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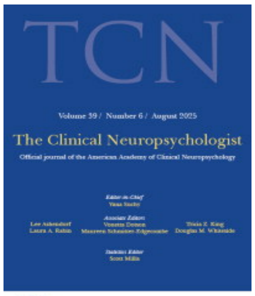

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Disinhibition, rather than moderate-to-severe traumatic brain injury, moderates the impact of anger provocation on subjective emotional experience

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ABSTRACT

Objective: Altered reactivity to emotional stimuli is common after traumatic brain injury (TBI), which is suggested to reflect difficulties with emotion regulation. While disinhibition is common after moderate-to-severe TBI, limited research has investigated the link between disinhibition and emotional reactivity in this clinical group. The aim of this research, therefore, was to investigate the relationship between disinhibition and TBI to anger provocation.
Method: Thirty-five individuals with moderate-to-severe TBI and thirty-one controls completed an anger induction task. Participants rated their experience of emotions and subjective arousal before and after the induction. Heart rate, respiration and skin conductance were also measured. Disinhibition was measured using the Frontal Systems Behavior Scale.
Results: In the full sample, the mood induction led to increased skin conductance, respiration, and self-reported anger, tension, arousal and negative mood. There were no differences between those with TBI and controls. Disinhibition interacted with the impact of anger provocation on subjective, but not objective, outcomes such that those elevated in disinhibition reported increased feelings of anger and tension in response to the mood induction. Disinhibition did not interact with TBI across any subjective and objective emotional measures examined in response to mood induction.
Conclusions: While anger causes emotional changes for all individuals, these changes are particularly pronounced among those who are disinhibited, irrespective of whether an individual has sustained a TBI. This is an important consideration when examining emotional regulation post-TBI as the degree of disinhibition appears to alter subjective interpretations of emotional events, which could lead to emotion dysregulation.

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Introduction

Traumatic Brain Injury (TBI) significantly contributes to global mortality rates and long-term disability. While TBIs are heterogeneous, moderate-to-severe TBIs typically lead to significant physical, psychosocial, and economical challenges for individuals and their psychosocial networks (Dewan et al., 2019), with difficulties with emotion dysregulation commonly reported and observed following moderate-to-severe TBI. Emotion regulation is defined as the ability to effectively manage, modulate, and respond to emotional, behavioural, and physiological reactions to given situations (Linehan, 2015). The capacity to use internal and external cues to modulate emotional states during normative conditions is salient for managing distress (Sarah et al., 2023). Emotion dysregulation after TBI is associated with an array of psychosocial difficulties, including increased risk of psychiatric disorders (Ashman et al., 2004), caregiver burden (Qadeer et al., 2017), and poor interpersonal outcomes (Winter et al., 2018). Emotion dysregulation following severe TBI is often expressed as irritability, anger, aggression, and agitation (Nash et al., 2019), and typically framed within the context of disinhibition.

Disinhibition refers to the inability to suppress automatic behavioural responses and involves the failure of top-down cognitive control processes required to supress a response to achieve goal-directed behaviour (Nigg, 2000). Damage to the fronto-temporal lobes, together with damage to the orbitofrontal cortex (OFC) and its connectivity to other brain regions, including the gyrus rectus, insula, amygdala, and anterior cingulate cortex (ACC), have been linked to acquired behavioural disinhibition (Osborne-Crowley & McDonald, 2018; Bush et al., 2000; Davidson et al., 2000). Interestingly, the OFC is particularly vulnerable to damage following brain insult, as acceleration/deceleration forces cause the bone cavities of the skull to directly impact brain tissue, resulting in multifocal lesions in the OFC (Levin & Kraus, 1994). Thus, behavioural disinhibition post moderate-to-severe TBI is common, with approximately 30% of TBI survivors experiencing disinhibition (Ciurli et al., 2011). A recent review by Filipčíková et al. (2021) found that participants with severe injury were three times more likely to develop social disinhibition compared to those with mild injuries, and such changes are associated with negative functional outcomes, particularly for caregivers. Indeed, families may struggle to adjust to changes in personality (Osborne-Crowley & McDonald, 2018), while relationship difficulties, poor social integration, suicidal ideation, and legal issues are further negative outcomes. Given that disinhibition involves several brain structures, including the reciprocal connection between the OFC and structures of the limbic system, the autonomic nervous system (ANS) has been implicated in acquired disinhibition.

Disruption to ANS functioning is common following TBI. There is evidence of dysregulation of the sympathetic nervous system (SNS), which is responsible for regulating reactivity to stress/threat and emotions (Lemke, 2004). For example, those with TBI have increased heart rate (Zheng et al., 2020) and reduced heart rate variability (HRV) (e.g. Baguley et al., 2006; Riganello et al., 2010; Vistisen et al., 2014) and lower resting skin conductance level (SCL) (e.g. Rushby et al., 2013a; Fisher et al., 2015). Indeed, higher HRV is associated with better physical health (Masi et al., 2007), cognitive functioning (Hansen et al., 2003), and emotion regulation (Thayer & Brosschot, 2005; Appelhans & Luecken, 2006). People with higher HRV have increased autonomic flexibility to respond to stress/threat and can better regulate emotional responses (Hourani et al., 2020). It follows, therefore, that the link between altered autonomic physiology and emotional outcomes after TBI have been investigated extensively.

Individuals with severe TBI have altered skin conductance when viewing emotion eliciting stimuli (e.g. images and video clips). For example, de Sousa et al. (2011) found that participants with severe TBI had lower skin conductance level than controls when observing angry faces, but not happy faces. Using the same sample, de Sousa et al. (2012) also found that those with severe TBI had lower SCL in response to unpleasant films compared to controls. Furthermore, they found significantly lower levels of self-reported arousal and reduced negative mood in response to the negative films in the TBI group. Additionally, a systematic review of studies investigating physiological outcomes at rest and in response to emotional stimuli after TBI reported several studies had found a reduction of SCL in those with TBI compared to controls (e.g. Fisher et al., 2015; Osborne-Crowley et al., 2020; Rushby et al., 2013a; Rushby et al., 2016; Sánchez-Navarro et al., 2005). While there is also evidence of no differences (e.g. Kelly et al., 2017; Krpan et al., 2011; Rushby et al., 2013b) or increased SCL (Aboulafia-Brakha et al., 2016), these studies predominately show that those with moderate-to-severe TBI may have attenuated subjective and objective responses to emotional inducing stimuli relative to controls.

While altered physiological responses to emotional stimuli have been associated with impaired emotional functioning after TBI, only one study to date has investigated the relationship between emotional reactivity and disinhibition. McDonald et al. (2010) investigated whether differences in subjective emotional responses were associated with control (i.e. loss of inhibition). They found no differences between groups for subjective emotional responses (i.e. anger/tension, anxiety, and depression) after watching a provocative film. However, for the TBI group, loss of inhibition was significantly associated with increased emotional experience to emotional films, specifically increased self-reports of anger and confusion. These findings suggest that a loss of inhibition can affect emotional processing after TBI. Given these findings, further investigation of the link between emotional output and disinhibition in those with TBI is warranted, as this could provide further evidence that disinhibition affects emotional processing and behaviour that could inform psychological interventions. While McDonald et al. (2010) investigated subjective emotional experiences, no studies have yet investigated the link between physiological outcomes and disinhibition after TBI. Furthermore, while studies have consistently reported altered physiological and subjective responses to emotion inducing stimuli, the directionality of these findings have been mixed. As such, it is possible that other factors, such as disinhibition, may be involved in subserving these divergent responses.

The aims of the current study were to: 1) determine whether participants with at least moderate-to-severe TBI have altered objective and subjective outcomes to a mood induction procedure compared to controls, and 2) whether disinhibition differentially affects this reaction to mood provocation for those with or without TBI. For objective responses, it was hypothesised that there will be 1) significantly lower SCL (de Sousa et al., 2011; de Sousa 2012), and 2) significantly lower HRV (Amorapanth et al., 2018) in response to a mood induction procedure for participants with TBI compared to controls. For subjective emotional experience, it was also hypothesised that TBI participants would report reduced subjective arousal and greater general mood in response to mood induction procedure relative to controls, suggesting there would be a reduced emotional response to the mood induction for those with TBI compared to controls (de Sousa et al., 2012). Furthermore, consistent with findings from McDonald et al. (2010), it was hypothesised that there would be no difference between groups in terms of self-reported anger and tension in response to the mood induction. It was also hypothesised that participants with clinically elevated disinhibition will have increased subjective emotional experiences in response to the mood induction, particularly within the context of anger, relative to those without elevated disinhibition. TBI participants who of at least moderate severity were of relevance to this study, as these individuals have greater functional and emotion dysregulation difficulties post-injury compared to those on the mild end of the severity spectrum.

Method

Participants

Participants were recruited from both the broader community and outpatient brain injury rehabilitation units in Sydney. To ensure that participants had sustained a TBI of at least moderate severity, their medical records were used to confirm several clinical indicators: (1) a period of post-traumatic amnesia (PTA) of greater than 24 h (Carroll et al., 2004), or (2) a coma longer than 24 h (as measured by the Glasgow Coma Scale), or (3) neuroimaging changes commensurate with traumatic damage (Malec et al., 2007). As GCS data was not available for many of the TBI participants, or in instances whereby there was a discrepancy between GCS and PTA scores, PTA was preferentially used in classifying the severity of TBI in our sample. Consistent with Carroll et al. (2004) guidelines, those were considered in the moderate range if they had a PTA lasting longer than 24 h but less than seven days, whereas those with a PTA greater than 7 days were of severe TBI classification. PTA had to be prospectively measured *via* the use of a validated measure (e.g. Westmead PTA scale), consistent with current recommended guidelines (Ponsford et al., 2023).

TBI participants were excluded if they had: (1) a history of a prior neurological disorder; (2) heart problems or abnormal ECG; (3) a TBI that was sustained before the age of 16 years; (4) a TBI of mild severity (i.e. PTA < 24 h as evidenced by their medical records); and (5) any communication issues. Participants were excluded if they were younger than 17 years or older than 75 years, or if they were not at least 12 months post-injury. Control participants consisted of healthy volunteers from the community recruited *via* online advertisements posted on social-media or advertisement platforms (e.g. Gumtree). They were excluded from participating if they met any of the following criteria: (1) language or perceptual difficulties that would hinder the completion of the tasks or cognitive testing, (2) a history of neurodevelopmental condition, psychiatric disorder or history of drug and alcohol dependence, and (3) a history of brain insult or neurodegenerative condition (i.e. dementia) or (4) uncorrected vision or hearing difficulties. Participants received reimbursement at a rate of approximately $20 AUD per hour.

In total, sixty-six participants were recruited to this study. They were aged between 20 to 70 years ($M = 47.09$, $SD = 14.24$). The TBI group consisted of 35 participants (10 females, mean age 47.80 years, range 21 - 70 years). They had a mean PTA duration of 66.03 days ($SD = 53.143$), with a range of PTA lasting between 9 and 186 days, meaning that our entire was predominately of severe TBI severity (Carroll et al., 2004). They had a mean GCS of 5.73 ($SD = 3.79$, range 3-14). At the time of participation, they were an average of 13.64 years ($SD = 11.98$, range 2—49 years) post-injury. The control group consisted of 31 individuals from the community (11 females, mean age 46.29 years, range 20-68 years). All participants provided informed consent and ethics was approved by Royal Prince Alfred Hospital (X16-0279) and ratified by Western Sydney University Human Research Ethics Committee (H15588) and the University of New South Wales (HREC/08/RPAH141).

Measures

Mood induction

To assess subjective emotional experience, each participant completed a mood induction that involved two parts. Firstly, participants viewed two movie clips from the films *My Bodyguard* and *Cry Freedom*, which have both been shown to elicit anger (Gross & Levenson, 1995; Wearne et al., 2021). The clip from *My Bodyguard* portrayed the scene of a young man getting bullied by a bodybuilder. The scene from *Cry Freedom* portrayed the attack by White South African soldiers on black protestors in the Soweto Massacre. To account for order effects, clip presentations were counterbalanced between participants. Next, participants completed the Paced Auditory Serial Addition Task (PASAT). This required participants to listen to a series of numbers, then continuously add the new digit to the prior one. The PASAT becomes increasingly difficult and elicits frustration and anger, and physiological arousal (Wearne et al., 2021). Subjective emotional experience was averaged across both aspects of the mood induction task.

Objective emotional responses

To assess objective outcomes, heart rate was recorded *via* three Ag/AgC1 sensors fitted to the participants wrists, and skin conductance level (SCL) was measured from sensors fitted to the second and third digits of participants' non-dominant hand. Raw data was transmitted to a laptop computer *via* a 10-channel FlexComp Infiniti Encoder system. Data was obtained using BioGraph Infiniti Software 6.0 (Thought Technology Ltd., Quebec, Canada). HRV was quantified using time-domain and frequency-domain analysis (Malik, 1996). For the time-domain, overall cardiovascular adaptability was measured by calculating the standard deviation of normal-to-normal intervals (SDNNs) (Malik, 1996). For the frequency domain, the absolute power spectral density (ms2) was quantified for the high frequency (HF) band (0.15-0.40 Hz) and low frequency (LF) band (0.04-0.15 Hz) (Malik, 1996). It has been considered that the HF band is an index of parasympathetic activity, and the LF band an index of sympathetic activity (Berntson et al., 1997). In sum, objective emotional reactivity was measured by comparing changes in SCL and HRV across two conditions, 1) at rest 2) and during the mood induction.

Subjective emotional experience

Two self-report questionnaires were administered to assess subjective mood changes in response to the mood induction procedure. The 24-item Profile of Mood States-Adolescent (POMS-A; Terry et al., 1999) is considered a valid index of adult mood states (Terry et al., 2003), and consists of six subscales: anger, vigor, depression, fatigue, confusion, and tension (4-items per subscale, maximum score of 16). Participants rated their present mood (e.g. angry) according to a 5-point Likert scale ranging from 0 (not at all) to 4 (extremely). In this study, only the anger and tension subscales were used. The Self-Assessment Manikin (SAM; Lang et al., 1997) was also used to assess valence/general mood (e.g. mood associated with a stimulus) and arousal during the resting state and in response to the mood induction. Graphic figures that portray values along each dimension on a 5-point scale is presented to record emotional responses. Greater scores indicated greater negative mood. The SAM has high reliability for both dimensions (Lang et al., 1997).

Disinhibition: frontal systems behavior scale (FrSBe)

The FrSBe is a 46-item behaviour rating scale that measures behavioural disturbances associated with damage to the frontal lobes and frontal brain systems (Stout et al., 2003). The FrSBe consists of a total score and three subscales: Apathy (14-items), Disinhibition (15-items), and Executive dysfunction (17-items). Item response is based on a 5-point Likert Scale (1 = *"Almost Never"* and 5 = *"Almost Always"*). The FrSBe has high internal consistency (self-rating form α = .88, informant-rating form α = .92) (Grace et al., 1999), good inter-rater reliability (total score: .83 to .89, subscales: .79 to .92) (Velligan et al., 2002), and is a useful measurement tool in the population of TBI (Niemeier et al., 2013). For the purpose of this study, the FrSBe was used to determine group membership, such that those with Disinhibition scores with a T-score of 65 or greater were allocated into the clinically elevated disinhibition group. The self-report version was used for control while the informant version was used for the severe TBI group (to minimise any issues with awareness and insight on the self-report of emotion dysregulation).

Procedure

Some of the TBI participants in the current study formed part of a larger research clinical trial (Wearne et al., 2021). First, prospective participants attended an assessment to determine eligibility, which consisted of an interview and review of medical records to obtain demographic and injury-related information (for the TBI-group). Next, they completed self-report measures before baseline objective (physiological) and subjective (self-report) data was measured to determine resting emotional state. During the resting state, participants sat comfortably with their eyes open for five minutes while physiological data was continuously measured to provide a baseline of physiological arousal. They then completed the SAM and POMS to provide a baseline of subjective resting emotional state. Participants subsequently engaged in the mood induction procedure whereby physiological data was continuously measured. Next, participants completed the self-report questionnaires to measure subjective emotional experience to the mood induction procedure.

Data analysis

For the objective and subjective emotional variables, the assumptions of normality and homogeneity of variances were violated on several factors. Skin conductance and HRV variables were consequently log transformed. Demographic and outcome baseline measures were assessed between the TBI and control group using independent samples t-tests. To examine objective and subjective emotional outcomes and the effect of disinhibition, data from each objective (SCL, HR, Respiration, HRV-HF, and HRV-SDNN) and subjective measure (POMS-Anger, POMS-Tension, SAM-Arousal, SAM-Valence) was subjected to a $2 \times 2 \times 2$ mixed ANOVA with Condition (Baseline vs Induction) as the within-subjects factor, and Group (TBI vs Control) and Disinhibition (Not Elevated vs Clinically Elevated) as the between subjects factors. Statistical analyses were completed using the Statistical Package for the Social Sciences (SPSS). Homogeneity of variance assumption was met for HR, SCL, POMS-Anger, SAM-Valence, and SAM-Arousal variables. ANOVA is quite robust to small-to-moderate violations of normality and homogeneity of variance, particularly if the sample size is large (Field, 2013). Therefore, with a sample size of 65, the departures from normality and homogeneity of variances were not a concern and analyses were conducted with the assumptions satisfied. A Bonferroni adjustment was made to correct for multiple comparisons such that alpha was adjusted to $p < .01$. Partial η^2 are reported (Partial eta squared, η^2; 0.01 = small, 0.06 = medium, 0.14 = large) (Lakens, 2013).

Results

Demographic and group characteristics

The means and standard deviations for all demographic and baseline outcome variables can be found in Table 1. The majority of the sample were of Australian background (71.4%) while the remaining identified as European (22.9%) or Asian (5.6%). Prior to sustaining their TBIs, the majority of the TBI group were students (22.9%) or worked in trades (28.6%) or professional positions (34.3%). Only 2.9% were unemployed prior to sustaining their injuries. However, 51.5% of the sample were unemployed or retired at the time of assessment, while 14.3% worked in trades and 20% worked in professional settings. Overall, this suggests that those with TBI had quite significant functional and occupational limitations post-injury. There were no significant differences between those with TBI and controls with respect to age ($t(64) = .427$, $p = .67$, $d = .11$), years of education ($t(64) = -1.66$, $p = .10$, $d = -0.41$) and gender distribution ([($\chi 2(1, n = 66) = .362$, $p = .55$). However, those with TBI had significantly greater total scores of disinhibition on the FRsBe compared to controls ($p < .05$, $d = .54$). However, there was no difference in the distribution of those with clinically elevated level of disinhibition in the TBI group compared to control participants ([($\chi 2(1, n = 66) = 2.629$, $p = .11$).

Baseline physiology & emotional factors

There were no significant group differences between those with TBI and controls with respect to baseline SCL ($t(64) = -1.38$, $p = .17$, $d = -0.34$), HR ($t(64) = -1.63$, $p = .11$,

Table 1. Demographic, resting, and disinhibition variables at baseline between those in the traumatic brain injury ($n = 35$) and control groups ($n = 31$).

Demographic and injury characteristics	TBI ($n = 35$) Mean (*SD*)	Control ($n = 31$) Mean (*SD*)	Sig.
Age (yrs)	47.80 (14.26)	46.29 (14.30)	.67
Biological sex (*n*)	$M = 25$, $F = 10$	$M = 20$, $F = 11$	.55
Education (yrs)	13.46 (2.83)	14.55 (2.47)	.10
PTA (days)	66.03 (53.14)		
GCS	5.73 (3.79)		
Time since injury (yrs)	13.64 (11.98)		
Resting physiology and emotional state			
HR	65.80 (9.76)	69.65 (9.35)	.11
SCL	1.28 (.91)	1.51 (.97)	.31
Respiration	14.36 (3.03)	13.76 (2.75)	.41
HRV – SDNN	3.88 (.76)	3.81 (.36)	.64
HRV - HF	4.71 (2.05)	4.33 (1.07)	.35
SAM – Valence	2.49 (.58)	2.48 (.63)	.98
SAM – Arousal	1.87 (.77)	1.39 (.62)	.01*
POMS – Anger	.70 (1.88)	.06 (.25)	.054
POMS – Tension	1.57 (2.27)	.77 (1.23)	.079
Disinhibition			
FrSbe	66.49 (19.59)	57.00 (14.84)	.03*
Clinically elevated (*n*)	$N = 17$	$N = 9$	.11
Not clinically elevated (*n*)	$N = 18$	$N = 22$	

Note. TBI: Traumatic brain injury; SD: standard deviation; PTA: Post Traumatic Amnesia; GCS: Glasgow Coma Scale; HR: Heart rate: SCL: skin conductance level; HRV: heart rate variability; SDNN: standard deviation of normal-to-normal intervals; HF: high frequency; SAM: Self-assessment manikin; POMS: Profile of Mood States; FrSbe: Frontal Systems Behavior System; yrs: years; n: number; sig: significance level.

$d = -0.40$), respiration rate ($t(64) = -0.830$, $p = .41$, $d = .25$) and HRV measures of SDNN ($t(49.99) = -0.472$, $p = .64$, $d = .11$) and HF-HRV ($t(52.83) = .94$, $p = .35$, $d = .22$). Those with TBI also reported no baseline differences with respect to general mood ($t(64) = .03$, $p = .98$, $d = .01$), anger ($t(35.35) = 1.99$, $p = .054$, $d = .46$) and tension ($t(64) = 1.73$, $p = .08$, $d = .43$). However, those with TBI reported elevated arousal compared to controls at baseline ($p < .01$, $d = .69$).

Effect of anger provocation: objective physiological outcomes

Heart rate

There was no main effects of anger induction ($F(1,62) = .210$, $p = .648$, partial $\eta2 = .003$), clinical group ($F(1,62) = 2.664$, $p = .108$, $\eta2 = .041$), or disinhibition ($F(1,62) = .036$, $p = .849$, $\eta2 = .001$) on HR. There were also no significant interactions between clinical group and anger induction ($F(1,62) = .046$ $p = .832$, $\eta2 = .001$), disinhibition and anger induction ($F(1,62) = .487$, $p = .488$, $\eta2 = .008$), and clinical group and disinhibition ($F(1,62) = .470$, $p = .496$, $\eta2 = .008$) on HR. There was also no significant three-way interaction between clinical group, disinhibition and anger induction on HR ($F(1,62) = .021$, $p = .885$, $\eta2 = .000$).

Skin conductance

There was a main effect of anger induction on SCL, $F(1,61) = 61.544$, $p < .0005$, partial $\eta2 = .502$, such that the anger induction produced significant increases to SCL compared to baseline. There was no main effect of clinical group ($F(1,61) = .770$, $p = .355$, η2 = .014) and there was no main effect of disinhibition ($F(1,61) = .879$, $p = .991$, η2 = .016). There was also no significant interaction between clinical group and anger induction ($F(1,61) = .936$, $p = .337$, η2 = .015), disinhibition and anger induction ($F(1,61) = 1.954$, $p = .167$, η2 = .031), and clinical group and disinhibition ($F(1,61) = .611$, $p = .688$, η2 = .011). There was also no significant three-way interaction between clinical group, disinhibition and anger induction on SCL ($F(1,61) = .207$, $p = .650$, η2 = .003).

Respiration

There was a main effect of anger induction on respiration rate ($F(1,61) = 5.806$, $p < .05$, partial η2 = .087), such that anger induction led to increased respiration rate. There were no significant main effects of clinical group ($F(1,61) = .607$, $p = .439$, η2 = .010), or disinhibition ($F(1,61) = .050$, $p = .824$, η2 = .001) on respiration. There were also no significant interactions between clinical group and anger induction ($F(1,61) = .191$. $p = .664$, η2 = .003), disinhibition and anger induction ($F(1,61) = .309$, $p = .580$, η2 = .005), and clinical group and disinhibition ($F(1,61) = 1.774$, $p = .188$, η2 = .028) on respiration. There was also no significant three-way interaction between clinical group, disinhibition and anger induction on respiration ($F(1,61) = .079$, $p = .779$, η2 = .001).

HRV-SDNN

There was a main effect of anger induction on SDNN ($F(1,62) = 7.507$, $p < .01$, partial η2 = .108), such that anger induction led to reduced SDNN. There were no significant main effects of clinical group ($F(1,62) = .741$, $p = .393$, η2 = .012), or disinhibition ($F(1,62) = 1.431$, $p = .236$, η2 = .023) on HRV-SDNN. There were also no significant interactions between clinical group and anger induction ($F(1,62) = 1.897$, $p = .173$, η2 = .030), disinhibition and anger induction ($F(1,62) = .015$, $p = .904$, η2 = .000), and clinical group and disinhibition ($F(1,62) = .429$, $p = .515$, η2 = .007) on HRV-SDNN. There was also no significant three-way interaction between clinical group, disinhibition and anger induction on HRV-SDNN ($F(1,62) = 1.425$, $p = .237$, η2 = .022).

HRV-HF

There were no main effects of anger induction ($F(1,61) = 1.504$, p=225, partial η2 = .024), clinical group ($F(1,61) = 2.514$, $p = .118$, η2 = .040), or disinhibition ($F(1,61) = .547$, $p = .462$, η2 = .009) on HRV-HF. There were also no significant interactions between disinhibition and anger induction ($F(1,61) = .073$, $p = .787$, η2 = .001), and clinical group and disinhibition ($F(1,61) = .817$, $p = .370$, η2 = .013) on HRV-HR. However, there was a significant interaction between clinical group and anger induction, although the interaction did not withstand a significance correction ($F(1,61) = 4.102$, $p = .047$, η2 = .063). There was also no significant three-way interaction between clinical group, disinhibition and anger induction on HRV-HF ($F(1,61) = 1.002$, $p = .321$, η2 = .016).

Effect of anger provocation: subjective emotional experiences

General mood

There was a main effect of anger induction on general mood ($F(1,62) = 167.887$, $p < .0005$, partial $\eta2 = .730$), such that anger provocation induced significant increased negative mood (Figure 1). There was also a main effect of disinhibition ($F(1,62) = 9.223$, $p < .005$, partial $\eta2 = .129$), such that those with clinically elevated disinhibition reported greater negative mood than those without disinhibition overall. There was no effect of clinical group on mood ($F(1,62) = .311$, $p = .569$, partial $\eta2 = .005$). There were also no significant interactions between clinical group and anger induction ($F(1,62) = .106$, $p = .746$, $\eta2 = .002$), disinhibition and anger induction ($F(1,62) = .881$, $p = .351$, $\eta2 = .014$), and clinical group and disinhibition ($F(1,62) = .032$, $p = .859$, $\eta2 = .001$) on mood. There was also no significant three-way interaction between clinical group, disinhibition and anger induction on general mood ($F(1,62) = .217$, $p = .643$, $\eta2 = .003$).

Subjective physiological arousal

There was a main effect of anger induction on subjective arousal ($F(1,62) = 75.931$, $p < .0005$, partial $\eta2 = .550$), such that anger provocation induced significant increased subjective appraisal of arousal. There was also a main effect of clinical group ($F(1,62) = 5.556$, $p < .05$, partial $\eta2 = .082$), such that those with TBI reported greater subjective arousal than controls (Figure 2). There was no main effect of disinhibition on subjective arousal ($F(1,62) = 3.561$, $p = .064$, partial $\eta2 = .054$). There were also no significant interactions between clinical group and anger induction ($F(1,62) = .076$, p

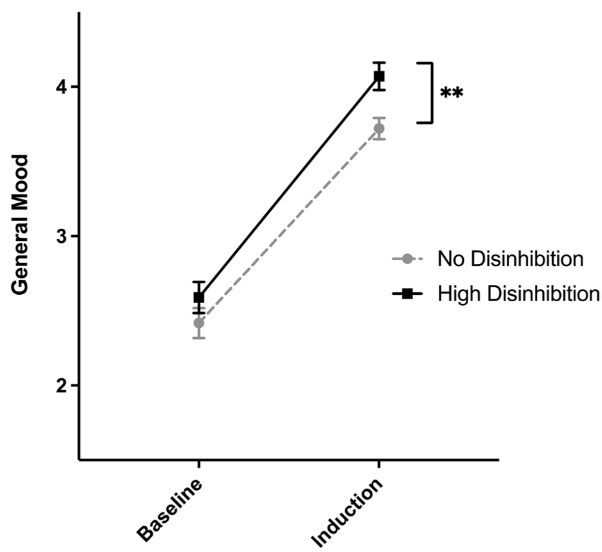

Figure 1. Changes in general mood between baseline and mood induction for those with and without elevated disinhibition. The mood induction produced greater negative mood (larger scores indicate worse mood). Those with clinical disinhibition reported greater negative mood overall compared to those without elevated disinhibition. These effects were consistent across brain injury and control groups. ** $p < .005$.

= .783, η2 = .001), disinhibition and anger induction ($F(1,62) = 3.378$, $p = .071$, η2 = .052), and clinical group and disinhibition ($F(1,62) = .468$, $p = .496$, η2 = .007) on subjective arousal. There was also no significant three-way interaction between clinical group, disinhibition and anger induction on arousal ($F(1,62) = .134$, $p = .715$, η2 = .002).

Self-Reported anger

There was a main effect of anger induction on the self-report of anger ($F(1,61) = 163.310$, $p < .0005$, partial η2 = .727), such that anger provocation induced significant increased subjective experience of anger. There was also a main effect of clinical group ($F(1,62) = 4.177$, $p < .05$, partial η2 = .064), such that those with TBI reported greater subjective anger than controls. Similarly, there was a main effect of disinhibition on the self-report of anger ($F(1,61) = 7.697$, $p < .01$, partial η2 = .112), such that those with clinically elevated disinhibition reported greater symptoms of anger. There were no significant interactions between clinical group and induction ($F(1,61) = 1.000$, $p = .321$, η2 = .016), and clinical group and disinhibition ($F(1,61) = .204$, $p = .653$, η2 = .003) on anger. However, there was a significant interaction between disinhibition and induction on the self-report of anger ($F(1,61) = 8.166$, $p < .001$, η2 = .118). As shown in Figure 3, while those with and without clinically elevated disinhibition reported similar levels of anger at baseline, those clinically elevated in disinhibition showed significantly greater self-report ratings of anger in response to mood induction. There was also no significant three-way interaction between clinical group, disinhibition and anger induction on anger ($F(1,61) = .560$, $p = .457$, η2 = .009).

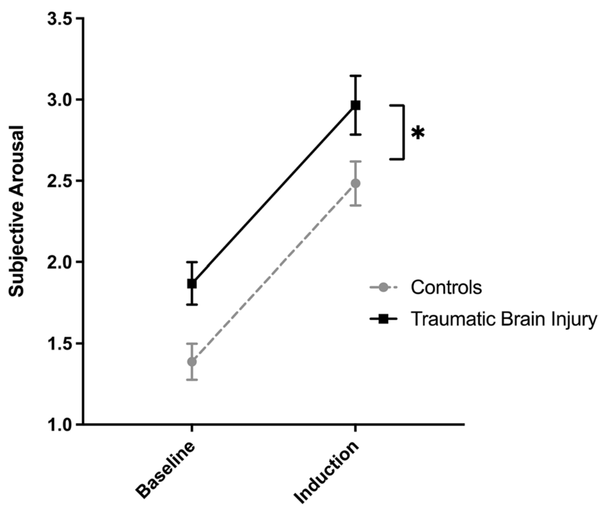

Figure 2. Changes in the self-appraisal of physiological arousal between baseline and mood induction for those with brain injury ($n = 35$) and controls ($n = 31$). The mood induction produced greater subjective experiences of physiological arousal. Those with brain injury reported greater arousal overall compared to controls. There was no effect of disinhibition. ** $p < .05$.

Self-reported tension

There was a main effect of anger induction on the self-report of tension ($F(1,61) = 61.103$, $p < .0005$, partial $\eta 2 = .500$), such that anger provocation induced significant increased subjective experience of tension. There was no main effect of clinical group

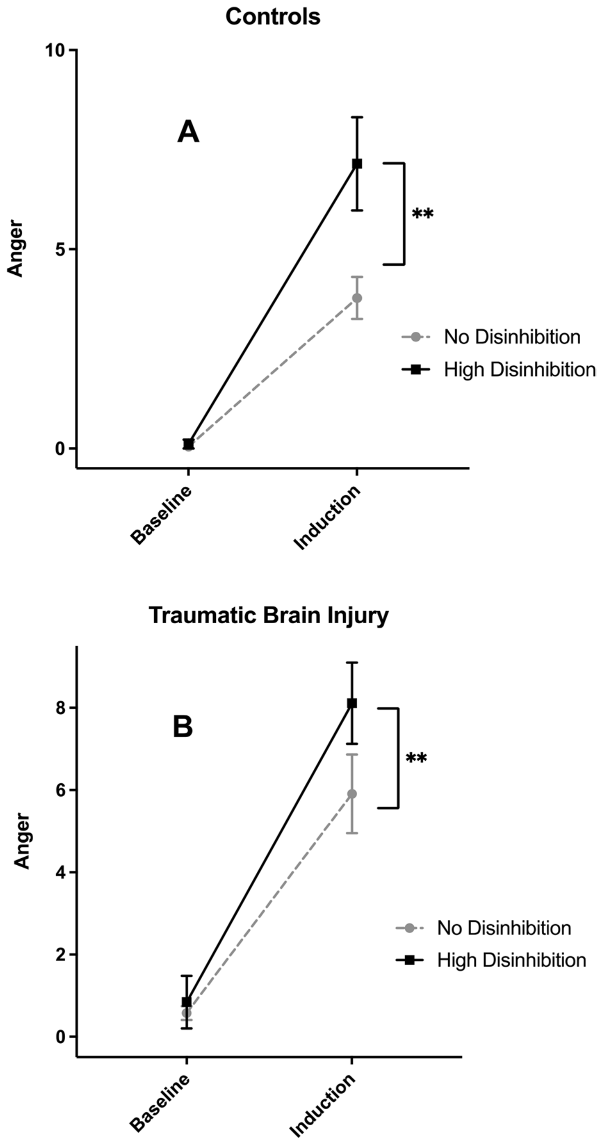

Figure 3. Changes in the self-report of anger between baseline and mood induction for those with elevated disinhibition groups compared those with no disinhibition. Figure 3A depicts the changes for those in the control group ($n = 31$). Figure 3B shows changes for those with severe Traumatic Brain Injury ($n = 35$). The mood induction produced greater feelings of anger overall. However, those with clinically elevated disinhibition reported greater feelings of anger in response to the mood induction procedure compared to those without disinhibition. These results were consistent across TBI and control groups. **p <.001.

(F(1,61) = 1.472, p = .230, partial η2 = .024) but there was a main effect of disinhibition on the self-report of tension (F(1,61) = 15.669, p < .0005, partial η2 = .204), such that those with clinically elevated disinhibition reported greater symptoms of tension. There were no significant interactions between clinical group and induction (F(1,61) = .043, p = .836, η2 = .001), and clinical group and disinhibition (F(1,61) = .564, p = .456, η2 = .009) on tension. However, there was a significant interaction between disinhibition and induction on the self-report of tension (F(1,61) = 6.604, p < .05, η2 = .098). As shown in Figure 4, while those with and without clinically elevated disinhibition reported similar levels of anger at baseline, those clinically elevated in disinhibition showed significantly greater self-report ratings of anger in response to mood induction. There was also no significant three-way interaction between clinical group, disinhibition and anger induction on tension (F(1,61) = .697, p = .407, η2 = .011).

Discussion

The first aim of the current study was to determine whether participants with moderate-to-severe TBI had altered objective and subjective experiences to a mood induction procedure. While the mood induction elicited significant increases in SCL, respiration and self-reported anger, tension, arousal, and general mood, together with decreases in HRV-SDNN—effectively showing an effective manipulation effect—we found no differences between TBI and control groups for any of the objective and subjective measures examined. The second aim was to determine whether disinhibition differentially affected the outcomes to anger provocation. Interestingly, the main findings of this study were that those with clinically elevated disinhibition reported elevated negative affect more generally than those not elevated in disinhibition. Additionally, while disinhibition did not affect physiological responses to the mood induction, it affected emotional subjective experience, such that the feelings of anger and tension in response to the mood induction were particularly pronounced among individuals who were disinhibited. These findings suggest that disinhibition affected subjective appraisal of the mood induction, particularly for negative affect. Importantly, these findings appear to be independent of whether an individual had previously sustained a TBI.

For objective outcomes, we found that the mood induction procedure resulted in significantly increased SCL and respiration across participants, and HRV-SDNN was reduced, while there were no significant changes to other physiological measures. Furthermore, we hypothesised that the TBI group would have significantly reduced SCL and HRV in response to the mood induction procedure relative to controls. Contrary to hypotheses, we failed to find group differences for most physiological measures. While some research has reported group differences in SCL after viewing emotion-eliciting stimuli (e.g. de Sousa et al., 2011, 2012), Rushby et al. (2013a) found no differences. Interestingly, both studies utilised the same emotion inducing films (i.e. *Bodyguard and Cry Freedom*) as the present study. Therefore, no group differences for SCL are consistent with findings from Rushby et al. (2013a). Additionally, two systematic reviews investigating physiological reactivity to emotional stimuli have reported that findings within the literature are mixed, however, there was more

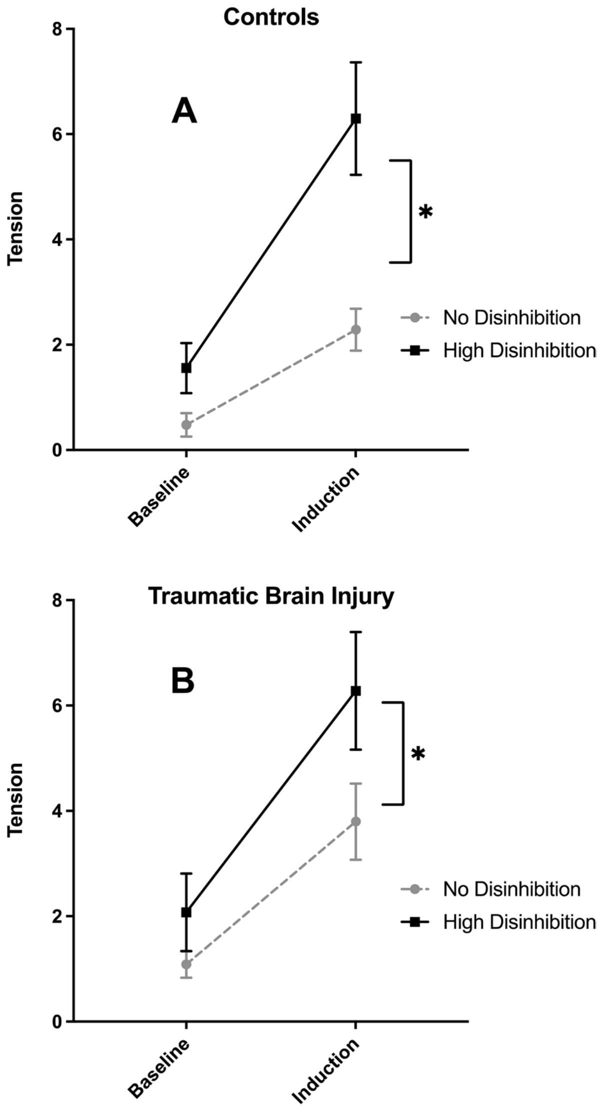

Figure 4. Changes in the self-report of tension between baseline and mood induction for those with elevated disinhibition groups compared those with no disinhibition. Figure 3A depicts the changes for those in the control group ($n = 31$). Figure 3B shows changes for those with severe Traumatic Brain Injury ($n = 35$). The mood induction produced greater feelings of tension overall. However, those with clinically elevated disinhibition reported greater feelings of tension in response to the mood induction procedure compared to those without disinhibition. These results were consistent across TBI and control groups. $^{**}p < .001$.

consistent evidence of reduced physiological reactivity in those with TBIs, particularly when viewing images and videos (Bodart et al., 2023; Sarah et al., 2023). While anger-inducing videos were used in this study, we also included the PASAT to induce stress and anger. Thus, the divergent results may be due to the addition of the cognitive task. Research investigating physiological responses to stressful tasks has been more consistently mixed, potentially due to differences in stress-inducing tasks and individual differences in the perceived threat of the stressor (Sarah et al., 2023).

Additionally, the lack of differences in HRV between groups for objective output is not consistent with findings from Amorapanth et al. (2018). In their sample, Amorapanth et al. (2018) included participants with self-reported cognitive and emotional difficulties. Therefore, it is possible that the emotional difficulties of their sample helped to explain the reduced HRV found in their study. This would also be consistent with findings that reduced HRV is associated with difficulties with emotion regulation (Cattaneo et al., 2021). Whilst almost 50% of our TBI sample met the diagnostic cut-off for elevated disinhibition, the variability in emotion dysregulation may have led to greater heterogeneity in our sample that limited the detection of group-based differences in HRV. Indeed, evaluation of the baseline HRV responses showed that the variability of HRV in the TBI group was almost double that of the control group, suggesting that the TBI group had greater within-group variability on the HRV measures. This greater variability within a TBI group has been demonstrated before (Francis et al., 2016). Overall, these mixed findings are unsurprising given that TBIs are heterogeneous, such that their idiosyncrasies are unlikely to produce a homogeneous physiological signature when viewing emotion inducing stimuli. Therefore, it is plausible to expect different findings between studies.

For subjective emotional experience, we found that the mood induction procedure resulted in significantly increased self-reported negative affect (e.g. general mood, anger, tension) and increased ratings of arousal. These findings suggest that the mood induction effectively elicited negative subjective feelings of emotion. In regard to group differences, it was hypothesised that the TBI group would have significantly lower self-reported arousal and reduced negative mood, and there would be no difference for anger and tension ratings in response to the mood induction relative to controls. Contrary to hypotheses, we did not find any TBI differences for subjective arousal and general mood in response to the mood induction. These findings were unexpected since de Sousa et al. (2012) found reduced arousal and reduced negative emotional response to unpleasant films in those with TBI compared to controls. The authors proposed that those with TBI have a reduced emotion recognition and therefore have reduced empathy and reactivity to emotional stimuli. However, our results are more consistent with independent findings that individuals with TBI are able to identify and differentiate the emotional intensity of various emotional stimuli despite having difficulty with emotion recognition (Wearne et al., 2020). Consistent with this latter observation, with intact ability to determine the emotional intensity of the scenes depicted in the films, participants with TBI could have used this information to produce responses when asked to rate how arousing or unpleasant the stimuli were for them. Although this is speculative, past research has found that participants with TBI exhibit a liberal bias when reporting emotional intensity of stimuli (Callahan et al., 2011). Alternatively, it is plausible that this sample with an average 13-years post-injury have developed feedback mechanisms to bring their subjective feelings of arousal to homeostasis. For example, being long-term post-injury they may have learnt to accurately attribute their personal experiences to physiology. As predicted, the mood induction did not result in any group differences for self-rated anger and tension. These findings are consistent with findings from McDonald et al. (2010). These results showed that both groups reported heightened feelings, arousal and negative mood to a similar degree.

Interestingly, we found that disinhibition significantly interacted with the exposure to anger provocation. However, this was specific to the subjective, but not the objective, outcomes of the study. That is, while negative mood increased following the anger induction for all participants, those with clinically elevated disinhibition showed greater self-reported anger and tension compared to those not elevated in disinhibition. Importantly, these findings were independent to changes in physiology, suggesting that disinhibition significantly affected the *interpretation* of the mood induction procedure, rather than affecting the response itself. Thus, our hypotheses of increased subjective emotional reactivity for those elevated in disinhibition was supported. These findings are aligned with previous research which has found that impaired control affects both emotional and cognitive processes (McDonald et al., 2010). Importantly, and unexpectedly, the relationship between high disinhibition and increased subjective emotional reactivity did not change as a function of having a TBI. This suggests that it is not necessarily having a TBI that impacts negative emotional appraisal but rather if someone has elevated disinhibition (Filipčíková et al., 2022). Given the lack of moderation between disinhibition and physiology, these findings suggest that disinhibition interacts specifically with the negative appraisal of emotion inducing stimuli.

These findings have several important clinical implications. First, the fact that disinhibition affected the negative appraisal of emotional material irrespective of TBI status suggests that baseline disinhibition may serve as an important prognostic factor in determining those who may be susceptible to emotion dysregulation following brain injury. It would therefore be important to assess disinhibition (both historical and current) when assessing emotional and behavioural outcomes after TBI. Further, since disinhibition was found to increase subjective reactivity, disinhibition may be an important component of emotion regulation interventions to consider after TBI, and more generally. For example, a study investigating disinhibition and emotion regulation in boys with ADHD found that those who were able to inhibit emotional responses when requested were more effective with emotion regulation than those with high disinhibition (Walcott & Landau, 2004). Therefore, improving inhibitory control could potentially ameliorate emotion dysregulation after TBI. Alternatively, given that these findings suggest that disinhibition led to greater interpretation biases in response to emotional provocation, cognitive and emotional reappraisal may be an effective strategy for those with elevated disinhibition scores, particularly for those following TBI.

There are also several limitations of this study that should be discussed. First, many of this sample are quite long-time post-injury, and therefore results could be different in the acute stages of recovery whereby individuals may be struggling to adjust to their new identity, ways of processing information, and how they respond emotionally and behaviourally to environmental cues. Regardless of severity, individuals can make functional improvements in the months and years following a TBI (Rosenbaum et al., 2018). Therefore, investigating the link between emotional reactivity and disinhibition in the acute stages of recovery may provide a different perspective, and is therefore a recommendation for future research. Second, the sample size is relatively small and may have lacked statistical power to detect other meaningful results. While we found no significant differences between those in the TBI and control groups, several of these outcomes were of small to medium effect size (i.e.

np^2s of 0.01 to 0.031). This suggests that our study was powerful enough to statistically identify differences that were of medium to large effect size. While smaller effect sizes raise questions about clinical usefulness, it may be that further results would have been identified with a larger sample. As such, these findings can be best described as exploratory and preliminary given these sample size concerns. Thirdly, while this study focused on disinhibition as a measure moderating reactivity to emotional stimuli, it is possible other factors could have affected these outcomes, such as attentional control. For example, it has been suggested that low attentional control - which is related to high disinhibition - has the potential to heightened emotions due to the inability to shift focus away from the threat (Wood & Worthington, 2017). Therefore, attentional control could also be a moderator in the relationship between elevated disinhibition and subjective emotional reactivity and should be explored in future research.

In conclusion, this study explored the relationship between disinhibition and severe TBI with respect to physiological and subjective responses to emotional provocation. We found that disinhibition moderated the impact of the anger provocation on subjective, but not objective, emotional experiences. This suggests that disinhibition, rather than TBI, may impact the negative appraisal of emotional events. As such, it cannot be assumed that having a severe TBI leads to emotional dysregulation, but rather it appears the degree of disinhibition may alter subjective interpretations of emotional events which could potentially lead to increased behavioural outbursts. This study therefore highlights the importance of interpretation and appraisal in the evaluation of emotional experiences, which may be important targets in the treatment of dysregulation for those with and without TBI.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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References

Aboulafia-Brakha, T., Allain, P., & Ptak, R. (2016). Emotion regulation after traumatic brain injury: Distinct patterns of sympathetic activity during anger expression and recognition. *The Journal of Head Trauma Rehabilitation*, *31*(3), E21–E31. https://doi.org/10.1097/HTR.0000000000000171

Amorapanth, P. X., Aluru, V., Stone, J., Yousefi, A., Tang, A., Cox, S., Bilaloglu, S., Lu, Y., Rath, J., Long, C., Im, B., & Raghavan, P. (2018). Traumatic brain injury results in altered physiologic, but not subjective responses to emotional stimuli. *Brain Injury*, *32*(13-14), 1712–1719. https://doi.org/10.1080/02699052.2018.1519598

Appelhans, B. M., & Luecken, L. J. (2006). Heart rate variability as an index of regulated emotional responding. *Review of General Psychology*, *10*(3), 229–240. https://doi.org/10.1037/1089-2680.10.3.229

Ashman, T. A., Spielman, L. A., Hibbard, M. R., Silver, J. M., Chandna, T., & Gordon, W. A. (2004). Psychiatric challenges in the first 6 years after traumatic brain injury: Cross-sequential analyses of Axis I disorders. *Archives of Physical Medicine and Rehabilitation*, *85*(4 Suppl 2), S36–S42. https://doi.org/10.1016/j.apmr.2003.08.117

Baguley, I. J., Heriseanu, R. E., Felmingham, K. L., & Cameron, I. D. (2006). Dysautonomia and heart rate variability following severe traumatic brain injury. *Brain Injury*, *20*(4), 437–444. https://doi.org/10.1080/02699050600664715

Berntson, G. G., Bigger, J. T., Jr, Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M., Nagaraja, H. N., Porges, S. W., Saul, J. P., Stone, P. H., & van der Molen, M. W. (1997). Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology*, *34*(6), 623–648. https://doi.org/10.1111/j.1469-8986.1997.tb02140.x

Bodart, A., Invernizzi, S., Lefebvre, L., & Rossignol, M. (2023). Physiological reactivity at rest and in response to social or emotional stimuli after a traumatic brain injury: A systematic review. *Frontiers in Psychology*, *14*, 930177. https://doi.org/10.3389/fpsyg.2023.930177

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences*, *4*(6), 215–222. https://doi.org/10.1016/S1364-6613(00)01483-2

Callahan, B. L., Ueda, K., Sakata, D., Plamondon, A., & Murai, T. (2011). Liberal bias mediates emotion recognition deficits in frontal traumatic brain injury. *Brain and Cognition*, *77*(3), 412–418. https://doi.org/10.1016/j.bandc.2011.08.017

Carroll, L. J., Cassidy, J. D., Peloso, P. M., Borg, J., von Holst, H., Holm, L., Paniak, C., & Pépin, M, WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. (2004). Prognosis for mild traumatic brain injury: Results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine*, *36*(43 Suppl), 84–105. https://doi.org/10.1080/16501960410023859

Cattaneo, L. A., Franquillo, A. C., Grecucci, A., Beccia, L., Caretti, V., & Dadomo, H. (2021). Is low heart rate variability associated with emotional dysregulation, Psychopathological dimensions, and prefrontal dysfunctions? An integrative view. *Journal of Personalized Medicine*, *11*(9), 872. https://doi.org/10.3390/jpm11090872

Ciurli, P., Formisano, R., Bivona, U., Cantagallo, A., & Angelelli, P. (2011). Neuropsychiatric disorders in persons with severe traumatic brain injury: Prevalence, phenomenology, and relationship with demographic, clinical, and functional features. *The Journal of Head Trauma Rehabilitation*, *26*(2), 116–126. https://doi.org/10.1097/HTR.0b013e3181dedd0e

Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the neural circuitry of emotion regulation–A possible prelude to violence. *Science (New York, N.Y.)*, *289*(5479), 591–594. https://doi.org/10.1126/science.289.5479.591

de Sousa, A., McDonald, S., & Rushby, J. (2012). Changes in emotional empathy, affective responsivity and behaviour following severe traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, *34*(6), 606–623. https://doi.org/10.1080/13803395.2012.667067

de Sousa, A., McDonald, S., Rushby, J., Li, S., Dimoska, A., & James, C. (2011). Understanding deficits in empathy after traumatic brain injury: The role of affective responsivity. *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior*, *47*(5), 526–535. https://doi.org/10.1016/j.cortex.2010.02.004

Dewan, M. C., Rattani, A., Gupta, S., Baticulon, R. E., Hung, Y.-C., Punchak, M., Agrawal, A., Adeleye, A. O., Shrime, M. G., Rubiano, A. M., Rosenfeld, J. V., & Park, K. B. (2019). Estimating the global incidence of traumatic brain injury. *Journal of Neurosurgery*, *130*(4), 1080–1097. https://doi.org/10.3171/2017.10.JNS17352

Field, A. (2013). *Discovering statistics using IBM SPSS statistics* (4th ed.). Sage.

Filipčíková, M., Wearne, T., & McDonald, S. (2022). Disinhibited and angry: Investigating the relationship between social disinhibition and the components of aggression following severe TBI. *Neuropsychological Rehabilitation*, *34*(1), 23–44. Advance online publication. https://doi.org/10.1080/09602011.2022.2149560

Filipčíková, M., Wearne, T., Li, R., & McDonald, S. (2021). The prevalence, predictors, associated symptoms, and outcomes of social disinhibition following moderate-to-severe TBI: A scoping review of quantitative evidence. *Journal of Clinical and Experimental Neuropsychology*, *43*(7), 716–736. https://doi.org/10.1080/13803395.2021.2000589

Fisher, A. C., Rushby, J. A., McDonald, S., Parks, N., & Piguet, O. (2015). Neurophysiological correlates of dysregulated emotional arousal in severe traumatic brain injury. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology*, *126*(2), 314–324. https://doi.org/10.1016/j.clinph.2014.05.033

Francis, H., Fisher, A., Rushby, J. A., & McDonald, S. (2016). Reduced heart rate variability in chronic severe traumatic brain injury: Association with impaired emotional and social functioning, and potential for treatment using biofeedback. *Neuropsychological Rehabilitation*, *26*(1), 103–125. https://doi.org/10.1080/09602011.2014.1003246

Grace, J., Stout, J. C., & Malloy, P. F. (1999). Assessing frontal lobe behavioral syndromes with the frontal lobe personality scale. *Assessment*, *6*(3), 269–284. https://doi.org/10.1177/107319119900600307

Gross, J. J., & Levenson, R. W. (1995). Emotion elicitation using films. *Cognition & Emotion*, *9*(1), 87–108. https://doi.org/10.1080/02699939508408966

Hansen, A. L., Johnsen, B. H., & Thayer, J. F. (2003). Vagal influence on working memory and attention. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, *48*(3), 263–274. https://doi.org/10.1016/S0167-8760(03)00073-4

Hourani, L. L., Davila, M. I., Morgan, J., Meleth, S., Ramirez, D., Lewis, G., Kizakevich, P. N., Eckhoff, R., Morgan, T., Strange, L., Lane, M., Weimer, B., & Lewis, A. (2020). Mental health, stress, and resilience correlates of heart rate variability among military reservists, guardsmen, and first responders. *Physiology & Behavior*, *214*, 112734. https://doi.org/10.1016/j.physbeh.2019.112734

Kelly, M., McDonald, S., & Rushby, J. (2017). Ostracism and physiological arousal following traumatic brain injury. *Brain Injury*, *31*(4), 550–559. https://doi.org/10.1080/02699052.2016.1271457

Krpan, K. M., Stuss, D. T., & Anderson, N. D. (2011). Planful versus avoidant coping: Behavior of individuals with moderate-to-severe traumatic brain injury during a psychosocial stress test. *Journal of the International Neuropsychological Society: JINS*, *17*(2), 248–255. https://doi.org/10.1017/S1355617710001499

Lakens, D. (2013). Calculating and reporting effect sizes to facilitate cumulative science: A practical primer for t-tests and ANOVAS. *Frontiers in Psychology*, *4*(4), 863. https://doi.org/10.3389/fpsyg.2013.00863

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1997). *International Affective Picture System (IAPS): Technical manual and affective ratings* (pp. 39–58). NIMH Center for the Study of Emotion and Attention.

Lemke, D. M. (2004). Riding out the storm: Sympathetic storming after traumatic brain injury. *The Journal of Neuroscience Nursing: Journal of the American Association of Neuroscience Nurses*, *36*(1), 4–9. https://doi.org/10.1097/01376517-200402000-00002

Levin, H., & Kraus, M. F. (1994). The frontal lobes and traumatic brain injury. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *6*(4), 443–454. https://doi.org/10.1176/jnp.6.4.443

Linehan, M. (2015). *DBT skills training manual* (2nd ed.). The Guilford Press.

Malec, J. F., Brown, A. W., Leibson, C. L., Flaada, J. T., Mandrekar, J. N., Diehl, N. N., & Perkins, P. K. (2007). The mayo classification system for traumatic brain injury severity. *Journal of Neurotrauma*, *24*(9), 1417–1424. https://doi.org/10.1089/neu.2006.0245

Malik, M. (1996). Heart rate variability: Standards of measurement, physiological interpretation and clinical use. Task force of the European Society of Cardiology the North American Society of Pacing Electrophysiology. *Circulation*, *93*, 1043–1065.

Masi, C. M., Hawkley, L. C., Rickett, E. M., & Cacioppo, J. T. (2007). Respiratory sinus arrhythmia and diseases of aging: Obesity, diabetes mellitus, and hypertension. *Biological Psychology*, *74*(2), 212–223. https://doi.org/10.1016/j.biopsycho.2006.07.006

McDonald, S., Hunt, C., Henry, J. D., Dimoska, A., & Bornhofen, C. (2010). Angry responses to emotional events: The role of impaired control and drive in people with severe traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, *32*(8), 855–864. https://doi.org/10.1080/13803391003596405

Nash, R. P., Weinberg, M. S., Laughon, S. L., McCall, R. C., Bateman, J. R., & Rosenstein, D. L. (2019). Acute pharmacological management of behavioral and emotional dysregulation following a traumatic brain injury: A systematic review of the literature. *Psychosomatics*, *60*(2), 139–152. https://doi.org/10.1016/j.psym.2018.11.009

Niemeier, J. P., Perrin, P. B., Holcomb, M. G., Nersessova, K. S., & Rolston, C. D. (2013). Factor structure, reliability, and validity of the Frontal Systems Behavior Scale (FrSBe) in an acute traumatic brain injury population. *Rehabilitation Psychology*, *58*(1), 51–63. https://doi.org/10.1037/a0031612

Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology: Views from cognitive and personality psychology and a working inhibition taxonomy. *Psychological Bulletin*, *126*(2), 220–246. https://doi.org/10.1037/0033-2909.126.2.220

Osborne-Crowley, K., & McDonald, S. (2018). A review of social disinhibition after traumatic brain injury. *Journal of Neuropsychology*, *12*(2), 176–199. https://doi.org/10.1111/jnp.12113

Osborne-Crowley, K., Wilson, E., De Blasio, F., Wearne, T., Rushby, J., & McDonald, S. (2020). Empathy for people with similar experiences: Can the perception-action model explain empathy impairments after traumatic brain injury? *Journal of Clinical and Experimental Neuropsychology*, *42*(1), 28–41. https://doi.org/10.1080/13803395.2019.1662375

Ponsford, J., Trevena-Peters, J., Janzen, S., Harnett, A., Marshall, S., Patsakos, E., Kua, A., McIntyre, A., Teasell, R., Wiseman-Hakes, C., Velikonja, D., Bayley, M. T., & McKay, A, INCOG Expert Panel. (2023). INCOG 2.0 guidelines for cognitive rehabilitation following traumatic brain injury, part I: Posttraumatic amnesia. *The Journal of Head Trauma Rehabilitation*, *38*(1), 24–37. https://doi.org/10.1097/HTR.0000000000000840

Qadeer, A., Khalid, U., Amin, M., Murtaza, S., Khaliq, M. F., & Shoaib, M. (2017). Caregiver's burden of the patients with traumatic brain injury. *Cureus*, *9*(8), e1590. https://doi.org/10.7759/cureus.1590

Riganello, F., Candelieri, A., Quintieri, M., Conforti, D., & Dolce, G. (2010). Heart rate variability: An index of brain processing in vegetative state? An artificial intelligence, data mining study. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology*, *121*(12), 2024–2034. https://doi.org/10.1016/j.clinph.2010.05.010

Rosenbaum, A. M., Gordon, W. A., Joannou, A., & Berman, B. A. (2018). Functional outcomes following post-acute rehabilitation for moderate-to-severe traumatic brain injury. *Brain Injury*, *32*(7), 907–914. https://doi.org/10.1080/02699052.2018.1469040

Rushby, J. A., Fisher, A. C., McDonald, S., Murphy, A., & Finnigan, S. (2013a). Autonomic and neural correlates of dysregulated arousal in severe traumatic brain injury. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, *89*(3), 460–465. https://doi.org/10.1016/j.ijpsycho.2013.05.009

Rushby, J. A., McDonald, S., Fisher, A. C., Kornfeld, E. J., De Blasio, F. M., Parks, N., & Piguet, O. (2016). Brain volume loss contributes to arousal and empathy dysregulation following severe traumatic brain injury. *NeuroImage. Clinical*, *12*, 607–614. https://doi.org/10.1016/j.nicl.2016.09.017

Rushby, J. A., McDonald, S., Randall, R., de Sousa, A., Trimmer, E., & Fisher, A. (2013b). Impaired emotional contagion following severe traumatic brain injury. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, *89*(3), 466–474. https://doi.org/10.1016/j.ijpsycho.2013.06.013

Sánchez-Navarro, J. P., Martínez-Selva, J. M., & Román, F. (2005). Emotional response in patients with frontal brain damage: Effects of affective valence and information content. *Behavioral Neuroscience*, *119*(1), 87–97. https://doi.org/10.1037/0735-7044.119.1.87

Sarah, A., Ownsworth, T., Clough, B., & Neumann, D. L. (2023). Impairments in physiological reactivity to emotive stimuli after traumatic brain injury: A systematic review of skin conductance and heart rate variability evidence. *The Journal of Head Trauma Rehabilitation*, *38*(3), 214–230. https://doi.org/10.1097/HTR.0000000000000801

Stout, J. C., Ready, R. E., Grace, J., Malloy, P. F., & Paulsen, J. S. (2003). Factor analysis of the frontal systems behavior scale (FrSBe). *Assessment*, *10*(1), 79–85. https://doi.org/10.1177/1073191102250339

Te Ao, B., Brown, P., Tobias, M., Ameratunga, S., Barker-Collo, S., Theadom, A., McPherson, K., Starkey, N., Dowell, A., Jones, K., & Feigin, V. L, BIONIC Study Group. (2014). Cost of traumatic brain injury in New Zealand: Evidence from a population-based study. *Neurology*, *83*(18), 1645–1652. https://doi.org/10.1212/WNL.0000000000000933

Terry, P. C., Lane, A. M., & Fogarty, G. J. (2003). Construct validity of the profile of mood states–adolescents for use with adults. *Psychology of Sport and Exercise*, *4*(2), 125–139. https://doi.org/10.1016/S1469-0292(01)00035-8

Terry, P. C., Lane, A. M., Lane, H. J., & Keohane, L. (1999). Development and validation of a mood measure for adolescents. *Journal of Sports Sciences*, *17*(11), 861–872. https://doi.org/10.1080/026404199365425

Thayer, J. F., & Brosschot, J. F. (2005). Psychosomatics and psychopathology: Looking up and down from the brain. *Psychoneuroendocrinology*, *30*(10), 1050–1058. https://doi.org/10.1016/j.psyneuen.2005.04.014

Velligan, D. I., Ritch, J. L., Sui, D., DiCocco, M., & Huntzinger, C. D. (2002). Frontal systems behavior scale in schizophrenia: Relationships with psychiatric symptomatology, cognition and adaptive function. *Psychiatry Research*, *113*(3), 227–236. https://doi.org/10.1016/S0165-1781(02)00264-0

Vistisen, S. T., Hansen, T. K., Jensen, J., Nielsen, J. F., & Fleischer, J. (2014). Heart rate variability in neurorehabilitation patients with severe acquired brain injury. *Brain Injury*, *28*(2), 196–202. https://doi.org/10.3109/02699052.2013.860477

Walcott, C. M., & Landau, S. (2004). The relation between disinhibition and emotion regulation in boys with attention deficit hyperactivity disorder. *Journal of Clinical Child and Adolescent Psychology: The Official Journal for the Society of Clinical Child and Adolescent Psychology, American Psychological Association, Division 53*, *33*(4), 772–782. https://doi.org/10.1207/s15374424jccp3304_12

Wearne, T. A., Logan, J. A., Trimmer, E. M., Wilson, E., Filipcikova, M., Kornfeld, E., Rushby, J. A., & McDonald, S. (2021). Regulating emotion following severe traumatic brain injury: A randomized controlled trial of heart-rate variability biofeedback training. *Brain Injury*, *35*(11), 1390–1401. https://doi.org/10.1080/02699052.2021.1972337

Wearne, T. A., Osborne-Crowley, K., Logan, J. A., Wilson, E., Rushby, J., & McDonald, S. (2020). Understanding how others feel: Evaluating the relationship between empathy and various aspects of emotion recognition following severe traumatic brain injury. *Neuropsychology*, *34*(3), 288–297. https://doi.org/10.1037/neu0000609

Winter, L., Moriarty, H. J., & Short, T. H. (2018). Beyond anger: Emotion regulation and social connectedness in veterans with traumatic brain injury. *Brain Injury*, *32*(5), 593–599. https://doi.org/10.1080/02699052.2018.1432895

Wood, R. L., & Worthington, A. (2017). Neurobehavioral abnormalities associated with executive dysfunction after traumatic brain injury. *Frontiers in Behavioral Neuroscience*, *11*, 195. https://doi.org/10.3389/fnbeh.2017.00195

Zheng, R. Z., Lei, Z. Q., Yang, R. Z., Huang, G. H., & Zhang, G. M. (2020). Identification and management of paroxysmal sympathetic hyperactivity after traumatic brain injury. *Frontiers in Neurology*, *11*, 81. https://doi.org/10.3389/fneur.2020.00081