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Effects of neuromodulation on cognitive and emotional responses to psychosocial stressors in healthy humans

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ABSTRACT

Physiological and psychological stressors can exert wide-ranging effects on the human brain and behavior. Research has improved understanding of how the sympatho-adreno-medullary (SAM) and hypothalamic-pituitary-adrenocortical (HPA) axes respond to stressors and the differential responses that occur depending on stressor type. Although the physiological function of SAM and HPA responses is to promote survival and safety, exaggerated psychobiological reactivity can occur in psychiatric disorders. Exaggerated reactivity may occur more for certain types of stressors, specifically, psychosocial stressors. Understanding stressor effects and how the body regulates these responses can provide insight into ways that psychobiological reactivity can be modulated. Non-invasive neuromodulation is one way that responding to stressors may be altered; research into these interventions may provide further insights into the brain circuits that modulate stress reactivity. This review focuses on the effects of acute psychosocial stressors and how neuromodulation might be effective in altering stress reactivity. Although considerable research into stress interventions focuses on treating pathology, it is imperative to first understand these mechanisms in non-clinical populations; therefore, this review will emphasize populations with no known pathology and consider how these results may translate to those with psychiatric pathologies.

1. Stress regulation

1.1. Overview

Stress is one of the most significant contributors to 21st century health problems. Stress reactivity occurs via multiple mechanisms (McEwen, 2007). In humans, stressors represent any threat to well-being, or any real or perceived disruption of physiological homeostasis (Goldstein and McEwen, 2002; Myers et al., 2012). Homeostasis is regulated by multiple brainstem nuclei that respond during departure from physiologic set-point (Chrousos and Gold, 1992; Park et al., 2020). Physiologic stressors (e.g. injury, exercise) often affect physiologic set-points, such as temperature, blood volume, blood pressure, and pH (Davies, 2016). Psychosocial stressors (e.g. public speaking) disrupt homeostasis indirectly by perturbing emotional balance, which then modifies physiological responses (McKlveen et al., 2013, 2015). In contrast to disruptions of physiological homeostasis, responses to psychosocial stressors begin in higher brain regions such as the prefrontal cortex (PFC) and inter-connected limbic nuclei. Despite this difference in origin, there is considerable overlap between the physiological systems and neurotransmitters involved in both physiological and psychosocial stress responses (Ulrich-Lai and Herman, 2009).

Responses to stressors are produced by the sympatho-adreno-medullary (SAM) axis and hypothalamic-pituitary-adrenocortical (HPA) axis. The SAM-axis mediates immediate responses to stressors (within seconds) via increased sympathetic nervous system (SNS) activation and decreased parasympathetic nervous system (PNS) activation (Carlson and Kraus, 2021; McCorry, 2007). The most abundant SNS neurotransmitters in the body are norepinephrine (NE) and epinephrine (E), which activate adrenergic receptors (Baak, 2001), and produce effects that depend on receptor subtype(s) and effector organ. Consistent acute responses include increased respiration, increased heart rate, blood pressure, and pupil dilation (Guyenet, 2006; Guyenet et al., 2013; Jänig, 2006; Molina, 2005; Ziemssen and Siepmann, 2019). In contrast to immediate SAM responses, HPA-axis responses occur over minutes to hours. During HPA axis activation, the adrenal cortex releases glucocorticoids, especially cortisol (Keller-Wood and Dallman, 1984).

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Glucocorticoids act on glucocorticoid receptors, ubiquitous throughout the brain and periphery, which regulate genes involved in development, inflammation, and immune response (Dickerson and Kemeny, 2004; Kadmiel and Cidlowski, 2013; Uchoa et al., 2014). Effects of glucocorticoids occur in two stages (Joëls et al., 2012). Immediate effects peak 20–30 min after stressor onset and reflect glucocorticoid binding to membrane-bound receptors, which induces rapid, non-genomic effects such as alterations in adrenergic receptor trafficking to the membrane (Joëls et al., 2013). Delayed genomic effects occur due to glucocorticoid binding to cytoplasmic receptors; these effects begin >1 h after stress onset and continue for several hours resulting in changes in gene transcription and translation (Groeneweg et al., 2012; Hermans et al., 2014).

1.2. Acute stress in humans

1.2.1. Neural regulation of acute stress response

The various neural mechanisms associated with acute stress are detailed elsewhere (Dedovic et al., 2009a; Herman et al., 2012; Myers et al., 2012; Shirazi et al., 2015; Ulrich-Lai and Herman, 2009). Here, we briefly outline key brain regions that regulate acute stress responses to provide a foundation for discussing the effects of neuromodulation. Neural regulation and responses to stressors may differ based on the stressor type; due to the substantial impact of psychosocial stressors on people with psychiatric disorders, this narrative review will focus on mechanisms that are relevant to psychosocial stressors in healthy persons (see Table 1 for a list of key psychosocial stressors). We know that neural responses to stressors can vary depending on underlying pathophysiology; therefore, it is important to review these mechanisms in healthy persons to ensure a thorough understanding of how different pathophysiology impacts these responses. Although SAM and HPA responses are differentially controlled, there is overlap in their regulation at a neural level, and changes in one system are typically mirrored by changes in the other.

The neural stress response can be broadly viewed as consisting of three levels of regulation (Ulrich-Lai and Herman, 2009). Fig. 1 shows details of key regions involved in this regulatory system. The first 'bottom-up' stage is monitored via the brainstem, which responds to signals of homeostatic imbalance, e.g. pain and inflammation (Myers et al., 2017; Petrovic et al., 2004; Salcido et al., 2018). Some imbalances can be life-threatening (e.g. hemorrhage) so immediate response is required and reflex arcs control rapid initial SNS responses (Ziemssen and Siepmann, 2019). Brainstem connections to sites in the midbrain and forebrain integrate, modulate, and monitor SAM responses (Herman et al., 2005; McKlveen et al., 2013, 2015; Ross and Van Bockstaele, 2020; Ulrich-Lai and Herman, 2009).

The hypothalamus plays a major role in the second or 'middle-management' level of psychosocially-mediated SAM and HPA responses (Ulrich-Lai and Herman, 2009). The hypothalamus and bed nucleus of the stria terminalis (BNST) are key integrators at this level. The BNST is a grey matter structure within the extended amygdala, which is a relay site for HPA-axis responses (Crestani et al., 2013; Dumont, 2009; Lebow and Chen, 2016). Limbic modulation of the HPA-axis is primarily mediated by BNST subregions that serve distinct roles in the stress response (Choi et al., 2007; Herman et al., 2003).

There is a complex interplay between signals that modulate SAM and HPA-axis basal tone and responses to stressors. Whereas the hypothalamus and BNST integrate these signals, forebrain areas including the PFC, amygdala, and hippocampus are responsible for 'top-down' regulation of these responses (Herman et al., 2005; Ulrich-Lai and Herman, 2009). These forebrain areas process higher-order sensory inputs alongside ascending inputs and modulate responses to physiological and psychological stressors. As psychosocial stressors indirectly influence physiological measures of homeostasis, there is no recognition of 'psychosocial stress' via brainstem pathways; however, SAM and HPA circuits are activated. Outputs from limbic areas such as the amygdala and hippocampus converge on subcortical sites responsible for 'middle management' of stress responses and their roles depend on the subregion activated and stressor type (Dayas et al., 1999, 2001; Herman and Mueller, 2006; Prewitt and Herman, 1997; Sawchenko et al., 2000; Xu et al., 1999).

Neural systems that regulate stress responding can be divided by the type of activity that is regulated (SAM or HPA-axis), type of stress recognized (physical vs. psychological), and direction of regulation (activation or inhibition) (Choi et al., 2007; Herman et al., 2003, 2005; Jacobson and Sapolsky, 1991; Pacak, 2000; Sawchenko et al., 2000; Ulrich-Lai and Herman, 2009). Importantly for this discussion, psychosocial stressors have a primarily 'top-down' effect, wherein forebrain areas process the stressful experiences first, resulting in indirect alteration of physiological measures of homeostasis. This top-down initiation of the stress response highlights an important mechanism through which neuromodulation could be used to modulate these stress responses. With this broad understanding of stressor response and regulation, we next focus on experimental induction of acute stress and its direct effects on physiological and psychological responses.

1.2.2. Importance of managing responses to stressors

Physiological responses to stressors can promote survival and safety, but can also produce negative psychobiological effects and facilitate development or exacerbation of psychiatric disorders (Jacobson, 2014; McEwen and Morrison, 2013; Pacák and Palkovits, 2001). Chronic stress-response activation through prolonged or repeated exposures can induce a different, sometimes opposite, series of effects. Although the role of chronic stress in psychiatric disorders and pathological behavior is integral to understanding stress reactivity, it is covered elsewhere (Conrad, 2010; Conrad et al., 2017; Herman, 2013; Lupien et al., 2018; Picard et al., 2021; Vyas et al., 2016).

This review focuses on the effects of acute psychosocial stressors and the potential for neuromodulation to alter reactivity to these stressors, thereby providing insights into the brain circuits that modulate stress reactivity. Considerable research into stress interventions focuses on treating or preventing pathology, but it is important to first understand these mechanisms in non-clinical populations; therefore, this review will emphasize populations with no known pathology and consider how these results may translate to those with different psychiatric pathologies.

2. Effects of acute stressors

2.1. Types of experimental stress

To investigate the effects of acute stressors on human physiological and behavioral responses, it is necessary to identify experimental interventions that recapitulate effects of ecological stress. Notably, studies show that destruction of ascending brainstem catecholaminergic neurons significantly reduces HPA-axis responses to stressors that cause *physiological* homeostatic imbalance; however, this destruction does not alter HPA-axis response to *psychological* stress, which illuminates distinct regulation of stress reactivity (Herman et al., 2003; Ritter et al., 2003). Psychosocial stress arises from the psychological need to be affiliated with others and is therefore defined as any type of social threat, which includes social evaluation, social exclusion, social defeat, and goal-focused performance evaluation (Kogler et al., 2015).

The development of reliable experimental psychological stressors is complicated because, compared to physical stressors, there is substantial interindividual response variability. Although precise methods of psychosocial stress induction vary, Table 1 shows the most common types of psychosocial stressor and their effects. The most experimentally advantageous psychosocial stress-induction procedures should induce stress responses (i.e. be effective) for the majority of the population. In general, evaluation of the efficacy of psychosocial stress induction focuses on key markers of SAM and HPA-axis activity: serum ACTH, cortisol, and catecholamines (Clemens Kirschbaum et al., 1993; Mutti

Table 1
Overview of methods and effects of common psychosocial stressors used to experimentally induce stress in human subjects.

Stressor	Category	Stressor Description	Control	Approximate Duration	Effects of Stressor on		Evidence of Habituation on:
					Affect/Cognition	Physiology	
Trier Social Stress Test (TSST)	Public speaking, mental arithmetic, anticipation, social evaluation	1. Prepare speech to give to a panel of judges	Friendly TSST	**Stressor:** 10 min anticipation, 10 min test	**Affective:** ↓ mood, ↑ stress and anxiety	**SAM:** ↑ HR & BP, inconsistent HRV findings	**Affect/Behavior:** No change in subjective stress
		2. Present speech to a panel of assessors				↑ salivary α-amylase, plasma adrenaline & noradrenaline	**Physiology:** ↓ HRV, HR, and HPA response; no change in SAM response
		3. Mental arithmetic task in front of judges		**Effects:** 30–60 min	**Cognitive:** Impaired working memory, cognitive flexibility, and cognitive inhibition	**HPA-Axis:** ↑ cortisol & ACTH	**NOTE:** modified rTSST created for repeated use
Critical Feedback	Social evaluation	Participants receive negative feedback during a trial, ranging from after completion of a task to self-criticism	Neutral or positive feedback	**Stressor:** 8 min	**Affective:** ↓ mood, ↑ stress and anxiety	**SAM:** ↓ HRV	**Affect/Behavior:** ?
				Effects: ~15 min	**Cognitive:** ?	**HPA-Axis:** No effect on cortisol	**Physiology:** ?
Paced Auditory Serial Addition Task (PASAT)	Mental arithmetic	1. Participants listen to audio recording and are given a number about every 3 s	N/A	**Stressor:** Up to 5 min	**Affective:** ↑ stress and anxiety	**SAM:** ↑ HR & BP, ↓ HRV	**Affect/Behavior:** ↓ stress & anxiety
		2. Must add the number they most recently heard with the one heard before		**Effects:** 20–30 min[a]	**Cognitive:** Impaired working memory, no effect on cognitive inhibition	**HPA Axis:** No cortisol response	**Physiology**: No change in cardiovascular reactivity
Cyberball	Social exclusion	1. Participants play an online ball/frisbee tossing game with fictitious others where they are typically "left out"	Inclusion or fair play	**Stressor:** 3 min per condition	**Affective:** ↓ mood and ↑ anxiety	**SAM:** ↑ HR & BP, ↑ respiratory rate & skin conductance, ↑ salivary α-amylase	**Affect/Behavior:** ?
		2. Complete questionnaire afterwards about how they felt during the game		**Effects:** 15–45 min[a]	**Cognitive:** Impaired working memory	**HPA-Axis:** ↑ salivary cortisol	**Physiology:** ?
Iowa Singing Social Stress Test (I-SSST)	Public "speaking", social evaluation	1. Participants presented with a series of neutral messages on a screen, with 1-min intervals between each message block	N/A	**Stressor:** ~15 min total, 20 s singing	**Affective:** ↓ mood, ↑ stress and anxiety	**SAM:** ↑ HR, no effect on BP ↑ in skin conductance; overall ↑ SAM activity	**Affect/Behavior:** ?
		2. Last message block instructs the participant to sing out loud		**Effects:** 30–45 min	**Cognitive:** ?	**HPA-Axis:** ↑ salivary cortisol	**Physiology:** No effect on cortisol response
Simple Singing Stress Test (SSST)	Public "speaking", social evaluation	1. Participant told to think of a song to sing to experimenter, given 60-sec to prepare	Reading lyrics out loud	**Stressor**: ~5 min	**Affective:** ↑ stress and anxiety	**SAM:** ↑ HR & BP, ↑ respiratory rate & skin conductance	**Affect/Behavior:** ?
		2. Participant recorded singing to experimenter					
		3. Portions of recording are played back		**Effects**: ~45 min	**Cognitive:** ?	**HPA-Axis:** ↑ salivary cortisol	**Physiology:** ?
		4. Participants informed that they would have to sing again at the end and be assessed					
Socially Evaluated Cold-Pressor Group Tests (SECPT)	Physiologically challenging, Social evaluation	While being recorded, participant instructed to submerge hand into ice water while silently staring into camera until told to stop	Warm water, no videotaping, duration is disclosed	**Stressor:** 3 min	**Affective:** ↓ mood, ↑ stress	**SAM:** ↑ HR & BP,	**Affect/Behavior:** No effect on subjective stress
				Effects: 60 min	**Cognitive:** Impaired working memory	**HPA-Axis:** ↑ cortisol & HPA axis activation	**Physiology:** ↓ HR reactivity, no effect on BP or cortisol

(*continued on next page*)

Table 1 (*continued*)

Stressor	Category	Stressor Description	Control	Approximate Duration	Effects of Stressor on		Evidence of Habituation on:
					Affect/Cognition	Physiology	
					and cognitive inhibition		
Montreal Imaging Stress Task (MIST)	Mental arithmetic, Social evaluation	1. Participants complete a series of mental arithmetic tasks with induced failure algorithm	Lack of social evaluative threat (e.g. no negative feedback)	**Stressor:** 2–6 min per run	**Affective:** ↓ mood, ↑ stress and anxiety	**SAM:** ↑ HR & BP, ↓ HRV ↑ stress-induced dopamine release; ↑ skin conductance	**Affect/Behavior:** No effect on mood or stress **Physiology:** ↓ in HR and HRV reactivity, ↓ HPA-axis
		2. Social evaluative threat presented by the investigator and within the program		**Effects:** ~45 min[a]	**Cognitive:** Impaired memory retrieval, no effect on short-term memory	**HPA-Axis:** ↑ cortisol[a]	**NOTE:** modified rMIST created for repeated use
Maastricht Acute Stress Test (MAST)	Physiologically challenging, Social evaluation	1.5-min preparation phase	Lukewarm water; Simple counting, no negative feedback	**Stressor:** 5 min preparation, 10 min exposure	**Affective:** ↓ mood, ↑ stress and anxiety	**SAM:** ↑ HR & BP,	**Affect/Behavior:** No effect on stress or mood
		2. Five SECPT-like trials from 60 to 90 s		**Effects:** ~30 min	**Cognitive:** No effect on working memory	↑ salivary α -amylase	
		3. Between immersion trials, TSST-like mental arithmetic trials with negative feedback				**HPA-Axis:** ↑ salivary cortisol	**Physiology**: No effects on α-amylase or cortisol
Yale Interpersonal Stressor Task (YIPS)	Social evaluation, Social exclusion	1. Discussion on a given topic presented by the experimenter to participant and 2 confederates	Given page of randomly typed letters and asked to circle every fifth "e" for 5 min	**Stressor:** Exp. #1: 5 min discussion	**Affective:** ↓ mood, ↑ stress and anxiety	**SAM:** ↑ HR & BP	**Affect/Behavior:** ?
		2. During discussion, confederates employ exclusion techniques against the participant		Exp. #2: 15 min			
		3. Participant completed BSPQ		**Effects:** ~30 min	**Cognitive:** ?	**HPA-Axis:** ↑ salivary cortisol	**Physiology:** ?

"?" indicates published data on these outcomes could not be identified by the authors at this time.
Abbreviations: HR: heart rate, HRV: heart rate variability, BP: blood pressure, ACTH: adrenocorticotropic hormone, HPA-axis: hypothalamic-pituitary-adrenocortical axis, SAM: sympatho-adreno-medullary axis.
References: TSST: Allen et al. (2014), 2017; Giles et al. (2014); Clemens Kirschbaum et al., 1993; Labuschagne et al. (2019); Narvaez Linares et al. (2020); N Y L Oei et al. (2006); Plessow et al. (2011), Critical Feedback: Chris Baeken et al., 2018; Chida and Hamer (2008); De Raedt et al. (2017); Nummenmaa and Niemi (2004), PASAT: Bachmann et al. (2019); Diehr et al. (1998); Gallagher et al. (2018); Hendrawan et al. (2012); Lockwood et al. (2004); Mathias et al. (2004); Tombaugh (2006), Cyberball: Eres et al. (2021); Helpman et al. (2017); K. D. Williams and Jarvis (2006); Williamson et al. (2018); Zadro et al. (2004); Zöller et al. (2010), I-SSST and SSST: Brouwer et al. (2018); Brouwer and Hogervorst (2014); Jump and Dockray (2021); Le et al. (2021); Reschke-Hernández et al. (2017); Sequeira et al. (2021); van der Mee et al. (2020); SECPT: Boyle et al. (2016); Giles et al. (2014); Meir Drexler et al. (2017); Minkley et al. (2014); Schwabe and Schächinger (2018), MIST: De Calheiros Velozo et al. (2021); Dedovic et al. (2005); Dedovic, D'Aguiar et al., 2009; Nair et al. (2020); Nitschke et al. (2020); Noack et al. (2019), MAST: Bali and Jaggi (2015); Meyer et al. (2013); C. W.E.M. Quaedflieg et al. (2017); Conny W.E.M. Quaedflieg et al. (2013); Shilton et al. (2017); Smeets et al. (2012), YIPS: Stroud et al. (2000); Zwolinski (2008)..
[a] indicates that effects were measured up to this time.

et al., 1989). Meta-analysis of psychological stress induction suggests the Trier Social Stress Test (TSST) may be the most reliable when considering these biomarkers (Allen et al., 2014; Birkett, 2011; Dickerson and Kemeny, 2004). However, neuroimaging studies demonstrate that different types of psychosocial stress induction may activate different neural regions depending on the type of threat, which provides some insight into the complexity of stress-related responses (Noack et al., 2019). Studies that have examined *subjective* measures of stressor effects (e.g. drug craving) across different populations have found varying results (Sinha, 2009; Sinha et al., 1999, 2011). These findings suggest optimal stress-induction methods may differ depending on the population and the outcomes of interest.

Despite improvements upon experimental methods of stress induction, one concern with both physical and psychosocial stressors is their inconsistency (Harris et al., 2005; Liu et al., 2017; Skoluda et al., 2015; Takai et al., 2004; van Stegeren et al., 2008). Although these methods have improved our basic understanding of stress responses, their utility is limited as effects of each stressor vary between and within subjects. Giles et al. (2014) compared responses to three commonly-used stressors (TSST, cold pressor test [CPT], and mental arithmetic) and found different biobehavioral responses to each stressor (Giles et al., 2014). This inconsistency between different stressors has been replicated in multiple contexts (Singh et al., 1999; Skoluda et al., 2015). As suggested by Skoluda et al., 2015) some of these differential responses may be partly due to distinct profiles of HPA and SAM activation. Furthermore, discordant findings between physiological indices (e.g. serum cortisol) and subjective indices (e.g. perceived stress), is unsurprising given the different ways in which the responses to stressors are regulated (Campbell and Ehlert, 2012). Discrepant outcomes between tasks and other major limitations such as lack of placebo-control, brief duration of effect, inability to manipulate stressor severity, and habituation contribute to disparate findings and small effect sizes reported in meta-analyses (Boesch et al., 2014; Gerra et al., 2001; Shields et al., 2016a).

Although there are limitations to experimentally-induced psychosocial stress and discrepancies between physiological and behavioral

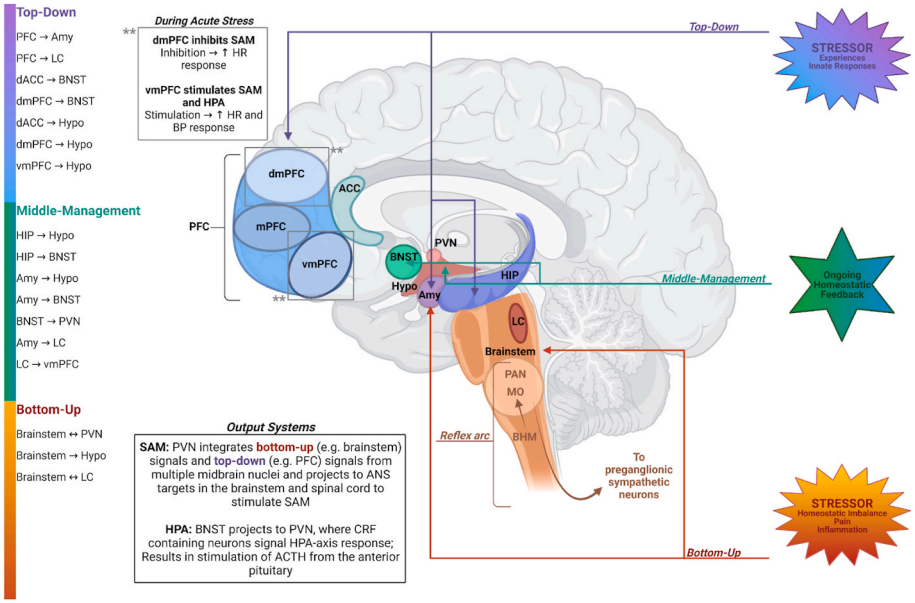

Fig. 1. Overview of key levels involved in neural regulation of responses to acute stressors
Abbreviations: mPFC: medial prefrontal cortex, vmPFC: ventromedial prefrontal cortex, dmPFC: dorsomedial prefrontal cortex, dlPFC: dorsolateral prefrontal cortex, ACC: anterior cingulate cortex, BNST: bed nucleus of the stria terminalis, Amy: amygdala, HIP: hippocampus, Hypo: hypothalamus, PVN: paraventricular nucleus of the hypothalamus, CRF: corticotropin-releasing factor, BHM: brainstem homeostatic monitors, MO: medulla oblongata, PAN: pre-autonomic neurons.

responses to these varying methods of inducing stress, all approaches have advanced understanding of the effects of acute stressors on physiological, behavioral, and cognitive outcomes. Next, we will evaluate effects of acute stressors on cognitive, and emotional outcomes and the theorized mechanisms for these effects.

2.2. *Effects of stressors on*

2.2.1. *Neural activity*

Characterizing neurophysiological responses to acute stressors may help explain certain cognitive and behavioral changes that occur under stress. Neural effects of stressors differ considerably depending on multiple factors including stress type and study population (Dedovic et al., 2009b; Noack et al., 2019; Wang et al., 2007); therefore, this review presents data only from healthy subjects unless otherwise specified. A condensed but comprehensive way to explore the CNS response to acute stressors is by reviewing key neural networks. The triple model of acute stress highlights three neural networks that play key roles in cognitive functioning (Menon, 2011). Fig. 2 illustrates the associated nodes of each network and their responses to acute stressors; each network will be briefly discussed (for an exhaustive review see: (van Oort et al., 2017).

The Default Mode Network (DMN) is active during rest and is important for self-referential mental activity (Menon, 2011; Raichle et al., 2001). The DMN consistently shows deactivation during goal-directed cognitive tasks (Hermans et al., 2014); this switch from DMN to other networks (e.g. Central Executive Network) appears to be mediated by resting activity in the fronto-insular cortex (rFIC) (Sridharan et al., 2008). Although the DMN is not typically considered integral to the stress response, neuroimaging studies demonstrate that under stress, there is consistent activation of the DMN (van Oort et al., 2017). Stress-induced DMN activation occurs even during situations of

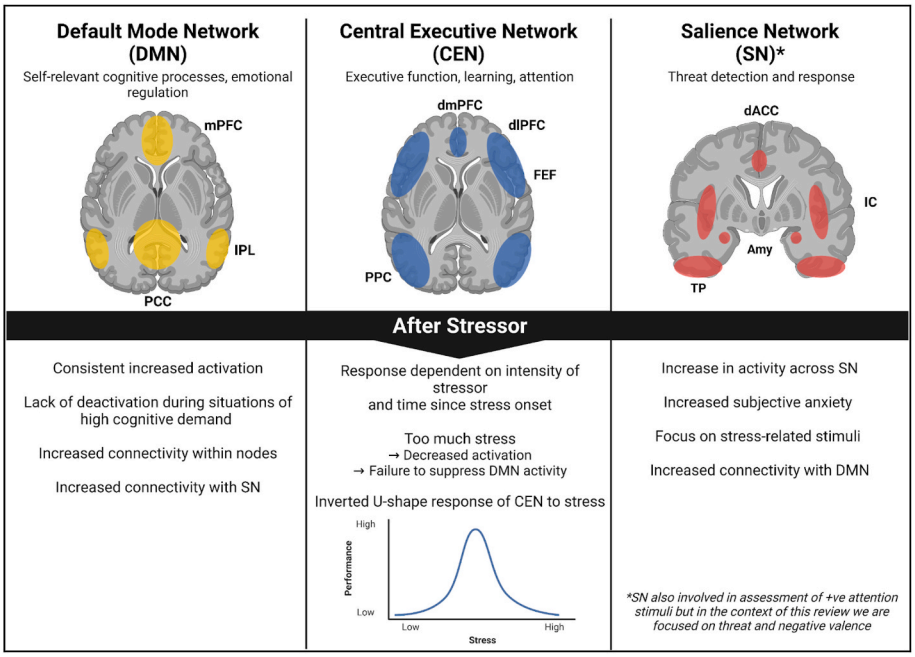

Fig. 2. Major neural networks and their roles at baseline and after exposure to psychosocial stressors
Abbreviations: mPFC: medial prefrontal cortex, IPL: inferior parietal lobule, PCC: posterior cingulate cortex, dmPFC: dorsomedial prefrontal cortex, dlPFC: dorsolateral prefrontal cortex, FEF: frontal eye fields, PPC: posterior parietal cortex, dACC: dorsal anterior cingulate cortex, IC: insular cortex Amy: amygdala, TP: temporal pole.

high cognitive demand when it would normally deactivate (Hermans et al., 2014). Given the current understanding that large-scale neural networks may compete for limited neural resources, excess DMN activity could contribute to stress-related cognitive deficits (Fox et al., 2009). Analyses of functional connectivity during stress induction have found that stressors increase nodal connectivity within the DMN as well as between the DMN and the Salience Network, which correlates with subjective stress response (Maron-Katz et al., 2016; Quaedflieg et al., 2015; Vaisvaser et al., 2013).

The Salience Network (SN) integrates threat detection and response to ensure survival during unsafe situations (Corbetta et al., 2008; Seeley et al., 2007). The SN reliably responds to salient stimuli, including stressors (Kober et al., 2008), with an immediate increase in amygdala activity (Oei et al., 2012; van Marle et al., 2009). High levels of catecholamines released in the amygdala and other limbic regions increase neuronal excitability (de Kloet et al., 2005), vital for detecting threats and regulating arousal and vigilance (Phelps and LeDoux, 2005). Other regions of the SN, e.g. thalamus and insula, demonstrate increased cerebral blood flow during acute stress, which positively correlates with subjective anxiety (Cameron et al., 2000). Acute stressors also increase activity in other regions of the SN, correlating with peripheral biomarkers such as heart rate variability and blood pressure (Ahs et al., 2009; Gianaros et al., 2008; Hermans et al., 2011; Pruessner et al., 2008; Wager et al., 2009). There may also be changes in functional connectivity between the SN and the DMN in response to stressors and these connectivity changes may in turn influence the activity of the Central Executive Network (Clewett et al., 2013).

The Central Executive Network (CEN) is based in frontoparietal brain regions and plays a pivotal role in executive function, learning, and attention; it is reliably activated during cognitively demanding tasks (Menon, 2011; Sridharan et al., 2008). The specific functional connectivity between nodes within the CEN may correspond to different facets of executive functioning (Nomi et al., 2017). Stress can impair activation of nodes within the CEN (van Oort et al., 2017). Acute stressors have been shown to reduce dorsolateral PFC (dlPFC) activation and impair task performance during certain executive function tasks (Qin et al., 2009; Schwabe et al., 2012; van Stegeren et al., 2010; Woodcock et al., 2019). This CEN deactivation is accompanied by failure to suppress DMN activity (Qin et al., 2009), and is partly mediated by activity of catecholamines in the PFC (Arnsten, 2009, 2015; Devilbiss et al., 2012). Furthermore, concurrent activation of the HPA-axis accentuates the negative effects of catecholamines on prefrontal nodes of the CEN (Myers et al., 2012). Notably, the effects of HPA-axis activation differ based on the time from initial stressor; specifically, longer-term genomic effects of glucocorticoids may actually improve dlPFC functioning in response to an acute stressor (Joëls et al., 2012; Yuen et al., 2009). Details of these long-term effects exceed the scope of this review, but are a reminder that baseline SAM and HPA-axis activity and responsiveness modulate individual responses to acute stressors.

Although these three neural networks do not represent all neural responses to acute stressors, they highlight key regions implicated in behavioral and emotional responses to stressors; specifically, executive functioning and emotional reactivity.

2.2.2. *Cognitive functioning: executive function*

Executive functioning (EF) refers to a complex set of neurocognitive processes that coordinate planning and goal-directed behavior (Suchy, 2009). Multiple domains of EF can be grouped into three overarching categories: working memory, cognitive inhibition, and cognitive flexibility (Diamond, 2013). Analysis of evidence surrounding effects of acute stressors on various facets of EF exceeds the scope of this review and study findings are heterogeneous (see: Klier and Buratto, 2020; Plieger and Reuter, 2020; Shields et al., 2015; Shields et al., 2016b). Meta-analyses of stressor effects on EF identified significant impairments in working memory, cognitive flexibility, and cognitive inhibition (i.e. interference control) but enhanced response inhibition after stress (Girotti et al., 2018; Shields et al., 2016). Studies find that different types of stressors variably influence facets of EF across populations (Demetriou et al., 2021; Girotti et al., 2018; Woon et al., 2017). Several possibilities underlie these differences. Some discrepancies between studies may be due to methodological inconsistencies, including mode of stress induction, assessment timing, and outcome measures (Becker and Rohleder, 2019; Henckens et al., 2011; Shields et al., 2015). Analyses of neural mechanisms by which stressors impact EF suggest a possible "inverted U" relationship between stress and EF, whereby moderate levels of stress may enhance EF, compared to very low levels (e.g. sedation or fatigue) and very high levels (Chamberlain et al., 2006; Lupien and McEwen, 1997; Sandi, 2013). These findings support theories that individual differences (see section 2.3) may drastically impact responses to stressors. Notably, these disparate findings also demonstrate the importance of investigating the effects of stress on EF domains separately. Prior reviews have highlighted neural correlates of these three categories of EF–working memory, cognitive inhibition, and cognitive flexibility–and the effects of stressors on relevant outcomes (Arnsten, 2009, 2015; Braem and Egner, 2018; Collette and Van der Linden, 2002; Funahashi, 2017; Kim et al., 2017; Shields et al., 2016; Uddin, 2021; Zhang et al., 2017).

The effects of acute stressors on working memory are perhaps the best characterized. Working memory refers to the ability to maintain and update information (Chai et al., 2018). For details of neural mechanisms involved in working memory, see Chai et al., 2018 and Funahashi (2017). In considering the effect of stressors, we must evaluate how stress impacts brain regions implicated in working memory; detailed mechanisms are elucidated by Arnsten (2009) and Arnsten (2015). It is theorized that exposure to an acute stressor may impair working memory performance by deactivating the dlPFC and entire CEN network. Studies in healthy subjects generally support these theories with the majority finding that physical, psychological, and pharmacological stressors impair dlPFC-dependent measures of working memory (Girotti et al., 2018; Shields et al., 2016; Woodcock et al., 2019).

Cognitive inhibition refers to a person's ability to inhibit certain actions (i.e. response inhibition) or thoughts (i.e. interference control) by focusing on task-relevant information or engaging in goal-directed, rather than habitual, behavior. For details of neural mechanisms involved in cognitive inhibition, see (Chambers et al., 2009; Wager et al., 2005; Zhang et al., 2018). The effects of acute stressors on cognitive inhibition are less well-characterized than the effects on working memory, but the evidence suggests that acute stressors may enhance response inhibition and impair cognitive inhibition (Shields et al., 2016). Although some data suggest cognitive and response inhibition are part of the same process, other evidence suggests these two types of inhibition may be dissociated in certain circumstances and pathologies (Friedman and Miyake, 2004; Johnstone et al., 2009). This differential effect of acute stressors on types of inhibition may result from how stressors can reallocate neural resources to focus attention on the cause of the stressor (LeBlanc, 2009; Plessow et al., 2011). Reallocation of cognitive resources would generally impair most types of EF, whereas selective attention would be improved (Schwabe et al., 2013; Shields et al., 2016); however, not all studies support this hypothesis (Sänger et al., 2014).

Cognitive flexibility refers to the ability to easily change between different rules or ways of thinking. For details of neural mechanisms involved in cognitive flexibility, see (Ionescu, 2012; Uddin, 2021; Wang et al., 2017). The effects of acute stressors on cognitive flexibility have been studied least of all EF domains; however, there are consistent findings suggesting that acute stressors impair cognitive flexibility (Shields et al., 2016). The effects of acute stressors on cognitive flexibility seem to be partly mediated by impairing PFC activity (Kalia et al., 2018) and modulated by SNS activation and not cortisol (Lapiz and Morilak, 2006; Marko and Riečanský, 2018; Shields et al., 2015). Specifically, stress-induced activation of the locus coeruleus-norepinephrine (LC-NE) system increases arousal and NE

release in the PFC. Medications that block effects of NE (e.g. sympatholytics) appear to block the deleterious effects of acute stressors on cognitive flexibility (Alexander et al., 2007; Girotti et al., 2018).

While acute stressors may impact other cognitive outcomes such as declarative memory (Ballan and Gabay, 2020; Cohen et al., 2020), learning (Becker and Rohleder, 2020; Wirz et al., 2018) and long-term memory (Henckens et al., 2009; Klier and Buratto, 2020), these major domains of EF are important in various aspects of healthy and pathological behavior and serve as useful markers for investigating effects of stressors on cognitive function. The PFC plays a major role in EF; however, specific PFC subregions and their related neural pathways differ depending on the domain of EF. Furthermore, the ways in which acute stressors impact EF and through which pathway (i.e. SNS or HPA-axis) varies, depending on the neural region and outcome measured. Although this section did not elaborate specific mechanisms behind these varying responses, this overview sets the stage for exploring how neuromodulation can modulate the effects of acute stressors on EF.

2.2.3. *Emotional reactivity*

Mood and emotional reactivity are key outcomes in therapeutic applications of stress amelioration. When exposed to stressors, individuals experience increased anxiety and negative affect (Campbell and Ehlert, 2012; Zapater-Fajarí et al., 2021). Negative mood and emotions caused by stressors are associated with a variety of additional negative outcomes (Du et al., 2018; Ford et al., 2018; Young et al., 2019). Although emotions can be described in various ways, they can generally be considered to be automatic psychobiological responses to real or imagined situations (Gross and Feldman Barrett, 2011). In one popular model, each basic emotion is considered to be a combination of different degrees of valence and arousal (Posner et al., 2009; Russell, 1980; Zald, 2003) wherein valence is defined as the (un)pleasantness of affective state and arousal reflects the level of mobilization towards or away from a particular stimuli (Lang and Davis, 2006). A stimulus that induces a strong negative response would be considered to have low valence and high arousal rating. Emotions are distinct from other related affective states, e.g. mood has a longer duration than an emotion and is usually not related directly to a specific event (Balzarotti et al., 2017; Gross, 2015). Although the specific definitions of affective states are not clearly agreed upon, affect may be considered as an overarching category for specific types of psychological states that can include: emotions, mood, and stress response (Gross, 2015; Russell, 2003). Despite the fact that stress responses are considered distinct from emotional states, one event can trigger both a stress response and a negative emotional state; this indicates there is considerable overlap between affective responses and, consequently, overlap between neural regions involved in these responses.

2.2.3.1. Mechanisms of emotional responses. Initially, the limbic system was considered responsible for emotional responses, whereas the PFC was responsible for higher-level cognition. This is now understood to be an overly simplistic approach, but the limbic system does play a key role in emotional responses (LeDoux, 2000). Within the PFC, the orbitofrontal (OFC) and ventromedial PFC (vmPFC) are associated with regulation of emotion (Arnsten et al., 2015). The vmPFC and anterior cingulate cortex (ACC) project to structures involved in limbic regulation, including the amygdala, ventral striatum, hypothalamus, and brainstem (Arnsten et al., 2015). It may also be possible to separate neural regions involved in emotion response based on valence and arousal networks (Posner et al., 2009). Extensive research has outlined the roles of specific SN nodes in emotional responses and regulation; Fig. 3 represents a synthesis of the literature on this topic (Anderson and Phelps, 2002; Arnsten et al., 2015; Davidson, 2002; Lang et al., 1998; Likhtik, 2005; Morris, 1998; Phan et al., 2004; Posner et al., 2009; Quirk et al., 2003; Richter-Levin, 2004; Thayer, 2006; Zald, 2003) and illustrates the roles of these neural regions in emotional responses, how they may be impacted by acute stressors, and the implications of this knowledge for interventions (e.g. neuromodulation).

The amygdala plays a key role in evaluating and responding to stimuli of high emotional salience, i.e. with very low or very high valence (Sergerie et al., 2008; Zald, 2003). It projects to higher-order sensory areas, the PFC, and hippocampus, enabling it to modulate responses to emotionally salient situations (Anderson and Phelps, 2001). Humans preferentially attend to stimuli of high emotional salience, including stressors; thus, one is more likely to focus longer on an unpleasant/stressful situation than a neutral one. This attentional modulation is primarily controlled by the amygdala (Aston-Jones et al., 1996; Lang and Davis, 2006; Morris, 1998; Zald, 2003). Exposure to aversive stimuli, including stressors, across any sensory modality leads to increased amygdala activation; these aversive stimuli may induce

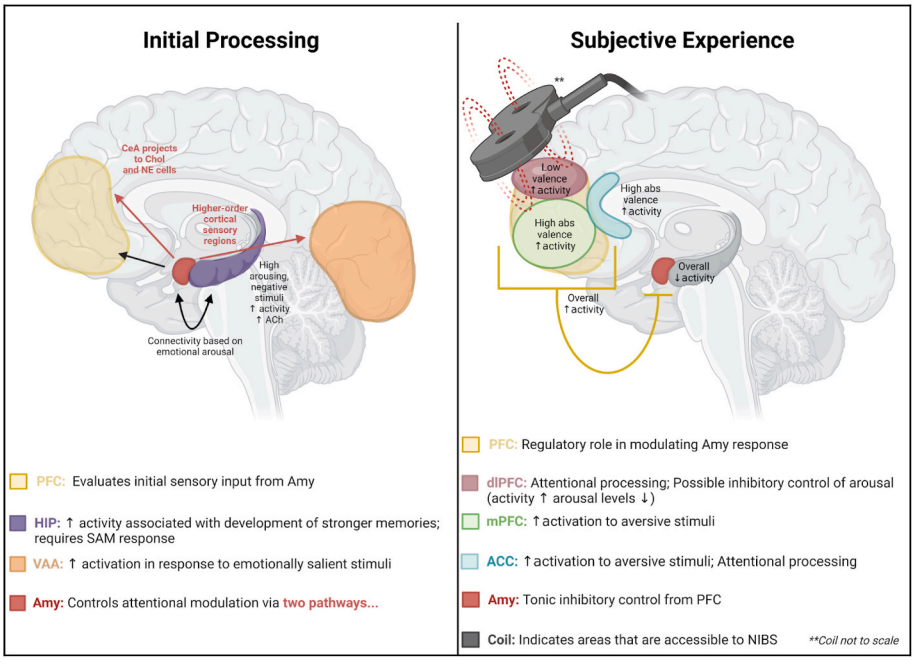

Fig. 3. Neural mechanisms of emotional responses and implications for neuromodulation interventions
Abbreviations: PFC: prefrontal cortex, CeA: central nucleus of the amygdala, NE: noradrenergic, Chol: cholinergic, ACh: acetylcholine, HIP: hippocampus, VAA: visual association area, Amy: amygdala, dlPFC: dorsolateral prefrontal cortex, dlPFC: dorsolateral prefrontal cortex, ACC: anterior cingulate cortex, abs: absolute, NIBS: Non-invasive brain stimulation.

feelings of disgust, anxiety, or ill-defined negative state (Zald, 2003). This increased amygdala response occurs to both psychological stressors (e.g. unpleasant images) and physical stressors (e.g. hypercapnia) (Brannan et al., 2001; Evans et al., 2002; Irwin et al., 1996; Lane et al., 1997). Although the amygdala modulates emotional responses, it may not be necessary for consciously evaluating and reporting subjective emotional experiences (Anderson and Phelps, 2002).

While the amygdala acts as a gate-keeper for processing emotionally salient stimuli, the understanding and subjective experiences of emotional responses would not be possible without prefrontal regions including the vmPFC and ACC. The vmPFC and ACC are integral for subjective evaluation of emotional states and have consistently demonstrated increased activation in response to aversive stimuli, findings that correlate with reports of subjective negative emotions (Phan et al., 2004). Evidence suggests the vmPFC regulates the amygdalar response to emotional stimuli to ensure affective responses are not excessive (Garcia et al., 1999; Ochsner et al., 2002; Posner et al., 2009). The amygdala is under tonic inhibitory control from the vmPFC, so changes in vmPFC activity can modulate output of the amygdala (Davidson, 2000; Thayer, 2006; Thayer et al., 2012). Stimuli with high arousal ratings (positive or negative valence) are associated with increased neural activity in the vmPFC and dACC, whereas activity in the dlPFC is correlated only with negative valence (i.e. aversive stimuli). Activity in the dlPFC inversely correlates with arousal ratings (as activity in the dlPFC increases and arousal levels decrease), which suggests the dlPFC exerts inhibitory control of arousal (Posner et al., 2009).

The importance of PFC subregions in subjective emotional responses and inhibitory control over autonomic arousal responses provides insights into how stressors may negatively impact responses to emotionally salient stimuli. In fact, the same neurochemical stress responses that can impair dlPFC function and working memory can actually strengthen amygdalar emotional responses. Thus, stress may switch control of behavior from the 'thoughtful PFC' to the more habitual, conditioned responses of the amygdala (Arnsten, 2009).

2.2.3.2. Emotional regulation. Importantly, while a situation–stressful or otherwise–may generate an emotional response, the way an individual evaluates (subconsciously and consciously) the situation, in light of their experiences, along with their innate biology modifies the final quality and magnitude of responding (Hooley and Gotlib, 2000; Zuckerman, 1999). Altering the ways in which a person experiences a situation can change the emotional response; these changes can occur through emotion regulation (Gross, 1999). Emotion regulation is distinct from emotion generation and is a critical skill. Emotion regulation is associated not only with changes to the immediate response to an event, but also with changes to how the event is encoded in memory (Hayes et al., 2010). Evidence suggests there are two key networks involved in cognitive reappraisal (Wager et al., 2008). The first involves the amygdala and other regions associated with negative emotional states whereas the second involves the nucleus accumbens and ventral striatum and is more associated with memory and action. Activity within the vlPFC appears to be correlated with activity in both of these networks, suggesting that the vlPFC plays a key role in emotion regulation and cognitive reappraisal (Wager et al., 2008). Effective emotion regulation is associated with enhanced psychological well-being and improved ability to cope with and respond to acute stressors (Balzarotti et al., 2016; Gross and Feldman Barrett, 2011; Gross and John, 2003; Haga et al., 2009).

This overview of mechanisms of emotion generation and regulation provides a foundational understanding that clarifies our use of these outcomes in studies of stress responses. It is clear that acutely stressful situations can lead to functional alterations resulting in increased responsiveness to stressors. Not only does the immediate response to acute stressors increase SAM activation and attention to negative, arousing stimuli but in some situations, it may also impair PFC activity, leading to emotion dysregulation. It is important to note that these responses to acute stressors are not inherently negative and are part of a complex series of adaptive processes that occur to protect the individual; nonetheless, these responses may occur inappropriately, especially among certain populations already exposed to chronic stressors or under psychological distress, resulting in maladaptive behavioral responses. Although this section did not discuss these mechanisms in detail, it demonstrates certain alternations that may occur under acute stress and offers insights into ways that interventions might be used to reduce adverse effects of acute stressors when they occur. The final step before we can discuss these potential interventions is to recognize how certain independent variables may impact these results and how this can complicate our understanding of the efficacy of these interventions.

2.3. Independent factors affecting stress response

It is evident that even among healthy subjects there is significant variation in responses to different types of stressors (e.g. psychological vs. physical). In addition to individual differences in the stress response itself, there are differences in many outcomes that may be measured (e.g. physiological, cognitive, and behavioral changes). This review does not consider all sources of variation, but offers a cursory overview of trait and state factors that may modify physiological and behavioral responses to stressors (for reviews, see: Kudielka et al., 2009; Sep et al., 2020). These variables can impact the specific stressor response, baseline activity, and outcomes in EF or emotional response tasks. These variables can be broadly divided into two groups: trait and state variables.

Key trait variables include sex/gender, age, life experiences, personality, and genetics. Sex differences have been found in stress responses both at the level of hormonal release and how stressors alter physiological and behavioral outcomes (McEwen et al., 2016; Merz and Wolf, 2017; Shields et al., 2016). Age also affects individual responses to stressors and relevant outcome measures. There are significant changes in neural development over time, which continue throughout adulthood (Brindle et al., 2014; Foley and Kirschbaum, 2010). Furthermore, new experiences (which come with age) also affect the stressor response. Even among healthy adults, some have experienced trauma or other adverse experiences during childhood or adolescence, which may not have led to overt psychopathology but may impact aspects of personality, including ways that the person responds to stressors (Raymond et al., 2021). Personality traits may also impact how a person perceives and responds to stressful stimuli. Studies have found that neuroticism, extraversion, and openness are associated with differing physiological, cognitive, and subjective responses to acute stressors (Hughes et al., 2011; Oswald et al., 2006; Schneider, 2004; Williams et al., 2009; Wirtz et al., 2007; Xin et al., 2017). Finally, genetics play an integral role in every aspect of human development and evidence suggests that many aspects of the stressor response are highly heritable, including SAM activity (Finley et al., 2004; Mueller et al., 2012), HPA-activity (Dedic et al., 2018; Derijk et al., 2008; Federenko et al., 2004; Tucker-Drob et al., 2017), and cardiovascular responses to stressors (Wright et al., 2007; Wu et al., 2010).

Key state variables include time of day, sleep, medications and drugs, psychological state, diet, and exercise. There is clear evidence that HPA-axis responses to acute stressors are time-dependent, with cortisol release following a circadian cycle (Chan and Debono, 2010; Oster et al., 2017); however, the evidence is less straightforward for SAM responses (Dunn and Taylor, 2014; Hissen et al., 2015; Scheer et al., 2010; Vitale et al., 2019). Sleep can also impact all of the outcomes discussed thus far, including response to stressors. Poor sleep quality or decreased sleep duration is associated with negative mood and increased rates of depression (Fang et al., 2021), rendering an individual more susceptible to acute stressors. Poor sleep is associated with HPA-axis dysregulation and exaggerated stress reactivity (Goodin et al., 2012; Massar et al., 2017). Sleep can affect a person's mood and mindset, how they respond

to stressors, and individual coping strategies can play a major role in the physiological, cognitive, and psychological effects of stressors (Crum et al., 2013; Radtke et al., 2020; Spada et al., 2008). Additionally, food and medications a person ingests may impact many outcomes discussed. It is clear how medications that interact directly with the SAM or HPA-axis can affect the response to an acute stressor; however, many other medications can also alter the stressor response (Brody et al., 2002; Kudielka et al., 2004; Kuhlmann and Wolf, 2005). Medications are not the only substances that impact responses to acute stressors. Most recreational drugs impact SAM and HPA-axis activity (al'Absi, 2006a; Fuxe et al., 1989; Kirschbaum et al., 1993; Lovallo et al., 1996, 2006; Matta et al., 1998; Shepard et al., 2000). Energy consumption, type of diet, and time of food ingestion all play a role in regulating HPA-axis responses to stressors (Gonzalez-Bono et al., 2002; Kirschbaum et al., 1997; Rohleder and Kirschbaum, 2007; Uçar et al., 2021). The role of physical activity on responses to acute stressors is complex with conflicting results; there is evidence that effects of physical activity may differ depending on the outcomes measured (Anderson and Wideman, 2017; Bernstein and McNally, 2017; De Geus and Van Doornen, 1993; Klaperski et al., 2013; Mueller, 2007; Tsatsoulis and Fountoulakis, 2006).

This section has only briefly discussed some individual differences. Furthermore, these factors were summarized in relative isolation; in reality they are all closely linked, e.g. one cannot evaluate the impact of sex without considering age, experience, and many other factors. The complexity of these responses cannot be overlooked and any analysis or review of the stress literature must attend to these variables. That being said, it is not always possible to control for all individual variation in studies of stress responses so we must remain cognizant of this limitation as we evaluate the literature.

3. Effects of neuromodulation

3.1. Non-invasive brain stimulation (NIBS)

Non-invasive brain stimulation (NIBS) techniques alter brain functioning using an external device. NIBS interventions often have immediate effects, which can be temporary or longer-lasting depending on the protocol used (To et al., 2018). The mechanisms of these prolonged effects are believed to be similar to long-term potentiation and long-term depression; for a more detailed review of these mechanisms see: (Chervyakov et al., 2015; Das et al., 2016; Di Lazzaro, 2013; Farzan et al., 2016). Due to their potential for long-lasting effects, there is a significant role for NIBS to serve as an important tool for both mechanistic exploration of neural pathways and the development of therapeutic interventions for neuropsychiatric disorders. At present, NIBS techniques are used across a wide variety of research fields to improve our understanding of neural circuitry and are FDA-cleared for treatment of migraines, major depression, smoking cessation, and obsessive-compulsive disorder. Additionally, there is evidence for their ability to advance mechanistic understanding and provide new therapeutic options for several other disorders (Davis and Gaitanis, 2020; Elias et al., 2021; J.-P. Lefaucheur et al., 2020; McClintock et al., 2019; Moisset et al., 2020; Yamamoto et al., 2021).

Common forms of NIBS include transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS). TMS applies a direct current pulse through an electromagnetic coil placed on the scalp to generate a momentary magnetic field. A magnetic field of sufficient amplitude will induce a momentary electrical field (E-field) in the neural region under the coil and cause neural membrane depolarization (Sack and Linden, 2003). Multiple pulses over a short period of time, or repetitive TMS (rTMS) (Klomjai et al., 2015), can be applied at different frequencies with different effects. Low frequency ($\leq$1Hz) stimulation leads to decreased cortical excitation and is considered inhibitory whereas high frequency ($\geq$5Hz) stimulation leads to increased cortical excitation and is considered excitatory (Chen et al., 1997; Fitzgerald et al., 2006). Theta burst stimulation (TBS) is a newer type of rTMS, in which three to five very high frequency ($\geq$50Hz) pulses are delivered in a 5Hz "bursting" pattern. If delivered continuously, (c)TBS leads to inhibition, if delivered intermittently, with 2 s of stimulation and 8 s of rest, (i)TBS leads to facilitation (Huang et al., 2005). One benefit of TMS is that there are various types of coils, enabling differential targeting of neural tissues (Deng et al., 2014). In contrast, tDCS is often less precise in its neural targeting (Bikson et al., 2013). With tDCS, two electrodes are placed on specific scalp locations and a low-amplitude direct current is passed between the electrodes. This direct current passes through the scalp area between the electrodes thereby altering the membrane potential in the neurons below (Medeiros et al., 2012). tDCS can be anodal or cathodal: anodal stimulation leads to neuronal depolarization and increased excitability, whereas cathodal stimulation leads to neuronal hyperpolarization and decreased excitability (Jacobson et al., 2012).

Despite key methodological differences between different forms of NIBS, consistent findings highlight their potential as both investigative and therapeutic interventions (Amidfar et al., 2019; Baptista et al., 2020; Elias et al., 2021; Gault et al., 2018; Kekic et al., 2016; Marques et al., 2019). Although there have been considerable advances in NIBS research, there are still a few major methodological inconsistencies (Broadbent et al., 2011; Ekhtiari et al., 2019; Guerra et al., 2020; Polanía et al., 2018; Sandrini et al., 2011) and a need for more clinical and safety guidelines (Brunoni et al., 2013; J. P. Lefaucheur et al., 2020; Matsumoto and Ugawa, 2017). As a result of wide-ranging variation in stimulation parameters, it can be difficult to critically evaluate the true efficacy of NIBS as a tool for mechanistic exploration and treatment.

3.2. Neuromodulation and acute stressors

We reviewed studies that evaluated effects of NIBS on behavioral and emotional responses to acute psychosocial stressors. Given highly variable methodology, we focused on studies that involved healthy volunteers, used a psychosocial stressor (see Table 1), used rTMS or tDCS, and evaluated at least one outcome related to executive function or emotional reactivity. Table 2 shows the full list of studies considered. Experimental designs, outcomes, and neural targets vary considerably between studies; because these studies are not directly comparable, we primarily outline relevant findings without in-depth interpretation and analysis. For outcomes that seemed comparable across multiple studies, we collected data regarding pre- and post-neuromodulation measures either from the article itself or via direct communication with the authors. This was possible for measures of emotional reactivity (Fig. 4) and salivary cortisol (Fig. 5).

3.2.1. Executive functioning

The effect of stressors on working memory is the most-studied and best-characterized among the three primary domains of EF. We found 4 studies that examined the effect of neuromodulation on working memory during psychosocial stressors; all used anodal tDCS of either the right or left dlPFC. Two studies used the TSST and all included a control condition (friendly TSST); these studies all targeted the right dlPFC with anodal tDCS, and one also used cathodal tDCS. Ankri et al., 2020 found no main effect of stressor or neuromodulation on working memory. Bogdanov and Schwabe (2016) found a main effect of stressor, leading to decreased working memory performance (Corsi block task and digit span backwards) and a main effect of tDCS on working memory (digit span backwards). Both studies found an interaction between neuromodulation and stressor, but in different directions. Ankri et al., 2020 found that tDCS led to decreased accuracy during the stress condition only. In contrast, Bogdanov and Schwabe, 2016 found that during stress conditions there was increased working memory performance (Corsi and digit span backwards) when receiving anodal tDCS vs. sham or cathodal tDCS. The within-session order of events is worth noting because Ankri et al., 2020 provided neuromodulation prior to stress induction, whereas Bogdanov and Schwabe, 2016 induced stress prior to

Table 2
Overview of all studies identified that explored the effects of combined neuromodulation and psychosocial stressors in healthy populations.

Stressor	Paper	Population (Healthy)	Design	NIBS/ Stress Order	Main Effects					Neuromodulation x Stress Effects		
					Type/Target	Details[a]	Executive Function	Emotional Reactivity	Biomarkers	Executive Function	Emotional Reactivity	Biomarkers
					tDCS/dlPFC (right)	Anodal 2 mA Cfade-in/fade-out ramp of 30 s 20mins	**Stress**: None	**Stress**: subjective stress reaction (STAI and VAS)	**Stress**: salivary cortisol	Stress only: tDCS (vs. sham) → ↓ n-back accuracy	No interaction effect (STAI or VAS)	Stress only: tDCS (vs. sham) → no stress-related ↑ cortisol
					- 10–20 system	Sham: Yes	**NIBS**: None	**NIBS**: None	**NIBS**: None			
	Bogdanov et al. 2016	♀ & ♂ 18–32 yrs "normal weight" (N = 120: 20 per group)	Between-subject	1. Stressor	tDCS/	Anodal vs. cathodal	**Stress**: ↓ working memory performance (Corsi block task and Digit span backwards)	**Stress**: ↓ mood and calmness (vs. control)	**Stress**: ↑ HR, DBP, SBP, salivary cortisol	Stress only: anodal tDCS (vs. sham or cathodal) → ↑ working memory performance	No data provided	No interaction effect (salivary cortisol)
			6 groups (stress vs. control, NIBS vs. sham, and anodal vs. cathodal)	2. NIBS	dlPFC (right)	1.075 mA 8 s fade-in and 5s fade-out Ended once working memory task completed						
			Control: Friendly	2a. EF task during NIBS (1 session)	- 10–20 system	Sham: Yes	**NIBS**: ↑ working memory performance	**NIBS**: Not provided	**NIBS**: None			
	Antal et al. (2014)	♂ 21–32 yrs (N = 60: 20 per group)	Between subjects	1. NIBS	tDCS/	Anodal vs. cathodal	**Stress**: N/A	**Stress**: subjective stress reaction (KAB and STAI)	**Stress**: ↑ salivary cortisol & medial frontal rCBF	N/A	No interaction effect (subjective stress)	Anodal: ↑ rCBF in right mPFC (vs. sham) & ↑ rCBF in right amygdala & right superior PFC (vs. cathodal)
			3 groups (anodal vs. cathodal vs. sham)	2. Stressor	mPFC (right)	1m Cfade-in/ fade-out ramp of 10s 20mins						
			Control: None	(1 session)	- 10–20 system	Sham: Yes	**NIBS**: N/A	**NIBS**: None	**NIBS**: Cathodal → ↑ cortisol & ↑ rCBF in right & left mPFC			
	Carnevali et al. (2020)	♂ (N = 30: 15 per group)	Between subjects	1. NIBS	tDCS/	Anodal 2 mA?	**Stress**: N/A	**Stress**: None	**Stress**: ↑ HR, ↓ HRV, ↑ cortisol	N/A	Stress only: tDCS (vs. sham) → ↓ anxiety	Stress: tDCS (vs. sham) → ↓ HR & ↑ HRV; no cortisol effect
			2 groups (anodal vs. sham)	1a. Stressor during NIBS (5mins after start)	dlPFC (left)	15mins						
			Control: None	(1 session)	- 10–20 system	Sham: Yes	**NIBS**: N/A	**NIBS**: ↓ anxiety (STAI)	**NIBS**: None			
				1. NIBS	rTMS/		**Stress**: N/A		**Stress**: ↓ HRV	N/A		

(*continued on next page*)

Table 2 (*continued*)

Stressor	Paper	Population (Healthy)	Design	NIBS/ Stress Order	Main Effects					Neuromodulation x Stress Effects		
					Type/Target	Details[a]	Executive Function	Emotional Reactivity	Biomarkers	Executive Function	Emotional Reactivity	Biomarkers
	Pulopulos et al. (2020)	♀ 18–35 yrs (N = 75)	Between subjects			20Hz 110% RMT 40 trains of 2s duration, ITI 12s 1600 pulses/ session?		**Stress**: ↑ stress and tension (VAS), ↓ happiness (VAS)			No interaction effect (VAS)	No interaction effect (HRV or salivary cortisol)
			2 groups (active vs. sham)	2. Stressor Control: None	dlPFC (left) (1 session)	Sham: Yes - Adjusted BeamF3 algorithm		**NIBS**: N/A	**NIBS**: None			
	NIBS: ↓ AUCi after active TMS (vs. sham)											
	de Wandel et al., 2020	♀ 18–27 yrs (N = 34)	Within subjects crossover (active vs. sham)	1. Stressor	rTMS/	50 Hz (burst freq 5Hz) 110% RMT 54 cycles, 10 burst of 3 pulses, train duration of 2s, ITI of 6 s 1620 pulses 2 sessions of 5mins	**Stress**: N/A	**Stress**: Cannot be assessed	**Stress**:↑ cortisol	N/A	No interaction effect (VAS)	No interaction effect (cortisol)
			Control: None	2. NIBS (2 sessions; 2nd occurred >1 week after 1st)	dlPFC (left) - Individual MRI navigation	Sham: Yes	**NIBS**: N/A	**NIBS**: None	**NIBS**: None			
	De Witte et al. 2019	♀ "young adults" (N = 38)	Within subjects crossover (active vs. sham)	1. Stressor	rTMS/dlPFC (left)	50 Hz (burst freq 5Hz) 110% RMT 54 cycles, 10 burst of 3 pulses, train duration of 2s, ITI of 6 s 1620 pulses 5mins	**Stress**: N/A	**Stress**: ↑ anger and tension & ↓ cheerful (VAS), ↑ momentary rumination	**Stress**: ↑ cortisol	N/A	No interaction effect (VAS or momentary rumination)	No interaction effect (cortisol)
			Control: None	2. NIBS (2 sessions; 2nd occurred >1 week after 1st)	- Individual MRI navigation	Sham: Yes	**NIBS**: N/A	**NIBS**: None	**NIBS**: None			
	Pulopulos et al. (2019)	♀ (N-35)	Within subjects crossover (active vs. sham)	1. Stressor	rTMS/dlPFC (left)	50 Hz (burst freq 5Hz) 110% RMT 54 cycles, 10 burst of 3 pulses each, with a train duration of 2s and ITI 6s 1620 pulses 5mins	**Stress**: N/A	**Stress**: ↑ vigorous, angry, tense (VAS), ↓ cheerful (VAS)	**Stress**: ↑ cortisol	N/A	No interaction effect on mood (VAS)	No interaction effect (cortisol)
Control: None			2. NIBS (2 sessions; 2nd	- Individual	Sham: Yes	**NIBS**: N/A	**NIBS**: None	**NIBS**: None				

(*continued on next page*)

Table 2 (*continued*)

Stressor	Paper	Population (Healthy)	Design	NIBS/ Stress Order	Main Effects					Neuromodulation x Stress Effects		
					Type/Target	Details[a]	Executive Function	Emotional Reactivity	Biomarkers	Executive Function	Emotional Reactivity	Biomarkers
			occurred >1 week after 1st)	MRI navigation								
Critical Feedback	De Raedt et al. (2017)	♀ Undergraduates (N = 32)	Within subjects crossover (active vs. sham)	1. NIBS	tDCS/dlPFC (left)	Anodal 1.5 mA 30s ramp up/ ramp down 20mins	**Stress**: N/A	**Stress**: VAS changes (↑ tension and anger; ↓ vigor and cheerfulness)	**Stress**: N/A	N/A	Stress only: tDCS (vs. sham) → ↓ in ruminative self-referential thinking; no VAS effects	N/A
			Control: Neutral & Praise	2. Stressor (2 sessions; 2nd occurred >48-hrs after 1st)	- Individual MRI navigation	Sham: Yes	**NIBS**: N/A	**NIBS**: None	**NIBS**: N/A			
	Baeken et al. (2018)	♀ 20–30 yrs (N = 30)	Within subjects crossover (active vs. sham)	1. NIBS	tDCS/dlPFC (left)	Anodal 1.5 mA 30s ramp up/ ramp down 20mins	**Stress**: N/A	**Stress**: VAS changes (↑ anger)	**Stress**: N/A	N/A	No interaction effect (VAS)	tDCS (vs. sham): Stressor → ↓ perfusion in right pgACC/ mPFC
			Control: Neutral & Praise	2. Stressor (2 sessions; 2nd occurred >48-hrs after 1st)	- Individual MRI navigation	Sham: Yes	**NIBS**: N/A	**NIBS**: None	**NIBS**: N/A			
	Remue et al. 2015	♀ Undergraduates (N = 38: 19 per group)	Between subjects 2 groups (left vs. right)	1. NIBS	rTMS/	20 Hz 110% RMT 40 trains of 1.9 s duration, separated by an ITI 12.1s, 1560 pulses per session. ~10 min.	**Stress**: N/A	**Stress**: ↑ negative mood (VAS Total)	**Stress**: ↑ HRV	N/A	No interaction effects for either side (mood (VAS total) or anxiety (STAI-S))	Stress only: left rTMS (vs. right or sham) → ↑ HRV
			Within subjects crossover (active vs. sham)	2. Stressor	dlPFC (left vs. right)							
			Control: None	(2 sessions; 2nd occurred >3 days after 1st)	- Individual MRI navigation	Sham: Yes	**NIBS**: N/A	**NIBS**: None	**NIBS**: None			
	Baeken et al. (2014)	♀ (N = 30)	Within subjects crossover (active vs. sham)	1. NIBS	rTMS/dlPFC (left)	20Hz 110% RMT 20 trains of 1.9 s duration, separated by an ITI 12.1 s 1560 pulses per session ?	**Stress**: N/A	**Stress**: ↓ vigor and cheerfulness (VAS), ↑ tension (VAS)	**Stress**: None	N/A	No interaction effect (VAS)	No interaction effect (salivary cortisol)
			Control: None	2. Stressor (2		Sham: Yes	**NIBS**: N/A	**NIBS**: None	**NIBS**: ↓ cortisol after			

(*continued on next page*)

Table 2 (*continued*)

Stressor	Paper	Population (Healthy)	Design	NIBS/ Stress Order	Main Effects					Neuromodulation x Stress Effects		
					Type/Target	Details[a]	Executive Function	Emotional Reactivity	Biomarkers	Executive Function	Emotional Reactivity	Biomarkers
				sessions; 2nd occurred >3 days after 1st)	- Individual MRI navigation				active vs. sham			
Psychosocial + Physical	Friehs et al. 2020	♀ & ♂ (N = 59: 29 and 30 per group)	Between subject	1. Stressor	tDCS/dlPFC (left)	Anodal	**Stress**: ↑ RT and accuracy (n-back)	**Stress**: N/A	**Stress**: ↑ cortisol, ↑ HR, HRV, ↑ PNS activation (LF power ↓ & HF power ↑)	No interaction effect (accuracy or RT)	N/A	No interaction effect (cortisol, HR, HRV, or LF/HF bands)
			2 groups (active vs. sham)	2. NIBS	- 10–20 system	0.5 mA 30s ramp up/ramp down						
			Control: None	(1 session)		19mins Sham: Yes	**NIBS**: None	**NIBS**: N/A	**NIBS**: LF/HF bands	NOTE: Only responders analyzed		
	De Smet et al. (2021)	♀ & ♂ 18–45 yrs (N = 69)	Within subjects crossover (active vs. sham tDCS) + active iTBS	1. NIBS 1	tDCS/dlPFC (bifrontal)	tDCS Anode and cathode were respectively placed over F3 and F4 2 mA 30s ramp up/ramp down 20mins Sham: Yes	**Stress**: N/A	**Stress**: ↑ perceived stress & negative affect	**Stress**: ↑ BP, HR, HRV, and EDA	N/A	No interaction effect (perceived stress and negative affect)	No interaction effect (BP, HR, HRV, or EDA)
			Control: None	2. NIBS 2	rTMS/dlPFC (left)							
				3. Stressor (2 sessions; 2nd occurred >1 week after 1st)	- Beam F3 localization system	rTMS 50 Hz (burst freq 5Hz) 110% RMT 54 cycles, 10 bursts of 3 pulses each, train duration of 2 s and with a cycling period of 8 s 1620 pulses 7mins Sham: No	**NIBS**: N/A	**NIBS**: None	**NIBS**: Lower HR in active tDCS vs. sham ↑ HRV in active tDCS vs. sham Lower EDA in tDCS vs. sham			
PASAT	Plewnia et al. (2015)	♂ (N = 28: 14 per group)	Between subjects 2 groups (anodal vs. sham)	1. NIBS 1a. Stressor during NIBS (5mins after start)	tDCS/dlPFC (left) - 10–20 system	Anodal 1 mA linear fade-in/ fade-out phase of 5 s 20mins Sham: Yes	**Stress**: Cannot be assessed	**Stress**: None	**Stress**: N/A	No interaction effect (ISI or errors)	Stress: tDCS (vs. sham) → ↓ negative affect (PANAS) No interaction effect on positive affect	N/A
			Control: None	(1 session)			**NIBS**: Anodal led to ↓ ISI (vs. sham)	**NIBS**: Negative affect (PANAS)	**NIBS**: N/A			
Cyberball	Riva et al. (2012)	♀ & ♂ (N = 79)	Between subjects 4 groups (active vs. sham and	1. NIBS 1a. Stressor during	tDCS/vlPFC (right) - 10–20 system	Anodal 1.5 mA ? 15mins Sham: Yes	**Stress**: None	**Stress**: ↑ social exclusion	**Stress**: N/A	No interaction effect (ball tosses identified)	Stress only: tDCS (vs. sham) → ↓ social exclusion, unpleasant & hurt feelings	N/A

(*continued on next page*)

Table 2 (*continued*)

Stressor	Paper	Population (Healthy)	Design	NIBS/ Stress Order	Main Effects					Neuromodulation x Stress Effects		
					Type/Target	Details[a]	Executive Function	Emotional Reactivity	Biomarkers	Executive Function	Emotional Reactivity	Biomarkers
			inclusion vs. exclusion) Control: Yes	NIBS (5mins after start) (1 session)			**NIBS**: None	**NIBS**: None	**NIBS**: N/A			
	Riva et al. 2014 (study 1)	♀ & ♂ University students (N = 82)	Between subjects	1. NIBS	tDCS/vlPFC (right)	Cathodal 1.5 mA ? 15mins Sham: Yes	**Stress**: ↓ correctly identified ball tosses	**Stress**: ↑ social exclusion, hurt feelings, & negative emotions	**Stress**: N/A	No interaction effect (ball tosses identified)	Stress only: tDCS (vs. sham) → ↑ Social exclusion, hurt feelings & negative emotions	N/A
			4 groups (active vs. sham and inclusion vs. exclusion) Control: Yes	1a. Stressor during NIBS (5mins after start) (1 session)	- 10–20 system		**NIBS**: None	**NIBS**: Cathodal → ↑ social pain	**NIBS**: N/A			
	Riva et al. 2014 (study 2)	♀ & ♂ University students (N = 40)	Between subjects 2 groups (active vs. sham) Control: Yes	1. NIBS 1a. Stressor during NIBS (5mins after start) (1 session)	tDCS/PPC (right) - 10–20 system	Cathodal 1.5 mA? 15mins Sham: Yes	**Stress**: Cannot be assessed **NIBS**: None	**Stress**: Cannot be assessed **NIBS**: None	**Stress**: N/A **NIBS**: N/A	No interaction effect (ball tosses identified)	No interaction effect (social exclusion, hurt feelings, or negative emotions)	N/A
	Fitzgibbon et al. (2017)	♀ & ♂ (N = 29: 16 and 13 per group)	Between subjects 2 groups (active vs. sham) Control: Yes	1. NIBS 2. Stressor (1 session)	rTMS/dlPFC (left) - Beam system	1 Hz 120% RMT 20 consecutive minutes 1200 pulses 20mins Sham: Yes	**Stress**: ↓ correctly identified ball tosses **NIBS**: ?	**Stress**: ↑ unpleasantness **NIBS**: ?	**Stress**: N/A **NIBS**: N/A	No interaction effect (ball tosses received)	No interaction effect (unpleasantness)	N/A

Abbreviations: TSST: Trier Social Stress Test, PASAT: Paced Auditory Serial Addition Task, NIBS: non-invasive brain stimulation, RMT: resting motor threshold, ITI: inter train interval STAI: State-Trait Anxiety Inventory, KAB: German version of the Short Questionnaire for Current Strain (Kurzfragebogen zur aktuellen Beanspruchung), VAS: Visual Analog Scale, PANAS: Positive Affect Negative Affect Scale, EF: executive functioning, ISI: inter-stimulus interval, HR: heart rate, HRV: heart rate variability, LF: low frequency, HF: high frequency, rCBF: resting cerebral blood flow, dlPFC: dorsolateral prefrontal cortex, mPFC: medial prefrontal cortex, vlPFC: ventrolateral prefrontal cortex, pgACC: pregenual anterior cingulate cortex, PPC: posterior parietal cortex.

[a] Neuromodulation details are given in the following order: tDCS parameters: 1) Stimulation frequency, 2) Stimulation intensity, 3) Stimulation parameters, 4) Total stimulation time; rTMS parameters: 1) Stimulation frequency, 2) Stimulation intensity, 3) Simulation parameters, 4) Total pulses, 5) Total stimulation time. "?" indicates that those details were not found within the publication.

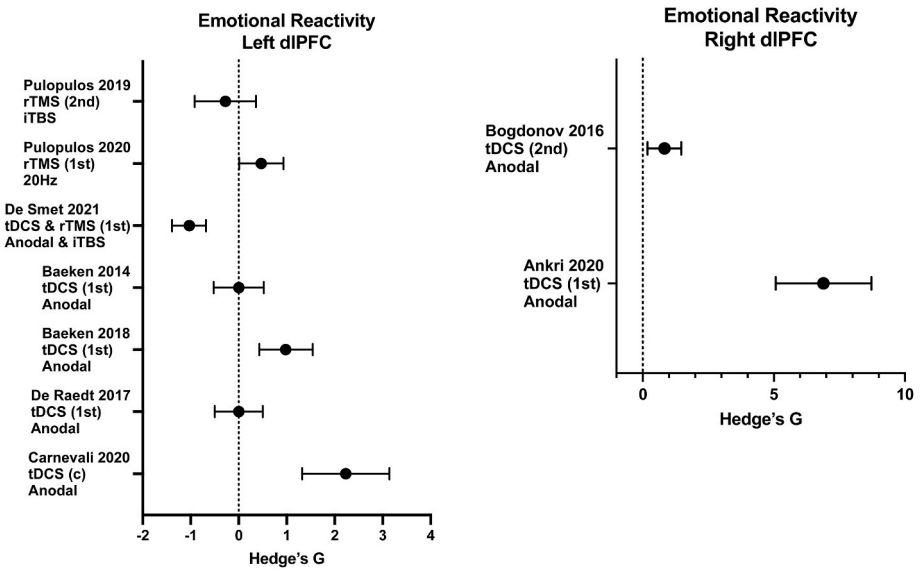

Fig. 4. Forest plots to show effect sizes for emotional reactivity measures separated by neural target for studies from Table 2 for which data were obtained
For each study, the following information is provided: 1) Neuromodulation type (rTMS and/or tDCS), 2) Order of neuromodulation and stressor (1st, 2nd, or concurrently [c]), 3) Neuromodulation frequency used.

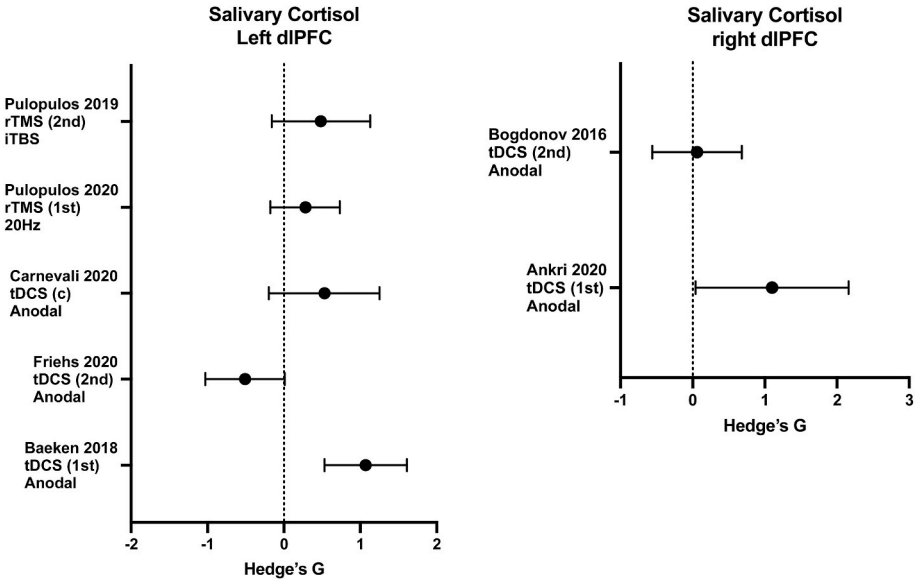

Fig. 5. Forest plots to show effect sizes for salivary cortisol separated by neural target for studies from Table 2 for which data were obtained
For each study, the following information is provided: 1) Neuromodulation type (rTMS and/or tDCS), 2) Order of neuromodulation and stressor (1st, 2nd, or concurrently [c]), 3) Neuromodulation frequency used.

neuromodulation. This difference in event order is also seen in the final 2 studies: one induced stress prior to neuromodulation (Friehs and Frings, 2020) and the other induced stress concurrently with neuromodulation (Plewnia et al., 2015). Both studies targeted the left dlPFC and used the socially evaluated cold pressor test (SECPT) or Paced Auditory Serial Addition Task (PASAT), respectively; neither included a control condition. Friehs et al., 2020 found that the stressor led to increased accuracy and reaction time (n-back task) but they found no main effect of tDCS or tDCS/stress interaction on measured EF outcomes. Notably, cortisol response was used as a marker of stress response and only "cortisol responders" were included in subsequent analyses of neuromodulation response. Plewnia et al., 2015 did not measure pre- and post-stressor EF outcomes so it is unclear whether there was an effect of stress on EF; however, they found tDCS led to decreased PASAT interstimulus interval (i.e. led to faster stimulus presentation speed in this adaptive performance task) relative to sham, but had no effect on errors.

Social exclusion induces psychosocial stress and is modeled in the cyberball task (Eisenberger et al., 2003; Williams et al., 2000). In the cyberball task, a virtual ball is tossed between several hypothetical players. The participant is either socially included to receive an equal percentage of ball tosses or socially excluded to receive a low percentage. The participant must monitor the percentage of ball tosses they receive during both conditions, which may induce cognitive inhibition of emotional reactivity to social exclusion. We found 4 studies (two within the same publication) that explored the effect of neuromodulation on cognitive inhibition during the psychosocial stressor. Each study used different neuromodulation protocols across 3 distinct

neural targets. Fitzgibbon et al., 2017 used 1Hz rTMS targeting the left dlPFC followed by stress induction using the cyberball task (Fitzgibbon et al., 2017). In the remaining 3 studies, neuromodulation and stress induction occurred concurrently with the cyberball task. Riva et al., 2012 used anodal tDCS over the right vlPFC, Riva et al., 2014 study 1 used cathodal tDCS over the right vlPFC, and study 2 used cathodal tDCS over the right posterior parietal cortex (PPC). Fitzgibbon et al., 2017 and Riva et al., 2014, study 1 found a main effect of stressor with the social exclusion condition leading to fewer correctly identified ball tosses compared to the social inclusion condition. None of the studies found a main effect of neuromodulation or an interaction effect on their EF outcome.

The 8 studies outlined here targeted 4 distinct neural locations: right dlPFC, left dlPFC, right vlPFC, and right PPC in both excitatory and inhibitory neuromodulation paradigms, with the majority using tDCS rather than rTMS. Although all studies used a between-subjects design, there were no other common design features and the wide-ranging approaches prohibit clear conclusions from these studies about potential optimal targets for modulating the effects of stress on working memory.

3.2.2. Emotional reactivity

Measures of emotional reactivity and responsiveness vary; however, they are most commonly measured via the State Trait Anxiety Inventory (STAI), Positive and Negative Affect Scale (PANAS), and different visual analog rating (VAS) scales. Although data suggest that various emotional responses (e.g. anxiety, depressed mood) may occur through slightly different neural pathways, findings presented in section 2.2.3.1 highlight common pathways involved in stress-induced emotional changes. Fig. 3 identifies key neural regions associated with some of these emotional responses, and highlights key locations that could serve as targets for NIBS. We will evaluate mood-related outcomes of the studies in Table 2 by target region.

The most commonly targeted location is the dlPFC. We found 3 studies targeting the right dlPFC, 11 targeting the left dlPFC, and 1 targeting both concurrently. All 3 studies targeting the right dlPFC found a main effect of stressor (TSST or Critical Feedback Task [CFT]) on at least one mood measure (Ankri et al., 2020 [tDCS]; Bogdanov & Schwabe [tDCS], 2016; Remue et al., 2016 [rTMS]); however, none of those studies reported any main effects of right dlPFC neuromodulation or interaction effects on mood measures. Of the 11 studies targeting the left dlPFC, 9 found a main effect of stressor (TSST, CFT, or cyberball) on at least one mood measure (Baeken et al., 2014, 2018; De Raedt et al., 2017; De Smet et al., 2021; De Witte et al., 2020; Fitzgibbon et al., 2017; Remue et al., 2016). Five of those studies used the TSST (Carnevali et al., 2020 [tDCS]; De Witte et al., 2020 [rTMS]; Pulopulos et al., 2019; 2020 [rTMS]; Wandel et al., 2020 [rTMS]) and only one found any effects of neuromodulation: Carnevali et al., 2020 used anodal tDCS on the left dlPFC; the group that received tDCS reported lower anxiety after stressor compared to sham. Four studies used critical feedback as the stressor; although all found a main effect of stressor on at least one mood measure, none found any effects of neuromodulation on mood (Baeken et al., 2014 [tDCS]; Baeken et al., 2018 [tDCS]; De Raedt et al., 2017 [tDCS]; Remue et al., 2016 [tDCS]). One study used the PASAT and, despite no significant main effect of stressor on overall mood, found that anodal tDCS of the left dlPFC blocked stressor-related increases in feeling 'upset' after performing the PASAT (Plewnia et al., 2015). Finally, one study targeted bilateral dlPFC using anodal tDCS alongside iTBS of the left dlPFC (De Smet et al., 2021); in this study, the stressor (Maastricht Acute Stress Test) increased perceived stress and negative affect; however, there was no effect of neuromodulation.

The vlPFC is another location associated with responses to stressors. Two studies explored the effect of either anodal (Riva et al., 2012) or cathodal (Riva et al., 2015) tDCS to the right vlPFC in conjunction with the cyberball task. Both studies found that the stress condition increased feelings of social exclusion and negative emotions. There was an interaction effect of *anodal* stimulation: social exclusion resulted in *less* unpleasantness and hurt feelings in the tDCS group compared to sham; no differences were seen during social inclusion (Riva et al., 2012). Interestingly, vlPFC *cathodal* stimulation showed the opposite interaction: during social exclusion there was an *increase* in hurt feelings and negative emotions in the tDCS group compared to sham; no differences were observed during social inclusion (Riva et al., 2015). Riva et al., 2014 also targeted the right PPC under the same conditions as an active control and found no effects of neuromodulation.

Although the mPFC is a key area in emotion regulation and stress response, we only found one study that targeted this location. Antal et al., 2014 examined how anodal or cathodal tDCS over the right mPFC impacted the responses to a subsequent stressor (TSST). There was a main effect of stressor on anxiety level (STAI) but no significant effects of neuromodulation on mood-related outcomes (Antal et al., 2014).

The 18 studies outlined here targeted 5 distinct neural locations: right dlPFC, left dlPFC, right vlPFC, right PPC, and right mPFC in both excitatory and inhibitory neuromodulation paradigms with the majority using tDCS rather than rTMS. Major differences in study designs make it very difficult to compare outcomes. We were able to collect emotional reactivity data from 9 of these studies to create Forest plots of effect sizes for the two represented regions (Fig. 4: left dlPFC, right dlPFC). Despite more data relating to mood outcomes than EF outcomes, the study design differences are more pronounced and outcomes even less consistent. Due to these differences, it is not possible to identify neural regions or NIBS parameters from these studies that are clear targets for future studies looking to modulate the effects of psychosocial stressors; however, the results of vlPFC stimulation are the most promising for future study, especially given evidence for the role of the vlPFC in cognitive reappraisal and emotion regulation (Feffer et al., 2018; Wager et al., 2008).

3.3. Independent factors affecting neuromodulation response

The considerable variability in experimental design (just discussed) also highlights areas where individual differences may impact responses and provides insights into potential best practices for future experimental designs. Many studies lacked a control condition (for stressor or neuromodulation) to appropriately evaluate a neuromodulation × stress interaction. Additionally, the order of events within sessions determines the effect of neuromodulation on stress reactivity: there is no consensus on whether neuromodulation should occur pre- or post-stressor for optimal effects. Finally, most studies discussed here use a between-subjects design, which may be problematic because many individual factors can affect responses to both the stressor and neuromodulation interventions. Given our understanding of the various individual differences that impact responses to stressors, it is unsurprising that these factors can also affect neural response to neuromodulation. This review does not consider all possible factors that affect responses to neuromodulation (others have already done so (see: (Valero-Cabré et al., 2017).

In addition to the obvious differences in study design affecting outcomes, various parameters of neuromodulation also differ between studies. These include variables that apply across multiple types of NIBS such as total number of sessions, session duration, and stimulation frequency along with the more NIBS-type specific parameters such as pulse morphology, pulse amplitude, and stimulation intensity (de Jesus et al., 2014; Niehaus et al., 2000; Rossi et al., 2021; Stokes et al., 2005). Although there are well-established safety guidelines for rTMS that dictate safe ranges for these parameters, there is still considerable variability within what is considered safe (Rossi et al., 2021). This wide range of NIBS parameters complicates comparison between studies and highlights an area in need of increased understanding and consistency within the field.

4. Future directions in research on neuromodulation & stress

4.1. State of current research

There is extensive research outlining the ways in which different types of stressors impact physiology and behavior along with a clear understanding of the varying mechanisms underlying these responses. In contrast, while research into NIBS mechanisms has expanded considerably in recent years (Beynel et al., 2020; Chervyakov et al., 2015), there remains much to uncover regarding the precise mechanisms and impacts of various methodologies. Thus, we are left with an expanding body of literature exploring ways that NIBS can affect stress response without the ability to appropriately compare and contrast between studies. Nonetheless, each study has the ability to highlight important experimental design details and identify potential targets for future study.

4.2. Promising neural circuits and targets

A theoretical, mechanistic-driven approach to neural targets may be the most effective. In developing this approach, we recognize that the negative effects of psychosocial stressors occur via multiple related but distinct neural pathways. Thus, it is fair to assume there may be multiple effective neuromodulation targets and their efficacy may depend on the outcome of interest. In this review, we focused on the EF and emotional impacts of psychosocial stressors, which led to the evaluation of multiple distinct neural circuits. Based on current evidence, the dlPFC—a key structure within the CEN—appears to be the best-supported target for addressing EF-related effects of stressors. In contrast, the vlPFC—a key structure in cognitive reappraisal circuits—may be the best-supported target to affect stress modulation of emotional responses.

In addition to the focused attention on theoretically-driven neural targets, we must also consider the role of biomarkers that may reflect changes in these neural circuits and can be used to identify efficacious neuromodulation therapy. Details of these biomarkers exceed the scope of this review (see (Cirillo et al., 2017; Kim et al., 2021), but it will be important to conceptualize and incorporate this information into future studies.

4.3. Individual differences

One major barrier in the current research in this field is the significant impact of individual differences on responses to both stressors and neuromodulation. As we highlighted, many state and trait variables may play a significant role in an individual's responses. It is not feasible to control and track all variables for every study; however, it is important they are controlled whenever possible and that any analysis and evaluation of intervention efficacy consider these issues. Where possible, fully within-subject study designs that include sham stimulation and protocol crossover will mitigate many of these concerns. Future studies should examine which of these factors explain the most variance, so that researchers can focus efforts on controlling and addressing those factors in their study designs.

4.4. Relevance to psychopathology

Stressors are not pleasant experiences for any individual; however, for individuals with existing psychopathology the addition of acutely stressful situations may significantly worsen outcomes. One key example comes from individuals with substance use disorders (SUDs). We know that people with SUDs experience impairments in EF and emotional regulation, which are associated with alterations in the same neural circuitry affected by acutely stressful situations (Bruijnen et al., 2019; Koob and Volkow, 2010; Madoz-Gúrpide et al., 2011). Stress-exposure is problematic for people trying to recover from any SUD because it weakens inhibition of automatic behaviors and may increase drug craving and likelihood of relapse (al'Absi, 2006b; Brady and Sinha, 2005; Brewer et al., 1998; Hyman et al., 2007; Kadam et al., 2017). Neurochemical theories of addiction suggest there may be dysfunction in two fronto-striatal circuits: (1) elevated activity in the limbic circuit resulting in hyper-sensitivity to drug cues; and (2) decreased executive control resulting in a diminished ability to resist drug-craving (Kravitz et al., 2015). For individuals with SUDs who are attempting to reduce their substance use, current treatments are insufficient for addressing the effects of stressors on these already dysfunctional neural pathways (Kotlyar et al., 2011; Leri et al., 2003; Ray et al., 2013). This understanding lays the theoretical groundwork for developing NIBS targets and protocols for treating SUDs, particularly stress-induced substance use.

The significant overlap between the pathways that are impaired in SUDs and those that are impacted by acute stressors provides a key example of the way in which these mechanistic findings can be translated to the treatment of psychopathology, but SUDs are by no means the only disorder that could benefit from these insights. A wide range of psychiatric disorders are characterized by inappropriate activation of the stress-response systems (e.g., anxiety disorders and post-traumatic stress disorder). Research has already demonstrated distinct impacts of acute stressors on populations with these disorders and understanding how NIBS can be used to modify stress responses in healthy populations could help in the development of interventions for these disorders. Furthermore, a more thorough mechanistic understanding of these effects could facilitate development of preventive interventions that may assist individuals in modulating their stress response in the aftermath of a trauma. Initial evidence examining pharmacological interventions that target ANS responses (e.g., beta-blockers) suggests the potential for medications that diminish the SNS response to reduce the consolidation of traumatic memories and later development of PTSD (Grillon et al., 2004; Krauseneck et al., 2010; Villain et al., 2016, 2018); understanding how NIBS influences these stress responses may allow for similar neuromodulatory interventions.

4.5. Intervention development

This review highlights significant inconsistency in the development of neuromodulation interventions. Even within interventions of the same modality (e.g. rTMS or tDCS) the duration and pattern of stimulation can vary considerably. The past two decades of neuromodulation research have demonstrated the importance of many of these neuromodulation variables (de Jesus et al., 2014; Lewis et al., 2016; Rossi et al., 2009; Valero-Cabré et al., 2017); as such, researchers should develop a consistent method of reporting these parameters to facilitate meaningful comparisons between studies.

One important parameter within neuromodulation research that has not been considered in most studies until recently is the timing of the NIBS intervention and any concurrent stimuli. It is clear that participant mental state during stimulation can significantly impact the outcome of the stimulation (Silvanto et al., 2007). It is thought that engaging the neural target during stimulation can modulate outcomes; for example, completing a working memory task while receiving excitatory NIBS to the EF circuit may increase EF improvement induced by the stimulation. Given this knowledge, it stands to reason that when NIBS is performed relative to stressor induction may impact outcomes. Furthermore, it is possible that introduction of behavioral stress reduction strategies during NIBS (e.g., mindfulness) may provide additional benefits. The studies presented here demonstrate considerable variability in the order of NIBS and stressor and this represents a key area for future research.

Of equal importance is the method of targeting the appropriate neural location. Until recently, scalp measurement and the EEG 10–20 system were standard (Herwig et al., 2003); however, we now have a concrete understanding of the variability that this type of targeting can create. At present, there are increasingly sophisticated techniques, including MRI-based structural and functional connectivity

neuronavigation that can significantly improve the accuracy of the neuromodulation targeting (Cole et al., 2022; Schönfeldt-Lecuona et al., 2010; Summers and Hanlon, 2017; Vila-Rodriguez and Frangou, 2021). Additionally, improved understanding and modeling of the E-field induced by TMS will provide a more precise understanding of both the specific locations impacted and the dose of the stimulation applied (Gomez et al., 2020). Most NIBS studies to date have focused on the dlPFC, an area for which there is already considerable research regarding optimal targeting methods and downstream effects of stimulation. Given the potential of other brain regions, such as the vlPFC, to serve as effective targets in stress modulation, more consideration should be given to ensuring these regions are targeted correctly and specifically. Furthermore, as highlighted in Section 2.2, no neural region functions in isolation and an improved understanding of functional connectivity within relevant brain networks will provide important guidance into the efficacy of certain NIBS paradigms (Cash et al., 2021). Future neuromodulation research should endeavor to use these more validated and precise techniques for ensuring precise neural targeting and recording the exact dose of stimulation applied to the target location.

5. Conclusions

Existing research in this field provides an important foundation for development of future work exploring the impacts of NIBS on stress responses and highlights several key areas for improvement in the field. Research in this area should use consistent, reliable methods for stress induction and NIBS targeting. Measuring key physiological outcomes related to the SAM-axis are vital for research seeking to identify methods to reduce stress reactivity. Furthermore, wherever possible control stressor and NIBS arms, ideally within subjects, should be used to strengthen interpretation of results. At present, it is difficult to identify clear clinical targets for NIBS-related stress reduction; however, initial studies alongside a theoretical understanding of cognitive and psychological responses to stressors highlight key pathways that are strong candidates for more rigorous exploration. Specifically, we believe that the dlPFC and vlPFC are the best-supported targets at this time due to their clear role in the respective EF and emotional regulation networks alongside the existing evidence that stimulation of these locations may modulate these stress responses. There is significant room for further study within this field and, with the implementation of a more standardized methodology and awareness of key variables leading to individual differences, there is a strong likelihood that reliable neural targets for NIBS intervention in stress responses will be identified.

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Contributors

TEHM took the lead in writing the manuscript and developing the Forest plots (Figs. 4 and 5). EG conceptualized and developed Figs. 1–3. TEHM and EG performed a primary review of the literature and extracted data from relevant studies. NM and MKG reviewed extracted data for completeness and accuracy. All authors provided critical feedback and contributed to writing the manuscript. All authors have reviewed the manuscript content and approved the final version for publication.

Declaration of competing interest

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