Investigating Difficulties in Emotion Regulation in Medically Unexplained Symptoms

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List of contents

Declaration

This work has not been submitted to any other institution or for any other qualification.

Thesis abstract

The thesis comprises a literature review and a research report. The review provides a critical evaluation and summary of literature pertaining to associations between emotion dysregulation and medically unexplained symptoms (MUS). Owing to ambiguities in the conceptualisation of emotion dysregulation, the way in which emotion dysregulation is being conceptualised in the MUS literature (e.g. which strategies are being investigated in the disorder) was investigated followed by an evaluation of the associations between difficulties in these emotion regulation strategies and MUS. The researcher concludes that further research is needed to improve our understanding of emotion dysregulation in MUS.

The research report investigated emotion dysregulation in psychogenic nonepileptic seizures (PNES). The aetiology of PNES is not well understood, research suggests that the aetiology involves a complex interplay of factors. Recently, high levels of emotion dysregulation have been reported in PNES. In addition, high rates of traumatic experiences have been reported in the disorder. The researcher hypothesised that high levels of emotion dysregulation may be associated with traumatic experiences in PNES. High levels of emotion dysregulation were reported in both participants with PNES and participants with epilepsy but not in healthy controls. Higher levels of traumatic experiences were reported by the participants with PNES in comparison with participants with epilepsy and healthy controls. The researcher's hypothesis was not supported; traumatic experiences did not account for the variance in emotion dysregulation, only anxiety accounted for this variance. The results are considered in relation to previous research and implications for practice and future research outlined.

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Section 1

Literature review

Is there an Association between Emotion Dysregulation and Medically Unexplained Symptoms? A Systematic Review of the Literature

Abstract

Objectives. Emotion dysregulation is manifested in many psychological disorders which have high co-morbidity with medically unexplained symptoms (MUS). Emotion dysregulation has been poorly defined in the literature; further conceptualisation of this is warranted. Whilst the relationship between alexithymia and MUS has been investigated extensively, less attention has been paid to the relationship between emotion dysregulation and MUS. The aim of this systematic literature review was to consider how emotion dysregulation is being conceptualised in MUS and to investigate associations between emotion dysregulation and MUS.

Methods. A systematic search of relevant databases (PsycINFO, CINAHL, Medline, and Web of Knowledge) was conducted using the following search terms: Emotion*, affect, regulation, dysregulation, emotion avoidance, re-appraisal of emotion, impulse control, emotion suppression, medically unexplained sympt*, unexplained medical sympt*, non-organ*, somatisation, somatization, psychophysiological, psychosomatic, psychogenic, hypochondria*, somatoform disorder*, functional disorder*, and conversion disorder*. The identified studies were critically evaluated and grouped into the emotion regulation strategies investigated.

Results. Eleven studies were identified. Emotion dysregulation was being conceptualised as difficulties in acceptance of emotions, suppression of emotions, avoidance of emotions, difficulty in dealing with negative emotions, difficulties in impulse control, and difficulties in engaging in goal directed behaviour. Difficulties in most emotion regulation strategies were found to be higher in MUS than in healthy controls but not higher than in other psychiatric disorders.

Conclusions. Whilst emotion dysregulation as a composite measure is associated with MUS, when specific emotion regulation strategies are investigated, the picture is mixed. Further research is needed taking into account the limitations of the existing research.

Practitioner points

Clinical implications

- In assessment, difficulties in specific emotion regulation strategies need to be investigated,
- The interaction between emotion dysregulation and alexithymia may be a target for therapy in MUS,
- The reasons behind emotion regulation strategies adopted should be considered, particularly regarding the cultural influences on this.

Limitations

- The review grouped together a number of MUS disorders which may not have been comparable,
- The majority of studies utilised self-report measures of emotion dysregulation, subject to bias (e.g. recall bias, social desirability bias),
- All studies were cross-sectional meaning that causality could not be inferred.

Introduction

Interest in emotion regulation has a long history dating back to Freud's studies of psychological defenses (Freud, 1926, pp.75-174) and research in the theoretical fields of stress and coping (Lazarus, 1966), attachment (Bowlby, 1969), and self-regulation (Mischel, Shoda, & Rodriguez, 1989). More recently, the field of emotion regulation has come into its own and the concept has received increased attention (Gross, 1998b).

Defining emotion regulation

Two researcher's definitions of emotion regulation are dominant in the literature; Thompson (1994) and Gross (1998b). Thompson (1994, pp. 27-28) proposed that "emotion regulation consists of intrinsic and extrinsic processes responsible for the monitoring, evaluating, and modifying of emotional reactions to accomplish one's goals". Gross (1998b, p.275) defined emotion regulation as "the process by which individuals influence which emotions they have, when they have them, and how they experience and express them".

Emotion regulation is therefore a complex process that involves initiating, inhibiting and modulating one's emotions in a given situation. Adaptive emotion regulation is considered to be necessary for daily functioning (Freud, 1961; Gross & Munoz, 1995). In the face of situations in which one would expect to experience felt emotion, aspects of emotion regulation can keep the individual within a 'window of tolerance'. In this 'window of tolerance' emotion can be processed without disrupting daily functioning; the 'window of tolerance' is where optimal social functioning is possible (Schore, 2003). Rigidity in, and over-reliance of emotion regulation strategies can result in emotions being experienced outside of this window of tolerance; functioning can then become impaired. If emotions are

under-regulated, they can be experienced as intense and overwhelming (termed hyperarousal); if they are over-regulated, emotions are suppressed and numbed (termed hypoarousal). Difficulties in adopting emotion regulation strategies to stay within the emotion 'window of tolerance' are defined as emotion dysregulation. Emotion dysregulation can disrupt daily functioning and manifest itself in a number of psychological disorders (Beauchaine, Gatzke-Kopp, & Mead, 2007; Gross 1998b).

Researchers have highlighted ambiguity in the conceptualisation of emotion dysregulation; firstly in the definitions of emotion dysregulation, and secondly in its association with alexithymia other emotion processing concepts (Cole, Martin, & Dennis, 2004). Definitional challenges in emotion dysregulation have been discussed in the literature, particularly with regards to the issue of various definitions of emotion regulation being used in research resulting in difficulties in comparing study findings (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Cole, Martin, & Dennis, 2004). As well as ambiguity in the definition of emotion dysregulation, there is also ambiguity in the differentiation between emotion dysregulation and other emotion related concepts, including alexithymia. When performing literature searches for emotion dysregulation in MUS, a large proportion of the literature relates to alexithymia. Alexithymia is a personality construct characterised by the inability to understand and express emotions (Sifneos, 1973). Whilst there is some overlap between the constructs of emotion dysregulation and alexithymia, they are largely considered to be distinct constructs representing independent domains of behaviour. For instance, it has been suggested that the ability to control impulsive behaviour may be specific to emotion dysregulation, whilst understanding emotions may be considered a component of both alexithymia and emotion dysregulation. (Pandey, Saxena, & Dubey, 2011). In a recent study using factor analysis, researchers found support for the notion that alexithymia and emotion dysregulation are independent constructs with minimum overlap (Pandey, Saxena, & Dubey,

2011). These findings highlight the need to clarify conceptualisations of emotion dysregulation in research and in clinical work. The introduction of specific measures of emotion dysregulation is enabling researchers and clinicians to move towards this.

Measuring emotion dysregulation.

In a review of measures of emotional responding, Sloan and Kring (2007) outlined the two most commonly used self-report measures of emotion dysregulation; The Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) and the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). The ERQ was designed to assess individual differences in the use of two emotion regulation strategies: cognitive reappraisal and expressive suppression. The DERS was based on several facets of emotion dysregulation. Six aspects of emotion dysregulation are measured: non-acceptance of emotional responses, difficulties in engaging in goal-directed behaviour, impulse control difficulties, lack of emotional awareness, limited access to emotion regulation strategies, and lack of emotional clarity.

Emotion dysregulation in psychological disorders.

Given increased research interest and the development of standardised measures, emotion dysregulation has been investigated in a number of psychological disorders. Emotion dysregulation has been associated with depression (Joorman & Gotlib, 2010), anxiety (Coan & Allen, 2004), post-traumatic stress disorder (PTSD; Boden et al., 2013; Wisco, Sloan, & Marx, 2013) and borderline personality disorder (BPD; Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2006; Gunderson, 2001). More recently, emotion dysregulation in medically unexplained symptoms (MUS) has been investigated.

Medically Unexplained Symptoms

The term MUS is used to capture patients with somatisation disorders, functional disorders, and psychosomatic disorders. MUS have been defined as "physical symptoms that prompt the sufferer to seek health care but remain unexplained after an appropriate medical evaluation" (Richardson & Engel, 2004). Whilst MUS may mimic physical disorders, when investigated, there is no conventional medical explanation for the symptoms; rather MUS are widely considered to be psychological in nature. In the MUS literature, the term is used to refer to both specific symptoms occurring in the absence of obvious pathology, and specific MUS syndromes.

Chronic fatigue syndrome (CFS) and fibromyalgia, and a number of functional neurological syndromes such as psychogenic nonepileptic seizures (PNES), are among a growing number of specific MUS syndromes (Clauw & Chrousos, 1997; Reuber, House, Pukrop, Bauer, & Elger, 2003; Wessely & Hotopf, 1999). Whilst some MUS are considered to be manifestations of psychological distress or perceptual or attentional abnormalities, the organic basis of these disorders cannot be ruled out. Some researchers consider that disorders classified as medically unexplained are unexplained purely due to limits in medical knowledge and available technology (Kirmayer, Groleau, Looper, & Dao, 2004). The reader should be mindful of this possibility throughout.

The aetiology of MUS.

There are a number of explanatory models of MUS. Whilst all have made contributions to the understanding of MUS, researchers have suggested that none have provided an adequate account, and further research is needed to improve our understanding of the aetiology of

these symptoms (Brown, 2004). Research has identified a number of risk factors for MUS including childhood or family illness (Hotopf, Mayou, Wadsworth, & Wessely, 1999), family stress (Moore, Baker, McDade, Chadwick, & Brown, 1994), and abuse history (Fry, Crisp & Beard, 1997). A number of psychological processes related to MUS have also been identified. This has included neuroticism (De Gucht & Heiser, 2003), alexithymia (Kooiman, 1998), and emotion dysregulation.

Difficulties in emotion regulation have been recognised as a mechanism or cause of MUS in a number of theoretical models including psychodynamic, cognitive and developmental theories (Taylor, Bagby and Parker, 1997; Waller & Scheidt, 2006). In psychodynamic theories of MUS, symptoms are viewed as a result of disturbances in the conscious regulation of emotions, emerging as a consequence of traumatic experiences (Freud & Breuer, 1991; Krystal, 1997). Cognitive theories suggest that individuals who have difficulties in regulating stress related emotions at the cognitive level may result in exaggerated physiological responses to stressful situations (Martin and Pihl, 1985, 1986). Developmental theories of MUS focus on the issue of emotion regulation and how it relates to attachment theory. The theory postulates that dismissing attachment styles result in children not developing emotion regulation strategies; difficult feelings are therefore expressed somatically as children are unable to express and regulate the emotions in other ways.

More recently, Brown (2004) proposed integrative conceptual model of medically unexplained symptoms combining existing theoretical approaches within a single explanatory framework. Within this model, emotion dysregulation is proposed as a predisposing factor in the development of MUS. The theory suggests that those who have difficulties in regulating their emotions are more likely to experience somatic symptoms in situations in which one would expect to feel emotions. Given difficulties in identifying and dealing with emotions, these symptoms would be more likely to be interpreted as illness.

In a review of the understanding and treatment of MUS, Burton (2003) suggested that some factors investigated have been too broad (e.g. neuroticism) and some too restricted (e.g. alexithymia) to be useful in a heterogeneous MUS population. This, in part, formed the rationale for this review.

Rationale for the systematic literature review

Patients with MUS are frequent health care attendees and the health care systems experience high costs as a result (Barsky, Orav, & Bates, 2005). More importantly, are the costs to patients. Unnecessary medical investigations and medication use can result in increased rates of iatrogenic complications (Bass & Benjamin, 1993; Fink, 1992). In addition, delay in the consideration of psychological causes can delay appropriate therapy which would be most beneficial to the patient (DeGruy, Columbia, & Dickinson, 1987). In order to reduce these costs to patients and to health care services, the aetiology of MUS needs to be further understood so that diagnostic accuracy and appropriate treatment can be provided.

Emotion dysregulation is particularly important as like in other disorders, it may play a causative or maintaining role in MUS as suggested in the theoretical models outlined above. Therapies incorporating emotion regulation skills training may therefore be applied to MUS, potentially improving the effectiveness of psychotherapeutic interventions and outcomes in this client group (Berking, Wupperman, Reichardt, Pejic, Dippel, & Znoj, 2008).

A lack of clarity in the definition of emotion dysregulation may be resulting in a lack of research in the area (Cole, Martin, & Dennis, 2004). In order to further understand the relationship between emotion dysregulation in MUS, further clarity needs to be sought in the way emotion dysregulation is being conceptualised. By investigating how emotion dysregulation is being conceptualised in MUS (e.g. by identifying which emotion regulation

strategies are being investigated in MUS research), it is hoped that the conceptualisation of emotion dysregulation in MUS can be tightened, encouraging further research in the area.

Given the general consensus that alexithymia and emotion dysregulation are distinct (but overlapping) constructs, the researcher will be searching for studies investigating emotion regulation strategies notwithstanding alexithymia. Furthermore, the relationship between alexithymia and MUS has already been investigated extensively in a recent meta-analysis (De Gucht & Heiser, 2003). On the whole, the review found support for a small to moderate relationship between alexithymia and MUS. With respect to total alexithymia scores, results were reasonably consistent; results for studies investigating the different dimensions of alexithymia were less consistent.

Aims

Aim 1: To clarify the current conceptualisation of emotion dysregulation in MUS.

Aim 2: To answer the question of whether emotion dysregulation is associated with MUS and to investigate which emotion regulation strategies people with MUS have difficulties with.

Method

Search strategy

The systematic literature search was conducted between March and April 2013 using PsychINFO, Web of Knowledge, CINAHL, and Medline databases. The MUS search terms were a combination of search terms used in other systematic reviews on the subject of MUS (Burton, 2003; Van Ravenzwaaig et al., 2010). The search terms are displayed in Table 1. The terms in each column were combined using the Boolean operator ‘OR’ and both columns were then combined using the Boolean operator ‘AND’.

Following searching the databases, reference lists of the relevant papers were manually screened to check for articles that had not been identified in the electronic search. Citations were examined and relevant journals and websites were searched for further papers. Studies were considered from peer reviewed journals, theses, and studies awaiting publication to avoid publication bias.

Table 1

Search terms

Emotion regulation	**MUS**
Emotion*	Medically unexplained sympt*
Affect	Unexplained medical sympt*
Regulation	Non-organ*
Dysregulation	Somatisation
Emotion avoidance	Somatization
Re-appraisal of emotion	Psyhophysiological
Impulse control	Psychosomatic
	Psychogenic
	Hypochondria*
	Somatoform disorder*
	Functional disorder*
	Conversion disorder*

Inclusion and exclusion criteria.

Figure 1 outlines the filtering process based on the PRISMA flow diagram (Moher, Liberati, Tetzlaff, & Altman, 2009). Following the removal of duplicate studies, titles and abstracts of identified records were screened for relevance. Papers were included or excluded according to the following criteria:

Inclusion criteria

- Sample included people with MUS,
- Study included a measure of emotion dysregulation,
- The relationship between emotion dysregulation and MUS was investigated,

- The paper was published in the English language.

Exclusion criteria

- Non-experimental studies (e.g. discussion papers),
- Investigated only alexithymia (e.g. included only an alexithymia measure).

Quality control

A quality control checklist for cross-sectional studies (Appendix 5) was adapted from Guyatt, Sackett, and Cook (1993) and the Critical Appraisal Skills Programme (2006) and systematically applied to studies included in the review. Higher scores were awarded for more methodologically rigorous studies. No studies were excluded due to their quality rating, but limitations are considered throughout the review. Due to the diversity in the emotion regulation measures adopted, a systematic literature review rather than a meta-analysis was conducted.

Figure 1: PRISMA Flow Diagram.

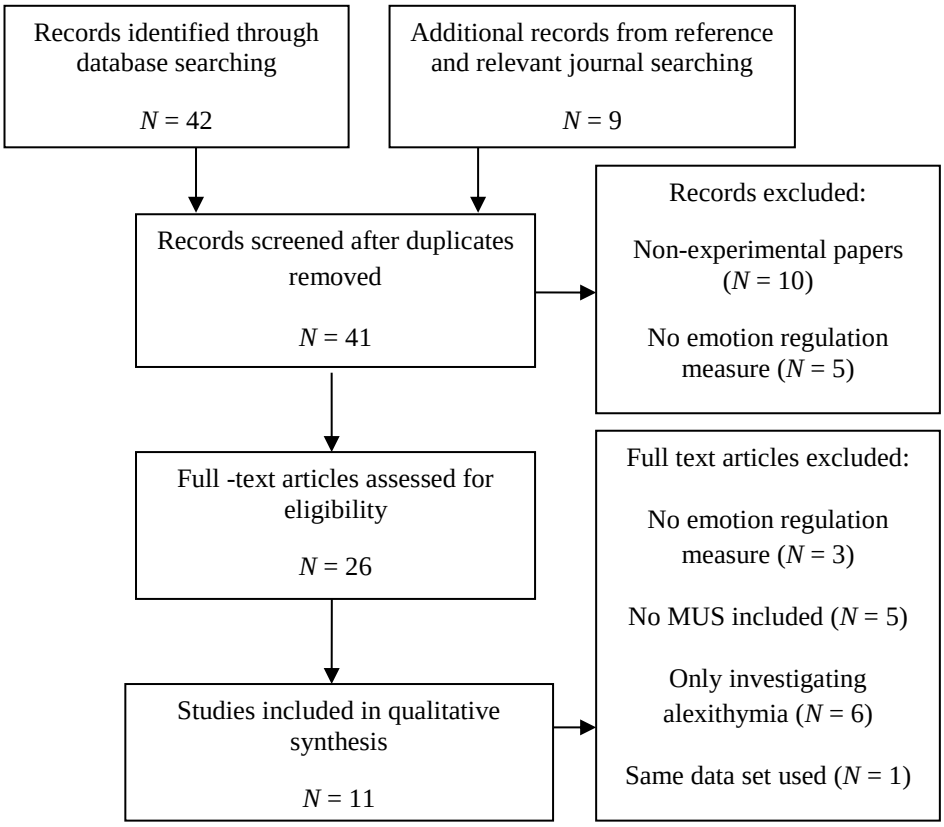

Table 2

Data Extraction Table

Authors/year/ country	**Aim(s)**	**Sample**	**Method**	**Measures used to assess MUS and ER[a]**	**Findings**	**QR[b]**
Brown et al. (unpublished doctoral thesis) UK	To investigate associations between ER, alexithymia, attachment and psychopathology in PNES	45 PNES patients and 24 epilepsy patients	Cross-sectional Questionnaires	**MUS:** PNES diagnosis from a neurologist or neuropsychologist **ER:** The DERS	Higher levels of emotion dysregulation were reported in the PNES group. Two clusters of PNES patients were identified: Cluster 1= high levels of psychopathology, alexithymia, most aspects of emotion dysregulation. Cluster 2 = high somatisation, normal levels of ER, alexithymia, and psychopathology	73%
Gilleland, Suveg, Jabob, & Thomassin (2009) USA	To examine predictors of children's somatic symptoms, including parental and child reports of ER	42 healthy child controls and 42 caregivers	Cross-sectional Questionnaires	**MUS:** Somatic scale of the Child Behaviour Checklist, somatisation scale of the Symptom Checklist-90-Revised **ER:** Awareness subscale of the Emotion Expressivity Scale for Children and the Emotion Regulation Checklist	Child's poor emotional awareness predicted their reports of somatisation. Parental reports of child's ER difficulties did not predict children's reports of somatisation	63%

Note: [a] ER = emotion regulation, [b] QS = quality score

Authors/year/ country	Aim(s)	Sample	Method	Measures used to assess MUS and ER[a]	Findings	QR[b]
Hambrook et al. (2011) UK	To explore how people with CFS and anorexia nervosa (AN), regulate, tolerate, manage, and express emotions	45 people with CFS , 40 people with anorexia nervosa and 48 healthy controls	Cross-sectional Questionnaires	**MUS**: Diagnosed by trained clinician **ER:** Distress Tolerance Scale, Beliefs about Emotions Scale, Silencing the Self Scale.	CFS and AN scored significantly higher on avoidance of affect and suppression of emotions than healthy controls but not different from each other.	78%
Lilly & Valdez (2012) USA	To examine relationships between ER, alexithymia, PTSD symptoms, and somatisation	248 university students	Cross-sectional Questionnaires completed online	**MUS:** The somatisation subscale of the Symptom Checklist 90-Revised. **ER**: DERS	Significant correlations between ER and somatic symptoms were found. ER difficulties were more highly correlated with somatisation for people who also reported greater alexithymia.	56%
Raval, Martini, & Raval (2010) India	To compare ER in Indian children experiencing internalising, externalising, or somatic problems	120 Gujarati children (aged 6-8) with internalising problems (*n* = 31), externalising problems (*n* = 32), somatic problems (*n* = 25), and healthy controls (*n* = 32)	Cross-sectional Questionnaires Emotion vignettes for children with questions from interviewer	**MUS**: Child Behaviour Checklist **ER**: Emotion vignettes	All 3 symptomatic groups reported ER problems. The internalising and externalising groups were associated with under-regulation. The somatic group were associated with over-regulation.	65%

Note: [a] ER = emotion regulation, [b] QS = quality score

Authors/year/ country	Aim(s)	Sample	Method	Measures used to assess MUS and ER[a]	Findings	QR[b]
Reuber, Pukrop, Bauer, Derfuss, & Elger (2004) Germany	To investigate whether people with PNES have maladaptive personality profiles (including emotion dysregulation)	85 PNES patients, 63 epilepsy patients and 100 healthy controls	Cross-sectional Questionnaires	**MUS:** PNES diagnosis confirmed by video-EEG, EEG, observation, and ictal examination **ER:** Dimensional Assessment of Personality Pathology – Basic Questionnaire	PNES patients had higher emotional dysregulation scores than healthy and epileptic control groups. 3 PNES clusters were revealed characterised by levels of emotion dysregulation and personality types	70%
Roberts et al. (2012) USA	To compare emotional responses among PNES and seizure free individuals with prior trauma exposure, and higher or lower levels of PTSD symptoms.	18 PNES patients, 18 individuals with elevated PTSD symptoms and 18 individuals with lower PTSD symptoms	Cross-sectional Questionnaires and physiological measures during and after an exercise involving exposure to emotional pictures	**MUS**: Diagnosed by epileptologists using video-EEG **ER:** DERS	PNES patients had higher levels of emotion dysregulation than individuals with low PTSD levels. Patients with PNES and individuals with high PTSD levels did not differ on emotion dysregulation	80%
Uliaszek, Prensky, & Baslet (2012) USA	To understand profiles of ER in PNES	55 PNES patients	Cross-sectional Questionnaires	**MUS**: Diagnosed by epileptologists using EEG or video-EEG. **ER**: DERS	2 PNES clusters were revealed. Cluster 1= high emotional dysregulation and psychopathology. Cluster 2 = low emotion dysregulation and psychopathology.	70%

Note: [a] ER = emotion regulation, [b] QS = quality score

Authors/year/ country	Aim(s)	Sample	Method	Measures used to assess MUS and ER[a]	Findings	QR[b]
Van der Kolk, Pelcovitz, Roth, Mandel, McFarlane, & Herman (1996) USA	To investigate relationships between PTSD, dissociation, ER and somatisation	395 patients with trauma related problems and 125 community controls who had all been exposed to stressors	Cross-sectional Questionnaires completed over the phone	**MUS**: Structured Interview for Disorders of Extreme Stress (SIDES). **ER**: SIDES	ER and somatisation were highly correlated in both groups. ER and somatisation were higher for PTSD sample	55%
Van Dijke, Ford, Van der Hart, Van Son, Van der Heijden, & Bühring (2010) Holland	To investigate under and over-regulation of emotions in patients with BPD, somatisation disorder (SoD), co-morbid BPD and SoD, and other psychopathology	472 participants: 120 BPD, 159 SoD, 129 BPD and SoD, and 64 other psychiatric disorders	Cross-sectional Questionnaires	**MUS:** Somatic scale of the Composite International Diagnostic Interview. **ER:** Under-regulation – SIDES. Over-regulation - Bermond Vorst Alexithymia Questionnaire	SoD was associated with over-regulation of affect and BPD with under-regulation. Participants with SoD and BPD reported more frequently both over and under-regulation of affect than participants diagnosed with BPD or SoD alone	75%
Van Middendorp, Lumley, Jacobs, Van Doornen, Bijlmsa, & Geenen (2008) Holland	To investigate how emotions and ER strategies relate to symptoms of fibromyalgia	403 women with fibromyalgia and 196 healthy controls	Cross-sectional Questionnaires	**MUS:** Using accredited fibromyalgia diagnostic criteria **ER:** Emotion Regulation Questionnaire	There were significant differences in the suppression (but not the reappraisal) component of ER between fibromyalgia and control participants	73%

Note: [a] ER = emotion regulation, [b] QS = quality score

Results

The data extraction table (Table 2) summarises the included studies. Where some studies had a broader focus, only the components related to emotion dysregulation in MUS are presented here. The table is accompanied by a narrative review of data extracted from the studies included in the review. The first part of the results section concerns the emotion regulation strategies included in the studies and their relationship to MUS. The second part concerns interesting themes that emerged from the studies.

Emotion regulation strategies

Composite scores of emotion dysregulation.

A number of the studies investigating emotion dysregulation in MUS have reported on general levels of emotion dysregulation (e.g. a composite of scores from emotion regulation measures). In two large scale studies that investigated emotion dysregulation and somatisation in healthy participants, significant strong positive correlations were reported between MUS and emotion dysregulation (r = .43, p <.05, Lilly & Valdez, 2012; r = .60, p <.05, Van der Kolk et al., 1996). Whilst this supports the notion that those with higher levels of emotion dysregulation are likely to have higher levels of MUS, it does not allow us to assess causality. In addition, it does not allow us to compare levels of emotion dysregulation between patient and healthy control groups, a finding with greater clinical and theoretical implications.

Other studies have compared general levels of emotion dysregulation with control groups of patients reporting similar symptoms caused by medical conditions, other psychiatric disorders, and healthy controls. On the whole, studies suggest that general levels of emotion dysregulation are higher in those with MUS disorders than in the healthy population (p <.001,

Reuber et al., 2004), and those with comparable medical disorders (p <.002, Reuber et al., 2004), but not higher than levels found in those with other psychiatric disorders, namely PTSD and BPD (p = .49, d = .24, Roberts et al., 2010; p >.05, n^2 = .03, Van Dijk et al., 2010).

One study included in the review corroborated the self-reported emotion dysregulation data with a physiological measure. As part of an emotion processing task, Roberts et al. (2010) investigated Respiratory Sinus Arrhythmia (RSA), considered a biomarker for emotion dysregulation, and found physiological support for the self-reported findings. Participants with MUS had significantly lower RSA (representative of higher levels of emotion dysregulation) in comparison with participants with low levels of PTSD symptomology (p = .05, d = 71) but not significantly different to participants with higher levels of PTSD symptomology (p = .61, d = .18).

Taken together, the results suggest that emotion dysregulation is associated with MUS and whilst levels of emotion dysregulation may be higher in people with MUS than in healthy controls and in people with comparable medical disorders, emotion dysregulation in people with MUS and psychiatric disorders (PTSD and BPD) may be similar.

Whilst investigating composite measures of emotion dysregulation gives us a broad overview of a person's emotion regulation difficulties, it does not allow us to explore specifically which emotion regulation strategies individuals may be having difficulties with. The remainder of the review focuses on associations between MUS and specific emotion regulation strategies.

Accepting emotions.

Non-acceptance of emotional responses refers to feelings of guilt, anger, embarrassment, and weakness in the face of felt negative emotion (e.g. 'when I get upset, I feel guilty'); three

studies included acceptance of emotions as an emotion regulation strategy; two investigating emotion dysregulation in PNES and one in chronic fatigue syndrome.

The results were similar to those describing composite levels of emotion dysregulation; non-acceptance of emotions was higher in patients with MUS than in healthy controls (p >.05, Hambrook et al., 2011), but no significant differences were found between MUS and patients with psychiatric disorders (p >.05, Hambrook et al., 2011), or comparable physical disorders (p >.05; Brown et al., unpublished doctoral thesis, 2010).

Recognising the clinical heterogeneity of a particular MUS disorder (PNES), studies have used cluster analysis to identify clusters of participants characterised by levels of psychological characteristics, including emotion dysregulation. Two studies have compared levels of difficulties in accepting emotions between clusters of patients. In both studies, two clusters of patients were identified, characterised by higher or lower levels of emotion dysregulation (Brown et al., unpublished doctoral thesis; Uliaszek et al., 2012). In both studies, the clusters characterised by higher levels of emotion dysregulation had significantly more problems with acceptance of emotions than clusters with lower levels of emotion dysregulation (p <.001, r = .54, Brown et al., unpublished doctoral thesis; p <.001; Uliaszek et al., 2012), normative data (p <.001; Uliaszek et al., 2012), and comparable medical disorders (p <.05, r = .46, Brown et al., unpublished doctoral thesis). Clusters associated with lower levels of emotion dysregulation were not significantly different to normative data (p >.05; Uliaszek et al., 2012) or to a comparable medical disorder, epilepsy (p >.05, r = .06, Brown et al., unpublished doctoral thesis).

Suppression of emotions.

The suppression of emotions has been investigated as an emotion regulation strategy in MUS. In the literature included in the review, suppression of emotions referred to the

internalising of negative emotions (e.g. ‘when angry, I’m angrier on the inside then I appear on the outside’). Three studies investigated suppression of emotions as an emotion regulation strategy.

The findings regarding suppression of emotions in MUS were mixed. In a large scale hospital study comparing the suppression of emotions between those with an MUS disorder (fibromyalgia) and healthy controls, the tendency to suppress emotions was significantly higher in people with MUS syndromes and in people with high levels of somatic symptoms respectively, than in healthy controls (p <.05; Raval, Martini, & Raval, 2010; p <.01, d^2 = .23; Van Middendorp et al., 2008). In a smaller scale hospital-based study, these findings were not supported; there were no significant differences in the suppression of emotion between MUS syndromes, psychiatric disorders (p >.05), or healthy controls (p >.05; Hambrook et al., 2011).

The researcher has considered possible reasons for these disparate results. The measure used to investigate suppression of emotion in the Hambrook et al. (2011) study concerned the suppression of emotion with regards to developing intimate relationships (e.g. ‘to preserve relationship harmony’). This may not be generalisable to everyday life situations, reducing the construct validity. In contrast, the measure used by Van Middendorp et al. (2008) measured the tendency to inhibit ones emotions in general everyday situations, which may be more generalisable with increased external validity. In addition, the data collection procedure in the study by Hambrook et al. (2011) was not clear (reducing the extent to which the validity and reliability of the study could be considered) and the sample size was relatively small, increasing the likelihood of a type II error.

Avoidance of emotions.

Whilst avoidance of emotions is considered to be an emotion regulation strategy in much of the emotion regulation literature (Gross & John, 2003), only one study investigated it as an emotion regulation strategy in MUS. Whilst superficially, avoidance of emotions may appear to be conceptually indistinguishable from suppression of emotions; on closer inspection, this is not the case. In the study that reported investigating avoidance of emotions as an emotion regulation strategy, this was conceptualised as avoiding situations that may trigger an emotional response (e.g. avoiding a social situation in which one might fear feeling a negative emotion). The literature suggests that the tendency to avoid emotions (e.g. avoiding emotional situations) is significantly higher in MUS disorders than healthy controls (p <.01), but not significantly higher than psychiatric disorder (p >.05) such as anorexia nervosa (Hambrook et al., 2011).

Dealing with negative emotions.

Dealing with negative emotions refers to having emotion regulation strategies one can access to deal with felt negative emotions and thus improve wellbeing. This includes feeling overwhelmed in the face of negative emotions and not having strategies to make oneself feel better in these situations (e.g. 'when I'm upset I believe that wallowing in it is all I can do' or 'when I want to feel less sadness, I change what I'm thinking about'). This has also been termed reappraisal of emotions.

There was no evidence from the studies included in the review that people with MUS have more difficulties in dealing with negative emotions in comparison with healthy controls (p >.05, r = .30, Brown et al., unpublished doctoral thesis; p >.05, Hambrook et al., 2011; p >.05, Van Middendorp et al., 2008) and psychiatric disorders (p >.05, Hambrook et al., 2011).

As found in the studies investigating the acceptance of emotions, of those studies investigating clusters of patients, the cluster characterised by high levels of emotion dysregulation did report more difficulties in accessing emotion regulation strategies (p <.001, Uliaszek et al., 2012) than the cluster characterised by lower levels of emotion dysregulation.

Impulse control difficulties.

In the emotion regulation literature, impulse control refers to the ability to control impulsive emotional behaviours (e.g. 'when I'm upset I have difficulties in controlling my behaviours'). Examples of this may be losing control over behaviours when upset or angry. Four studies included impulse control as an emotion regulation strategy.

The results suggest that people with MUS have difficulties in controlling impulsive behaviours in the face of felt negative emotion. People with MUS disorders have significantly more difficulties with controlling emotionally impulsive behaviours than people with comparable physical disorders (p < .005, r = .36; Brown et al., unpublished doctoral thesis), healthy controls (p <.05, Van der Kolk et al., 1996), normative data (p <.05, Uliaszek et al., 2012) and patients with psychiatric disorders (p <.05, Van Dijk et al., 2010).

Whilst other studies have found no significant differences in emotion dysregulation between people with MUS and people with clinical level psychiatric disorders, the Van Dijke et al. (2010) study found no support for this. This may suggest that difficulties in impulse control as a specific emotion regulation strategy, is specific to MUS. However, considering the nature of the sample in this study, these findings should be interpreted with caution. Recruiting from an inpatient facility, the study may have been subject to selection bias and demand characteristics, thus reducing validity. In addition, the findings may not be generalisable to other settings.

Engaging in goals.

The final sub-set of emotion regulation strategies relate not to how the person regulates the emotions they feel, but rather how the emotion regulates them (i.e. how their felt emotions interferes with their behaviours and goals). The DERS terms this 'difficulties in engaging in goal directed behaviour' (e.g. 'when I'm upset, I have difficulty getting things done').

The studies investigating the extent to which people with MUS can engage in goal-directed behaviour in light of negative emotions, suggest people with MUS disorders have significantly more difficulties in this domain in comparison with a comparable disorder with a physical basis (p <.001, r = .43; Brown et al., unpublished doctoral thesis) and normative data (p <.001, Uliaszek et al., 2012).

Emerging themes

In addition to data regarding the relationship between difficulties in emotion regulation strategies and MUS, themes emerged from the studies that yielded interesting findings regarding emotion dysregulation in MUS.

The role of alexithymia.

An interesting theme emerging from the studies concerned the role of alexithymia in the relationship between emotion dysregulation and MUS (Hambrook et al., 2011; Lilly & Valdez, 2012; Van Middendorp, et al., 2008). In a large scale study investigating emotion dysregulation and its relationship to MUS and to PTSD, the researchers found that alexithymia significantly moderated the relationship between emotion regulation difficulties (as a composite measure) and somatisation (p <.001; Lilly & Valdez, 2012), greater emotion dysregulation conferred less risk for MUS in the absence of heightened levels of alexithymia.

In a smaller scale study investigating emotion dysregulation in fibromyalgia, emotion dysregulation was related to higher levels of somatic symptoms only in those patients who lacked the ability to identify or describe emotions, akin to alexithymia (p <.001; Van Middendorp et al., 2008). Furthermore, in a study comparing emotion dysregulation between MUS and a psychiatric disorder (anorexia nervosa), whilst comparable levels of emotion dysregulation between the two groups were reported, the only factor that differentiated the two groups was their beliefs about emotions, specifically, maladaptive beliefs about expressing emotions (e.g. "it is not ok to say exactly how I'm feeling"). The researchers suggested that this may be indicative of alexithymia. Whilst this alone did not predict whether a person had MUS or anorexia nervosa, the researchers hypothesised that it is the combination of emotion dysregulation and alexithymia that may result in MUS.

Although the search criteria did not include alexithymia, a number of studies included measures of expression of emotions, conceptualising these as emotion dysregulation strategies. It is the expression of emotions that has been considered as a component present in both emotion dysregulation and alexithymia by some researchers (Pandey, Saxena, & Dubey, 2011). Considering expression of emotion, the results were mixed. Whilst some studies found higher levels of difficulties in the expression of emotions in MUS compared to healthy controls (p <.001, n^2 = .16, Raval & Martini, 2010; p <.001, Reuber et al., 2004), other studies found no support for this (p >.05, Gilleland et al., 2009; p <.001, d^2 = .18, Van Middendorp et al., 2008).

The researcher considered possible reasons for these disparate results, namely the methodological limitations in the Van Middendorp et al. (2008) and Gilleland et al. (2009) studies. Firstly, in the Van Middendorp et al. (2008) study, the control sample were not screened for somatic symptoms. Whilst this may be more representative of the general population (and thus yield improved external validity), this may have reduced the internal

validity of the study and the reliability of comparisons between clinical and healthy controls may have been compromised. This may have resulted in false positive findings. In relation to the Gilleland et al. (2009) study, the findings relating to difficulties in emotion expression were based solely on children's own reports of their emotional expression. The reliability and credibility of children's reports in research has been questioned (Bruck, Ceci, & Hembrooke, 1998). Whilst obtaining a child's viewpoint is important, the need to corroborate this with parental reports has also been suggested (Punch, 2002). Although levels of emotion dysregulation in children were corroborated by parental reports in the Gilleland et al. (2009) study, as different measures were used investigating different strategies, this rendered the two reports of emotion dysregulation non comparable.

Over and under-regulation of emotions.

An interesting theme that emerged in the literature included in the review relates to the over and under regulation of emotions (also termed hyper and hypoarousal of emotions). Whilst some researchers reported that MUS was more associated with the under-regulation of emotions than the over-regulation of emotions (Raval, Martini, & Raval, 2010); others reported that MUS was more associated with the over-regulation of emotions than the under-regulation of emotions (Van Dijk et al., 2010). It should be noted that in the Van Dijk et al. (2010) study, over-regulation of emotions were measured using an alexithymia measure reducing the validity of the findings. Taking the study findings as a whole, the majority of emotion regulation strategies investigated were akin to the under regulation of emotions (e.g. non-acceptance of emotions, dealing with negative emotions, impulse control and engaging in goal directed behaviour) as opposed to over regulation of emotions (e.g. difficulties in the suppression and avoidance of emotions). The findings most supported the relationship between MUS and the under-regulation of emotions (no support was found for either of the strategies relating to the over regulation of emotions). This may be because more strategies

involved in the under-regulation of emotions have been investigated in the literature included in the review.

Beliefs about emotions.

Two of the studies included in the review investigated beliefs about emotions in relation to the emotion regulation strategies adopted (Hambrook et al., 2011; Raval, Martini, & Raval, 2010). In a study investigating emotion dysregulation in children living in rural India (Raval, Martini, & Raval, 2010), those with MUS and high emotion dysregulation reported being influenced by parental and cultural norms in their expression of emotions. These children reported the over-use of a number of emotion regulation strategies as a result of this, including the suppression of emotions. In a similar sized study utilising adult samples in the UK, high levels of maladaptive emotion beliefs were reported in the MUS sample. Whilst similar levels of emotion dysregulation were reported in the MUS and in the psychiatric samples, only these maladaptive beliefs (including "If I lose control of my emotions in front of others, they will think less of me" and "I should not let myself give into negative feelings") separated the two participant groups. These findings yielded interesting insights into effortful as opposed to automatic emotion dysregulation and the reasons behind the over-reliance on emotion regulation strategies.

Discussion

The aim of this systematic literature review was twofold; firstly to investigate how emotion dysregulation is being conceptualised in MUS and secondly to investigate the extent to which difficulties in the emotion regulation strategies investigated are associated with MUS.

Summary of evidence

Eleven studies were included in the review, five investigated the association between emotion dysregulation and the number of somatic symptoms, and six compared emotion dysregulation between specific MUS disorders and control groups. Nine of the studies used adult samples and two used child samples. Eight studies focused on clinical samples and two on non-clinical samples. All but one of the studies were conducted in western cultures.

Whilst all studies reported to be investigating emotion dysregulation, there was discrepancy amongst the studies with regards to how researchers were conceptualising emotion dysregulation and only two studies included a definition of the concept. This echoes other researcher's views who have suggested that despite rapidly evolving literature in the area, emotion dysregulation is measured inconsistently across MUS studies, with little regard to whether different approaches capture the same construct (Vasilev, Crowell, Beauchaine, Mead, & Gatzke-Kopp, 2009). Whilst some studies used alexithymia measures alongside emotion regulation measures, viewing the two concepts as distinct (Hambrook et al., 2011; Lilly & Valdez, 2010), one study used an alexithymia measure as a measure of emotion dysregulation (Van Dijk et al., 2010). This provides some evidence for ambiguity in the conceptualisation of emotion dysregulation, in both the definition of the concept and its relationship with other emotion processing variables.

The emotion regulation strategies being investigated in MUS fall into the following areas: acceptance of emotions, suppression of emotions, avoidance of emotions, dealing with negative emotions, impulse control and engaging in goal directed behaviour. These emotion regulation strategies fit with the two most dominant definitions of emotion dysregulation (Gross, 1998b, p.275; Thompson, 1994, pp.27-28). However, whilst acceptance of emotions

was included in studies investigating emotion dysregulation in MUS, this is not included in these definitions.

This conceptualisation fits with the DERS measure of emotion dysregulation, although avoidance of emotion is not included in this measure. Whilst this may be due to many of the studies included in this review using the DERS to measure emotion dysregulation (45% of the studies included in the review), a number of the other studies investigated these emotion regulation strategies as part of other measures or using measures solely designed to measure these strategies. This suggests that the DERS may be the best available measure of emotion dysregulation in this population, capturing the majority of strategies in the current conceptualisation of emotion dysregulation in MUS.

Whilst composite scores of emotion dysregulation are associated with MUS and scores are higher in MUS than in the healthy population (but not in patients with psychiatric disorders), when considering individual emotion regulation strategies, the picture is more mixed. This suggests that whilst considering composite scores of measures may be beneficial as a screening tool, further investigation into specific emotion regulation strategies need to be investigated.

The literature suggests that people with MUS may be more likely to have difficulties in accepting their emotions (i.e. feeling guilty when feeling upset) and may have difficulties in controlling impulsive behaviours in the face of felt negative emotions. Additionally, people with MUS have difficulties in engaging in their goal directed behaviour when feeling negative emotions. Some support was found for the notion that people with MUS may suppress their emotions (e.g. internalise their emotions) although this was non-conclusive. No support was found for people with MUS avoiding their emotions or not being able to deal

with negative emotions (i.e. not knowing what to do to make oneself feel better or dealing with emotions in a self-defeating manner) in comparison with the healthy population.

Limitations

Before the theoretical and clinical implications of the findings are discussed, the limitations need to be considered. There are limitations of the literature review itself that should be considered when generalising the findings to research or clinical practice. Firstly, the review did not search for studies using purely physiological measures of emotion dysregulation. Given limitations in self-reported measures, inclusion of studies using physiological measures may have triangulated the data, improving validity of the reviews findings. Additionally, synthesis of data into the emotion regulation strategies investigated resulted in a number of MUS disorders being grouped together, these disorders may not be completely comparable. For instance, patients with PNES may have difficulties in different emotion regulation strategies than patients with CFS. In addition, in including studies reporting to be investigating emotion dysregulation (to investigate how emotion dysregulation is being conceptualised in MUS), studies investigating strategies of emotion regulation but not explicitly stating this may have been missed. Including both child and adult literature in the review allowed for a thorough investigation of how emotion dysregulation is being conceptualised in MUS across all population groups. However, the child and adult emotion dysregulation data may not be comparable (in comparison with adults emotion regulation strategies, children's emotion regulation strategies may not be fully developed). Now more clarity has been sought on how emotion dysregulation is being conceptualised, future research should consider the relationships between emotion regulation and MUS in child and adult samples independently.

Researcher rated quality ratings for each study can be seen in Table 2. Quality of studies ranged from 55% to 80% on the researcher rated quality ratings. Whilst some limitations were unique to some studies (as outlined in the results section), a number of limitations were similar and were found across all of the studies. The majority of limitations will therefore be discussed in this section.

Whilst there were a number of strengths including the accurate and reliable diagnoses of MUS, the use of well-matched control groups, and the use of both clinical and healthy control comparison groups in a number of studies, there were also a number of limitations. Of particular note was the lack of definition of emotion dysregulation and the lack of justification for the emotion dysregulation measure adopted. As already mentioned, this is likely to be due to the ambiguity in the definition of emotion regulation which highlights an area which warrants further research. The disparities in the conceptualisations of emotion dysregulation limited the reliability and validity of the study findings and the extent to which findings could be generalised to clinical practice. The majority of studies were marked down on the researcher rated quality ratings on question one of the quality rating scale ("Did the study address a clearly focused research question?") as a result of this.

All studies utilised self-report measures of emotion regulation. Given findings regarding the use of avoidant emotion regulation strategies, the same people may be unaware or reluctant to report emotion regulation difficulties. However, only two of the studies triangulated the data, one included parental reports (Gilleland, Suveg, Jacob, & Thomassin, 2009) one included physiological markers of emotion dysregulation (Roberts et al., 2012). The use of solely self-report measures may have resulted in social desirability bias and demand characteristics, threatening validity. Relating to the measurement of MUS specifically, of the studies that investigated associations between emotion dysregulation and the number of somatic symptoms, the questionnaires used only checked for the presence of

somatic symptoms, not whether these symptoms were medically unexplained or not. This may have lead to an overestimation of somatisation. The studies investigating specific MUS disorders may have therefore yielded more valid and reliable results.

All of the studies included in the review utilised cross-sectional designs. Although this allows for multiple outcomes to be assessed in a relatively short time reducing the load on participants, this design does not allow for conclusions about cause or effect or sequence of events. The design is also prone to selection and measurement bias. However, in a relatively new research area, this design allows for hypotheses to be made that can be explored further in future research.

Theoretical implications

Limitations aside, the review has highlighted some interesting theoretical implications, all of which should be considered in light of the limitations of the research. The findings provide some support for models of MUS that suggest that a complex interplay of factors contribute to the development and maintenance of MUS (Deary, Chalder, & Sharpe, 2007). Whilst emotion dysregulation is associated with MUS (although causality cannot be inferred), the interplay of emotion dysregulation and alexithymia appear to strengthen this association.

By investigating how emotion dysregulation has been conceptualised in MUS, it is hoped that this review will be helpful to future researchers in this area. Whilst aetiological models of MUS have suggested emotion dysregulation is a contributing factor, this study highlights that it may only be some elements of emotion dysregulation (non-acceptance of emotions, impulse control, engaging in goal directed behaviour, and possibly suppression of emotions) that people with MUS have difficulties with. Rather than using the general term emotion dysregulation, researchers should therefore specify which emotion regulation strategies they

are referring to. However, given limitations in the research, this needs to be investigated further.

Clinical implications

A number of potential clinical implications can be drawn from this systematic literature review. Whilst clinicians may screen for emotion regulation difficulties using composite scores from emotion regulation measures, the importance of investigating individual emotion regulation strategies has been highlighted.

Whilst possible mediating factors have potential theoretical implications, they also have potential clinical implications. In assessment and formulation, therapists may consider alexithymia, and the relationship they may be playing in perpetuating MUS. In addition, these mediating factors may be considered targets for therapy.

In addition, the review highlighted possible difficulties in a number of emotion regulation strategies that clinicians should be aware of. One area of difficulty is the acceptance of emotions. Clinicians should be aware of the possibility of this and be mindful of this during assessments of emotions, utilising a number of methods of assessing emotions to ensure validity. In addition, clinicians can normalise these feelings and work on these feelings in therapy. With regards to impulse control, when working with potential behavioural problems in this client group, the underlying emotion dysregulation should be explored. Similarly, clinicians should consider emotion regulation skills training in enabling goal directed behaviour and wellbeing in this client group.

Furthermore, interesting clinical implications came from the studies that considered the reasons behind adopting particular emotion regulation strategies. Expressions of emotions may be influenced by a number of factors including parenting and cultural norms, which

should be investigated in clinical work with people with MUS displaying emotion regulation difficulties.

Conclusions and future directions

The review has added to literature in the area by highlighting how emotion dysregulation is being conceptualised in MUS. In addition, it has highlighted the specific emotion regulation strategies that people with MUS have difficulties in. The review discussed the significant limitations in the existing research and areas that need to be investigated further.

Principally, the review highlighted the need for further research investigating emotion dysregulation in MUS. Prospective and longitudinal studies are needed to further explore the association between emotion dysregulation and MUS; this will provide a better understanding of the relationship between this association and whether emotion dysregulation serves as a predisposing or a maintaining factor. The differential associations between the constituting emotion regulation strategies should be further investigated. Furthermore, research investigating the relationship between emotion dysregulation and alexithymia is warranted. Studies that partial out the effects of the two constructs would add to the aetiological understanding of the disorder.

Future research needs to utilise clear definitions of emotion dysregulation to improve construct validity. Using standardised dedicated measures of emotion dysregulation alongside objective measures of emotion dysregulation (e.g. implicit or physiological measures) would be advantageous. In addition, research needs to move towards the use of measures that allow us to make distinctions between somatic symptoms that are medically explained and those which are not (as suggested by De Gucht and Heiser, 2003).

As is evident in the limited number of studies included in this review, there is a paucity of research investigating emotion dysregulation in MUS. It is hoped that this review may go some way to encourage more high quality studies in the area.

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Section 2

Research Report

Investigating the Relationship between Emotion Dysregulation and Trauma in Psychogenic Nonepileptic Seizures: An Exploratory Study

Abstract

Objectives. Research suggests that emotion dysregulation may contribute to the development and maintenance of psychogenic nonepileptic seizures (PNES). Despite the high prevalence of traumatic experiences in PNES, no researchers have investigated associations between emotion dysregulation and traumatic experiences in this population. The current study sought to address this gap to further understand the aetiology of PNES.

Design and methods. Participants with PNES (N = 25), epilepsy (N = 23) and healthy controls (N = 27) completed measures of emotion dysregulation, traumatic experiences, and anxiety and depression. Patient participants also provided information on seizure frequency and severity.

Results. There were no significant differences in levels of emotion dysregulation between PNES and epilepsy participants. Participants with epilepsy and participants with PNES reported higher levels of emotion dysregulation than healthy controls. PNES participants reported experiencing significantly more traumatic events than epilepsy or healthy control participants. PNES and epilepsy participants reported significantly higher levels of anxiety and depression than healthy controls, but did not differ from each other. Only anxiety (not traumatic experiences) significantly predicted variance in emotion dysregulation in the three participant groups. Three clusters of participants were identified characterised by diagnosis, and higher and lower levels of emotion dysregulation, psychopathology, and experiences of trauma.

Conclusions. High levels of emotion dysregulation have been reported in PNES and in epilepsy. This is not associated with experiences of trauma but with levels of anxiety. Further research to explore the nature of the relationship between anxiety, trauma, and emotion dysregulation is needed going beyond self-report methodology.

Practitioner points

Clinical Implications

- Psychological therapy focusing on the interaction between anxiety and emotion dysregulation may be beneficial to patients with PNES and patients with epilepsy,
- Experiences of trauma may be a risk factor for patients with PNES; this should be explored in assessment, formulation and intervention.

Limitations

- The study has a relatively small sample size, relies on self-report measures, and utilises cross-sectional methodology so causality cannot be inferred.

Introduction

Psychogenic non-epileptic seizures (PNES) are defined as episodes of altered movement, sensation or experience similar to epilepsy, but caused by psychological processes not associated with epileptiform discharges in the brain (Lesser, 1996). The current nosologies do not agree on how PNES are best categorised. The Diagnostic and Statistical Manual of Mental Disorders-5 (DSM-5; American Psychiatric Association, 2013) classifies PNES as a somatoform disorder and the ICD-10 (World Health Organisation, 1992) classifies PNES as a dissociative disorder. There is no reliable information regarding the prevalence of PNES in the general population. Studies based on the prevalence of PNES in people attending neurological clinics for diagnosis have suggested incident rates of 4.90 per 100,000 per year (Duncan, Razvi, & Mulhern, 2011).

Diagnosing PNES

The introduction of video electroencephalography (vEEG) into clinical practice, which involves capturing seizures on video and EEG simultaneously, has improved the diagnostic accuracy of PNES (Mostacci et al., 2011). Despite diagnostic advances, there is often delay in the correct diagnosis of PNES; it takes a mean of 7.2 years for a correct diagnosis of PNES to be made (Reuber, Fern-Andez, Bauer, Helmstaedter, & Elger, 2002). Over this time, tangible and intangible costs add up for the patient and for health care services (Martin, Gilliam, Kilgore, Faught, & Kuzniecky, 1998). For instance, many people with PNES are initially prescribed anticonvulsant medication which can have serious iatrogenic effects (Reuber et al., 2003). Furthermore, the failure to recognise the psychological basis of PNES can cause problems with engagement in appropriate psychological interventions (Bowman & Markand, 1996).

The prognosis for PNES patients remains poor (Durrant, Rickards, & Cavanna, 2011; Reuber, et al., 2003) and the aetiology of PNES remains uncertain; a better understanding of the aetiology would allow treatment to be better tailored to the needs of patients with PNES, which may improve prognosis for this population.

The Aetiology of PNES

Research suggests that PNES disorders do not have a single aetiology; rather, a number of different contributing predisposing, precipitating and perpetuating factors tend to interact in individual patients (Reuber, 2009). Several possible factors have been investigated, and research has begun to focus on how these factors may interact in PNES. The factors investigated have included co-morbid psychopathology, a history of traumatic experiences, and emotion regulation difficulties.

In a multifactorial model of the aetiology of PNES, Reuber (2009) postulated that a number of factors codetermine whether PNES will develop in an individual. The model suggests that experiences of trauma in early or later life may predispose a person to developing PNES. This experience, along with a genetic constitution of vulnerability or limited resilience may in part, result in emotion regulation difficulties. Stressful life experiences, along with mental health problems are suggested as possible precipitating factors in PNES. The model suggests that these factors (along with other possible physical factors) may interact and result in a presentation of PNES. Resultant anxiety and depression (along with other potential factors) may perpetuate PNES. The three main factors (co-morbid psychopathology, trauma history and emotion regulation difficulties) are outlined below.

Co-morbid psychopathology.

Co-morbid psychopathology has been suggested as both a predisposing and perpetuating factor in PNES (Reuber & Elger, 2003). High levels of co-morbid psychopathology have been reported in patients with PNES (Bowman & Markand, 1996; Reuber, Pukrop, Bauer, Helmstaedter, & Elger, 2003). These co-morbid disorders include other somatic disorders (Bowman, 1999), anxiety disorders (Alper, Devinsky, Perrine, Vazquez, & Luciano, 1995), depressive disorders (Mökelby et al., 2002), post-traumatic stress disorder (PTSD; Dikel, Fennell, & Gilmore, 2003) and personality disorders (Bowman & Markand, 1996). Whilst some psychiatric disorders are more common in PNES than in epilepsy (i.e. PTSD and personality disorders), similarly high levels of anxiety and depression have been reported in people with epilepsy and people with PNES (Arnold & Privitera, 1996; Tojek, Lumley, Barkley, Mahr, & Thomas, 2000).

Traumatic experiences.

Traumatic experiences have been investigated as a possible risk factor for developing PNES (Bakvis et al., 2009; Fiszman, Alves-Leon, Nunes, D'Andrea, & Figueira, 2004). People with PNES have been shown to have experienced high rates of trauma (40-100% of PNES participants), 15-40% higher rates than those found in healthy control groups (Fiszman et al., 2004). Researchers have suggested that PNES may occur as a somatic expression of distress related to experiences of trauma (Fiszman et al., 2004).

Research into the nature of the trauma experienced by people with PNES suggests that a wide range of traumatic experiences including sexual or physical abuse in childhood (Rosenberg, Rosenberg, Williams, & Wolford, 2000), social and family conflicts (Wood, McDaniel, Burchfiel, & Erba, 1998), and bullying (Duncan & Oto, 2008) may be associated with the development of PNES. Whilst researchers have investigated levels of traumatic

experience in PNES, there has been little research regarding how these experiences of trauma are associated with other psychological factors prevalent in PNES, including difficulties in emotion regulation.

Difficulties in emotion regulation.

Emotion regulation is defined as "the process by which individuals influence which emotions they have, when they have them, and how they experience and express them" (Gross, 1998, p.275). Difficulties in emotion regulation are often termed emotion dysregulation. A recent literature review (Wilkinson, unpublished doctoral thesis) found that in medically unexplained symptoms, emotion dysregulation is being conceptualised as: Difficulties in acceptance of emotions, suppression of emotions, avoidance of emotions, dealing with negative emotions, impulse control, and engaging in goal directed behaviour.

Adaptive emotion regulation allows a person to experience felt emotion in their 'window of tolerance', enabling engagement in daily functioning. Emotion dysregulation may manifest as excessive intensification of emotion or excessive suppression of emotion due to over-reliance or rigidity in the use of emotion regulation strategies. Emotion dysregulation has been associated with a number of psychological disorders including anxiety (Coan & Allen, 2004), depression (Joorman & Gotlib, 2010), and other medically unexplained symptoms (Wilkinson, unpublished doctoral thesis).

Researchers have suggested that emotion dysregulation may be a possible predisposing and perpetuating factor in PNES; due to paucity of longitudinal studies in the area, the exact contribution of emotion dysregulation in the aetiological model is unknown (Roberts et al., 2012). Four studies investigating self-reported emotion dysregulation in PNES have been identified; Reuber, Pukrop, Bauer, Derfuss, & Elger (2004), Uliaszek, Prensky, and Baslet (2010), Roberts et al. (2012) and Brown et al. (unpublished doctoral thesis). The researchers

found higher levels of emotion dysregulation in PNES patients in comparison with people with epilepsy (Reuber et al., 2004; Brown et al., unpublished doctoral thesis), healthy controls (Reuber et al., 2004; Roberts et al., 2012), and normative samples (Uliaszek et al., 2010). However, Brown et al. (unpublished doctoral thesis) found that only two aspects of emotion dysregulation reached significance (difficulties in engaging in goal directed behaviour and impulse control difficulties) and although the quality of studies were generally high (Wilkinson, unpublished doctoral thesis), there were some limitations of note. Limitations across the studies included the lack of vEEG diagnosis, not including epilepsy and healthy control groups, and not using dedicated emotion regulation measures. Further exploration of emotion dysregulation in PNES is therefore warranted.

Cluster analysis has also been used to investigate potential clusters of PNES participants. At least two clusters of patients with PNES have been identified, characterised by higher and lower levels of emotion dysregulation and psychopathology. To date, no researchers have investigated clusters of mixed diagnoses participants (e.g. PNES, epilepsy, and healthy controls in one cluster analysis). It may be that clusters characterised by diagnosis (e.g. a PNES cluster, epilepsy cluster, and healthy control cluster) and unique groups of symptoms for each diagnosis emerge. This would add to the aetiological understanding as specific patterns of symptoms may be identified in PNES.

Associations between trauma and emotion dysregulation.

Associations between emotion dysregulation and traumatic experiences have been investigated widely in the literature. Research suggests that traumatic experiences can result in difficulties in emotion dysregulation (Briere & Rickards, 2007; Kim & Chicchetti, 2010; Van der Kolk et al., 1996). This can be a result of early attachment disruption (and thus not developing emotion regulation strategies from caregivers), underdevelopment of brain

structures involved in emotion regulation (e.g. due to developmental trauma), and effects on the autonomic nervous system, which regulates our emotional and physiological states in the face of stress (Monson, Price, Rodriguez, Ripley, & Warner, 2004; Tull, Barrett, McMillan, & Roemer, 2007). Given the high prevalence of traumatic experiences in PNES, traumatic experiences may be associated with emotion dysregulation in the disorder. However, to date, no studies have investigated the association between experiences of trauma and emotion dysregulation in PNES.

Rationale for this research

We need to understand the aetiology of PNES further if we want to be able to develop more effective treatments (Fiszman et al., 2004). A greater understanding of the psychological basis of PNES could improve PNES diagnosis and ensure that appropriate psychological therapies are offered to patients with PNES without delay. This could result in reduced tangible and intangible costs for patients, medical professionals and health care systems.

Further understanding of the relationship between emotion dysregulation and PNES would contribute to understanding of the aetiology of PNES. Whilst researchers have postulated that the aetiology involves a complex interplay of factors, to date, no researchers have investigated the association between emotion dysregulation and traumatic experiences, anxiety and depression, and seizure frequency and severity, although all are common in PNES. Research in this area, taking into account the limitations of previous research investigating emotion dysregulation in PNES, is therefore warranted.

Aims and hypotheses

Aims.

The present study aims to increase our understanding of the aetiology of PNES by exploring associations between emotion dysregulation, and trauma history, anxiety and depression in three participant groups; patients with PNES, a disease control group of patients with epilepsy and a healthy control group. In participants with seizures, relationships between emotion dysregulation and seizure frequency and severity were also studied.

Aim 1: To investigate differences between levels of self-reported emotion dysregulation, traumatic experiences, and anxiety and depression, between three participant groups (patients with PNES, patients with epilepsy, and healthy controls).

Aim 2: To investigate the extent to which variance in self-reported emotion dysregulation is associated with experiences of trauma, anxiety, and depression in the three groups of participants (patients with PNES, patients with epilepsy and healthy controls). Seizure frequency and severity will also be considered in patients with PNES or epilepsy.

Aim 3: To explore any potential clusters of participants characterised by diagnosis (i.e. PNES, epilepsy, or healthy controls) and levels of emotion dysregulation, traumatic experiences, anxiety, or depression to explore whether there are unique groups of symptoms associated with PNES, epilepsy, and healthy controls.

Hypotheses.

Hypothesis 1: Patients with PNES will have significantly higher levels of emotion dysregulation, and traumatic experiences than participants with epilepsy and healthy controls. Levels of anxiety and depression will not differ between participants with PNES and

participants with epilepsy, both will report higher levels of anxiety and depression than healthy controls.

Hypothesis 2: In participants with PNES, emotion dysregulation will be associated with experiences of trauma.

Hypothesis 3: There will be at least two clusters of participants identified characterised by higher and lower levels of emotion dysregulation.

Method

Participants

Patient participants were 48 patients with PNES (N = 25) or epilepsy (N = 23) recruited over an eight-month period from the weekly seizure clinic at The Royal Hallamshire Hospital, Sheffield. A consultant neurologist at The Royal Hallamshire Hospital identified patient participants who were suitable for inclusion in the study in accordance with the following inclusion criteria:

- A v-EEG documented clinical diagnosis of PNES or epilepsy,
- Over the age of 18,
- Sufficient English language skills to complete self-report measures,
- Able to give informed consent.

Participants were excluded from the study if they had a mixed seizure disorder or if they had not had a seizure in the past 12 months. A letter inviting potential participants to take part (Appendix 6) and an information sheet (Appendix 7) were sent to potential participants two weeks prior to their appointment at the epilepsy clinic. On receipt of these, participants were invited to contact the researcher if they had any questions about the research. When potential patient participants attended their appointment at the epilepsy clinic, they were approached

by the researcher and asked if they would like to take part in the research. Those who wished to take part were asked to re-appraise the information sheet and sign the consent form (Appendix 8).

The healthy control participants were 27 student and non-academic staff from The University of Sheffield. Control participants were recruited through email (Appendix 9) or recruitment posters (Appendix 10). A recruitment email was sent to all students and non-academic staff at The University of Sheffield through the student volunteers' service. Recruitment posters were placed around university buildings. Potential participants were invited to contact the researcher if they met the inclusion criteria and were interested in taking part. Participants were invited to take part if they met the following inclusion criteria:

- Over the age of 18,
- Sufficient language skills to complete questionnaires,
- Able to give informed consent.

Participants were excluded if they had ever experienced a blackout or seizure. Control participants were sent a participant information sheet (Appendix 11) and invited to attend an appointment at The University of Sheffield two weeks following this. On attendance of the appointment, the participants were asked to re-appraise the information sheet and sign a consent form (Appendix 12).

Participant characteristics.

Table 1 outlines the participant characteristics. A Chi-square test of goodness-of-fit was performed to determine whether the demographic variables were equal between participant groups. Gender ($X^2(2, N = 75) = 7.40$, $p = .03$, *Cramér's V* =.31), age ($X^2(12, N = 75) = 29.03$, $p < .01$, *Cramér's V* =.44) and education level ($X^2(12, N = 75) = 26.76$, $p < .01$,

Cramér's V =.43) were not equally distributed between groups. The PNES and control groups were well matched; there were no statistically significant differences in gender, age, or education level. There were significantly more females in the PNES group compared to the epilepsy group, but no statistically significant differences in age or education level. The epilepsy group had fewer females, were older, and educated to a lower level than the control group. T-tests were conducted to investigate differences in seizure frequency and severity; there were no significant differences in seizure frequency ($t(35) = .50$, $p = .96$, $r = .08$) or severity ($t(39) = -.51$, $p = .96$, $r = .08$) between PNES and epilepsy participants.

Design

This study utilised a between-subject cross-sectional design using self-report questionnaires.

Measures

Demographics.

A demographic questionnaire devised by the researcher was used to obtain information on gender, age and education level (Appendix 13).

Seizure severity.

Seizure frequency and severity was assessed using the Liverpool Seizure Severity Scale-Revised (LSSS-2; Baker, Smith, Jacoby, Hayes, & Chadwick, 1998). The LSSS-2 (Appendix 14) was designed to quantify the frequency and severity of seizures. Frequency of seizures over the past four weeks is recorded and questions regarding the severity of these seizures are completed. The severity scale is termed the ictal scale. The ictal scale has been found to have good internal consistency ($\alpha = .86$). The test-retest scores demonstrated very good reliability ($\rho = .93$, $p < .01$; Baker et al., 1998). The LSSS-2 has been used in other studies with PNES

populations (Whitehead, Kandler, & Reuber, 2013). Scale reliability for the ictal scale across the entire sample in this study was acceptable (α = .74).

Emotion dysregulation.

Emotion dysregulation was assessed using the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2003). This 36-item self-report scale measures overall emotion regulation difficulties and six sub-scales: non-acceptance, goals, impulse, awareness, strategies, and clarity (Appendix 15). The overall DERS score has excellent internal consistency (α = .93) and the subscales have good internal consistency (α > .80; Gratz & Roemer, 2003). Test-retest studies demonstrated good reliability (ρ = .88, p <.01). The DERS is thought to be the measure that captures most of the common conceptualisations of emotion dysregulation in medically unexplained symptoms (Wilkinson, unpublished doctoral thesis). Scale reliability across the entire sample in this study was good (α = .86). Sub-scale reliabilities across the entire sample ranged from poor to good (Nonacceptance = .88; Goals = .52; Impulse = .77; Awareness; .68; Strategies = .77; Clarity = .56).

Table 1: *Sample Characteristics: Frequencies and Percentages, Chi-Square Test of Goodness of Fit and Effect Sizes for Participant Groups*

	PNES[a]		Epilepsy[b]		Control[c]		PNES vs. Controls		PNES vs. Epilepsy		Epilepsy vs. Control	
	N	%	*N*	%	*N*	%	X^2	*CV*[d]	X^2	*CV*	X^2	*CV*
Gender							2.47	.22	7.64**	.43	7.39**	.38
Male	6	(24)	12	(52)	5	(19)						
Female	19	(76)	11	(48)	22	(81)						
Age group							7.70	.39	4.61	.34	7.70	.68
18-21	3	(12.00)	3	(13.04)	1	(3.70)						
22-25	4	(16.00)	2	(8.70)	7	(25.93)						
26-30	6	(20.00)	2	(8.70)	14	(51.86)						
31-40	4	(16.00)	1	(4.35)	1	(3.70)						
41-50	4	(16.00)	5	(21.74)	2	(7.41)						
51-60	2	(8.00)	9	(39.13)	1	(3.70)						
61+	2	(8.00)	1	(4.35)	1	(3.70)						
Education							6.70	.36	6.26	.40	17.94**	.60
<GCSE's	6	(24.00)	6	(26.09)	1	(3.70)						
GCSE's	3	(12.00)	5	(21.74)	3	(11.11)						
A levels	5	(20.00)	7	(30.43)	3	(11.11)						
Diploma	2	(8.00)	2	(8.70)	1	(3.70)						
Degree	7	(28.00)	2	(8.70)	8	(29.64)						
Masters	2	(4.00)	0	(.00)	10	(37.04)						

Note: [a] *N* = 25, [b] *N* = 23, [c] *N* = 27, [d] = Cramérs V effect size, small = .1, medium = .3, large = .5 effects. *p <.05, **p<.01

Trauma history.

Trauma history was measured using the Traumatic Experiences Checklist (TEC; Nijenhuis, Van der Hart, & Kruger, 2002). This is a retrospective 29-item self-report measure of traumatic experiences. The TEC includes questions regarding emotional neglect, emotional abuse, physical abuse, sexual harassment, sexual abuse, and bodily threat. Scores for each item are rated as 1 if they apply and 0 if they do not apply. An example item is "physical abuse (e.g. being hit, tortured, or wounded) by your parents, brothers or sisters". Nijenhuis, Van der Hart and Kruger (2002) found the TEC to have excellent internal consistency (α = .90) and excellent reliability (r = .91, p <.01). The TEC format also allows for trauma area severity scores detailing age at onset, duration of the trauma and subjective response. To reduce question load and potential distress for participants, at the request of the ethics board, the trauma area severity scores were not included in this study. These were removed from the questionnaire and an adapted TEC was produced to include only the "did this happen to you" questions (Appendix 16). The questionnaire has been used in this format in other research in this population (Reuber, Monzoni, Sharrack, & Plug, 2009). Although internal consistency has previously been reported for this measure, the researcher considered it inappropriate given the nature of the scale items (i.e. being sexually abused by a family member may not make it more likely that you were abused by a non-family member).

Anxiety and depression.

The Hospital Anxiety and Depression Scale (HADS; Zigmund & Snaith, 1983) was used to assess anxiety and depression. The HADS is a 14-item questionnaire with two subscales measuring anxiety and depression (Appendix 17). Each subscale contains seven items scored on four point likert scales to indicate degree of psychological distress. A recent literature review (Bjelland, Dahl, Haug, & Neckelman, 2002) reported good internal consistency for

the anxiety and depression subscales with a mean Cronbach's alpha of .83 and .82 respectively. The scale has been found to have good test-retest reliability (ρ = .84, p <.01; Herrmann, 1997). Scale reliability across the entire sample in this study was excellent (α = .94). Sub-scale reliabilities across the entire sample ranged from good to excellent (Anxiety = .92; Depression = .88).

Procedure

After giving informed consent, participants were asked to complete the questionnaires. A quiet room had been made available for patient participants to complete questionnaires in private at the epilepsy clinic. The healthy control participants completed their questionnaires in a room in the Clinical Psychology department of The University of Sheffield. Participants were debriefed following completing the questionnaires and asked if they wished to be informed of the findings. The completion of the questionnaires took approximately 20 minutes. Participants were offered reimbursement for parking.

Ethical considerations

The research proposal was subject to an internal review by the Department of Psychology Research Ethics Committee at The University of Sheffield (Appendix 2). The study was given favourable ethical opinion by the Yorkshire and the Humber Research Ethics Committee (Appendix 3). Research governance approval was provided by the Sheffield Teaching Hospitals NHS Foundation Trust Research Department (Appendix 4).

The researcher was available throughout the research procedure to offer support to participants. All patient participants had the opportunity to discuss any concerns with the researcher or their neurologist at their outpatient appointment. Some of the PNES patients had already received psychotherapy and many were on the psychotherapy waiting list.

Leaflets signposting participants in the right direction for further psychological support were available for all participants.

Statistical approach

The statistical approaches are outlined under the study aims below. An alpha level of .05 was used for all statistical tests. IBM SPSS Statistics for Windows, Version 21.0 (IBM Corporate, 2012) was used for analyses.

Results

Data screening

Normality was assessed for each of the variables through inspection of histograms, interpretation of skewness and kurtosis and interpretation of Kolmogorov-Smirnov tests (Field, 2005). All variables were normally distributed with the exception of the DERS, TEC, and the frequency domain of the LSSS-2. Given the positive skew, log transformations of the skewed scales were performed (Tabachnick & Fidell, 2007). This corrected the distributions. The log transformed data was used for all analyses for the DERS, TEC, and frequency domain of the LSSS-2. There were five missing data points (individual question answers missing) for the DERS data and two for the HADS data, these were excluded listwise due to the relatively small sample.

Correlations

The researcher conducted Pearson product-moment correlations to examine relationships between factors investigated in the sample as a whole, and in the PNES, epilepsy and healthy control groups separately.

In all participant groups, anxiety and depression were significantly correlated with each other. For the sample as a whole, a significant positive correlation was found between emotion dysregulation and traumatic experiences, such that the more traumatic events experienced, the higher the levels of emotion dysregulation the person reported. Significant positive correlations were also found between traumatic experiences, anxiety and depression (the more traumatic events experienced, the higher the levels of anxiety and depression reported; see Table 2).

Table 2

Inter-correlations for the TEC sum, HADS Anxiety, and HADS Depression Scales for the Whole Study Sample

	DERS sum	TEC sum	Anxiety	Depression
DERS sum	----	.31***	.83***	.58***
TEC sum		----	.39***	.29*
Anxiety			----	.70***
Depression				----
Severity				
Frequency				

Note: *p<.05, **p<.01, ***p<.001

For PNES participants, significant positive correlations between emotion dysregulation and anxiety and depression were found. Significantly positive correlations were found between experiences of trauma and depression, but not anxiety. Significant positive correlations were found between depression and seizure severity for PNES participants (see Table 2a).

Table 2a

Inter-correlations for the DERS sum, TEC sum, HADS Anxiety, HADS depression, and LSSS-2 Ictal and Frequency Scales for the PNES Sample

	DERS sum	TEC sum	Anxiety	Depression	Severity	Frequency
DERS sum	----	.28	.89***	.72***	.38	.41
TEC sum		----	.39	.55***	.32	.34
Anxiety			----	.71***	.38	.48
Depression				----	.65*	.31
Severity					----	.32
Frequency						----

Note: *p<.05, **p<.01, ***p<.001

In the epilepsy sample, significant positive correlations were found between emotion dysregulation; anxiety and depression (see Table 2b).

Table 2b

Inter-correlations for the TEC sum, HADS Anxiety, HADS depression, and LSSS-2 Ictal and Frequency Scales for the Epilepsy Sample

	DERS sum	TEC	Anxiety	Depression	Severity	Frequency
DERS sum	----	-.27	.86***	.66***	.44	.05
TEC sum		----	.72	-.13	.35	.07
Anxiety			----	.83***	.47	.16
Depression				----	.20	.05
Severity					----	-.09
Frequency						----

Note: *p<.05, **p<.01, ***p<.001

In the healthy control participants, significant positive correlations were found for all factors investigated; higher levels of emotion dysregulation were significantly correlated with higher levels of traumatic experiences, anxiety, and depression. Furthermore, statistically significant positive correlations were found between experiences of trauma and both anxiety and depression.

Table 2c

Inter-correlations for the TEC sum, HADS Anxiety, and HADS depression Scales for the Healthy Control Sample

	DERS sum	TEC sum	Anxiety	Depression
DERS sum	----	.69***	.72***	.61***
TEC sum		----	.70***	.67***
Anxiety			----	.83***
Depression				----

Note: *p<.05, **p<.01, ***p<.001

Aim 1: Investigating differences between self-reported emotion dysregulation, traumatic experiences, and anxiety and depression between participants with PNES, participants with epilepsy, and healthy controls.

Emotion dysregulation.

Emotion dysregulation was measured using the DERS. Higher scores indicated greater emotion dysregulation. There is no reliable normative data available for this measure and no published clinical cut-offs.

A one-way, between subjects, analysis of variance (ANOVA) was performed to investigate differences in levels of self-reported emotion dysregulation between the three participant groups. The Levene's test indicated that there was no homogeneity of variance; the assumptions of the ANOVA were met. All participants reported some difficulties with emotion dysregulation. There were no significant differences between gender (F(1,68) = .31, p = .58, n^2 = .00), age (F(6,62) = 1.80, p = .11, n^2 = .15) or education levels (F(6,62) = 1.71, p = .13, n^2 = .14) in levels of emotion dysregulation reported.

Table 3 presents the DERS data between participant groups. There were significant differences in overall emotion dysregulation, impulse control, and awareness of emotions

sub-scales of the DERS between the three participant groups. Post-hoc analyses were performed for significant results using Scheffé post-hoc criterion for significance. There were no significant differences between the self-reported emotion dysregulation scores for people with PNES and people with epilepsy. Participants with epilepsy (*M* = 91.10, *SD* = 26.85) and participants with PNES (*M* = 87.86, *SD* = 33.72) reported significantly higher overall emotion dysregulation than the healthy controls (*M* = 71.93, *SD* = 26.68). Differences between PNES and healthy controls did not reach significance (*p* = .07).

People with epilepsy had significantly more difficulties with emotional awareness (*M* = 17.95, *SD* = 4.08) and non-acceptance of emotional responses (*M* = 15.29, *SD* = 6.46) than the healthy controls (*M* = 14.33, *SD* = 4.40; *M* = 10.39, *SD* = 5.48). The PNES group reported significantly higher difficulties in impulse control (*M* = 14.05, *SD* = 7.78) and non-acceptance of emotional responses (*M* = 15.36, *SD* = 7.59) than the healthy controls (*M* = 9.48, *SD* = 5.26).

Table 3

Means, Standard Deviations, F-ratios, and Effect Sizes for the DERS Scores

	PNES *N* = 25	Epilepsy *N* = 23	Control *N* = 27	*F*	n^2 [a]
Sum	87.86 (33.72)	91.10 (26.85)	71.93 (26.68)	3.59*	.10
Non-accept	15.36 (7.59)	15.29 (6.46)	10.93 (5.48)	3.88*	.10
Impulse	14.05 (7.78)	13.19 (6.60)	9.48 (5.26)	4.12*	.11
Goals	14.59 (6.35)	14.52 (5.70)	13.85 (8.97)	.35	.01
Awareness	16.09 (4.87)	17.95 (4.08)	14.33 (4.40)	4.33*	.11
Strategies	17.54 (9.76)	19.05 (7.88)	14.11 (6.55)	1.13	.03
Clarity	8.50 (3.14)	8.86 (3.14)	7.52 (2.94)	1.13	.03

Note: [a]effect size, 0-.1 = weak effect, .1-.3 = modest effect, .3-.5 = moderate effect, >.5 = strong effect, *$p<.05$, Given inadequate to poor internal consistency, all but the DERS sum and the non-acceptance scales should be interpreted with caution.

Traumatic experiences.

Self-reported traumatic experiences were measured using the TEC. Higher scores indicated higher frequency of traumatic events experienced. The majority of participants (81%) reported experiencing at least one traumatic event; 88% of people with PNES, 91% of people with epilepsy, and 63% of healthy controls had experienced at least one traumatic event in their lifetime. A one-way, between subjects, analysis of variance (ANOVA) was performed to investigate differences in reports of traumatic experiences between the three participant groups. The Levene's test indicated that there was no homogeneity of variance; the assumptions of the ANOVA were met. There were no significant differences between gender ($F(1,73) = 1.77$, $p = .19$, $n^2 = .02$), age ($F(1,73) = 2.32$, $p = .06$, $n^2 = .17$), or education level ($F(6,67) = 1.79$, $p = .11$, $n^2 = .14$) considering the sample as a whole.

There were statistically significant differences in the overall self-reported traumatic experiences between the three participant groups. There were also statistically significant differences in the frequency of self-reported experiences of emotional neglect and sexual abuse between the three participant groups (see Table 4). Post-hoc analyses were performed for significant results using Scheffé post-hoc criterion for significance. The findings showed that overall exposure to traumatic experiences was highly elevated in participants with PNES. There were no significant differences between people with epilepsy and healthy controls in their experiences of trauma. Participants with PNES reported significantly higher levels of overall trauma ($M = 5.63$, $SD = 4.73$), emotional neglect ($M = .52$, $SD = .82$), and sexual abuse ($M = .48$, $SD = .77$) than people with epilepsy ($M = 3.22$, $SD = 3.21$; $M = .09$, $SD = .29$; $M = .17$, $SD = .49$) and healthy controls ($M = 2.26$, $SD = 3.51$; $M = .33$, $SD = 1.11$; $M = .11$, $SD = .42$).

Table 4

Means, Standard Deviations, F Ratios and Effect Sizes for the TEC Scores

TEC domains	PNES *N* = 25	Epilepsy *N* = 23	Control *N* = 27	*F*	n^{2a}
Sum	5.64 (4.73)	3.22 (3.21)	2.26 (3.51)	6.62***	.15
Emotional neglect	.52 (.82)	.09 (.29)	.33 (1.11)	3.32*	.08
Emotional abuse	.68 (.75)	.22 (.67)	.89 (3.29)	2.30	.06
Bodily threat	1.04 (1.02)	.65 (.78)	.48 (.89)	2.30	.07
Sexual harassment	.28 (.54)	.13 (.46)	.07 (.27)	1.70	.48
Sexual abuse	.48 (.77)	.17 (.49)	.11 (.42)	3.42*	.08

Note: [a] effect size, 0-.1 = weak effect, .1-.3 = modest effect, .3-.5 = moderate effect, >.5 = strong effect **p<.01

Anxiety and depression.

Self-reported anxiety and depression were measured using the HADS. Higher scores indicated higher levels of self-reported anxiety and depression. The results were first performed using clinical-cut offs (e.g. dichotomous scores indicating the presence or absence of anxiety or depression) and then using HADS scores to represent levels of anxiety and depression.

Clinically significant levels of anxiety and depression.

Forty two percent of the sample reached clinically significant levels of anxiety, 44% reached clinically significant levels of depression, and 39% reached clinically significant levels of both anxiety and depression. A Chi-square test of goodness-of-fit was performed to determine whether there were differences in clinically significant levels of anxiety and depression between gender, age, and education levels of participants. There were no significant differences between genders ($X^2(1, N = 75) = .00$, $p = .98$, *Cramér's V* =.00), ages

($X^2(6, N = 75) = 9.65$, $p = .14$, *Cramér's V* =.37), or education levels ($X^2(6, N = 75) = 4.58$, $p = .60$, *Cramér's V* =.26).

A Chi-square test of goodness-of-fit was performed to investigate differences between the three participant groups. There were statistically significant differences in anxiety ($X^2(2, N = 75) = 10.38$, $p = .01$, *Cramér's V* =.38), depression ($X^2(2, N = 75) = 18.91$, $p < .01$, *Cramér's V* =.52), and those with both anxiety and depression ($X^2(2, N = 75) = 11.14$, $p < .01$, *Cramér's V* =.40) between the three participant groups. Further Chi-square tests of goodness-of-fit were performed to investigate these differences further. None of the post hoc chi-square goodness-of-fit results reached significance (see Table 5).

Table 5

Presence of Clinical Levels of Anxiety, Depression, and Both Anxiety and Depression in the Three Participant Groups: Frequency, Percentages, Chi-Square Values and Effect Sizes

	PNES		Epilepsy		Controls		PNES vs. Control		Epilepsy vs. Control		PNES vs. Epilepsy	
	N	%	N	%	N	%	X^2	CV	X^2	CV	X^2	CV
Anxiety	14	56	11	48	4	15	1.48	.25	1.04	.21	4.51	.31
Depression	14	56	14	61	2	7	1.04	.21	2.85	.35	4.24	.30
Both	13	52	11	48	3	11	5.41	.31	1.87	.39	2.96	.25

Note: effect size, 0-.1 = weak effect, .1-.3 = modest effect, .3-.5 = moderate effect, > 5 = strong effect

Levels of anxiety and depression.

A one-way, between subjects, ANOVA was performed to investigate differences in levels of self-reported anxiety and depression between the three participant groups. There were no significant differences between levels of anxiety between genders ($F(1,69) = .00$, $p = .95$, $n^2 = .00$), age ($F(6,63) = 2.84$, $p = .17$, $n^2 = .21$), or education ($F(6,63) = 2.01$, $p = .07$, $n^2 = .38$) and no significant differences between levels of depression between genders ($F(1,69) = .94$, p

= .34, n^2 = .01), age ($F(6,63) = 3.00$, p = .12, n^2 = .22), or education level ($F(6,63) = 3.28$, p = .07, n^2 = .23). There were statistically significant differences in self-reported anxiety and depression between the three participant groups (see Table 6).

Table 6

Means, Standard Deviations, F-ratios and Effect Sizes for the HADS scores

HADS	PNES *N* = 25	Epilepsy *N* = 23	Control *N* = 27	*F*	n^{2a}
Anxiety	10.09 (6.56)	9.71 (6.49)	4.26 (4.83)	7.60**	.18
Depression	9.71 (6.50)	9.95 (5.00)	3.26 (3.43)	14.32**	.30

Note: *p<.05, **p<.001, [a] effect size, 0-.1 = weak effect, .1-.3 = modest effect, .3-.5 = moderate effect, > 5 = strong effect

Post-hoc analyses were performed for the significant results using Scheffé post-hoc criterion for significance. There were no significant differences between levels of self-reported anxiety or depression between participants with PNES and participants with epilepsy. Participants with PNES and participants with epilepsy reported significantly higher levels of anxiety (M = 10.09, SD = 6.56; M =9.71, SD = 6.49) and depression (M = 9.71, SD = 6.50; M =9.95, SD =5.00) than the control group (M = 4.26, SD = 4.83; M = 3.26, SD = 3.43).

Aim 2: To investigate the extent to which variance in self-reported emotion dysregulation is associated with experiences of trauma, anxiety, and depression in the three groups of participants (patients with PNES, patients with epilepsy, and healthy controls). Seizure frequency and severity will also be considered in patients with PNES or epilepsy.

Hierarchical multiple regression analyses were performed to explore the extent to which the independent variables (anxiety, depression, seizure frequency and severity, and

experiences of trauma) accounted for variation in emotion dysregulation (the dependent variable) in participants with PNES, participants with epilepsy, and healthy control participants. Levels of anxiety as opposed to clinical cut offs were used in all further analyses.

Prior to performing a hierarchical multiple regression analysis, the assumptions of the statistical analysis were tested. Firstly, inter-correlations between predictor variables were assessed for each participant group (see Tables 2, 2a and 2b). There were some significant correlations between predictor variables. However, as the collinearity statistics (VIF and tolerance) were within acceptable limits, the assumption of multi-collinearity was considered to have been met (Field, 2005). Sample sizes of 25 (PNES), 23 (epilepsy) and 27 (healthy control) were deemed adequate as 5 (3 for the control group) independent variables were to be entered into the analysis, based on the minimum sample size requirement of five to one (Tabachnick, & Fidell, 2001). Mahalanobis distance scores indicated no multivariate outliers and scatter and residual plots indicated that the assumptions of normality, linearity and homoscedasticity were satisfied (Pallant, 2001). The Durbin-Watson statistics indicated that the assumption of independent errors was acceptable (Field, 2005).

Predictor variables were entered into the model in their order of theoretical importance and guided by preliminary analyses. In preliminary analyses, all possible independent variables were entered into the regression analysis independently. Only anxiety emerged as a significant predictor, this was therefore a known predictor and was entered into the model first. New predictors were then entered hierarchically in accordance with the researcher's hypotheses. Depression, seizure frequency and severity and trauma history were then entered into the model. These were entered into the regression analysis last so that the extent to which they predicted variance in emotion dysregulation scores could be investigated once known variables had been controlled for.

PNES.

For the PNES participants, the full model of anxiety, depression, seizure frequency and severity, and trauma history, to predict emotion dysregulation (model 4) was statistically significant, $R^2 = .82$, $F(5,19) = 17.29$, $p < .001$, $f^2 = 4.56$; adjusted $R^2 = .77$. Following the addition of anxiety in model 1 which was statistically significant $R^2 = .79$, $F(1,23) = 86.70$, $p < .001$, $f^2 = 3.76$; adjusted $R^2 = .78$, and accounted for 79% of variance in emotion dysregulation, the addition of depression added 1% of variance, seizure frequency and severity added no further variance, and trauma history added an additional 2 % of variance. None of these increases in variance reached significance, only anxiety made a significant contribution to the final regression equation. Table 8 gives details of the regression models.

Table 8

Hierarchical Multiple Regression Predicting Emotion Dysregulation from Anxiety, Depression, Seizure Severity and Frequency, and Trauma History in PNES Participants

	Emotion dysregulation							
	Model 1		Model 2		Model 3		Model 4	
Variable	B	ß	B	ß	B	ß	B	ß
Constant	45.46		43.41		44.36		46.72	
Anxiety	4.39	.89	3.88	.79	3.90	.79	3.84	.78
Depression			.82	.15	.78	.14	1.30	.23
Ictal[c]					.00	.00	-.01	-.02
Freq[d]					-.02	-.01	-.01	-.01
Trauma							-1.10	-.17
R^2	.79		.80		.80		.82	
F	86.70***		44.25***		2.01***		17.29***	
ΔR^2	.79		.01		.00		.02	
ΔF	86.70*		1.17		.01		1.97	

Note. $N = 25$. *$p < .05$, **$p < .01$, ***$p < .001$

Epilepsy.

For the epilepsy participants, the full model of anxiety, depression, seizure frequency and severity, and trauma history to predict emotion dysregulation (model 4) was statistically significant, $R^2 = .55$, $F(5,17) = 5.76$, $p < .001$, $f^2 = 1.22$; adjusted $R^2 = .52$. Anxiety accounted for 55% of the variance in emotion dysregulation (model 1). Depression, seizure frequency and severity, and traumatic experiences added no further statistically significant variance. Only anxiety made a significant contribution to the final regression equation. Table 9 gives full details of the regression models.

Table 9

Hierarchical Multiple Regression Predicting Emotion Dysregulation from Anxiety, Depression, Seizure Severity and Frequency, and Trauma History in Epilepsy Participants

	Emotion dysregulation							
	Model 1		Model 2		Model 3		Model 4	
Variable	B	ß	B	ß	B	ß	B	ß
Constant	59.43		62.58		57.68		59.98	
Anxiety	3.14	.74	3.61	.86	3.91	.94	4.18	.99
Depression			- .77	-.13	-1.05	-.53	-1.48	-.26
Ictal[c]					-.05		-.39	-.06
Freq[d]					.14		.27	.14
Trauma							-1.94	-.25
R^2	.55		.56		.57		.63	
F	26.06**		12.71**		5.96		5.76	
ΔR^2	.55		.01		.01		.06	
ΔF	26.07**		.26		.22		2.69	

Note. $N = 23$. [a] = depression, [b] = psychopathology, [c] = seizure severity, [d] = seizure frequency.*$p < .05$, **$p < .01$

Healthy controls.

For the healthy control participants, the full model of anxiety, depression, and trauma history to predict emotion dysregulation (model 3) was statistically significant, $R^2 = .59$, $F(4,22) = 3.23$, $p < .001$, $f^2 = 1.44$; adjusted $R^2 = .54$. Anxiety accounted for 59% of the variance in emotion regulation scores (model 1). Depression and trauma added no further statistically significant significance. Only anxiety made a significant contribution to the final regression model. Table 10 gives full details of the regression models.

Table 10

Hierarchical Multiple Regression Predicting Emotion Regulation from Anxiety, Depression, and Trauma History in Healthy Control Participants

	Emotion dysregulation					
	Model 1		Model 2		Model 3	
Variable	B	ß	B	ß	B	ß
Constant	54.90		54.72		54.99	
Anxiety	4.00	.72	3.84	.70	2.92	.53
Depression			.26	.34	-.53	.07
Trauma					2.75	.36
R^2	.52		.52		.63	
F	27.50**		13.22**		10.98**	
ΔR^2	.52		.52		.59	
ΔF	27.50**		.02		3.62	

Note. $N = 27$.*$p < .05$, **$p < .01$

All regression analyses were checked for mediation using the "four steps in establishing mediation" method (Baron & Kenny, 1986). No mediators were identified. For all hierarchical multiple regression analyses, gender, age and education were entered into an initial model. None had a predictive value of over 5%. Due to limitations in the number of

predictor variables that could be entered, they were eliminated before the final analyses were carried out.

Hierarchical multiple regression for TEC subscales and non-acceptance of emotions.

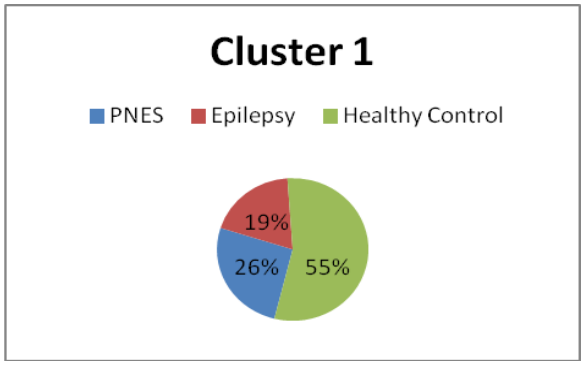
The same method of entry was used to investigate the extent to which variations in emotion dysregulation was associated with experiences of emotional neglect, emotional abuse, bodily threat, sexual harassment, and sexual abuse. In addition, the extent to which variance in non-acceptance of emotions (a DERS sub-scale) was associated with the independent variables was investigated (other DERS subscales were not included due to inadequate to poor internal consistency).

For all traumatic experiences in all participant groups, only anxiety significantly predicted variance in emotion dysregulation. The exception to this was experiences of emotional abuse for participants with PNES; both anxiety and experiences of emotional abuse significantly predicted variance in emotion dysregulation (R^2 = .98, $F(5,4) = 38.34$, p <.001, f^2 =49; adjusted R^2 = .95). Anxiety accounted for 91% of the variance in emotion dysregulation, depression and seizure frequency and severity added no further variance, experiences of emotional abuse added a further 7% of variance which reached significance (p = .02). Only anxiety predicted variance in difficulties in non-acceptance of emotions in all participant groups (see Appendix 18).

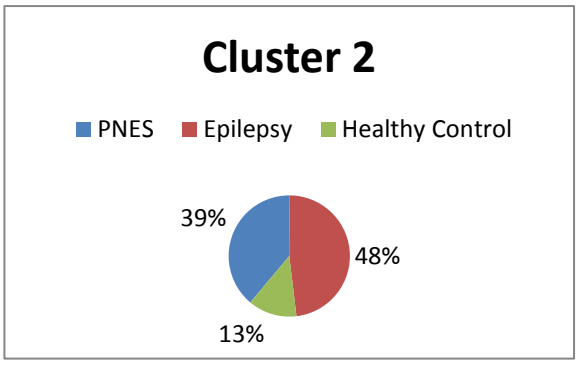
Aim 3: To explore potential clusters of participants characterised by levels of emotion dysregulation, traumatic experiences and anxiety and depression which could be investigated further with larger sample sizes.

Agglomerative hierarchical cluster analysis based on between-groups linkage using Squared Euclidean Distance as the distance measure was performed on all total scores for the

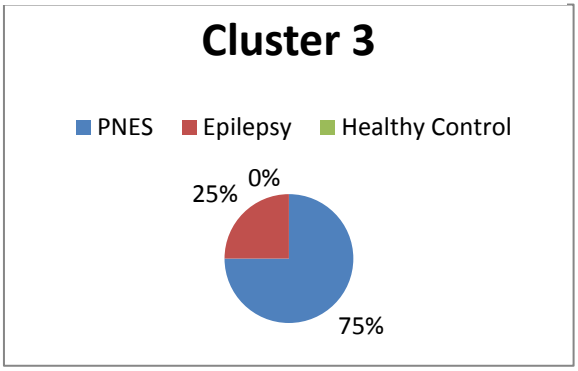
DERS, TEC, and HADS data for all participants. Z-transformed values were used to remove the effects of scaling differences. Three clusters emerged from the analysis; Cluster 1 characterised by primarily healthy control participants, cluster 2 by epilepsy participants and cluster 3 by PNES participants (see Figure 1).



Characterised by low levels of emotion dysregulation, traumatic experiences, anxiety and depression



Characterised by high levels of emotion dysregulation, medium levels of traumatic experience and high anxiety and depression



Characterised by medium levels of emotion dysregulation, high levels of traumatic experience, medium levels of anxiety and high levels of depression

Figure 1: Pie Charts Illustrating the Percentage of PNES, Epilepsy, and Healthy Control Participants in Each Cluster.

One-way, between subjects ANOVA's were performed to investigate differences in levels of self-reported emotion dysregulation, experiences of trauma, anxiety, and depression between the three PNES clusters. There were significant differences in the total emotion dysregulation, traumatic experiences, anxiety, and depression between the three PNES clusters (Table 11 outlines the results). The three clusters did not differ with respect to gender, age, or education level.

Post-hoc analyses were performed using Scheffé post-hoc criterion for significance for significant one-way ANOVA results. Cluster 1 had significantly lower levels of emotion dysregulation (M = 64.83, SD = 15.59) than cluster 2 (M = 113.78, SD = 22.29) which had significantly higher levels of emotion dysregulation than cluster 3 (M = 75.50, SD = 20.57). There were no significant differences between cluster 1 and cluster 3. Cluster 2 had significantly higher levels of depression (M = 13.13 SD = 3.00) than cluster 1 (M = 3.09 SD = 2.86) and cluster 3 (M = 11.25 SD = 2.87). There were no significant differences in the reported levels of depression between cluster 1 and cluster 3. There were significant differences between all clusters on the number of traumatic experiences and the levels of anxiety.

Table 11

Means, Standard Deviations, F-ratios, and Effect Sizes for Cluster Scores

	Cluster 1 N = 42	Cluster 2 N = 23	Cluster 3 N = 4	F	n^2 [a]
ED	64.83(15.59)	113.78(22.29)	75.50(20.57)	53.26***	.62
Trauma	1.97(1.94)	4.78(4.22)	14.50(2.65)	35.92***	.52
Anxiety	3.33(2.85)	15.65(3.08)	8.75(6.13)	113.78***	.77
Depression	3.09(2.86)	13.13(3.00)	11.25(2.87)	93.07***	.73

Note: [a]effect size, 0-.1 = weak effect, .1-.3 = modest effect, .3-.5 = moderate effect, <.5 = strong effect, *p<.05, **p<.01, ***p<.001.

Power analysis.

A post-hoc power analysis was conducted using the software G*Power 3 (Faul, Erdfelder, Lang, & Buchner, 2007). For the ANOVA analyses, the power for the total scores for the DERS, TEC, HADS anxiety, and HADS depression scales was 71%, 90%, 93%, and 99% respectively. For the hierarchical multiple regression analyses, the power of findings was 99% for all participant groups.

Discussion

The present research investigated the association between emotion dysregulation and traumatic experiences in PNES. The researcher hypothesised that participants with PNES would have significantly higher levels of emotion dysregulation and would have experienced significantly more traumatic events than the participants with epilepsy and the healthy controls. Furthermore, participants with PNES and participants with epilepsy would report similar levels of anxiety and depression, both higher than the healthy controls. Furthermore, the researcher hypothesised that in participants with PNES, emotion dysregulation would be associated with traumatic experiences. The results provided some support for these hypotheses. Exploratory cluster analyses were also performed to identify potential clusters of participants.

There were no statistically significant differences between people with PNES and people with epilepsy in their self-reported levels of emotion dysregulation. Participants with epilepsy and participants with PNES reported significantly higher levels of emotion dysregulation than healthy controls, but the difference in the level of emotion dysregulation was only significant between participants with epilepsy and healthy controls. As hypothesised, the participants with PNES reported significantly higher levels of traumatic experiences than participants with epilepsy and healthy controls. There were no significant differences between

participants with PNES and participants with epilepsy in levels of self-reported anxiety and depression; both reported higher levels than healthy controls. Variation in emotion dysregulation was explained by levels of anxiety, not traumatic experiences, in all participant groups. Three clusters were identified in the cluster analysis characterised by levels of emotion dysregulation, traumatic experiences, anxiety, depression and diagnosis.

Emotion dysregulation in people with PNES, people with epilepsy, and healthy controls

To the researcher's knowledge, only two studies have compared levels of emotion dysregulation between people with PNES and people with epilepsy; Reuber et al., (2004) and Brown et al. (unpublished doctoral thesis). Given the discrepancies between the way in which emotion dysregulation has been conceptualised in this study and the study by Reuber et al. (2004), the results of this study may only be comparable with the Brown et al. (unpublished doctoral thesis) findings. The results of this study supported the findings of Brown et al. (unpublished doctoral thesis) who found no statistically significant differences in overall levels of emotion dysregulation between participants with PNES and participants with epilepsy, as measured by the DERS.

The finding relating to emotion dysregulation in epilepsy is interesting and warrants further investigation in larger scale studies. To the researcher's knowledge, no studies to date have specifically investigated emotion dysregulation in epilepsy (although some studies have investigated emotional intelligence and alexithymia). However, studies have investigated affective disorders in epilepsy, and the findings of these studies may also relate to emotion dysregulation. Studies have investigated affective disorders in epilepsy in relation to the distress of experiencing seizures (De Souza & Salgado, 2006; Vasquez & Devinsky, 2003) and organic causes of affective disorders due to structural abnormalities in the brain (Charney, 2003; Trimble & Van Elst, 2003). Structures which may be relevant in affective

disorders (e.g. the amygdala) are also central in the regulation of emotions (Davidson, Putnam, & Larson, 2000; Goldin, McRae, Ramel, & Gross, 2008) and commonly implicated in focal epilepsy. Emotion dysregulation in epilepsy may therefore be organic in nature. In addition, given findings regarding associations between anxiety and emotion dysregulation, it may be that the comparable levels of anxiety in the epilepsy and PNES samples explain the levels of emotion dysregulation.

On the whole, studies have found higher levels of emotion dysregulation in PNES than in healthy controls (Reuber et al., 2004; Roberts et al., 2012). These finding were only partially supported in this study. Whilst emotion dysregulation was higher in the PNES sample, this did not reach significance. Furthermore, the effect sizes for significant results were largely weak. The researcher has considered possible reasons for these disparate results.

Firstly, lower than expected scores on measures of psychological or emotional distress are often thought to be due to emotional avoidance and/or failure to recognise psychological distress in PNES. Avoidance tendencies have been documented in PNES (Goldstein & Mellers, 2006) and may represent a trait in these patients. Patient participants in this study were recruited from a seizure clinic as opposed to a psychological facility. The patient participants may have therefore represented a treatment refractory group of patients (following diagnosis and psychological therapy, they were still having seizures). Lack of insight and acceptance in this participant sample may have therefore been more probable than in people with PNES recruited from other settings. However, there were no statistically significant differences in non-acceptance of emotions as measured by the DERS between participants with PNES and participants with epilepsy. In addition, non-acceptance of emotion did not significantly account for variance in levels of emotion dysregulation in PNES participants.

Related to the insight are possible issues with the emotion dysregulation measure used in this study. There may be limitations inherent in the use of self-report tools to measure emotion dysregulation. The extent to which individuals can accurately self-report on their emotion regulation strategies has certainly been questioned (Koster, Soetens, Braet, & De Raedt, 2008; Robinson & Clore, 2002). Deficiencies of the DERS may have led to under-reporting of emotion dysregulation in all (or some of the) samples. In addition, the fact that participants psychotherapy history was not controlled for may have confounded the results. Participants with PNES may have received more psychotherapy than the other participant groups; they may have therefore learned emotion regulation strategies and developed more insight into these strategies. This may have resulted in them scoring more highly on the DERS. Furthermore, due to inadequate to poor internal consistency on all but the DERS sum and non-acceptance subscales, the DERS sum was used for the majority of analyses. This may not have been a true reflection of participants' emotion dysregulation as difficulties in particular strategies may have yielded different results.

In addition, the PNES sample was investigated as a whole group, not considering possible sub-groups of PNES participants. Research suggests that there are different sub-groups of participants characterised by different levels of emotion dysregulation (Brown et al., unpublished doctoral thesis; Reuber et al., 2004); investigating this heterogeneous population as a whole may have therefore confounded the results.

Whilst anxiety and depression were measured, other psychiatric diagnoses were not accounted for. Levels of emotion dysregulation in the three participant groups may have therefore been related to co-morbid psychiatric disorders as opposed to being a result of the factors investigated in this study.

Finally, given the relatively small sample size and the fact that differences between people with PNES and healthy controls neared significance, this may have simply been a false negative finding. Replication with a larger sample size would therefore be recommended.

Experiences of trauma in people with PNES, people with epilepsy, and healthy controls

As hypothesised, the participants with PNES reported significantly higher levels of traumatic experiences than participants with epilepsy and healthy controls. This fits with the findings of other studies reporting higher overall levels of traumatic experiences in patients with PNES (Myers, Perrine, Lancman, Fleming, & Lancman, 2013; Sharpe & Faye, 2006).

Participants with PNES reported significantly higher levels of emotional neglect and sexual abuse than participants with epilepsy and healthy control participants, the other traumatic experiences investigated did not reach significance. The relationship between experiences of sexual abuse in PNES has been widely reported in the literature (Bowman, 1993; Rosenberg, Rosenberg, Williamson, & Woodford, 2000) as has the relationship between emotional neglect and PNES (Procenca, Castro, Jorge, & Marchetti, 2011).

Limitations in the trauma measure used may have resulted in some traumatic experiences not reaching significance and the effect sizes being largely weak to moderate. The removal of the qualitative aspect of the trauma measure may have resulted in people not answering "yes" to particular questions as they felt the question did not wholly apply to them. Giving the individualised nature of peoples traumatic experiences this is highly probable.

Anxiety and depression in people with PNES, people with epilepsy, and healthy controls

Significant positive correlations were found between anxiety and depression in all participant groups. However, anxiety and depression were independently associated with the

psychological factors investigated. Levels of anxiety were significantly associated with levels of emotion dysregulation in all participant groups. Only depression was significantly correlated with trauma and with severity of seizures in PNES.

Given the prevalence of anxiety (Alper, 1994; Bowman & Markand, 1996) and depression (Lempert & Schmidt, 1990; Mökelby et al., 2002) in PNES and in epilepsy (Tojek et al., 2000; Wood, McDaniel, Burchfiel, & Erba, 1998), the researchers hypothesised that the anxiety and depression levels would be higher in participants with PNES and participants with epilepsy than controls but that the two patient groups would not differ significantly from each other. This hypothesis was supported for levels of anxiety of depression in the sample. The researcher also investigated clinical cut-offs of anxiety and depression in the sample, whilst there were differences between groups, these did not remain significant in post-hoc tests. This may suggest that whilst levels of anxiety and depression are aetiological factors in PNES, they may not necessarily reach clinically significant levels.

The study provided support for other studies that have found little or no difference between anxiety and depression in people with PNES and people with epilepsy (Arnold & Privitera, 1996; Wood, McDaniel, Burchfiel, & Erba, 1998). Whilst this may be an accurate reflection of levels of anxiety and depression in PNES and epilepsy, it may be that self-report measures of anxiety and depression are less reliable in patients with seizures as the experience of having seizures may account for some of the replies given. Question 6 of the HADS is a good example: "I get palpitations, or sensations of 'butterflies' in my stomach or chest", could refer to anxiety symptoms or symptoms of an epileptic seizure aura (Manford & Shorvon, 1992). The construct validity of such measures in this population may therefore be limited.

Whilst the levels of depression were similar in participants with PNES and participants with epilepsy, it was only in the participants with PNES that depression was significantly correlated with trauma and with severity of seizures. This is an interesting finding and may suggest that the way in which depression interacts with other aetiological factors in PNES, may be unique to the disorder.

Explaining levels of emotion dysregulation in PNES, epilepsy, and healthy controls

Whilst the cross-sectional nature of this study did not allow us to infer causality, the researcher hypothesised that in PNES, emotion dysregulation would be associated with experiences of trauma. This hypothesis was not supported. Only anxiety significantly accounted for variance in levels of emotion dysregulation in the three participant groups. Anxiety accounted for 79%, 55%, and 59% of variance in emotion dysregulation as measured by the DERS for participants with PNES, participants with epilepsy, and healthy controls, respectively.

There are a number of possible explanations for these findings. Firstly, the findings may be due to limitations in the traumatic experience measure used. The trauma measure did not grade scores on severity of traumatic experience. For instance, people scoring on questions relating to emotional abuse (e.g. being belittled, teased, or unjustly punished by your parents, brothers or sisters) or questions relating to sexual abuse (e.g. unwanted sexual acts involving physical contact by your parents, brothers, and sisters) would both be rated as having the same amount of traumatic experience where subjectively, experiences of sexual abuse may be considered more traumatic. A further limitation of the TEC relates to the adapted version of used in this study. Whilst the measure in its original form allows for exploration of the age at which the traumatic event happened and the subjective impact this event had on the person, in the current form, only whether or not the event was experienced is investigated. The use of

the measure in the original form would have enabled the researcher to explore the severity of the traumatic experience (as rated by the participant) and the age at which the trauma was experienced may have resulted in different findings. The age at which the trauma occurred may be particularly significant given findings suggesting that earlier experiences of trauma result in greater emotion regulation difficulties (Cole, Michel, & Teti, 1994; Van der Kolk, 2005).

A second possibility is limitations in the way in which emotion dysregulation was investigated. It is possible that the relationship between emotion-regulation strategies and psychopathology may be inflated due to item overlap. The DERS correlated highly with anxiety, this may suggest that the DERS is measuring anxiety as well as emotion dysregulation. Researchers have suggested that the assessment of self-reported emotion may be confounded by distress (Stanton, Dannof-Burg, Cameron, & Ellis, 1994). However, the DERS also correlated highly with depression and traumatic experiences and these factors did not add any statistically significant variance to the final models.

Anxiety is widely viewed as being the result of difficulties in regulating emotions (Campbell-Sills & Barlow, 2007). This may explain the relationship between anxiety and emotion dysregulation in this sample (discussed further in the theoretical implication section). Finally, the possibility of this being a false negative finding due to the relatively small sample size should be considered.

Clusters of participants

Cluster analysis revealed three identifiable subgroups within the whole sample. Cluster 1 characterised by primarily healthy control participants, lower levels of emotion dysregulation, low levels of trauma and low levels of anxiety and depression. Cluster 2 was characterised by primarily participants with epilepsy, high levels of emotion dysregulation, medium levels of

trauma, and high anxiety and depression. Cluster 3 was characterised by primarily PNES participants, medium emotion dysregulation, high levels of traumatic experiences, medium levels of anxiety and higher levels of depression.

To the researcher's knowledge, this is the first study to investigate clusters of PNES, epilepsy and healthy control participants together. The three clusters that emerged were primarily characterised by diagnosis and by patterns of symptoms. Given the relatively small sample size and the small cluster numbers (particularly cluster 3), these results should be interpreted with caution and should be replicated with larger sample sizes.

Theoretical implications

Only relatively recently have empirical studies been conducted to determine the nature of emotion dysregulation in PNES. The findings of this study add to our understanding of the wider aetiology of PNES and more specifically, of emotion dysregulation in PNES and in epilepsy.

Reuber (2009) proposed a multifactorial model of PNES aetiology. The model proposes that there are several interacting causes in PNES which can be categorised into predisposing factors, perpetuating factors, and triggering factors. Trauma has been suggested as a possible predisposing factor in PNES. The presence of elevated levels of experiences of trauma in the participants with PNES in this study supports this notion. Researchers suggest that childhood traumatic experiences can be linked to other precipitating and perpetuating factors causing PNES to develop later in life (Holman, Kirkby, Duncan, & Brown, 2008; Salmon, Al-Marzooqi, Baker, & Reilly, 2003). One of the factors that have been suggested is emotion dysregulation (Bakvis et al., 2009). This association has been well supported (Heim & Nemeroff, 2001; Schore, 2001, 2002; Van der Kolk, 2005), this therefore seems plausible. However, this study found no support for this.

Having said that, the findings do provide support for the notion that traumatic experiences are a risk factor in PNES (Duncan & Oto, 2008; Harden, 1997). The over-representation of trauma in the PNES group supports theories of PNES being a form of dissociation, keeping the traumatic memory out of conscious awareness (Fiszman et al., 2004). Experiences of trauma were the only factor investigated that differentiated people with PNES from people with epilepsy; although there were no significant differences in levels of anxiety, depression and emotion dysregulation between people with PNES and people with epilepsy. Also, cluster analysis identified a cluster of primarily PNES participants characterised by high levels of traumatic experience. This may suggest that anxiety, depression and emotion dysregulation may have different causes in people with PNES, people with epilepsy and healthy controls and there may be unique patterns of symptoms characteristic in each diagnosis (as demonstrated in the cluster analysis). The fact that there was a correlation between trauma and emotion dysregulation in the healthy controls but not in other participant groups would be in keeping with this explanation. However, this adds no strength to the hypothesis that trauma causes emotional dysregulation in people with PNES. There was no correlation in the PNES participant group between trauma and emotion dysregulation which is thought to underpin PNES by many.

The findings of this study suggested that levels of anxiety are associated with emotion dysregulation. Whilst the direction of this relationship cannot be inferred, the results provide support for emotion dysregulation models of anxiety (e.g. Mennin et al., 2007). These theories postulate that general anxiety disorders are characterised by dysfunctional emotion regulation strategies (Campbell-Sills & Barlow, 2007; Mennin et al., 2007). This would explain why comparable levels of emotion dysregulation were reported in PNES and in epilepsy, both of which reported high levels of anxiety.

A further implication comes from findings regarding the association between trauma and depression in people with PNES. Trauma was significantly correlated with depression and depression was correlated with seizure severity in the PNES group. In addition, the cluster made up of primarily PNES participants was characterised by high levels of traumatic experiences and depression. Although causality cannot be inferred, this may suggest that experiences of trauma result in depression in these patients which in turn is related to somatisation (i.e. severity of seizures). This warrants further investigation utilising longitudinal methodology.

Clinical implications

Although most experts consider PNES to be psychogenic, many patients perceive their problems as physical and can struggle to understand the relevance of emotional difficulties to their attacks (Monzoni, Duncan, Grünewald, & Reuber, 2011; Stone, Binzer & Sharpe, 2004). Psychological treatment is recommended by most experts (LaFrance, Rusch & Machan, 2008). This study adds to existing literature regarding the aetiology of PNES. Further understanding of the aetiology of the disorder may help clinicians to explain the condition to patients which may improve outcome (Ettinger, Devinsky, Weisbrot, Ramakrishna, & Goyal, 1999; Monzoni, Grünewald, & Reuber, 2011) and to increase acceptance of the psychological basis of the disorder and thus increase acceptance of psychological interventions (Reuber & Elger, 2003).

A further implication comes from findings regarding the role of anxiety. Researchers have suggested that psychotherapy focusing on anxiety in PNES is beneficial to PNES participants (Cramer & Brandenburg, 2005). This research supported the importance of the role of anxiety in PNES and in epilepsy. In PNES, the interaction between factors is an important focus of therapy and researchers have suggested that the interaction of factors is more readily

addressed in PNES therapy than the specific factors themselves (Carson et al., 2012). Alongside psychoeducation, the association between anxiety and emotion dysregulation (and depression and seizure severity in PNES) could be an initial target for therapy in PNES. Furthermore, research suggests that once perpetuating factors have been addressed and seizures have been reduced, it is important to address the underlying issue to improve quality of life and reduce social and financial dependence (Reuber, Mitchell & Elger, 2005). The findings of this study suggest that trauma may be the underlying issue in PNES.

The findings also highlighted the importance of psychological therapy for people with epilepsy. Anxiety disorders can go undetected and untreated in epilepsy (Beyenberg et al., 2005; Devinsky, 2003), yet high anxiety levels may lead to a higher frequency of epileptic seizures (Thapar, Kerr & Harold, 2009; Vazquez & Devinsky, 2003). The initial stages of therapy for PNES focusing on psychoeducation (e.g. the effects of anxiety and seizures on epilepsy) and emotion regulation skills training may therefore also be beneficial for patients with epilepsy as well as patients with PNES.

Limitations

The findings should be considered in light of several limitations. Firstly, the data were based on self-reports and were not validated by physical or psychiatric examination; therefore the results may be subject to recall bias, social desirability bias and demand characteristics. This may have been particularly salient given the sensitive topic of traumatic experiences and the potential for people with PNES to view the disorder as stigmatising (Freidl et al., 2007; Stone et al., 2003). Furthermore, self-report measures of emotion dysregulation are limited in the extent to which they can measure automatic, physiological responses to emotions which would be considerably harder to self-report.

Whilst the PNES and control groups were well matched, the PNES and epilepsy groups were less well matched. This is likely to be due to the large amount of females in the PNES sample. Given that three quarters of people with PNES are women (Lesser, 1996), this is likely to be representative of the PNES population. Matching on all participant variables may have led to selected and ungeneralisable samples.

Data regarding seizure types was not available for all of the participants with epilepsy; as a result, comparisons between seizure types could not be made. Given the associations between seizure types, brain foci and psychological presentations, this is a limitation of the study. Future, larger scale research should therefore consider comparisons between the psychological presentations of people with PNES and people with different epilepsy seizure types.

Whilst levels of anxiety and depression were investigated in all participant groups using a self-report measure, formal psychiatric diagnostic data were not available. This may have limited the external validity and generalisability of the findings. In future larger scale studies, psychiatric diagnoses should be controlled for in all participant groups.

"Reliable data regarding participants receiving psychotherapy was available for some, but not all participants. Furthermore, the researchers could not rule out the possibility of participants having received psychotherapy outside of the NHS. As a result, researchers could not control for this factor in the analysis. It is likely that those who had engaged in psychotherapy may have learned emotion regulation strategies and may have more insight into the strategies they adopt, being more able to report them accurately. This may have limited the validity and reliability of the findings, future research should control for psychotherapy history to overcome this limitation".

Additionally, the sample size of the study was relatively small and some effect sizes were weak. However, it is comparable with sample sizes in similar studies (Roberts et al., 2012; Tojek, Lumley, Barkley, Mahr, & Thomas, 2000) and the power analysis demonstrated generally high levels of power. High power was demonstrated for the TEC and DERS data, reducing the probability of type II errors. However, the power for the HADS data was lower. This is a limitation of the study and suggests that higher sample sizes would be needed in future research.

Finally, the study was cross-sectional; therefore causality between the factors investigated could not be inferred.

Conclusions and future research directions

Although preliminary, these findings add to our understanding of the aetiology of PNES. This was the first study to investigate emotion dysregulation using a dedicated measure of the construct comparing levels between people with PNES, people with epilepsy, and healthy controls. In addition, this was the first study to investigate association between experiences of trauma and emotion dysregulation in this patient group and the first to conduct a cluster analysis with PNES, epilepsy, and healthy control participants in one analysis. Whilst most studies describe group differences rather than the positive or negative predictive value of a particular feature (Reuber & Elger, 2003), this study furthered our understanding of emotion dysregulation by investigating which psychological factors were associated with emotion dysregulation.

The research highlighted interesting findings including the similarity in levels of emotion dysregulation between patients with PNES and patients with epilepsy, associations between trauma and depression, and depression and seizure severity in people with PNES, and the

unique symptom clusters specific to people with PNES, people with epilepsy, and the healthy population.

It appears that common in PNES, epilepsy and healthy controls is the association between anxiety levels and emotion dysregulation. Whilst experiences of trauma were much higher in people with PNES and is clearly an aetiological factor in the disorder, this study provides no evidence suggesting that experiences of trauma are related to self-reported emotion dysregulation in PNES.

Further research is needed in the area to investigate relationships between emotion dysregulation, traumatic experiences, anxiety, and depression. Future research should utilise physiological measures of emotion dysregulation to corroborate self-reported emotion dysregulation data. In addition, trauma should be investigated more thoroughly, investigating the age of trauma and the severity of the trauma more comprehensively using longitudinal methodology.

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Appendices

Appendix 1

A note about ethics approval

On the ethics approval letters and participant information sheets and consent forms, there is mention of measuring Heart Rate Variability (HRV). Due to issues with the equipment used to calculate the HRV from the obtained ECG outputs and a number of participants agreeing to complete the questionnaires but declining to have an ECG, this part of the project was not included in the thesis. Reference to HRV and to the measures associated with this (caffeine consumption questionnaire), are therefore not relevant to this project.

Appendix 2

University of Sheffield Ethics Approval Letter

The University Of Sheffield.

Department Of Psychology.
Clinical Psychology Unit.

Doctor of Clinical Psychology (DClin Psy) Programme
Clinical supervision training and NHS research training & consultancy.

Clinical Psychology Unit
Department of Psychology
University of Sheffield
Western Bank
Sheffield S10 2TP UK

Telephone: 0114 2226570
Fax: 0114 2226610
Email: dclinpsy@sheffield.ac.uk
Please address any correspondence to Ms. Christie Harrison, Research Support Officer

9th March 2012

To: Research Governance Office

Dear Sir/Madam,

RE: Confirmation of Scientific Approval of enclosed Research Project

Project title: Investigating associations between autonomic function and emotions in people with or without seizures

Investigators: Katy Wilkinson (DClin Psy Trainee, University of Sheffield); Dr Claire Isaac (Academic Supervisor, University of Sheffield); Dr Markus Reuber (NHS supervisor; Royal Hallamshire Hospital)

I write to confirm that the enclosed proposal forms part of the educational requirements for the Doctoral Clinical Psychology Qualification (DClin Psy) run by the Clinical Psychology Unit, University of Sheffield.

Three independent reviewers appointed by the Clinical Psychology Unit Research Sub-committee have scientifically reviewed it.

I can confirm that all necessary amendments have been made to the satisfaction of the reviewers, who are now happy that the proposed study is of sound scientific quality. Consequently, the University will also be happy to indemnify it and to act as research sponsor once ethical approval has been gained.

Given the above, I would remind you that the Unit already has an agreement with your office to exempt this proposal from further scientific review. However, if you require any further information, please do not hesitate to contact me.

Yours sincerely

Dr. Andrew Thompson
Director of Research Training

Cc. Katy Wilkinson; Dr Claire Isaac; Dr Markus Reuber

Appendix 3: Yorkshire and the Humber Research Ethics Committee Approval

NHS
Health Research Authority

NRES Committee Yorkshire & The Humber - Sheffield
Yorkshire and the Humber REC Office
First Floor, Millside
Mill Pond Lane
Meanwood
Leeds
LS6 4RA

Telephone: 0113 3050127
Facsimile: 0113 8556191

09 August 2012

Dr Markus Reuber
Reader in Neurology
University of Sheffield
Department of Neurology
Royal Hallamshire Hospital
Glossop Road, Sheffield
S10 2JF

Dear Dr Reuber

Study title:	**Investigating associations between autonomic function and emotions in people with or without seizures.**
REC reference:	**12/YH/0322**
Protocol number:	**STH16441**

Thank you for your letter of 31 July 2012, responding to the Committee's request for further information on the above research and submitting revised documentation.

The further information has been considered on behalf of the Committee by the Chair.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised, subject to the conditions specified below.

Ethical review of research sites

NHS sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

Non-NHS sites

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of

the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission ("R&D approval") should be sought from all NHS organisations involved in the study in accordance with NHS research governance arrangements.

Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at http://www.rdforum.nhs.uk.

Where a NHS organisation's role in the study is limited to identifying and referring potential participants to research sites ("participant identification centre"), guidance should be sought from the R&D office on the information it requires to give permission for this activity.

For non-NHS sites, site management permission should be obtained in accordance with the procedures of the relevant host organisation.

Sponsors are not required to notify the Committee of approvals from host organisations

It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document	*Version*	*Date*
Advertisement	1	28 May 2012
Covering Letter		07 June 2012
Investigator CV		
Letter of invitation to participant	1	28 May 2012
Other: Student CV: Katy Wilkinson		
Other: Academic Supervisor CV: Claire Isaac		
Other: Email invitation for student volunteers	1	28 May 2012
Other: Research approval form		
Participant Consent Form: Patient	2	11 July 2012
Participant Consent Form: Control	2	11 July 2012
Participant Information Sheet: Control	2	11 July 2012
Participant Information Sheet: Patient	3	31 July 2012
Protocol	2	02 February 2012
Questionnaire: Liverpool Seizure Severity Scale	2.0	
Questionnaire: Core-10		01 February 2006
Questionnaire: Difficulties in Emotion Regulation Scale (DERS)		
Questionnaire: Hospital Anxiety and Depression Scale (HADS)		
Questionnaire: Demographic Questionnaire	1	10 December 2011
Questionnaire: Caffeine Consumption Questionnaire	1	10 December 2011
Questionnaire: Traumatic Experiences Checklist (TEC)	3	31 July 2012
REC application		11 June 2012
Referees or other scientific critique report		09 March 2012
Response to Request for Further Information		12 July 2012

Response to Request for Further Information		31 July 2012

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Reporting requirements

The attached document *"After ethical review – guidance for researchers"* gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Notification of serious breaches of the protocol
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

Feedback

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

Further information is available at National Research Ethics Service website > After Review

12/YH/0322	**Please quote this number on all correspondence**

With the Committee's best wishes for the success of this project

Yours sincerely

pp
Dr Basil Sharrack
Chair

Email: anne.ward7@nhs.net

Enclosures: "After ethical review – guidance for researchers"

Copy to: *Ms Ramila Patel, STH Research Department*

Appendix 4: Sheffield Teaching Hospitals NHS Foundation Trust Ethics

intelligence dr foster 2011 trust of the year (North)

Sheffield Teaching Hospitals NHS
NHS Foundation Trust

Ref: STH16441/RP

24 Aug 12

Dr Markus Reuber
Consultant Neurologist
N Floor
Royal Hallamshire Hospital
Glossop Rd
Sheffield,
S10 2JF

Dear Dr Reuber

Authorisation of Project

STH ref: STH16441
Study title: Investigating associations between autonomic function and emotions in people with or without seizures

Chief Investigator: Dr Markus Reuber, University of Sheffield
Principal Investigator: Ms Katy Wilkinson, Trainee Clinical Psychologist

Sponsor: Sheffield Teaching Hospitals NHS Foundation Trust
Funder: Un-funded

The Research Department has received the required documentation for the study as listed below:

1.	**Sponsorship IMP studies (non-commercial)**	N/A
	Sponsorship responsibilities between institutions	N/A
	Responsibilities of investigators	N/A
	Monitoring Arrangements	N/A
2.	**STH registration document: completed and signed**	NHS R&D Form, V3.4:
		C Isaac, 05 Jul 12
		D Patel, 30 May 12
3.	**Evidence of favourable scientific review**	N/A
4.	**Protocol – final version**	Version 2.0, 02 Feb 12
5.	**Participant Information sheet – final version**	
	Patient	Version 2.0, 11 Jul 12
	Control	Version 2.0, 11 Jul 12

Appendix 5: Researcher rated quality scores

Item	Criteria	Scoring		
		No	Partly	Yes
Research question				
1	Did the study address a clearly focused research question? (e.g. clearly focused population, disorder, and aims).	0	1	2
2	Was an appropriate design used to address the research question? (e.g. was a cross-sectional methodology appropriate?).	0	1	2
Sample				
3	Were the participants recruited in an appropriate way? (e.g. were the sample representative of the defined population, was everybody included that should have been included?).	0	1	2
4	Was the diagnosis of the sample robust? (e.g. how was the diagnosis of patient participants determined? Was the gold standard method of diagnosis used? Were diagnoses checked by medical and/or mental health professionals? Were standardised measures used?).	0	1	2
5	Was a control group used? (score 1 for another symptomatic group, score 2 for a healthy control group and/or another symptomatic group).	0	1	2
Data collection				
6	Were the data collected in a way that addressed the research question? (e.g. Was the setting for the data collection justified? Is it clear how the data were collected? Were the research methods made explicit?).	0	1	2

7	Were ethical issues considered? (e.g. issues around informed consent, how researchers have handled the effects of the study on participants)	0	1	2
Measures				
8	Were the measures used subjective or objective? (Score 0 for purely subjective or purely objective, score 1 for partly subjective, partly objective, Score 2 for inclusion of subjective and objective measures).	0	1	2
9	Have the measures been validated? (Score 0 for no validation, score 1 for validated in general or other populations, score 2 for validated in the population being studied).	0	1	2
10	Was reliability of the measures reported? (Score 0 for no reports, score 1 for reliability reports included from other studies, score 2 if reported reliability for the study in question).	0	1	2
11	Did the emotion regulation measure(s) corroborate with the Gross (1998b, p.275) definition of emotion regulation?	0	1	2
Results				
12	Did the study have enough participants to minimise the play of chance? (e.g. Was a power calculation reported?)	0	1	2
13	Were statistical techniques appropriate? (e.g. Were statistical techniques justified? Was sufficient detail given so statistical techniques could be replicated, such as correcting skewed data, a-priori and post-hoc techniques used? Were missing data accounted for, and did the researchers report how missing data was dealt with in analysis?).	0	1	2
14	Were effect sizes reported?	0	1	2
Conclusions				
15	Was there a clear statement of findings? (e.g. Were findings explicit?)	0	1	2
16	Is there adequate discussion of evidence both for and against the findings?	0	1	2

17	Does the researcher discuss the credibility of their findings? (e.g. limitations, triangulation).	0	1	2
Value of the research				
18	How valuable is the research? (e.g. does the researcher discuss the contribution the study makes to existing research?)	0	1	2
19	Do the researchers identify new areas where research is needed?	0	1	2
20	How generalisable are the results to the local population?	0	1	2
			Total score	**/40**

Appendix 6: Patient participant letter

Version 1: 28/05/2012

Sheffield Teaching Hospitals NHS
NHS Foundation Trust
Excellence as standard

The University Of Sheffield.

Dear Patient,

You are going to have an appointment in the neurology outpatient clinic at the Royal Hallamshire Hospital in Sheffield.

We are currently carrying out a research project in this clinic, which we would like to inform you about.

You are under no obligation whatsoever to take part in this study and your standard of care will not be affected in any way should you choose not to participate.

We would be grateful if you could have a look at the enclosed information sheet about the study and think about whether you would like to take part or not.

You do not have to take any other action at present. You will have the opportunity to ask any questions about the project from a member of the research team when you come for your appointment. If you decide to take part you can give your consent when you come to the hospital for your appointment.

Thank you for taking the time to consider taking part in this study.

Yours sincerely,

Katy Wilkinson
Researcher
Trainee Clinical Psychologist

Dr Markus Reuber
Research Supervisor
Reader and Consultant Neurologist

Appendix 7: Patient Participant Information Sheet

Version 3: 31/07/2012

Sheffield Teaching Hospitals NHS
NHS Foundation Trust
Excellence as standard

The University Of Sheffield.

PARTICIPANT INFORMATION SHEET

Title of Project: Investigating associations between autonomic functioning and emotions in people with or without seizures.

Name of Researchers: Katy Wilkinson, Markus Reuber and Claire Isaac.

We would like to invite you to take part in a research study. Before you decide whether to take part, you should understand why the research is being done and what it would involve for you. Please take time to read the following information carefully. Talk to others about the study if you wish. Please contact us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part. Thank you for reading this.

Background

The autonomic nervous system is a part of the nervous system that regulates key involuntary functions of the body. Heart rate variability (HRV) is a measure of variation in heart rate. Measures of HRV give us reliable information about the functioning of the autonomic nervous system. Recent research has explored HRV in people with non-epileptic attack disorder and epilepsy.

What is the purpose of the study?

This study is designed to find out about more about how heart rate variability is associated with non-epileptic attack disorder and epilepsy. We are interested in how HRV is associated with subtypes of non-epileptic attack disorder and how it is associated with self-report measures of psychological characteristics and seizure frequency and severity.

Why have I been asked to take part?

We are approaching people who have experienced seizures and who have been a patient at The Royal Hallamshire Hospital in Sheffield. We are asking people with non-epileptic seizures to take part in this study as well as people with epileptic seizures and people who do not experience seizures.

Do I have to take part?

It is up to you to decide whether or not to take part. If you have any questions about this study at any time, you can contact us and we will answer them. If you do decide to take part you are free to withdraw at any time, without giving a reason. This would not affect the standard of care you receive.

What will happen to me if I take part?

We will arrange an appointment for you at the clinic where your seizures were investigated (or the clinic where your relatives seizures were investigated if you do not have seizures yourself), at a time that is convenient for you. When you attend the appointment, you will have the chance to ask questions, and we will ask you to sign a consent form to show that you agree to take part. We will then ask you to complete six questionnaires which should take no longer than 30 minutes. Your HRV will be assessed using a simple ECG monitor (this should take about 20 minutes, 10 minutes of which will be you resting beforehand). The appointment should take about an hour.

What are the possible benefits of this study?

This study will add to our understanding of people with seizures. We hope that this will help us to find out what types of treatment are most useful for people who have non-epileptic seizures. We also hope the study will help us to identify ways to make the diagnosis of non-epileptic attacks quicker and more accurate.

What are the possible risks of taking part in this study?

One of the questionnaires asks you about history of traumatic experiences. The questionnaire covers sensitive topics which you may find intrusive and distressing. The researcher is a Trainee Clinical Psychologist who will be available for support should you require it. The Consultants in the department have extensive experience of dealing with seizures and working with people with non-epileptic attack disorder and epilepsy. Details of organisations you can contact for further support will be provided if you wish.

Should an abnormality be found in your ECG recording, you will be informed and will be referred for further investigation.

Will my taking part in this study be kept confidential?
All the information that is collected about you during this study will be kept strictly confidential. We will keep your personal details, such as name, address and telephone number, separately and locked in a secure location. This means that your identity will be kept private. Any personal details held by us will be destroyed once the study has finished.

What will happen to the results of the study?
We will publish the results of the study in a scientific journal. You will not be identified individually in the write-up. If you would like a summary of the results of the study once it is complete, please let us know.

What if I change my mind?
You do not have to take part in this study. If you have agreed to take part, you can stop at any time without giving your reasons. This will have no effect on any services you are receiving.

Who should I contact if I have a question or need more information?

Miss Katy Wilkinson
Clinical Psychology Department
Department of Psychology
The University of Sheffield
Western Bank
Sheffield
S10 2TN
Email: k.wilkinson@sheffield.ac.uk

Alternatively, call Ms Christie Harrison (Research Support Officer at The University of Sheffield) on 0114 2226650.

What if something goes wrong?

If you have a concern about any aspect of this study, you should ask to speak to the researchers who will do their best to answer your questions. If they are unable to resolve your concern or you wish to make a complaint regarding the study, please contact Sheffield Patient Services Team (previously known as PALS) on 0114 2712400 or Dr Philip Harvey (Registrar and Secretary, University of Sheffield) on registrar@sheffield.ac.uk or 0114 222 1101.

PLEASE DO NOT HESITATE TO ASK IF YOU HAVE ANY MORE QUESTIONS, EITHER NOW OR LATER

Appendix 8: Patient Participant Consent Form

Version 2: 11/07/2012

Sheffield Teaching Hospitals NHS
NHS Foundation Trust
Excellence as standard

The University Of Sheffield.

CONSENT FORM - Patient Participant

Title of Project: Investigating associations between autonomic functioning and emotions in people with or without seizures.

Name of Researchers: Katy Wilkinson, Markus Reuber and Claire Isaac.

Please initial box

1. I confirm that I have read and understand the information sheet for the above study and have had the opportunity to ask questions. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason, without my medical care or legal rights being affected. ☐

3. I agree to my medical notes being accessed if necessary. ☐

4. I agree to take part in the above study and understand that the data will be used as part of a Doctorate in Clinical Psychology degree thesis. ☐

_____	_____
Name	Signature
_____	_____
Today's date	Your date of birth
_____	_____
Name of person taking consent	Signature

Appendix 9: Control Participant Recruitment Email

Version 2: 08/12/2012

Dear students,

I am a doctoral student at the Department of Clinical Psychology, conducting research into autonomic functioning and emotion in people with or without seizures.

I would like to invite you to participate in this study. I am hoping to recruit a group of students and non-academic university staff who will act as a control group. The study will involve 1 face to face meeting at The Royal Hallamshire Hospital, Glossop Road, Sheffield. The meeting will last no longer than one hour.

At this meeting you will be asked to:

- Complete questionnaires about demographic details, psychopathology, trauma history and the way you regulate your emotions
- Have your heart rate variability measured. This will involve resting for 10 minutes and being attached to an ECG monitor for 10 minutes whilst resting

Participants must:

- Be over 18
- Have sufficient English language skills to complete the questionnaires without help
- Must never have experienced a blackout or seizure

If you are interested in taking part please contact me on pcp10kw@sheffield.ac.uk

All parking costs will be reimbursed.

For information about this email list, including how to remove your name, please visit http://www.shef.ac.uk/cics/email/distributionlists.html and click the list name.

Kind regards,

Katy Wilkinson

Trainee Clinical Psychologist

The University of Sheffield

Appendix 10: Control Participant Recruitment Email

Version 2: 08/12/12

Sheffield Teaching Hospitals NHS
NHS Foundation Trust
Excellence as standard

The University Of Sheffield.

Participants needed

We are looking for student and non-academic staff participants to take part in a study examining the relationship between the autonomic nervous system and the way people regulate emotions in PEOPLE WHO DO NOT EXPERIENCE SEIZURES.

As a participant in this study you would attend the neurology clinic at The Royal Hallamshire Hospital. The research would take no longer than 45 minutes.

Participating in the research involves having your heart rate variability measured (by being attached to an ECG monitor for 10 minutes) and completing self-report questionnaires.

Participants must be: over 18 years old, have sufficient English language skills to complete questionnaires and must never have experienced seizures or blackouts.

If you are interested in taking part or would like further information please contact me on pcp10kw@sheffield.ac.uk

Appendix 11: Control Participant Information Sheet

Sheffield Teaching Hospitals NHS
NHS Foundation Trust
Excellence as standard

The University Of Sheffield.

Version 3: 08/12/12

PARTICIPANT INFORMATION SHEET

Title of Project: Investigating associations between autonomic functioning and emotions in people with or without seizures.

Name of Researchers: Katy Wilkinson, Markus Reuber and Claire Isaac.

We would like to invite you to take part in a research study. Before you decide whether to take part, you should understand why the research is being done and what it would involve for you. Please take time to read the following information carefully. Talk to others about the study if you wish. Please contact us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part. Thank you for reading this.

Background

The autonomic nervous system is a part of the nervous system that regulates key involuntary functions of the body. Heart rate variability (HRV) is a measure of variation in heart rate. Measures of HRV give us reliable information about the functioning of the autonomic nervous system. Recent research has explored HRV in people with non-epileptic attack disorder and epilepsy.

What is the purpose of the study?

This study is designed to find out about more about how heart rate variability is associated with non-epileptic attack disorder and epilepsy. We are interested in how HRV is associated with subtypes of non-epileptic attack disorder and how it is associated with self-report measures of psychological characteristics and seizure frequency and severity.

Why have I been asked to take part?

We are recruiting a group of student and non-academic university staff volunteers who do not experience seizures to take part in the study. This is so we can compare HRV and self-reported measures of psychological characteristics in people who do not experience seizures with people who do experience seizures.

Do I have to take part?

It is up to you to decide whether or not to take part. If you have any questions about this study at any time, you can contact us and we will answer them. If you do decide to take part you are free to withdraw at any time, without giving a reason. This would not affect the standard of care you receive.

What will happen to me if I take part?

We will arrange a research appointment for you at The Royal Hallamshire Hospital, Glossop Road, Sheffield at a time that is convenient for you. When you attend the appointment, you will have the chance to ask questions, and we will ask you to sign a consent form to show that you agree to take part. We will then ask you to complete five questionnaires which should take no longer than 25 minutes. Your HRV will be assessed using a simple ECG monitor (this should take about 20 minutes, 10 minutes of which will be you resting beforehand). The whole appointment should take about an hour.

What are the possible benefits of this study?

This study will add to our understanding of people with seizures. We hope that this will help us to find out what types of treatment are most useful for people who have non-epileptic seizures. We also hope the study will help us to identify ways to make the diagnosis of non-epileptic attacks quicker and more accurate.

What are the possible risks of taking part in this study?

One of the questionnaires asks you about history of traumatic experiences. The questionnaire covers sensitive topics which you may find intrusive and distressing. The researcher is a Trainee Clinical Psychologist who will be available for support should you require it. Details of organisations you can contact for further support will be provided if you wish.

Should an abnormality be found in your ECG recording, you will be informed and will be referred for further investigation.

Will my taking part in this study be kept confidential?
All the information that is collected about you during this study will be kept strictly confidential. We will keep your personal details, such as name, address and telephone number, separately and locked in a secure location. This means that your identity will be kept private. Any personal details held by us will be destroyed once the study has finished.

What will happen to the results of the study?
We will publish the results of the study in a scientific journal. You will not be identified individually in the write-up. If you would like a summary of the results of the study once it is complete, please let us know.

What if I change my mind?
You do not have to take part in this study. If you have agreed to take part, you can stop at any time without giving your reasons.

Who should I contact if I have a question or need more information?
Miss Katy Wilkinson
Clinical Psychology Department
Department of Psychology
The University of Sheffield
Western Bank
Sheffield

S10 2TN
Email: k.wilkinson@sheffield.ac.uk
Alternatively, call Ms Christie Harrison (Research Support Officer at The University of Sheffield) on 0114 2226650.

What if something goes wrong?
If you have a concern about any aspect of this study, you should ask to speak to the researchers who will do their best to answer your questions. If they are unable to resolve your concern or you wish to make a complaint regarding the study, please contact Dr Philip Harvey (Registrar and Secretary, University of Sheffield) on registrar@sheffield.ac.uk or 0114 222 1101.

PLEASE DO NOT HESITATE TO ASK IF YOU HAVE ANY MORE QUESTIONS, EITHER NOW OR LATER

Appendix 12: Control Participant Consent Form

Version 2: 11/07/2012

Sheffield Teaching Hospitals NHS
NHS Foundation Trust
Excellence as standard

The University Of Sheffield.

CONSENT FORM – Healthy Control Participant

Title of Project: Investigating associations between autonomic functioning and emotions in people with or without seizures.

Name of Researchers: Katy Wilkinson, Markus Reuber and Claire Isaac.

Please initial box

1. I confirm that I have read and understand the information sheet for the above study and have had the opportunity to ask questions. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason, without my medical care or legal rights being affected. ☐

3. I agree to take part in the above study and understand that the data will be used as part of a Doctorate in Clinical Psychology degree thesis. ☐

Name	Signature	Today's date
Name of Person taking consent	Signature	Today's date

Version 1: 10/12/11 Appendix 13: Demographic questionnaire

Sheffield Teaching Hospitals NHS
NHS Foundation Trust
Excellence as standard

The University Of Sheffield.

Demographic Questionnaire

Please tick

1. Are you male or female?

☐ Male ☐ Female

2. What is your age?

☐ 18-21 ☐ 22-25

☐ 26-30 ☐ 31-40

☐ 41-50 ☐ 51-60

☐ 61+

3. What is the highest level of education you have?

☐ Less than GCSE/O Levels

☐ GCSE/O Levels

☐ A level

☐ Diploma

☐ Bachelors Degree

☐ Masters Degree

☐ Doctoral Degree

Other.....

Appendix 14: Liverpool Seizure Severity Scale-2

Liverpool Seizure Severity Scale 2.0

So we can better understand the severity of your seizures, please complete the following questionnaire thinking about the *most severe seizure* you experienced during the past 4 weeks. (This may be different for each individual, but is based on your most severe seizures over the past 4 weeks.) Your responses are a very important part of this study and will be kept strictly CONFIDENTIAL. No one but the research staff will see your responses. If results of this study are published, only aggregate data will be used; names and any other identifying information will not be reported.

How many seizures have you experienced during the past 4 weeks?	_____seizures	Note: Please enter '0' if you have not experienced any seizures in the last 4 weeks and do not complete the remainder of the questionnaire. If you cannot remember the exact number of seizures you've experienced, please estimate based on the number you usually had during a single day or week.

Please answer each question based on the most severe seizure you have experienced in the past 4 weeks. Circle only one answer for each question.

1. I feel that my most severe seizures have mostly been:	Very severe 0	Severe 1	Mild 2	Very Mild 3		
2. Most commonly when I blank out/lose consciousness:	I blank out for less than 1 minute 1	I blank out for between 1-2 minutes 2	I blank out for between 3 and 5 minutes 3	I blank out for more than 5 minutes 4	I never blank out/lose consciousness 0	
3. When I have my most severe seizures, I smack my lips, fidget, or behave in an unusual way:	Always 0	Usually 1	Sometimes 2	Never 3		
4. After my most severe seizures:	I feel very confused 0	I feel fairly confused 1	I feel slightly confused 2	I do not feel confused at all 3		
5. After my most severe seizures my confusion lasts for:	Less than 1 minute 1	Between 1 and 5 minutes 2	Between 6 minutes and 1 hour 3	1 to 2 hours 4	More than 2 hours 5	I never feel confused 0

6. When I have my most severe seizures:	I always fall to the ground 0	I usually fall to the ground 1	I sometimes fall to the ground 2	I never fall to the ground 3		
7. After my most severe seizures:	I always have a headache 0	I usually have a headache 1	I sometimes have a headache 2	I never have a headache 3		
8. After my most severe seizures:	I always feel sleepy 0	I usually feel sleepy 1	I sometimes feel sleepy 2	I never feel sleepy 3		
9. After my most severe seizures:	I always find that I have wet myself 0	I usually find that I have wet myself 1	I sometimes find that I have wet myself 2	I never find that I have wet myself 3		
10. After my most severe seizures:	I always find that I have bitten my tongue 0	I usually find that I have bitten my tongue 1	I sometimes find that I have bitten my tongue 2	I never find that I have bitten my tongue 3		
11. After my most severe seizures:	I always find that I have injured myself (other than biting my tongue) 0	I usually find that I have injured myself (other than biting my tongue) 1	I sometimes find that I have injured myself (other than biting my tongue) 2	I never find that I have injured myself (other than biting my tongue) 3		
12. After my most severe seizures I can usually return to what I am doing in:	Less than 1 minute 0	Between 1 and 5 minutes 1	Between 6 minutes and 1 hour 2	1 to 2 hours 3	More than 2 hours 4	

Appendix 15: Difficulties in Emotion Regulation Scale

Difficulties in Emotion Regulation Scale (DERS)

Response categories:

1 Almost never (0-10%)
2 Sometimes (11-35%)
3 About half the time (36-65%)
4 Most of the time (66 – 90%)
5 Almost always (91-100%)

1. I am clear about my feelings.
2. I pay attention to how I feel.
3. I experience my emotions as overwhelming and out of control.
4. I have no idea how I am feeling.
5. I have difficulty making sense out of my feelings.
6. I am attentive to my feelings.
7. I know exactly how I am feeling.
8. I care about what I am feeling.
9. I am confused about how I feel.
10. When I'm upset, I acknowledge my emotions.
11. When I'm upset, I become angry with myself for feeling that way.
12. When I'm upset, I become embarrassed for feeling that way.
13. When I'm upset, I have difficulty getting work done.
14. When I'm upset, I become out of control.
15. When I'm upset, I believe that I will remain that way for a long time.
16. When I'm upset, I believe that I'll end up feeling very depressed.
17. When I'm upset, I believe that my feelings are valid and important.
18. When I'm upset, I have difficulty focusing on other things.
19. When I'm upset, I feel out of control..
20. When I'm upset, I can still get things done.
21. When I'm upset, I feel ashamed with myself for feeling that way.
22. When I'm upset, I know that I can find a way to eventually feel better.
23. When I'm upset, I feel like I am weak.
24. When I'm upset, I feel like I can remain in control of my behaviors.
25. When I'm upset, I feel guilty for feeling that way.
26. When I'm upset, I have difficulty concentrating.
27. When I'm upset, I have difficulty controlling my behaviors.
28. When I'm upset, I believe there is nothing I can do to make myself feel better.
29. When I'm upset, I become irritated with myself for feeling that way.
30. When I'm upset, I start to feel very bad about myself.
31. When I'm upset, I believe that wallowing in it is all I can do.
32. When I'm upset, I lose control over my behaviors.
33. When I'm upset, I have difficulty thinking about anything else.
34. When I'm upset, I take time to figure out what I'm really feeling.
35. When I'm upset, it takes me a long time to feel better.
36. When I'm upset, my emotions feel overwhelming.

Appendix 16: Traumatic Experiences Checklist

Version 3: 03/07/2012

T. E. C.

People may experience a variety of traumatic experiences during their life. We would like to know if you have experienced any of the following 29 events:

If you do not wish to answer a question, please leave it blank.

1. Having to look after your parents and/or brothers and sisters when you were a child.	☐ No	☐ Yes	(Please tick)
2. Family problems (e.g., parent with alcohol or psychiatric problems, poverty).	☐ No	☐ Yes	
3. Loss of a family member (brother, sister, parent) when you were a CHILD.	☐ No	☐ Yes	
4. Loss of a family member (child or partner) when you were an ADULT.	☐ No	☐ Yes	

5. Serious bodily injury (e.g., loss of a limb, mutilation, burns).

☐ No ☐ Yes

6. Threat to life from illness, an operation, or an accident.

☐ No ☐ Yes

7. Divorce of your parents

☐ No ☐ Yes

8. Your own divorce

☐ No ☐ Yes

9. Threat to life from another person (e.g., during a crime).

☐ No ☐ Yes

10. Intense pain (e.g., from an injury or surgery).

☐ No ☐ Yes

11. War-time experiences (e.g., imprisonment, loss of relatives, deprivation, injury).	☐ No	☐ Yes
12. Second generation war-victim (war-time experiences of parents or close relatives)	☐ No	☐ Yes
13. Witnessing others undergo trauma.	☐ No	☐ Yes
14. Emotional neglect (e.g., being left alone, insufficient affection) by your parents, brothers or sisters.	☐ No	☐ Yes
15. Emotional neglect by more distant members of your family (e.g., uncles, aunts, nephews, nieces, grandparents).	☐ No	☐ Yes

16. Emotional neglect by non-family members (e.g., neighbours, friends, step-parents, teachers). ☐ No ☐ Yes

17. Emotional abuse (e.g., being belittled, teased, called names, threatened verbally, or unjustly punished) by your parents, brothers or sisters. ☐ No ☐ Yes

18. Emotional abuse by more distant members of your family. ☐ No ☐ Yes

19. Emotional abuse by non-family members. ☐ No ☐ Yes

20. Physical abuse (e.g., being hit, tortured, or wounded) by your parents, brothers, or sisters. ☐ No ☐ Yes

21. Physical abuse by more distant members of your family. ☐ No ☐ Yes

22. Physical abuse by non-family members. ☐ No ☐ Yes

23. Bizarre punishment ☐ No ☐ Yes

24. Sexual harassment (acts of a sexual nature that DO NOT involve physical contact) by your parents, brothers, or sisters. ☐ No ☐ Yes

25. Sexual harassment by more distant members of your family. ☐ No ☐ Yes

26. Sexual harassment by non-family members.

☐ No ☐ Yes

27. Sexual abuse (unwanted sexual acts involving physical contact) by your parents, brothers, or sisters.

☐ No ☐ Yes

28. Sexual abuse by more distant members of your family.

☐ No ☐ Yes

29. Sexual abuse by non-family members.

☐ No ☐ Yes

Thank you very much for your cooperation.

Appendix 17: Hospital Anxiety and Depression Scale

Hospital Anxiety and Depression Scale (HADS)

nferNelson
understanding potential

Name: _____ Date: _____

Clinicians are aware that emotions play an important part in most illnesses. If your clinician knows about these feelings he or she will be able to help you more.

This questionnaire is designed to help your clinician to know how you feel. Read each item below and **underline the reply** which comes closest to how you have been feeling in the past week. Ignore the numbers printed at the edge of the questionnaire.

Don't take too long over your replies, your immediate reaction to each item will probably be more accurate than a long, thought-out response.

FOLD HERE

A D

I feel tense or 'wound up'
- Most of the time
- A lot of the time
- From time to time, occasionally
- Not at all

I still enjoy the things I used to enjoy
- Definitely as much
- Not quite so much
- Only a little
- Hardly at all

I get a sort of frightened feeling as if something awful is about to happen
- Very definitely and quite badly
- Yes, but not too badly
- A little, but it doesn't worry me
- Not at all

I can laugh and see the funny side of things
- As much as I always could
- Not quite so much now
- Definitely not so much now
- Not at all

Worrying thoughts go through my mind
- A great deal of the time
- A lot of the time
- Not too often
- Very little

I feel cheerful
- Never
- Not often
- Sometimes
- Most of the time

I can sit at ease and feel relaxed
- Definitely
- Usually
- Not often
- Not at all

I feel as if I am slowed down
- Nearly all the time
- Very often
- Sometimes
- Not at all

I get a sort of frightened feeling like 'butterflies' in the stomach
- Not at all
- Occasionally
- Quite often
- Very often

I have lost interest in my appearance
- Definitely
- I don't take as much care as I should
- I may not take quite as much care
- I take just as much care as ever

I feel restless as if I have to be on the move
- Very much indeed
- Quite a lot
- Not very much
- Not at all

I look forward with enjoyment to things
- As much as I ever did
- Rather less than I used to
- Definitely less than I used to
- Hardly at all

I get sudden feelings of panic
- Very often indeed
- Quite often
- Not very often
- Not at all

I can enjoy a good book or radio or television programme
- Often
- Sometimes
- Not often
- Very seldom

Now check that you have answered all the questions

A D

TOTAL

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Appendix 18

Hierarchical multiple regression outputs for the TEC sub-scales

The tables below outline the hierarchical multiple regression outputs for each participant group (PNES, epilepsy and healthy controls). Each table relates to the prediction of emotion dysregulation from anxiety, depression, seizure frequency and severity, and emotional neglect, emotional abuse, bodily threat, sexual harassment, and sexual abuse respectively.

PNES participants

Emotional Neglect: PNES Participants

	Emotion dysregulation							
	Model 1		Model 2		Model 3		Model 4	
Variable	B	ß	B	ß	B	ß	B	ß
Constant	39.61		40.09		46.19		39.27	
Anxiety	4.76	.96	4.87	.98	5.21	1.05	4.89	.98
Depression			-.19	-.03	-.78	-.13	.44	.07
Ictal[c]					.02	.08	.00	.06
Freq[d]					-.11	-.10	-.29	-.02
Emotional neglect							-6.40	-.18
R^2	.91		.91		.92		.94	
F	82.04***		36.09***		14.52***		12.51**	
ΔR^2	.91		.00		.00		.00	
ΔF	82.04***		.04		.29		1.27	

Note. N = 25. *p <.05, **p<.01, ***p<.001

Emotional Abuse: PNES Participants

	Emotion dysregulation							
	Model 1		Model 2		Model 3		Model 4	
Variable	B	ß	B	ß	B	ß	B	ß
Constant	39.61		40.09		46.18		52.39	
Anxiety	4.76	.96	4.86	.97	5.21	1.05	3.67	.74
Depression			.18	.04	.78	.12	1.90	.31
Ictal[c]					.01	.08	.09	.11
Freq[d]					.12	.11	.02	1.12
Emotional abuse							17.04	.02
R^2	.91		.91		.92		.98	
F	82.04***		36.09***		14.52***		38.33**	
ΔR^2	.91		.00		.01		.06	
ΔF	82.04***		.04		.29		11.51*	

Note. N = 25. *p <.05, **p<.01, ***p<.001

Bodily Threat: PNES Participants

	Emotion dysregulation							
	Model 1		Model 2		Model 3		Model 4	
Variable	B	ß	B	ß	B	ß	B	ß
Constant	39.61		40.08		46.19		47.58	.97
Anxiety	4.76	.95	4.87	.97	5.21	1.05	4.82	.00
Depression			.19	.03	.78	.13	.04	.04
Ictal[c]					.02	.09	.00	.08
Freq[d]					.12	.11	.09	.08
Bodily threat							3.50	.11
R^2	.90		.89		.86		.84	
F	82.00***		36.09***		14.52***		10.06*	
ΔR^2	.91		.00		.00		.00	
ΔF	82.03***		.04		.29		.30	

Note. N = 25. *p <.05, **p<.01, ***p<.001

Sexual Harassment: PNES Participants

	Emotion dysregulation							
	Model 1		Model 2		Model 3		Model 4	
Variable	B	ß	B	ß	B	ß	B	ß
Constant	39.61		40.09		46.19		45.76	
Anxiety	4.76	.96	4.86	.97	5.21	1.05	3.69	.74
Depression			.19	.03	.78	.13	1.30	.21
Ictal[c]					.02	.09	.00	.01
Freq[d]					.12	.11	.02	.02
Sexual harassment							11.44	.22
R^2	.91		.91		.92		.92	
F	82.04***		36.09**		14.52**		7.76*	
ΔR^2	.91		.00		.00		.00	
ΔF	82.04***		.03		.29		.19	

Note. N = 25. *p <.05, **p<.01, ***p<.001

Sexual Abuse: PNES Participants

	Emotion dysregulation							
	Model 1		Model 2		Model 3		Model 4	
Variable	B	ß	B	ß	B	ß	B	ß
Constant	39.61		40.09		46.19		47.03	
Anxiety	4.76	.96	4.87	.98	5.21	1.05	4.29	.86
Depression			.19	.03	.78	.13	.57	.09
Ictal[c]					.02	.09	.00	.04
Freq[d]					.12	.11	.08	.24
Sexual abuse							5.35	11.56
R^2	.91		.91		.92		.93	
F	82.04***		36.09***		14.52***		9.83*	
ΔR^2	.91		.00		.00		.00	
ΔF	82.04***		.04		.29		.21	

Note. N = 25. *p <.05, **p<.01, ***p<.001

Epilepsy participants

Emotional Neglect: Epilepsy Participants

	Emotion dysregulation							
	Model 1		Model 2		Model 3		Model 4	
Variable	B	ß	B	ß	B	ß	B	ß
Constant	58.38		67.80		62.69		58.39	
Anxiety	2.80	.87	4.71	1.46	5.28	1.64	5.08	1.58
Depression			3.24	.68	3.84	.81	3.57	.75
Ictal[c]					.03	.11	.06	.18
Freq[d]					.15	.15	.29	.29
Emotional neglect							18.17	.29
R^2	.76		.87		.90		.95	
F	18.91***		17.71***		6.76		8.08	
ΔR^2	.76		.12		.02		.05	
ΔF	18.91***		4.71		.36		2.23	

Note. N = 25. *p <.05, **p<.01, ***p<.001

Emotional Abuse: Epilepsy Participants

	Emotion dysregulation							
	Model 1		Model 2		Model 3		Model 4	
Variable	B	ß	B	ß	B	ß	B	ß
Constant	58.32		67.80		62.67		59.39	
Anxiety	2.80	.87	4.71	1.46	5.28	1.64	5.08	1.58
Depression			3.24	.68	3.84	.81	3.57	.75
Ictal[c]					.03	.11	.06	.19
Freq[d]					.15	.15	.29	.29
Emotional abuse							5.06	.29
R^2	.76		.88		.90		.95	
F	18.98***		17.71***		6.77		8.07	
ΔR^2	.76		.88		.90		.95	
ΔF	18.98***		4.70		.36		2.23	

Note. N = 25. *p <.05, **p<.01, ***p<.001

Bodily Threat: Epilepsy Participants

	Emotion dysregulation							
	Model 1		Model 2		Model 3		Model 4	
Variable	B	ß	B	ß	B	ß	B	ß
Constant	58.38		67.80		62.68		107.40	
Anxiety	2.80	.87	4.71	1.46	5.28	1.64	.21	.06
Depression			3.24	.68	3.84	.81	1.02	.22
Ictal[c]					.03	.11	.05	.15
Freq[d]					.15	.15	.34	.34
Bodily threat							32.85	.79
R^2	.76		.88		.90		.98	
F	18.98***		17.71***		6.76		20.57	
ΔR^2	.76		.12		.02		.08	
ΔF	18.98***		4.71		.36		8.41	

Note. N = 25. *p <.05, **p<.01, ***p<.001

Sexual Harassment: Epilepsy Participants

	Emotion dysregulation							
	Model 1		Model 2		Model 3		Model 4	
Variable	B	ß	B	ß	B	ß	B	ß
Constant	58.31		67.80		62.69		59.39	
Anxiety	2.80	.87	4.71	1.45	5.28	1.64	5.08	1.58
Depression			3.24	.68	3.84	.81	3.57	.75
Ictal[c]					.03	.11	.06	.19
Freq[d]					.15	.15	.29	.29
Sexual harassment							9.09	.29
R^2	.76		.88		.90		.95	
F	18.98***		17.71**		6.77		8.07	
ΔR^2	.76		.12		.02		.05	
ΔF	18.98***		4.71		.36		2.23	

Note. N = 25. *p <.05, **p<.01, ***p<.001

Sexual Abuse: Epilepsy Participants

	Emotion dysregulation							
	Model 1		Model 2		Model 3		Model 4	
Variable	B	ß	B	ß	B	ß	B	ß
Constant	38.39		67.80		62.69		59.59	
Anxiety	2.80	.87	4.71	1.46	5.28	1.64	5.08	1.58
Depression			3.24	.68	3.84	.81	3.57	.75
Ictal[c]					.03	.11	.06	.19
Freq[d]					.15	.15	.29	.29
Sexual abuse							9.09	.29
R^2	.76		.88		.90		.95	
F	18.98***		17.71***		6.78		8.07	
ΔR^2	.76		.12		.02		.05	
ΔF	18.98***		4.71		.36		2.23	

Note. N = 25. *p <.05, **p<.01, ***p<.001

Healthy control participants

Emotional Neglect: Healthy Control Participants

	Emotion dysregulation					
	Model 1		Model 2		Model 3	
Variable	B	ß	B	ß	B	ß
Constant	54.90		54.72		54.66	
Anxiety	3.99	.72	3.84	.70	3.50	.63
Depression			.26	.03	.13	.02
Emotional neglect					5.89	.25
R^2	.52		.52		.58	
F	27.50***		13.28***		10.50***	
ΔR^2	. 52		.00		.05	
ΔF	27.50***		.02		2.94	

Note. N = 27. *p <.05, **p<.01

Emotional Abuse: Healthy Control Participants

	Emotion dysregulation					
	Model 1		Model 2		Model 3	
Variable	B	ß	B	ß	B	ß
Constant	54.90		54.72		53.76	
Anxiety	3.99	.72	3.84	.70	3.89	.71
Depression			.26	.03	.03	.00
Emotional abuse					1.69	.21
R^2	.52		.52		.57	
F	27.50***		13.22***		10.02***	
ΔR^2	.52		.00		.04	
ΔF	27.50***		.02		.04	

Note. $N = 27$. *$p < .05$, **$p < .01$

Bodily Threat: Healthy Control Participants

	Emotion dysregulation					
	Model 1		Model 2		Model 3	
Variable	B	ß	B	ß	B	ß
Constant	54.90		54.72		56.15	
Anxiety	3.40	.72	3.84	.70	3.00	.54
Depression			.26	.03	.53	.07
Bodily threat					9.71	.33
R^2	.52		.52		.57	
F	27.40***		13.21***		10.19***	
ΔR^2	.52		.00		.05	
ΔF	27.50***		.02		2.50	

Note. $N = 27$. *$p < .05$, **$p < .01$

Sexual Harassment: Healthy Control Participants

	Emotion dysregulation					
	Model 1		Model 2		Model 3	
Variable	B	ß	B	ß	B	ß
Constant	54.90		54.72		54.79	
Anxiety	3.40	.72	3.84	.70	3.61	.65
Depression			.26	.03	.34	.04
Sexual harassment					9.12	.09
R^2	.52		. 52		.53	
F	27.50***		13.22***		8.69***	
ΔR^2	.52		.00		.01	
ΔF	27.50***		.02		.35	

Note. N = 27. *p <.05, **p<.01

Sexual Abuse: Healthy Control Participants

	Emotion dysregulation					
	Model 1		Model 2		Model 3	
Variable	B	ß	B	ß	B	ß
Constant	54.90		54.72		56.80	
Anxiety	3.99	.72	3.84	.70	2.38	.43
Depression			.26	.00	.77	.09
Sexual abuse					22.57	.35
R^2	.52		.52		.61	
F	27.50***		13.22***		11.84***	
ΔR^2	.52		.00		.08	
ΔF	27.50***		.02		4.85*	

Note. N = 27. *p <.05, **p<.01

Hierarchical multiple regression outputs for the non-acceptance of emotion sub-scale

The tables below outline the hierarchical multiple regression outputs for each participant group (PNES, epilepsy and healthy controls). Each table relates to the non-acceptance of emotions from anxiety, depression, seizure frequency and severity, and traumatic experiences.

PNES participants

	Non-acceptance of emotions							
	Model 1		Model 2		Model 3		Model 4	
Variable	B	ß	B	ß	B	ß	B	ß
Constant	6.22		6.81		3.81		8.88	
Anxiety	.79	.84	.93	.99	.86	.92	1.46	1.56
Depression			.23	.20	.03	.03	1.66	1.42
Ictal[c]					.00	.04	.00	.26
Freq[d]					.04	.20	.06	.32
Trauma							.97	.90
R^2	.71		.73		.75		.88	
F	19.71***		9.49*		3.75		6.34*	
ΔR^2	.71		. 02		.02		.14	
ΔF	19.71***		.50		.19		4.97	

Note. $N = 25$. *p <.05, **p<.01, ***p<.001

Epilepsy participants

	Non-acceptance of emotions							
	Model 1		Model 2		Model 3		Model 4	
Variable	B	ß	B	ß	B	ß	B	ß
Constant	10.03		13.97		18.09		18.72	
Anxiety	.40	.57	1.19	1.69	.99	1.40	.89	1.26
Depression			1.34	1.23	1.12	1.87	1.00	.97
Ictal[c]					.01	.19	.01	.20
Freq[d]					.07	.33	.06	.26
Trauma							.32	.22
R^2	.33		.74		.87		.90	
F	2.88***		7.18*		5.09		3.72	
ΔR^2	.32		.42		.14		.03	
ΔF	2.88***		.04		.35		.50	

Note. $N = 25$. *p <.05, **p<.01, ***p<.001

Healthy control participants

	Non-acceptance of emotions					
	Model 1		Model 2		Model 3	
Variable	B	ß	B	ß	B	ß
Constant	7.33		7.18		7.22	
Anxiety	.84	.75	.71	.62	.55	.49
Depression			.23	.14	.08	.05
Trauma					.48	.31
R^2	.55		.56		.61	
F	31.14***		15.31***		13.88***	
ΔR^2	.55		.01		.05	
ΔF	31.14***		.32		2.76	

Note. $N = 25$. *p <.05, **p<.01, ***p<.001