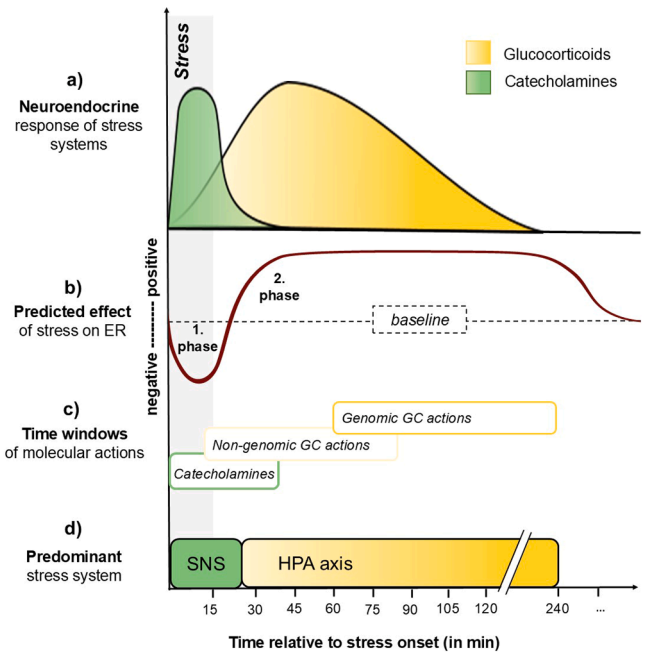

Fig. 3. Predominant Stress System Underpins Regulation of Emotions (PRESSURE) model illustrating the biphasic process of stress effects on emotion regulation (ER) outcomes. Stress instantly increases levels of catecholamines (i.e., adrenaline, noradrenaline, dopamine) and slowly triggers the secretion of glucocorticoids (GCs; cortisol in humans; **a**). GCs modify neural activity via rapid, non-genomic and slow, genomic actions leading to three different time windows of stress effects on brain activity (**c**). Whereas catecholaminergic and non-genomic GC effects persist as long as the respective physiological mediator is elevated, genomic GC actions take at least 60 min to initiate and then continue for several hours. Timing differences in the physiological responses of the two major stress determines stress system dominance (**d**). Superiority of the sympathetic nervous system (SNS) leads to predominant catecholaminergic actions that may reduce ER performances (first phase; **b**). After some delay, predominance of the hypothalamus-pituitary-adrenocortical (HPA) axis foster ER attempts via (non-genomic and genomic) GC effects (second phase; **b**) that may persist as long as genomic actions continue. Together, changes in the predominant stress system over time may guide variations in stress effects on ER to occur.

whereas HPA axis dominance reverses these initial effects by opposing neuroendocrine actions on the ER network.

To the best of our knowledge there is no study to date that assessed ER performances in time windows > 90 min after stress / pharmacological manipulation. It is thus not clear how long genomic GC actions may continue to improve ER performances after stress. Inspired by animal research showing that genomic GC actions continue at least for four hours (Joëls et al., 2012), Henckens et al. (2011) examined neural effects of cortisol on cognitive testing 240 min after pharmacological administration in a human sample. As expected, cortisol intake increased dlPFC activation and improved cognitive control performance during a working memory task four hours after pill intake. Given that ER relies on similar cognitive control functions (Pruessner et al., 2020), it can be speculated that cognitive regulatory control is improved for hours after stress before returning to baseline as soon as genomic GC actions have been subsided (Fig. 3b). Future studies extending the delay between stress / pharmacological manipulation and ER assessment are thus warranted to shed light on changes of genomic GC effects on ER over time.

It has to be noted though that there are some studies that found no significant effect of stress on self-reported, electrodermal, or neural markers of ER (Hamza et al., 2024; Sandner et al., 2021; Shermohammed et al., 2017; cf. 3.2.3). In these studies, the ER task took place ~18–40 min after stress onset or in alternating blocks of stress and ER

during which biomarkers of the SNS (heart rate) and the HPA axis (salivary cortisol) were both elevated. According to the PRESSURE model, these null findings might result from a balanced activation of the two stress systems in time intervals where opposing actions of catecholamines and GCs should act on the emotion regulatory network and thus could have canceled each other out. Future studies are needed in which dominance of the SNS or HPA axis after stress is experimentally manipulated (e.g. via pharmacological blockade or activation of GC receptors or α- / β-adrenoceptors; cf. 5.2) to draw conclusions on its causal role for the direction and strength of stress effects on the cognitive control of emotions.

Besides timing, we identified some moderating factors (sex, stimulus intensity, ER strategy) in the literature that may influence effect sizes. These moderators will thus be discussed in the following section.

4.2. Moderators of stress effects on emotion regulation

4.2.1. Sex hormones

It is known that the physiological response to acute stress (Kudielka and Kirschbaum, 2005) and its influence on cognition and emotion (Jentsch et al., 2022) differ between men and women. Beyond, there is initial evidence for sex differences in the effective use of ER strategies (Goubet and Chrysikou, 2019) and its modulation by acute stress (Kinner et al., 2014; Langer et al., 2023, 2020). In more detail, stress has been repeatedly shown to increase capacities to downregulate negative emotions in men but not in women (Langer et al., 2023, 2020). Consistently, pharmacological cortisol elevations led to diminished subjective emotional reactivity (Jentsch et al., 2019) and increased activation in the dorsomedial PFC (Ma et al., 2017) when men - but not women - were exposed to emotional pictures. These data suggest that men profit more from the beneficial effects of stress on ER than women which may contribute to reduced effect sizes in samples with a high proportion of female participants (Hamza et al., 2024).

Different possible underlying mechanisms should be discussed. First, men have repeatedly been shown to exhibit larger salivary cortisol responses to psychosocial stressors as compared to women (Kirschbaum et al., 1999; Kudielka and Kirschbaum, 2005) suggesting a larger proportion of GCs to arrive at target tissue in the brain. In view of the inverted U-shaped dose-response curve between GCs and cognitive functioning (Joëls, 2006), stress actions on the ER network might depend on a certain magnitude of cortisol levels to exert their full-blown effects. It is worth noting that in the study by Langer et al. (2020) stressed men exhibited a significant larger cortisol increase than women which was accompanied by ER improvements after stress in men only. In another study, however, in which no sex differences in ER outcomes after stress were found, men and women showed similar physiological stress responses (Langer et al., 2021b). Therefore, sex-specific stress effects on ER might at least in parts be explained by sex differences in HPA axis reactivity. In addition, there are hints in the literature suggesting a stronger excitability of the locus coeruleus (major source of catecholamines in the brain; Roosevelt et al., 2006) in women compared to men which might make women more susceptible to the catecholaminergic-driven ER impairments after stress (Langer et al., 2023). Taken together, previous research suggests that men and women differ in the ratio of SNS to HPA axis reactivity, which may account for sex-specific susceptibility to the detrimental or beneficial effects of stress on ER.

However, there are also studies showing sex differences in emotional reactivity (Jentsch et al., 2019) and ER outcomes after stress (Kinner et al., 2014; Langer et al., 2023) despite similar stress hormone levels in men and women. Additional hormonal mechanisms should thus be taken into account: For example, cognitive and emotional functioning after stress have been linked to sex hormone variations between men and women which may alter the responsiveness of the brain to the same amount of neuroendocrine stress signals (Jentsch et al., 2022). These sex differences in neural responsiveness may be explained by research suggesting that female sex hormones - such as estradiol and progesterone - interact with the density and functionality of GC receptors (Ter Horst et al., 2012). For instance, estradiol has been shown to inhibit expression and functionality of the GR (Krishnan et al., 2001) which is essential for efficient stress coping and the return to homeostasis (De Kloet et al., 1998; Oitzl et al., 2010). In addition, there is evidence for female sex hormones to influence MR expression and affinity (Ter Horst et al., 2012) which is related to the early initiation of the stress response. Animal research indicates that the MR is more frequently expressed (Lin et al., 2011) and has a threefold higher binding affinity in males compared to females (Turner, 1997). In line with these findings, there is evidence for oral contraceptive usage (suppressing the typical secretion pattern of female sex hormones over the menstrual cycle) to moderate stress effects on a variety of cognitive-emotional processes (Jentsch et al., 2022). In addition to effects on GC receptor density and functionality, hormonal contraception has been shown to upregulate cortisol-binding globulin (CBG) in human blood samples (van der Vange et al., 1990). CBG is the primary cortisol-binding protein, determining the proportion of free, unbound cortisol available to cross the blood-brain barrier. Therefore, the female sex hormone status may account for variations in the amount of biologically active cortisol levels thereby affecting human brain functioning after stress.

Taken together, complex sex-stress hormone interactions may influence the ratio between SNS and HPA axis reactivity, GC receptor density and functionality as well as the amount of free, unbound cortisol that in turn may guide the magnitude and direction of stress effects on ER to occur. However, given little number of studies analyzing potential sex differences and lack of studies in which sex hormone levels are measured or even experimentally manipulated, interpretation of underlying mechanisms should be treated with caution until more data is available.

4.2.2. Emotional intensity

A substantial body of research has shown that ER strategy choice and its effectiveness rely on the intensity of the emotional stimulus (Shafir et al., 2015; Sheppes, 2020). Building on this earlier work, there are initial hints in the reviewed literature for stress effects on ER to interact with the intensity of the emotional material used. In detail, acute stress exposure (Langer et al., 2022) and cortisol administration (Langer et al., 2021a) led to improvements in ER outcomes when dealing with high-intensity, but not low-intensity negative emotions. These findings suggest that beneficial stress effects on ER depend on a certain threshold of emotional activation. Of note, some studies postulate that the demand for cognitive resources increases with rising stimulus intensity (Shafir et al., 2015; Silvers et al., 2015). Since stress can impair top-down control (Arnsten, 2009), stronger beneficial stress effects on ER for high-intensity stimuli may seem counterintuitive. However, Langer et al. (2020) showed that stress may improve reappraisal success, probably due to increased cognitive regulatory engagement that was driven by a cortisol-driven boosted recruitment of prefrontal control areas (Jentsch et al., 2019). Therefore, beneficial stress effects on ER may be most evident when the PFC is heavily recruited, as expected during explicit regulation of highly intense emotions. However, due to the lack of studies varying the intensity of emotional stimuli, the neural mechanisms underlying emotional intensity-dependent stress effects on ER remain speculative. Additionally, it remains unclear whether the relationship between stimulus intensity and cortisol-driven stress effects on ER follows a positive linear dose-response curve, or if there is a level of emotional activation at which beneficial stress effects on ER are reversed. Supporting this idea, studies reporting ER improvements or no effects after stress generally used pictures to induce negative emotions (Jentsch et al., 2019; Sandner et al., 2021) while studies reporting stress-induced regulatory impairments used either combined visual and painful stimuli (Raio et al., 2013) or an interpersonal anger-provocation procedure (Zhan et al., 2017). It is reasonable to assume that such interactive social procedures, but also more complex multisensory

stimuli create a more vivid and intense emotional experience than pictures alone. Stress effects on ER might thus be moderated by the potency of the stimulus material to provoke negative emotions in the first place.

4.2.3. Emotion regulation strategy

Reviewed studies that examined both, reappraisal and distraction performances, revealed strategy-dependent stress effects on ER outcomes (Table 2; Kinner et al., 2014; Langer et al., 2023; Langer et al., 2021b; Langer et al., 2020). Accordingly, stress may influence regulatory efficacy as a function of the ER strategy used. But what predicts which strategy is influenced by stress and in which direction? Except for one study (Kinner et al., 2014), stress was repeatedly shown to enhance the regulatory impact of distraction on negative emotional responses. In these studies, participants were asked to actively distract themselves from negative emotional stimuli by thinking about unrelated, neutral situations (active distraction). In contrast, Kinner et al. (2014) used a mathematical task presented as an overlay on the emotional stimuli to passively distract participants from the emotional input (passive distraction). Active distraction likely requires more cognitive effort than passive distraction. Given findings indicating that beneficial stress effects on ER rely on a certain degree of cognitive engagement (Langer et al., 2020), passive distraction may profit less from a cortisol-driven enhancement of cognitive control resources. Differences in the implementation of distraction may therefore moderate stress effects on regulatory outcomes.

However, there are studies that used the same instructions for distraction and reappraisal and yet differed in the target strategy improved by stress (Langer et al., 2023, 2020). One further interacting factor might be the intensity of the emotional stimuli used (cf. 4.2.2). Whereas stress promoted distraction - but not reappraisal - performances when participants dealt with high-intensity emotional stimuli (Langer et al., 2023), the opposite was observed (improvement of reappraisal, but not distraction) in a study that used emotional stimuli of lower intensity (Langer et al., 2020). Typically, distraction is more effective for downregulating emotions of high intensity (Shafir et al., 2015) and requires fewer cognitive resources than reappraisal (Strauss et al., 2016). Stress has been shown to favor the selection of low-demand yet effective cognitive strategies (Schwabe and Wolf, 2013), such as distraction over reappraisal, when regulating high-intensity emotions (Langer et al., 2022). This stress-induced shift towards distraction is thought to help maintain regulatory performance even if cognitive regulatory resources are limited in early time windows after stress. Consequently, one may hypothesize that stressed participants are more motivated to put effort into distracting themselves from high-intensity stimuli rather than reappraising the presented situation, ultimately leading to better regulatory outcomes.

In addition to strategy instructions and emotional intensity as potential moderators, timing-dependent neurobiological effects of stress in the brain might account for differences in the target strategy. Apart from a common neural network of regulatory control areas, reappraisal and distraction recruit different brain regions within this core network respectively associated with reevaluation of emotional meaning (vlPFC, orbitofrontal cortex, inferior temporal cortex) and attentional shifting (dorsal anterior cingulate cortex, dorsomedial PFC, parietal cortex; Kanske et al., 2011; McRae et al., 2009). Studies either reporting stress effects on reappraisal or distraction differ in timing between stress exposure and ER assessment (Langer et al., 2023, 2021b, 2020). Therefore, strategy-specific stress effects might result from differences in sensitivity of underlying neural structures to molecular actions varying over time. In support of this idea, imaging data indicate that delayed GC effects reduce activity in the cuneus and impair amygdala connectivity to the insula, likely hindering stimulus-driven, bottom-up attentional processing (Henckens et al., 2012). Given that the neuroanatomical substrates of top-down and bottom-up processes have been shown to be distinct (Hahn et al., 2006), delayed cortisol effects may specifically facilitate top-down control of emotions by reducing attentional interference, which in turn might explain improvements in distraction - but not reappraisal - in later time windows after stress exposure (Langer et al., 2021b). However, given lack of fMRI data on different ER strategies after stress with varying delays, potential neural mechanisms that explain strategy-specific effects remain speculative.

Despite multiple hints for stress effects on the ability to downregulate negative emotions, stress did not alter regulatory outcomes when participants were asked to upregulate negative emotions (Kinner et al., 2014; Langer et al., 2021b, 2020). These findings suggest that stress effects on ER performances might be restricted to pro-hedonic regulatory goals such as the downregulation of negative emotions. When exposed to acute stress, not only negative affect but also the need to downregulate the upcoming emotional pressure increases (Feldman Barrett et al., 2001). Given that acute stress initiates self-regulatory processes (De Kloet et al., 2005), it is reasonable to assume that stress encourages individuals to follow pro-hedonic regulatory goals which in turn may favor regulatory performances. It has to be noted though, that there is only a small number of studies that include contra-hedonic instructions (e.g., upregulation of negative emotions) and to the best of our knowledge there is no study to date that investigated stress effects on

Table 2

Stress and cortisol effects on emotion regulation performance of different strategies. Stress and pharmacological (cortisol) studies are listed alphabetically (according to the first author) together with the respective significant positive / negative effects on performances of the examined emotion regulation strategies.

Publication	Emotion regulation strategy				
(first author, year)	Reappraisal (down)	Reappraisal (up)	Distraction (active)	Distraction (passive)	Acceptance
ACUTE STRESS					
Hamza, 2024	-	n/a	n/a	n/a	-
Kinner, 2014	**positive**	-	n/a	**negative**	n/a
Langer, 2023	-	n/a	**positive**	n/a	n/a
Langer, 2020	**positive**	-	-	n/a	n/a
Langer, 2021b	-	-	**positive**	n/a	n/a
Langer, 2022	**positive**	n/a	**positive**	n/a	n/a
Raio, 2013	**negative**	n/a	n/a	n/a	n/a
Sandner, 2021	-	n/a	n/a	-	n/a
Shermohammed, 2017	-	n/a	n/a	n/a	n/a
Wessa, 2024	**negative**	n/a	n/a	n/a	n/a
Zhan, 2017	**negative**	n/a	n/a	n/a	n/a
PHARMACOLOGY					
Jentsch, 2019	**positive**	n/a	**positive**	n/a	n/a
Langer, 2021a	**positive**	n/a	**positive**	n/a	n/a
Pan, 2023	**negative / positive (down minus up)**		n/a	n/a	n/a

Note: n/a = no data assessed, - = no significant effects of stress / cortisol on ER outcomes

Table 3
Most important open research questions and examples for experimental designs either using a pharmacological or stress induction approach.

Research question	Hypotheses	Pharmacological approach	Stress induction approach
1. Do catecholamines and GCs exert opposing effects on ER?	Adrenergic receptor activity impairs ER MR/GR activity improves ER	Administration of specific receptor agonists (e.g., adrenergic receptors: yohimbine vs. GC receptors: hydrocortisone vs. both) vs. placebo	Calculating a ratio of delta values (peak - baseline) between biomarkers of the stress systems and including it as a predictor in the statistical model
2. Does the predominant stress system determine the direction of stress effects on ER?	SNS dominance causes ER impairments HPA dominance causes ER improvements	Administration of specific receptor antagonists prior to stress to experimentally induce dominance of one stress system over the other (e.g., adrenergic receptors: propranolol vs. GC receptors: metyrapone vs. both)	Variation in timing of ER assessment relative to stress exposure (e.g., ER in anticipation of or during stress exposure vs. $\geq$ 25 min after stress)
3. Do beneficial cortisol effects on ER rely on a boosted recruitment of the PFC?	Deactivation of the PFC reduces ER performances especially after stress/ cortisol manipulation	*Neuroscientific approach:* rTMS stimulation of the dlPFC (inhibition) or sham condition during ER assessment 25 min after stress/ cortisol manipulation vs. control condition *Behavioral approach:* Manipulation of PFC resources by cognitive exhaustion (e.g., difficult WM task vs. easy task) during ER assessment after stress/cortisol manipulation	
4. Do delayed, genomic GC effects on ER continue for hours after stress?	Delayed GC effects benefit ER both, 90 and 240 min after experimental manipulation	Administration of a GC agonist (e.g., hydrocortisone) or a placebo either 90 or 240 min prior to ER	Exposure to acute stress or a control condition either 90 or 240 min prior to ER
5. Do stress (hormone) effects depend on the regulatory goal?	Stress (hormones) alter downregulation of negative emotions / upregulation of positive emotions (pro-hedonic) but not the opposite direction	Stress (hormone) manipulation or control condition prior to an ER task in which participants are asked to up- and downregulate negative and positive stimuli with different strategies	
6. Do stress (hormone) effects depend on the emotional stimuli?	Stress (hormone) effects on ER vary as a function of stimulus intensity (inverted U-shape relationship)	Stress (hormone) manipulation or control condition prior to an ER task which varies in emotional stimulus intensity and its vividness: e.g., low and high intensity pictures, scripts, videos and emotional challenging interpersonal interactions	

Note: rTMS = repetitive transcranial magnetic stimulation; PFC = prefrontal cortex; ER = emotion regulation; GC = glucocorticoids; MR = mineralocorticoid receptor; GR = glucocorticoid receptor; sAA = salivary alpha-amylase; SNS = sympathetic nervous system; HPA axis = hypothalamus-pituitary-adrenocortical axis

the up-/downregulation of positive emotions. Therefore, future research is needed to determine whether and how the effects of stress on ER are limited to specific regulatory goals.

5. Concluding remarks and future perspectives

5.1. Conclusion

In this systematic review, we summarize and discuss the current literature on the effects of acute stress and hormonal responses on cognitive ER. Findings on the effects of acute stress are mixed, showing beneficial, impairing, or no effects on the ability to downregulate negative emotions. Pharmacological studies provide evidence that cortisol administration facilitates the downregulation of negative emotions. Moreover, biomarkers of the two major stress systems - the SNS (sAA, cardiovascular activity) and the HPA axis (cortisol) - have been associated with ER performance in opposite directions. Given that the two stress systems differ in timing of their molecular actions in the brain, variations in the delay between stress exposure and ER assessment across studies may explain heterogeneous findings. We thus propose the *Predominant Stress System Underpins Regulation of Emotions* (PRESSURE) model, which postulates that the direction and magnitude of stress effects on ER depend on the relative predominance of one stress system over the other (SNS vs. HPA axis). Accordingly, the predominance of catecholaminergic actions, which rapidly impair ER performance following stress, is later counteracted by beneficial (non-genomic and genomic) GC actions on the ER network. These stress hormonal mechanisms may interact with interindividual and contextual factors such as participant sex, emotional stimulus intensity, and ER strategy, collectively enabling successful adaptation to emotional challenging environments. The PRESSURE model serves as a framework for future studies to advance our understanding of the complex interplay between neuroendocrine actions and ER processes in the aftermath of stress.

5.2. Limitations, open questions and future research directions

This review reveals some limitations of the current state of research and methodological challenges that need to be addressed in future studies. First, we did not identify any study that used a pharmacological approach to examine the effects of noradrenergic activity on ER processes. Therefore, it remains unclear whether the noradrenergic system exerts effects on ER attempts exclusively or in interaction with other stress hormones. Future studies selectively activating α- or β-adrenergic receptors in comparison to and in combination with manipulation of the GC system, are necessary to test its causal role for ER processes (Table 3; research question 1). Additionally, evidence for the effects of SNS dominance on ER is constrained by the rapid return of SNS activity to baseline following stress offset. In the present studies, the ER task was scheduled after stress exposure, when SNS activity had already diminished. This issue could be solved by experimental designs in which the ER task is conducted concurrently with the stress induction procedure (e.g., foot (SE)CPT during a computer-based ER task; alternating blocks of stress and ER) or during the anticipation phase of an imminent stressful situation. Of note, pharmacological suppression of one stress system (e.g., HPA axis) prior to stress exposure has been shown to boost reactivity of the other system (e.g., SNS; Ali et al., 2020). Future studies could make use of this mechanism by experimentally manipulating dominance of one stress system over the other via administration of a GC and noradrenergic receptor antagonist to test for predictions of the PRESSURE model presented in Section 4.1 (Table 3; research question 2).

There are several hints in the literature for beneficial stress effects on ER to rely on a boosted recruitment of prefrontal control resources (Jentsch et al., 2019; Langer et al., 2020). Future research could test this idea using a neuroscientific approach, such as manipulating dlPFC activation through repetitive transcranial magnetic stimulation (***rTMS;*** cf. glossary) while participants engage in an ER task after stress. Alternatively, available PFC resources could be manipulated by inducing

cognitive exhaustion, having participants complete a challenging cognitive task prior to or during the ER paradigm (Table 3; research question 3)

Furthermore, existing studies examining delayed, genomic cortisol effects on ER consistently used a 90 min delay before ER assessment. In the pharmacological studies, cortisol levels remained elevated at the beginning of the ER task (Jentsch et al., 2019; Langer et al., 2021a; Pan et al., 2023) suggesting that non-genomic GC actions may still have influenced ER outcomes. Therefore, a 90 min delay may be insufficient to fully isolate the two molecular action pathways. Additionally, animal research has shown that genomic GC actions persist for at least four hours (Joëls et al., 2012). Given the lack of data on ER effects > 90 min after cortisol manipulation, the duration and trajectory of cortisol-driven effects on ER remain unclear. Extending the delay between stress induction or pharmacological challenge and ER assessment in future research may help gaining knowledge on genomic GC effects on ER over time (Table 3; research question 4).

Another limitation of the current research is the high variability in methodological approaches, which hampers synthesis of findings across studies. For instance, stress induction methods vary among the TSST, CPT, SECPT, and ScanStress-C. These stressors differ in their potential to stimulate the SNS and HPA axis (Schwabe and Schächinger, 2018). Given the PRESSURE model's assumption of opposing effects between the two major stress systems, variations in the ratio of SNS to HPA axis reactivity may influence ER performance following stress.

To address this issue and improve comparability across studies, future research may benefit from calculating a ratio between delta values (peak – baseline) of biomarkers from each stress system and including this ratio as a moderator of stress effects on ER in the statistical model. Moreover, different outcome measures are used to quantify ER performance: self-report instruments, physiological measures such as SCR, HR, pupil dilation and EMG responses and neural activity via EEG or fMRI. These measures differ in their sensitivity to changes in emotional arousal and valence during ER processes. Whereas autonomic measures (SCR, HR and pupillometry) appear to reflect changes in emotional arousal, EMG responses are more sensitive to changes in valence. Beyond, psychophysiological measures differ in their suitability for depicting ER performances. Whereas the effects of reappraisal and suppression on autonomic measures are rather small, EMG responses appear to be more sensitive to regulatory changes in emotions at least when applying reappraisal (see meta-analyses by Zaehringer et al., 2020). It is worth noting that there are several hints in the literature that pupil sizes are more sensitive to the regulatory effort than changes in emotional activation during cognitive ER (Kinner et al., 2017; Langer et al., 2021a, 2020) reducing comparability with other autonomic measures. Moreover, the reviewed studies have focused on the up-/downregulation of negative emotional stimuli, whereas positive stimuli have never been used so far. Even though existing data indicate that stress effects on ER are restricted to pro-hedonic regulatory goals (e.g., downregulation of negative emotions), it is completely unclear whether this is valid for the up-/downregulation of positive stimuli (Table 3; research question 5). Furthermore, existing ER tasks are limited by the examined strategies (reappraisal and distraction) and the emotional stimuli used (most often pictures) challenging ecological validity. Given initial hints for stimulus intensity to affect stress effect sizes on ER, future studies are needed comparing a variety of emotional stimuli (e.g., pictures, scripts, videos, interpersonal interactions) to test whether there is a linear or rather inverted U-shaped relationship between stimulus intensity and ER performances after stress (Table 3; research question 6).

Notably, available studies have primarily focused on regulatory performance assessed through paradigms in which participants are instructed to apply a fixed strategy. However, ER competencies in daily life critically depend on the ability to flexibly choose and switch between different regulatory strategies (Aldao et al., 2015), a skill that may be particularly challenged in stressful situations. Langer et al. (2022) were the first to show that acute stress may promote a preference for distraction over reappraisal, especially when dealing with high-intensity emotions. It remains completely unknown, however, whether participants who initially select a non-optimal strategy (e.g., reappraisal for high-intensity emotions) are yet flexible to switch to a more effective one (e.g., distraction for high-intensity emotions) when given the option. Moreover, it is still an open question whether stress effects on strategic regulatory decisions in fact alter ER performances. Thus, future studies assessing ER flexibility - both in terms of strategy selection and switching - and its impact on ER performance under stress may offer valuable advancements for the field.

Taken together, research on the effects of acute stress and stress hormones on ER has expanded considerably in recent years, showing great potential for further advancements in the future. Although still in its early stages, the current body of research demonstrates that ER processes can indeed be influenced by hormonal mechanisms in response to stressful situations. The PRESSURE model offers the first integrative framework for understanding the effects of stress on ER, explaining heterogeneous findings through timing differences in ER assessments following stress. However, further steps are needed to deepen our understanding of these effects, including more precise delineation of the roles of specific hormones, exploration of the timing and duration of their impact on ER, and examination of individual and contextual factors that may moderate these effects. Advancing research in this area is essential for the development of clinical interventions aimed at improving ER skills in stressful situations when they are needed the most.

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Glossary

Acceptance: A cognitive emotion regulation strategy that involves allowing emotions to be present without trying to change, suppress, or judge them.
Agonist: A substance that binds to a specific receptor type and activates it, mimicking the action of a natural substance to stimulate a biological response.
Emotion regulation: Emotion regulation (ER) refers to the process of influencing the intensity, duration, or expression of one's emotions, either by changing how one experiences or reacts to emotional situations. It involves strategies like reappraisal, distraction, and acceptance to manage emotional responses.
Emotion regulation network: A system of brain regions, including the prefrontal cortex, amygdala, and insula, that work together to manage and control emotional responses. This network determines how emotions are experienced, expressed, and controlled.
Catecholamines: A group of neurotransmitters, including dopamine, adrenaline and noradrenaline that are involved in the body's response to stress, regulating functions such as heart rate, blood pressure, and mood. In the periphery, they are released by the adrenal medulla and in the brain by the locus coeruleus.
CPT: The Cold-Pressor Test (CPT) is a standardized procedure to induce acute physical stress. Participants are asked to immerse their hand or foot in ice-cold water for three minutes.
Distraction: An emotion regulation strategy that involves redirecting attention away from an emotional trigger to reduce its impact, often by focusing on a neutral or pleasant activity or thought.
dlPFC / vlPFC: Dorsolateral (dl) and ventrolateral (vl) regions of the prefrontal cortex (PFC) located in the frontal lobe of the brain, involved in higher cognitive functions such as decision-making, working memory, and emotion regulation. It plays a key role in controlling thoughts and behaviors, including deliberate attempts to alter emotional responses.
fight-or-flight response: A body's acute stress reaction that prepares it to either confront or flee from a perceived threat. It involves activation of the sympathetic nervous system, increasing heart rate, blood flow, and energy, while inhibiting non-essential functions.
fMRI: Functional magnetic resonance imaging (fMRI) is a neuroimaging technique that measures brain activation by detecting changes in blood flow, allowing researchers to observe which areas of the brain are active during specific tasks or stimuli.
Genomic GC actions: Delayed effects of glucocorticoids (GCs) that involve binding to intracellular receptors, leading to changes in gene expression. These actions take at least one hour to initiate and then continue for several hours.
Glucocorticoids (GCs): A class of steroid hormones produced by the adrenal glands that play a key role in regulating metabolism, reducing inflammation, and managing the body's response to stress. Cortisol is the primary glucocorticoid in humans.
Glucocorticoid receptor (GR): A type of protein found in cells that binds to glucocorticoids, allowing these hormones to regulate gene expression and influence various physiological processes, including stress response, immune function, and metabolism.
HPA axis: The HPA axis (Hypothalamus-Pituitary-Adrenocortical axis) is a central stress

response system involving the hypothalamus, pituitary gland, and adrenal glands. It regulates the body's reaction to stress by controlling the release of cortisol and other stress hormones, affecting mood, immune function, and energy levels.

Locus coeruleus (LC): A small cluster of neurons in the brainstem that is the primary source of noradrenaline in the brain. It plays a key role in regulating arousal, attention, and the stress response.

Mineralocorticoid receptor (MR): A protein found in cells that binds to mineralocorticoids, such as aldosterone, to regulate salt and water balance, blood pressure, and electrolyte levels in the body.

Non-genomic GC actions: Quick effects of glucocorticoids (GCs) on neural activity without altering gene expression that occur as long as GC levels are elevated. These actions typically involve the activation of cellular signaling pathways, leading to immediate changes in cell function, such as altering neurotransmitter release or modulating ion channels.

Reappraisal: A cognitive emotion regulation strategy that involves changing one's interpretation of a situation to alter its emotional impact. This can either happen by reframing the situation within another context or thinking about another ending.

rTMS: repetitive Transcranial Magnetic Stimulation (rTMS) is a non-invasive brain stimulation technique that uses magnetic fields to stimulate or inhibit nerve cells in specific brain regions. By delivering repeated magnetic pulses through a coil placed on the scalp, rTMS can modulate neural activity.

SECPT: The Socially Evaluated Cold-Pressor Test (SECPT) is a variation of the Cold-Pressor Test in which participants immerse their hand or foot in ice-cold water for three minutes while being observed by a reserved experimenter, adding a social evaluation component.

SNS: The SNS (Sympathetic Nervous System) is part of the autonomic nervous system responsible for the body's rapid, involuntary response to stressful or emergency situations, often called the "fight-or-flight" response. It increases heart rate, blood flow, and energy availability to prepare the body for action.

TSST: The Trier Social Stress Test (TSST) is an instrument to induce acute psychosocial stress in the laboratory. It simulates a mock-job interview in front of a reserved committee including a public speech and a mental arithmetic task while being evaluated by the committee.