TANYA SUHALITKA BSc (Hons) MSc

THE IMPACT OF TRAUMA IN FUNCTIONAL NEUROLOGICAL DISORDER

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Summary of the Major Research Project

Section A is a narrative review that examined a) the differences in psychological characteristics between individuals with FND based on the presence or absence of trauma-exposure and b) the psychological correlates of trauma-exposure in FND. The review found that exposure to traumatic events, especially in childhood, was associated with greater psychological distress, such as depression, anxiety, PTSD, dissociation, and emotional dysregulation. Subgroup analyses found significant differences on most psychological characteristics except somatoform dissociation and alexithymia, which may reflect a common underlying mechanism for all individuals with FND. This review highlighted the relevance of trauma-exposure in this population, which should be integrated into treatment and inform future research. Clinical and research implication are provided.

Section B is a multiple baseline single-case experimental design (SCED) study that examined the effectiveness of EMDR in alleviating FND symptomatology, functional impairment, and psychological distress in participants with FND who also reported a history of trauma. The findings presented a mixed picture regarding treatment effectiveness. Most participants reported significant treatment gains with relatively few sessions on all outcome measures, while some participants reported little or no improvement. Despite methodological limitations and inconclusive findings, this study offers tentative evidence for the use of EMDR in this client group that warrants further empirical investigation. Clinical and research implications are provided.

Section C is a list of appendices.

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SECTION A: LITERATURE REVIEW

THE IMPACT OF TRAUMA-EXPOSURE IN FUNCTIONAL NEUROLOGICAL DISORDER

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Abstract

Background: Functional Neurological Disorder (FND) is a complex and heterogenous presentation. Despite the high prevalence of trauma-exposure in individuals with FND, little is known about the impact of traumatic events on psychological functioning in individuals with FND.

Aims: The aim of this review was to conduct a systematic search of available literature to examine a) the differences in psychological characteristics between individuals with FND based on the presence or absence of trauma-exposure and b) the psychological correlates of trauma-exposure in FND.

Methodology: A systematic search of four electronic databases retrieved eighteen eligible papers.

Results: Findings indicated that a history of trauma, especially childhood emotional maltreatment, was associated with greater mental health difficulties in individuals with FND including anxiety, depression, dissociation, and emotional dysregulation. However, subgroup analyses based on trauma-exposure found no differences on alexithymia or somatoform dissociation between those with and without trauma, which may reflect a common underlying mechanism across individuals with FND.

Implications: Individuals with FND who report a history of trauma may have a more a complex psychological profile than those without trauma, which may require a psychological assessment and intervention. Further investigation into alexithymia and somatoform dissociation is needed to clarify whether both constructs are specific to FND irrespective of trauma-exposure. Future studies would benefit from the inclusion of psychiatric control groups, broader FND populations, and multivariate designs.

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Introduction

Definition of Functional Neurological Disorder

Functional neurological disorder (FND) encompasses symptoms that superficially resemble neurological disorders but without comparable organic neuropathology (Lehn et al., 2016). FND can be categorised into several major subtypes: functional seizures (FS), functional motor (FMD) and sensory disorders, which are further distinguished by negative (e.g., loss of sensation) and positive (e.g., seizures) symptoms. The burden of FND is comparable to organic conditions, such as multiple sclerosis, resulting in high rates of disability, unemployment, and health care utilisation (A. Carson et al., 2011). Individuals with FND are also likely to have concurrent organic conditions and other functional symptoms (Fobian & Elliott, 2019).

FND is a complex and heterogenous presentation, without consensus on nosology, aetiology, or underlying mechanism. This is reflected in the multiple terms used to describe symptoms, including hysteria, conversion disorder, psychogenic/dissociative neurological disorder. This review will use "functional" as the preferred prefix for different symptoms, as it is both more acceptable by patients (Kozlowska et al., 2021) and reflective of theoretical uncertainty.

Diagnosis of FND

Recent advancements in FND have enabled a rule-in diagnosis to be made by identifying and specifying positive markers of functional symptoms. Despite the high validity and reliability these makers (Aybek & Perez, 2022), establishing a diagnosis in clinical practice continues to be fraught with difficulties. Possible reasons include fear of making clinical errors, misconceptions about FND among medical professionals (Shneker & Elliott, 2008), and poor cross-discipline collaboration (McMillan et al., 2014).

Epidemiology of FND

FND is a frequent referral to neurology clinics, with an estimated prevalence rate of 4-12 per 100,000 (Fobian & Elliott, 2019); however, due to underreporting, this is likely to be a lot higher (Nicholson et al., 2011). A majority of FND cases occur in women (60-75%) (Espay et al., 2018), which may be linked to the higher prevalence of sexual and physical abuse, chronic pain, and psychiatric diagnoses reported by women than men with FND (Thomas et al., 2013). FND has been associated with anxiety and depression (Walsh et al., 2018), dissociation (Pick et al., 2020), emotional dysregulation (Sojka et al., 2018), lower socioeconomic status (Smakowski et al., 2021), lower education attainment (Jennum et al., 2019), lower social support and high rates of childhood and lifetime trauma (Levita et al., 2020). However, there is also significant heterogeneity across psychosocial factors in FND (Aybek & Perez, 2022) that highlighted the importance of examining distinct clinical subgroups based on various psychological factors, such as trauma-exposure and emotional regulation, to further elucidate aetiology and mechanisms of FND (Afari et al., 2014; Uliaszek et al., 2012).

Prognosis of FND

The largest prospective FND study with over 700 participants found that 67% reported poor outcomes after a 1-year-follow-up (Sharpe et al., 2010). A 14-year prospective cohort study of functional limb weakness found that 80% of participants experienced symptoms 14 years later

(Gelauff et al., 2019); and a review on the prognosis for FS showed that only 40% of adults achieved remission in their seizures (Durrant et al., 2011).

Theories of FND

Some of the earliest accounts of FND originated in Egyptian and Greek texts which conceptualised FND as a supernatural phenomenon or a female ailment brought on by uterine dysfunction. More contemporary theories of FND, however, can be traced back to the neurological advances of the latter 19th century, which classified FND, then known as hysteria (from the Greek *hystera*, meaning uterus), under *névroses* or neuroses, which were clinically, but not anatomically, identifiable disorders (Raynor & Baslet, 2021). The French neurologist Jean-Martin Charcot, who pioneered the nosology of neurological disorders, described hysteria as a dynamic and hereditary neurological disorder that was provoked and exacerbated by environmental factors such as emotional stress and physical trauma (Goetz, 2016). Charcot's acknowledgement of stress-related factors in hysteria generated scientific interest in the psychological explanation of FND, specifically the role of trauma, and transformed FND from a purely neurological to a predominantly psychogenic condition.

At the turn of the 20th century, FND was explained by two dominant theories, iterations of which are still prevalent today, dissociation (Janet, 1889, 1907) and conversion (Breuer & Freud 1893-1895; 1982). Although both theories diverged on the mechanism, they contextualised FND within a diathesis-stress framework, where physical symptoms occurred in response to emotional stress. Janet proposed that exposure to traumatic events could cause an 'emotional shock' so overwhelming that cognitive, affective, and somatosensory components of the event could become separated (compartmentalised) from conscious awareness. These

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compartmentalised states would then intrude into consciousness when reminded of trauma, and affect mental, emotional, physical, and behavioural functioning. In this way, FND symptoms could be viewed as compartmentalised somatic fragments of a traumatic experience -- a kind of somatic flashback.

More recent models, such as Hilgard's (1977) neo-dissociation theory proposed that consciousness is divided into two hierarchically organised systems: the central executive, which is under conscious and effortful control, and a lower-level system that operates largely outside of awareness (i.e., dissociated) that is responsible for routine functions, such as breathing and driving. In this way, dissociation can be explained as a shift in the conscious control of functions. Brown (2016) however, indicated that many of these lower-level dissociated functions can in fact be brought back into conscious control when needed, such as suddenly stopping a car in an emergency, which is distinct from FND symptoms that cannot be controlled at will. He therefore proposed that FND is a compartmentalisation phenomenon, characterised by a reduction or loss of voluntary psychological or somatoform functioning, due to dissociation of perceived or actual control over lower-level processing by the central executive. Detachment, which relates to altered states of consciousness, such depersonalisation and derealisation (Holmes et al., 2005) has also been associated with FND, however, findings have been more inconsistent (Brown & Reuber, 2016).

Conversion theory proposed by Freud (Breuer & Freud, 1982) did not view functional symptoms as dissociated states, but rather as "abnormal expressions of emotion". While Freud acknowledged the emotional impact of trauma, he also incorporated more subtle emotional experiences and unconscious mental conflicts in his theory, all of which could be converted to physical symptoms as a way of ameliorating anxiety and psychic distress. Although conversion

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theory mostly fell into disregard, partly due to its unfalsifiable claims, current models, nevertheless, incorporate psychological distress in the development and maintenance of FND. For example, FND is often associated with somatisation, which is the tendency to express somatic symptoms in response to psychosocial stress (Lipowski, 1988). One explanation is that individuals with FND struggle to process and regulate emotional states. FND is frequently associated with alexithymia (Demartini et al., 2014), which relates to difficulties with identifying and differentiating between emotions and physical sensations, and externally oriented thinking. FND is also linked to unhelpful coping strategies, such suppression and avoidance of emotion (Williams et al., 2018).

Another contemporary model that links FND to stress and negative emotional experiences is the stress-system model, which posits that FND symptoms occur when the burden of cumulative stressful experiences, big or small, trigger the chronic over-activation of the body's stress system (Kozlowska et al., 2020). This results in aberrant processing of body states, and an increased activation of sensorimotor components in response to negative emotional stimuli (Kozlowska, 2005, 2017).

Neurobiological predictive processing (PP) models (e.g., Edwards et al., 2012) and the Integrative Cognitive Model (ICM) (Brown, 2004; Brown & Reuber, 2016b) of FND offer broader biopsychological frameworks which have been particularly influential in the conceptualisation of FND in the recent years.

PP models posit that predictions about the world, including somatosensory functioning, are shaped by prior experiences and expectations which influence how current and future somatosensory functioning is experienced. As such, FND symptoms could arise from 'inference failures' whereby increased attention to implicit beliefs about somatic sensations could induce symptoms that are experienced as real and involuntary (Edwards et al., 2012; Henningsen et al., 2018).

The ICM further elaborates on this model by proposing that FND symptoms are generated and maintained by maladaptive or 'rogue' mental representations (similar to PP's predictions) that have developed from previous experiences, such as trauma or exposure to familial experiences of seizures/tremors. These representations comprise cognitive, emotional, and physiological components that become activated via internal and external cues. These cues are often associated with enhanced threat processing and anxious arousal. Like dissociation theories, the ICM emphasises that the activation of rogue representation is preconscious and therefore not experienced as voluntary. Like PP, increased attention to rogue representations is implicated in the development and maintenance of symptoms, however, other attentional difficulties are also highlighted, such as the inability to inhibit rogue representations which can maintain symptoms. (Brown & Reuber, 2016b).

Trauma-exposure and FND

The perception and understanding of the role of trauma-exposure in FND has changed considerably over the last two decades. Prior to this, dominant theories of the 19th and 20th century emphasised trauma-exposure as critical in the development of FND, however, over time this became more dubious as many individuals with FND do not present with such histories (Roelofs & Pasman, 2016). Much of the current research in this area has, therefore, shifted from a focus on aetiological to more mechanistic models of FND.

Nevertheless, the association between trauma-exposure and FND remains important. A recent meta-analysis of over 1,400 individuals with FND and over 2,000 healthy and clinical

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controls (psychiatric and neurological) by Ludwig et al. (2018) found that individuals with FND were more likely to report a history of emotional neglect (49% for cases vs 20% for controls), physical abuse (30% vs 12%), and sexual abuse (24% vs 10%). Authors concluded that individuals with FND were eight times more likely to report trauma-exposure than healthy controls, and twice as likely as clinical populations. Other studies found that trauma-exposure predicted the likelihood of an FND diagnosis over and above other risk factors, such as emotional dysregulation, anxiety, and depression (Karatzias et al., 2017). Trauma-exposure was linked to greater FND symptom severity and worse psychosocial and physical outcomes (van der Feltz-Cornelis et al., 2020).

The current consensus on the role of trauma-exposure in FND is that it is an important but not essential - risk factor (Ludwig et al., 2018). Given the psychological and clinical heterogeneity in FND, it is possible that trauma-exposure influences the development of distinct psychological profiles that may be important in informing clinical interventions (Karatzias et al., 2017).

Trauma-exposure is a conceptually ambiguous construct, and subject to much debate (Hyland et al., 2021). The central argument hinges what constitutes an event to be 'traumatic'. Diagnostic criteria of PTSD in the current Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) defines trauma as exposure to direct or indirect *"actual or threatened death, serious injury, or sexual violence"*. However, there is substantial evidence that experiences outside of this definition can produce the same detrimental effects (Larsen & Pacella, 2016). As such, this review recognises and incorporates a broader definition of 'trauma-exposure' to include any direct or indirect event that has the potential to cause substantial psychological and/or physical harm.

Aim of this review

Previous reviews have primarily focused on the prevalence of trauma-exposure and its utility in differentiating FND from other conditions. With one notable exception which focused exclusively on FS (Beghi et al., 2015), less is known, about the relationship between traumaexposure and psychological functioning in FND. Furthermore, to the best of the author's knowledge, no review currently exists that explored psychological differences between individuals with FND based on trauma-exposure. To address these gaps in the literature, the review was guided by two main questions:

- 1. Is there a difference in psychological characteristics between individuals with FND based on trauma-exposure?
- 2. What are the psychological correlates of trauma-exposure in FND?

Method

Search strategy

A systematic literature search was conducted in July 2021, using four electronic databases: Psychinfo, MEDLINE, ASSIA, and Web of Science. Search terms were developed after an initial scoping search of the relevant literature in this subject area and listed in Table 1. A manual search of grey literature (Open Grey) and Google Scholar was also conducted. Titles and abstracts were then screened based on the inclusion/exclusion criteria (Table 2). Once eligible studies were identified, their reference lists were examined, and two additional studies added to the review. A PRISMA flow diagram (Liberati et al., 2009) in Figure 1 illustrates the search process.

Table 1

Search terms

functional neurologic* OR conversion disorder OR unexplained neurologic* OR non-organic neurologic* OR FND OR NEAD OR FNSD OR non-epileptic OR nonepileptic OR functional motor OR functional sensory OR functional symptom* OR dissociat* seizure* OR functional dystonia OR functional tremor OR functional movement OR psychogenic movement OR psychogenic speech

AND

trauma* OR sex* abuse OR physical abus* OR emotion* abus* OR neglect OR assault OR rape OR abus*

Table 2

Inclusion and exclusion criteria

Adults over 18 years old with a diagnosis of Neurobiological studies FND Neuropsychological studies Trauma-exposure assessed by validated Neuropsychological studies outcome measure or clinical interview Case studies Papers examining the relationship between trauma exposure and psychological factors Papers comparing psychological characteristics Sector S
Neuropsychological studiesTrauma-exposure assessed by validated outcome measure or clinical interviewCase studiesPapers examining the relationship between trauma exposure and psychological factors
outcome measure or clinical interview Case studies Papers examining the relationship between trauma exposure and psychological factors Papers comparing psychological characteristics East the studies
Papers examining the relationship between trauma exposure and psychological factors Papers comparing psychological characteristics
Papers comparing psychological characteristics
in subgroups based on trauma-exposure
Use of validated measures for psychological variables
Peer-reviewed research
English language
Quantitative papers

Figure 1

PRISMA Diagram



Quality assessment

The studies included in this review were cross-sectional and case-control in design. As such, the quality was assessed by two critical appraisal tools: the AXIS tool for cross-sectional studies (Downes et al., 2016) (Appendix A) and the Joana Briggs Checklist for Case-Control studies (Moola et al., 2017) (Appendix B). Although critical appraisal tools can be used for inclusion/exclusion purposes, given the relatively small number of identified studies, none of the studies were judged to have methodological issues necessitating exclusion.

Structure of the review

A summary of the studies is presented in Table 3. Firstly, study and trauma-exposure characteristics are presented. Thereafter, the main results are first organised and synthesised by the psychological factor/s examined, and secondly by the review question they answered. This approach allowed for clarity and relative robustness of findings to be considered. Some studies were discussed more than once if they examined multiple factors or related to both review questions. After this, a critique of key methodological issues is presented, followed by a discussion of results with reference to relevant research, clinical and research implications, and limitations of this review.

Table 3

Summary of studies

Study	Design	Sample (Number, mean/median age, female %, setting)	Trauma measure	Measures of key psychological factors	Main findings
Bodde et al., 2013	Cross- sectional	FS N=40 M=30 F=80% Tertiary epilepsy centre, Netherlands	TEC	DIS-Q SDQ-20 CERQ Short MMPI Short TCI	 Trauma-exposure subgroup scored higher on MMPI's 'negativism' (t = 2.030; p = .049) and 'shyness' (t=-2.891; p=.006). No differences on psychopathology, somatisation, and extraversion scales. Trauma-exposed subgroup scored higher on TCI's 'harm avoidance' (t = -2.304; p = .027) and less on 'self-directedness' (t = 2.343; p=.024). Trauma-exposed subgroup scored higher scored on DIS-Q 'identity confusion and depersonalization' (t = -2.166; p = .037), but no difference on SDQ-20. Trauma-exposed subgroup scored higher on CERQ 'blaming others' cognitive coping strategy (t = -2.490; p = .018).
Boesten, Myers & Wijnen, 2019	Cross- sectional	FS N=217 M=38 F=83% Epilepsy clinic, USA	Unvalidated clinical interview	TSI 1/TSI-2 QOLIE-31-P	 Trauma-exposed subgroup scored higher on almost all TSI scales (except suicidality, somatic preoccupation, and dysfunctional sexual behaviours) suggesting greater trauma-related symptoms and psychological distress. Trauma-exposed subgroup scored lower on the total QOLIE-31-P (p=.008) and on subscale of 'energy' (p=.021). Age and education but not sex were significant covariates on both TSI and QOLIE-31-P.

Hingray et al., 2011	Cross- sectional	FS <i>N</i> =25 Trauma subgroup <i>Mdn</i> =28 No trauma subgroup <i>Mdn</i> =35 F=80% Neurology Department, France	CTQ and clinical interview for adult trauma- exposure	DES TAS-20 MADRS HAM-A MINI- DSMIV-TR	•	Only one person in the no-trauma subgroup had a psychiatric diagnosis (histrionic personality), whereas everyone in the trauma-exposed subgroup had at least one current or historic psychiatric diagnosis. No difference on total score of TAS-20 (p=.192) however, the trauma-exposed subgroup scored significantly higher on the dimension of "difficulty describing feelings" (p=.033). Trauma-exposed subgroup scored significantly higher on the DES global scores (p=.000) and on all three sub-dimensions (p<.01). All forms of trauma-exposure (emotional, physical, and sexual) correlated with DES ($r > .58$, p<.001). Trauma-exposure correlated with suicide attempts ($r > .60$, p<.001), psychiatric antecedents and comorbidity ($r > .05$, p<.01) higher MADRS ($r > .06$, p<.01), and higher levels on HAM-A ($r > .05$, p<.05).
Hingray et al., 2017	Cross- sectional	FS N=31 Trauma subgroup M=30 No trauma subgroup M=36.2 F=80% Neurology Department, France	CTQ and clinical interview for adult trauma- exposure	DES MADRS HAM-A MINI- DSMIV-TR	•	35% of repeated childhood trauma-exposed subgroup also had PTSD compared to 0% in no-trauma subgroup. The repeated childhood trauma-exposed subgroup scored higher on both DES total (40.8 versus 21.8, p<.001) and all DES subscales. Repeated childhood trauma-exposed subgroup had higher scores on MADRS (17.9 versus 6.1, p<.001), HAM-A (21.5 versus 11.9, p<.01), and higher somatic anxiety on subscales of cardiovascular, gastrointestinal, genitourinary, autonomic symptoms. Repeated childhood trauma-exposed subgroup reported more current and past mood disorders, anxiety disorders, suicide attempts, and PTSD.

Kienle et al., 2017	Case- control	FND N=60 M=42.6 F=75% PTSD controls N=39 M=41.3 F=84% Healthy controls N=40 M=40.6 F=85% Neurological rehabilitation centre, Germany	KERF	DES SDQ-20 TAS-26 PDS	•	FND+PTSD had higher TAS-26, SDQ-20, PDS, and DES than FND without PTSD. FND+PTSD also had higher emotional abuse but not sexual/physical abuse than FND without PTSD. FND+PTSD did not differ from PTSD controls on SDQ- 20 or PDS, but FND+PTSD had lower DES scores. PTSD controls had more sexual and physical abuse than FND+ PTSD, however, no differences found on physical, emotional maltreatment, and lifetime trauma. Positive association found for the number of lifetime traumatic events, PDS, and SDQ-20 for the two subsamples of FND participants. TAS-26 correlated with PDS ($r=.31$, $p=.02$). Childhood adversity, number of lifetime trauma and PDS explained 30% variance of SDQ-20, however, childhood abuse and number of lifetime events did not explain variance in addition to PDS ($\beta = .38$, p < .001).
Lally et al., 2010	Case- control	FS N=27 M=33 F=81% Epilepsy controls N=39 M=39 F: 82% Regional Neurology Centre, Northern Ireland, UK	THQ	BSI	•	No difference on trauma-exposure events tally (p=0.5) or type of trauma (p=.13) between FS and epilepsy controls. THQ total trauma (ρ =0.63, p=.01), THQ general trauma (ρ =0.66, p=.01), THQ sexual and physical trauma (ρ =0.448, p=.05) correlated with General Severity Index of BSI in the FS group but not in the ES group.

Martino et al., 2020	Cross- sectional	FS N=63 sexual abuse subgroup M=38.93 No-trauma subgroup M=34.19 F=77% Epilepsy centre, Italy	TEC	DES SDQ-20 BDI-2 TAS-20 HAM-A	•	Subgroup with sexual trauma scored higher on DES (p=.003) and BDI-2 (p=.001) than the no-sexual trauma subgroup. No differences between subgroups on HAM-A (p=.130), TAS-20 (p=.137) or SDQ-20 (p=.486).
Myers et al., 2013a	Case- control	FS N=66 M=38.4 F=91% Epilepsy controls N=35 M=42.1 F=60% Epilepsy clinic, USA	Unvalidated clinical interview	TSI-2 TAS-20	•	Positive correlations between TSI-2 subscales and TAS- 20, including Anxious Arousal ($r = .497$, p<000), Intrusive Experiences ($r = .541$, p<.000), Dissociation ($r = .421$, p<.001), Defensive Avoidance ($r=.444$, p<.000) No significant correlation between TAS-20 and age of trauma-exposure, physical trauma, or sexual trauma. TSI-2 subscales of Defensive Avoidance ($t=-3.34$, p=.002), Intrusive Experiences ($t=5.50$, p = .0001) independently predicted TAS-20.

Myers et al., 2013b	Cross- sectional	FS N=82 M=39.7 F= 87% (60 participants from previous study used) Epilepsy clinic, USA	Unvalidated clinical interview (only enquired about physical and sexual abuse)	TSI-2 CISS	 Trauma-exposure was not associated with any CISS coping styles. CISS emotion-focused coping subscale correlated with TSI-2 subscales of Anxious Arousal (<i>r</i>=.702, p=.001), Depression (<i>r</i>=.682, p=.001), Intrusive Experiences (<i>r</i>=.622, p=.001), Dissociation (<i>r</i>=.537, p=.001), and Tension Reduction Behaviours (<i>r</i>=.389, p=.001). Three trauma-related TSI-2 scales were independent predictors of CISS emotion-focused coping: Depression (<i>t</i> = 3.62, p = .001), Intrusive Experiences (<i>t</i> = 4.40, p = .001), Tension Reduction Behaviours (<i>t</i> = -2.52, p = .016), No TSI-2 subscales were associated with CISS task-oriented or avoidance-oriented coping.
Myers et al., 2013c	Cross- sectional	FS N=61 M = 37.5 F=86% Epilepsy clinic, USA	Unvalidated clinical interview (only enquired about physical and sexual abuse)	TSI-2 MMPI-2-RF	 Trauma-exposed subgroup were significantly more likely to be diagnosed with a mood/bipolar disorder (p = .005, χ = 8.69) and PTSD (p = .04, χ = 3.93). Trauma-exposed subgroup scored higher than the notrauma subgroup all TSI-2 subscales; and the trauma-exposed subgroup differed significantly on the tally of clinically elevated (T ≥ 65) (t = 5.295 and p = .001) TSI-2 scales. On the MMPI-2-RF, the trauma-exposed subgroup scored significantly higher than the no-trauma subgroup on the Demoralization scale (t = -2.280, p=.028). Higher tally of trauma events correlated significantly with the 'PTSD likely' status (t = - 5.295, p = .001). PTSD subgroup had higher rate of depression/bipolar diagnoses (37.8% vs. 0%), and a higher incidence of suicide attempts (63.6% vs. 36.3%).

Pick, Mellers & Goldstein, 2017	Case- control	FS N=40 Mdn=40 F= 80% Health Controls N=43 Mdn=36 F=81.4% Neuropsychiatry Clinic, UK	TEC	MDI SDQ-20 PDS HADS	•	FS participants reported more sexual and physical abuse, but not emotional abuse, than healthy controls. 66.7% of FS participants met criteria for PTSD compared to 0% in the control group. TEC total positively correlated with MDI subscales of Depersonalisation (r =.444, p=.005) and Emotional Constriction (r =.433, p=.006). TEC sexual abuse positively correlated with MDI subscale of Identity Dissociation (r =.483, p=.002). Sexual abuse significantly predicted SDQ-20 and HADS depression. SDQ-20 fully mediated the relationship between sexual abuse and FS diagnosis (depression was not a significant mediator).
Rosales et al., 2020	Cross- sectional	FS N=143 M=39 F=83% Caucasian=73% Neuropsychiatric clinic, USA	Unvalidated interview	TIMMS ASQ	•	Those with a history of childhood abuse scored lower on emotional attention (p=.04) and clarity (p=.001) (TIMMS), adjusting (p=.02) and tolerating (p=.02) (ASQ) than those without a history of childhood abuse. Differences between physical and sexual abuse exposure: Physical abuse led to lower scores on emotion clarity (p=.02) and higher scores on emotion concealing (p=.02) No differences in scores for sexual abuse. PTSD subgroup scored lower on emotion clarity (p=.02), adjusting (p = .009), and tolerating (p = .01), and scored higher on the emotion concealing (p = .02) than the no- PTSD subgroup.

Sar, Islam & Öztürk, 2009	Mixed FND N=32 (70.6% FS) M=33.3 F=80% Psychiatric outpatient	СТQ	DES STQ-20 CADSS DDIS DSM- IV	 Childhood emotional abuse predicted DES (β = .54, p=.002) and SDQ-20 scores (β = .44, p=.013). CADSS scores were predicted both by emotional abuse (β = .86, p=.001) and neglect (β =47, p < .014).
	service for dissociative disorders, Turkey	;		
Spinhoven et al., 2004	Three FND samples: FS N=63 M=31.5 F=77% Motor and sensory FND 1st sample: N=102, M=39.1 F=74%	STI TEC STQ	SDQ-20 DIS.Q DES SCL-90	 FS sample: SDQ-20 was associated with sexual abuse (r=.37, p<.001) and physical abuse (r=.27, p<.05). Physical abuse correlated with DIS.Q (r=.28, p<.05). FND sample 1: Sexual abuse correlated with DIS.Q (r=26, p<.05), but no significant correlation with DES (r=.04, p>.05). No correlation between physical abuse and DIS.Q or DES (p>.05). FND sample 2: Physical abuse correlated with SDQ-20 (r=.26, p<.05). SCL-90 fully mediated all relationships, except for one partial correlation between physical abuse and SDQ-20, (r=.27, p<.05) in FND sample 2.
	Motor and sensory FND 2nd sample: N=54 M=37.7 F=83% Tertiary services, Netherlands			

Steffen et al., 2015	Case- control	Sensory/Motor FND N=45 M=40.4 F=71% Healthy Controls N=45 M=44.8 F=68% Neurorehabilitation clinic, Germany	ETI	SDQ-20 PSS-I LEQ TAS-26	 FND participants reported more general and emotional abuse/neglect than controls, but no difference found on physical and sexual abuse. FND had more co-occurring PTSD (24% vs 6%) than controls. In the FND group SDQ-20 correlated with emotional abuse/neglect (<i>r</i> = 0.41, p<.001) and general traumata (<i>r</i> = 0.39, p<.001) but not physical or sexual abuse. Independent predictors of SDQ-20: negative life events (β- = 0.37, p < 0.01), emotional childhood abuse (β = 0.21, p < 0.05) and alexithymia (β = 0.28, p < 0.05), adjusted R² = 0.4; p < 0.01. The relationship between emotional abuse and SDQ-20, was partially mediated by alexithymia and negative life events.
Steffen- Klatt et al., 2019	Case- control	Motor/sensory FND N=82 M=41.63 F=73% Healthy Controls N=82 M=45.94 F=71% Neurological rehabilitation centre, Germany	KERF	PDS BDI-2 SDQ-20 TAS-26	 Correlations: Total KERF and BDI-2 (r=.34, p=.001). KERF abuse and SDQ-20 (r=.30, p=.044). KERF neglect and BDI-2 (r=.38, p<.001). PDS correlated with overall KERF (r=.39, p<0.001), KERF abuse (r=.41, p<0.01) and KERF neglect (r=.38, p<0.001). PDS and SDQ-20 (r=.39, p<0.001). PDS and BDI-2 (r=.49, p<0.01). Results for whole sample only (not significant for subgroups): TAS-26 and total KERF (r=.32, p<0.001), KERF abuse (r=.42, p<0.001). TAS-26 and PDS (r=.42, p<0.001). TAS-26 partially mediated relationship between total KERF was and SDQ-20.

Williams et al., 2019		Motor FND N=56 (comorbid FS in 20) M=40.2 F=73% FND Clinic, USA	CTQ	RSQ LEC	 Fearful attachment positively correlated with LEC 'happened to me' (r=.37, p=.005), CTQ- abuse (r=.55, p<.001), CTQ-neglect (r=.34, p=.01). CTQ sexual abuse (β=.29, p=.03) and CTQ emotional abuse (β=.49, p=.002) independently predicted fearful attachment. This model explained 37% of the variance in fearful attachment style scores.
Zeng, Myers & Lacman, 2018	Cross- sectional	FS <i>N</i> =156 <i>M</i> =37.87 F= 85% Epilepsy clinic, USA	Unvalidated clinical interview	TSI-2 TAS-20 CISS	 No differences between subgroups (FS with PTSD, FS no PTSD, and FS no trauma) on task-oriented coping strategies. No differences between subgroups on avoidance-oriented coping. Large group differences on alexithymia (η2=0.19) and medium group difference on emotion-focused coping (η2=0.13). Post-hoc tests revealed that FS+PTSD subgroup had higher alexithymia scores than the other two subgroups (which did not differ from each other). FS+PTSD subgroup was also more likely to use emotion-coping than the other two groups.

Note. **TEC**= The Traumatic Experiences Checklist; **DIS.Q**= The Dissociation Questionnaire; **SDQ-20**= The Somatoform Dissociation Questionnaire; **CERQ**= Cognitive Emotion Regulation Questionnaire; **Short MMPI**=Shot version of the Minnesota Multiphasic Personality Inventory (Dutch); **Short TCI**=Short version of Temperament and Character Inventory (Dutch); **TSI-2**= Trauma Symptom Inventory-2; **QOLIE-31-P**= Quality of Life in Epilepsy Inventory; **CTQ**= The Childhood Trauma Questionnaire; **DES**= The Dissociative Experiences Scale; **TAS-20/26**= Toronto Alexithymia Scale; **MADRS**= Montgomery-Asberg Depression Rating Scale; **HAM-A**= Hamilton Anxiety Rating Scale; **MINI-DSMIV-TR**= The Mini-International Neuropsychiatric Interview; **KERF**= The 'Maltreatment and Abuse Chronology of Exposure' (MACE) Scale for the Retrospective Assessment of Abuse and Neglect During Development (MACE) (KERF German version); **PDS**= Post-Traumatic Stress Diagnostic Scale; **THQ**= Trauma History Questionnaire; **BSI**= The Brief Symptom Inventory; **BDI-2**; Beck Depression Inventory; **CISS**= The Coping Inventory for Stressful Situations; **MMPI-2-RF**= Minnesota Multiphasic Personality Inventory-2-RF; **MDI**= Multiscale Dissociation Inventory; **HADS**= Hospital Anxiety and Depression Scale; **ASQ**= Affective Styles Questionnaire; **TIMMS**= Trait Meta Mood Scale; **CADSS**= The Clinician-Administered Dissociative State Scale; **DDIS(DSM-IV)**= The Dissociative Disorders Interview Schedule DSM-IV; **STI**= The Structured Trauma Interview; **STQ**= Short Trauma Questionnaire; **SCL-90**= The Symptom Checklist-90; **ETI**= Early Trauma Inventory; **PSS-I**= PTSD Symptom Scale - Interview Version; **LEQ**= Life Events Questionnaire; **RSQ**= The Relationship Scales Questionnaire

Results

Study characteristics

The search yielded 18 eligible studies for the purpose of this review. Studies were conducted in the USA (N=7), UK (N=2), Germany (N=3), Netherlands (N=2), Turkey, (N=1) France (N=2) and Italy (N=1). Twelve studies were cross-sectional and six were case control. Control groups included participants with epilepsy, PTSD, and healthy volunteers. Twelve studies only had participants with FS, three had FND other than FS, and three had a mixed sample. Participants' mean age ranged from 30 to 42 years. Of the studies that reported on the ethnicity of their participants, 73-80% were Caucasian. The mean number of years in education for participants with FND ranged from 7 to 14 years. All participants were recruited from specialist or tertiary clinical services.

Trauma-exposure characteristics

The prevalence of self-reported trauma-exposure ranged from 63% to 97% in the reviewed studies. Of those that reported on individual trauma types, sexual abuse prevalence ranged from 9% to 82%, physical abuse and neglect ranged from 12% to 62%, and emotional abuse and neglect ranged from 24% to 70%. Studies which assessed the tally of trauma-exposure reported that between 28% to 100% of their participants reported multiple traumatic events.

Case-control studies that compared trauma-exposure rates between FND and controls found that FND participants had higher rates of trauma-exposure than healthy controls (Kienle et al., 2017; Steffen et al., 2015; Steffen-Klatt et al., 2019), similar rates to epilepsy controls (Lally et al., 2010) and lower rates than PTSD controls (Kienle et al., 2017). Mean age of initial trauma reported across studies ranged from 11 to 26 years.

Measurement of trauma-exposure

Ten studies used psychometrically validated self-report measures of trauma-exposure, six studies used unvalidated clinical interviews, and two studies used both. Studies assessed either childhood or lifetime trauma-exposure, and experiences of direct interpersonal trauma-exposure (sexual, physical, emotional) were the most common.

Four studies (Hingray et al., 2011, 2017; Sar et al., 2009; Williams et al., 2019) assessed childhood trauma-exposure using the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1994). The CTQ is a measure of adverse childhood experiences that covers five domains: physical abuse and neglect, emotional abuse and neglect, and sexual abuse. Four studies (Bodde et al., 2013; Martino et al., 2020; Pick et al., 2017; Spinhoven et al., 2004) used the Traumatic Experiences Checklist (TEC; Nijenhuis et al., 2002) which measures lifetime experiences of neglect, sexual, physical, and emotional abuse. Two studies (Kienle et al., 2017; Steffen-Klatt et al., 2019) used the Maltreatment and Abuse Chronology of Exposure (MACE; German version KERF; Isele et al., 2014) which measures different forms of childhood sexual trauma, physical and emotional abuse, and neglect. One study (Lally et al., 2010) used the Trauma History Questionnaire (THQ; Hooper et al., 2011) which assesses physical and sexual trauma, emotional neglect but not emotional abuse. One study (Steffen et al., 2015) used the Early Trauma Inventory (ETI; Bremner et al., 2000) which assesses sexual, physical, emotional, and general trauma. One study (Spinhoven et al., 2004) used the Structured Trauma Interview (STI; Draijer & Langeland, 1999) which assesses childhood parental dysfunction, physical and sexual abuse, but not emotional abuse or neglect, and the Short Trauma Questionnaire (STQ; Dalle Grave et al., 1996) which assesses lifetime experiences of physical, sexual, emotional abuse and neglect, and war.

Studies that assessed trauma-exposure using unvalidated interviews varied in their definition and classification of trauma-exposure. Four studies (Hingray et al., 2011, 2017; Myers et al., 2013b, 2013c) did not assess emotional abuse or neglect; two studies assessed general trauma (e.g., bereavement), physical, sexual, and emotional abuse. One study (Myers et al., 2013a) did not specify trauma types.

Synthesis of key findings

Due to the heterogeneity in the quality, design, and analysis of the studies in this review, a meta-analysis was not possible. Therefore, a narrative review was deemed appropriate to summarise and synthesise findings. The term 'trauma-exposure' relates to the collective experience of lifetime traumatic events, unless otherwise specified.

Dissociation

Twelve studies examined dissociation in relation to trauma-exposure; four of which measured psychoform dissociation (i.e., detachment) only, two measured somatoform dissociation (i.e., compartmentalisation) only, and six measured both types.

Psychoform dissociation was measured by the Dissociative Experiences Scale (DES), Trauma Symptom Inventory (TSI/TSI-2), Dissociation Questionnaire (DIS.Q), and Multiscale Dissociation Inventory (MDI). Somatoform dissociation was measured by the Somatoform Dissociation Questionnaire (SDQ-20).

Subgroup comparison on dissociation

Six studies explored subgroup differences on psychoform dissociation. All studies found that the trauma-exposed subgroup reported significantly higher scores than the no-trauma subgroup. Martino et al. (2020) found the highest scores in those with a history of sexual abuse. The severity of dissociation varied by the outcome measure used; studies that used the DES reported clinically elevated scores, whereas studies that used the TSI reported mixed results. Boesten et al. (2019) found a significant difference for age and years of education (but not sex), which suggests that those with a higher age and lower education reported higher psychoform dissociation scores. Of note, all these studies reported on participants with FS, which limits the generalisability to the wider FND population.

Two studies compared subgroups on somatoform dissociation based on either lifetime trauma-exposure (Bodde et al., 2013) or sexual trauma (Martino et a., 2020). Neither study found a significant difference between subgroups. Further examination of somatoform dissociation scores across studies revealed that all but one study (Bodde et al., 2013) found clinically elevated scores, irrespective of trauma-exposure, which may suggest universality of somatoform dissociation in this client group.

Relationship between trauma-exposure and dissociation

Seven studies explored the relationship between trauma-exposure and dissociation. Two studies had FS samples, three had non-FS samples, and two had mixed samples.

Studies which used the DES reported mixed results; Hingray et al. (2011) found moderate associations between the DES and lifetime exposure to sexual, physical, and emotional trauma, whereas Sar et al. (2009) only found a positive association for childhood emotional abuse and

neglect, but not childhood physical or sexual abuse. Spinhoven et al. (2004) also did not find an association between physical and sexual trauma-exposure on the DES, but they did on the DIS.Q; however, this was no longer significant once psychological distress was controlled for. Pick et al. (2017) found a moderate association between total trauma-exposure and subscales of depersonalisation and emotional constriction on the MDI, whereas sexual abuse had a moderately strong association the identity dissociation subscale.

Five studies examined the relationship between trauma-exposure and somatoform dissociation. Childhood emotional abuse was significantly associated with somatoform dissociation (Sar et al., 2009; Steffen et al., 2015; Steffen-Klatt et al.,2019); however, results for childhood physical and sexual abuse were conflicting. Spinhoven et al. (2004) did find an association between physical and sexual abuse and somatoform dissociation, however, after controlling for psychological distress, only one (of three) semi-partial correlations remained for physical abuse and SDQ-20, suggesting that the relationship between trauma-exposure and somatoform dissociation can be partly explained by the severity of psychological distress.

Alexithymia was also found to partially mediate the relationship between overall childhood trauma-exposure (Steffen-Klatt et al., 2019), childhood emotional abuse (Steffen et al., 2015) and somatoform dissociation. However, in the Steffen-Klatt et al. (2019) study, this effect was only significant for whole sample (FND and healthy controls) which may be due to low power to detect a subgroup effect, or the conservative adjustments used for multiple comparisons. Kienle et al. (2017) found that total childhood trauma-exposure was not a significant predictor of somatoform dissociation severity once PTSD was controlled for, suggesting that PTSD symptoms, rather than trauma-exposure, are crucial in the development of somatoform dissociation.
Conclusion

The trauma-exposed subgroup reported more severe and frequent experiences of psychoform dissociation than the no-trauma subgroup. Those with a history of sexual abuse reported more severe psychoform dissociation than those without. Higher age and lower education were associated with higher psychoform dissociation scores. No difference was found on somatoform dissociation between subgroups.

Cumulative childhood trauma-exposure, particularly emotional abuse, was associated with more severe psychoform and somatoform dissociation, whereas results for physical and sexual abuse were mixed depending on the study and the outcome measure used. Three possible mediators were identified to explain the relationship between trauma-exposure and somatoform dissociation: psychological distress, alexithymia, and PTSD.

PTSD

Eight studies reported that between a quarter and a third of their sample met the criteria for current PTSD. Seven of these studies had FS-only participants. One study (Pick et al., 2017) reported PTSD in over two thirds of their trauma-exposed subgroup, however, the PTSD measure was only administered to those who reported a moderate-to-severe impact of their traumatic experiences, which likely inflated this result.

There was a difference in the analysis and reporting of PTSD across studies; some examined overall PTSD severity, some examined PTSD symptoms and symptom clusters, while others dichotomised participants based on the presence or absence of a PTSD diagnosis.

Outcome measures of PTSD were based on DSM-IV or DSM-5 criteria and included: PTSD Symptom Scale-Interview Version (PSS-I), The Mini-International Neuropsychiatric Interview (MINI-DSMIV-TR), Post-Traumatic Stress Diagnostic Scale (PDS), Posttraumatic Stress Disorder Checklist-5 (PTSD-CL5), and the Trauma Symptom Inventory (TSI/TSI-2).

Subgroup comparison on PTSD

Seven studies examined differences on cognitive-emotional processing, coping styles, and psychological distress between three subgroups: PTSD, trauma-exposed without PTSD, and no-trauma. The outcome measures used were the Toronto Alexithymia Scale (TAS-20); Trait Meta Mood Scale (TIMMS); Affective Styles Questionnaire (ASQ); The Coping Inventory for Stressful Situations (CISS); and patient medical records.

The PTSD subgroup was characterised by greater childhood emotional abuse and neglect (which differentiated those with and without PTSD), psychoform and somatoform dissociation (Kienle et al., 2017), mood related diagnoses and suicide attempts (Myers et al., 2013c). On measures of emotional processing and coping styles, the PTSD subgroup reported higher alexithymia (Kienle et al., 2017; Zeng et al., 2018) even after controlling for years of education. The PTSD subgroup reported greater use of emotion-focused coping strategies (Zeng et al., 2018).

Furthermore, Rosales et al. (2020) found that both the PTSD and the childhood traumaexposed subgroup reported less emotional clarity, a lower ability to tolerate and adjust to emotional states, and a higher use of suppression and avoidance strategies than the no-trauma subgroup. No differences were reported between PTSD and childhood trauma-exposed subgroups. Of note, only the 'adjusting to emotions' subscale was significantly different from normative data.

Relationship between trauma-exposure and PTSD

Four studies examined the relationship between trauma-exposure and PTSD. Findings revealed that trauma-exposure was associated with a higher likelihood of a PTSD diagnosis (Myers et al., 2013c) and greater PTSD severity (Kienle et al., 2017; Steffen-Klatt et al., 2019). Steffen-Klatt et al. (2019) found that experiences of childhood abuse and neglect were associated with greater PTSD severity in the FND group but not in healthy controls. They also found that alexithymia partially mediated the relationship between overall childhood trauma-exposure and PTSD, however, this was only significant for the whole sample (FND and healthy controls) and not individual subgroups.

Conclusion

Between a quarter and a third of individuals with FND had co-occurring PTSD, although most had FS. Most studies found that the PTSD subgroup reported more psychological distress, somatoform and psychoform dissociation, alterations in emotional processing and an overreliance on emotion focused coping styles. PTSD status and severity varied with cumulative trauma-exposure (especially childhood abuse and neglect), alexithymia, and emotion-focused coping. Alexithymia was identified as a possible mediator between childhood trauma-exposure and PTSD.

Emotional processing and regulation

Ten studies examined emotional processing and regulation. Seven had FS-only participants, and three had motor/sensory FND. The outcome measures used were the Cognitive Emotion Regulation Questionnaire (CERQ), TIMMS, ASQ, TAS-20/TAS-26, and CISS.

Subgroup comparison on emotional processing and regulation

Four studies compared alexithymia severity between subgroups. No difference was found on alexithymia severity between subgroups (Hingray et al., 2011; Kienle et al., 2017; Martino et al., 2020; Zeng et al., 2018), unless PTSD was also present, in which case, the scores were significantly higher (Kienle et al., 2017; Zeng et al., 2018). Alexithymia scores across studies were in the 'possible alexithymia' range.

Rosales et al. (2020) found that the childhood trauma-exposed subgroup scored lower on emotional clarity, attention to emotions, ability to tolerate and adjust to emotional states; and had a greater propensity for relying on emotional concealing strategies, such as suppression and avoidance. The trauma-exposed subgroup also reported a higher use of the 'blaming others' coping strategy, however, this result did not significantly differ from normative data (Bodde et al., 2013).

Relationship between trauma-exposure and emotional processing and regulation

Three studies examined the relationship between trauma-exposure and alexithymia, and one study assessed associations between trauma-exposure and coping styles. Two studies (Steffen et al., 2015; Steffen-Klatt et al., 2019) found that cumulative childhood adversity, especially childhood emotional abuse/neglect, was associated with more severe alexithymia. Conversely, Myers et al. (2013a, 2013b) found no association between alexithymia and traumaexposure (physical and sexual abuse), but moderate associations were found for PTSD (Kienle et al., 2017; Myers et al., 2013a, 2013b). Similarly, Myers et al. (2013b) found no association between trauma-exposure and any coping style, but a positive association was found between PTSD and emotion-focused coping.

Conclusion

No significant difference on the severity of alexithymia was found between subgroups, except in cases of co-occurring PTSD, which had significantly higher scores. However, childhood trauma-exposure, especially emotional maltreatment, was related to more severe alexithymia. The childhood trauma-exposed subgroup had more difficulties with attending to, identifying, and tolerating their emotions. They were more likely to regulate distress by externalising and blaming others, or by concealing their emotions. However, no association was found between physical/sexual abuse and copying styles. Most emotional processing and regulation characteristics were not outside the normative range, except alexithymia. This suggests that trauma-exposure may have more of an impact on emotional processing than regulation.

Mental health and personality characteristics

Eleven studies explored mental health and/or personality characteristics. The outcome measures used were: TSI/TSI-2; Montgomery-Asberg Depression Rating Scale (MADRS), Beck Depression Inventory (BDI-2), Hospital Anxiety and Depression Scale (HADS), Hamilton Anxiety Rating Scale (HAM-A) Minnesota Multiphasic Personality Inventory-2-RF (MMPI-2-RF), Quality of Life in Epilepsy Inventory (QOLIE-31-P), Mini-International Neuropsychiatric Interview (MINI-DSMIV-TR) and medical records.

Subgroup comparison on mental health and personality characteristics

Five studies compared subgroups on mental health characteristics, and two studies on personality traits. All studies had FS-only participants. The trauma-exposed subgroup reported significantly higher rates of psychiatric diagnoses, suicide attempts (65% vs 7%) (Hingray et al., 2017), and a lower quality of life (Boesten et al., 2019). The most common psychiatric diagnoses were related to mood, anxiety, and PTSD (Hingray et al., 2011, 2017; Myers et al., 2013c).

The trauma-exposed subgroup reported more severe depression (especially for those with sexual abuse) and anxiety (Boesten et al., 2019; Hingray et al., 2017; Martino et al., 2020). Scores on somatisation and physical symptoms of anxiety, however, were mixed (Boesten et al., 2019; Hingray et al., 2017; Martino et al., 2020). One explanation for this may that some of the items measuring physical anxiety and somatisation were conflated with core FND symptoms.

Two studies compared trauma-related symptomatology on the TSI/TSI-2, which assesses the impact of cumulative, rather than index, trauma. Myers et al. (2013c) and Boesten et al. (2019) found that their trauma-exposed subgroup had more severe symptoms of anxious arousal, intrusive experiences, avoidance, impaired self-reference, psychoform dissociation, and anger. Conflicting results were found for dysfunctional sexual behaviours and suicidality.

Assessment of personality traits revealed that the trauma-exposed subgroup was characterised by greater demoralisation (Myers et al., 2013c), negativism (e.g., feelings of dissatisfaction and hostility) shyness, harm-avoidance attitudes and behaviours (e.g., excessive worry, fearfulness), and less self-directedness (e.g., self-acceptance, resourcefulness) (Bodde et al., 2013).

Relationship between trauma-exposure and mental health difficulties

Six studies reported on associations between trauma-exposure and mental health difficulties. Moderate-to-large associations were found between trauma-exposure and fearful attachment (Williams et al., 2019), depression, anxiety, suicide attempts, PTSD, and the

likelihood of having a mental health difficulty (Hingray et al., 2011; Kienle et al., 2017; Pick et al., 2017; Steffen-Klatt et al., 2019). Lally et al. (2010) found an association between different types of trauma-exposure and overall psychological distress in participants with FS but not in epilepsy controls, even though trauma-exposure rates were similar. Alexithymia was found to partially mediate the relationships between overall adverse childhood experiences and depression for the whole sample (FND and healthy controls) (Steffen-Klatt et al., 2019).

Conclusion

Individuals with FND presented with significant co-occurring mental health issues, such as depression, anxiety, and PTSD. When the sample was further analysed by trauma-exposure, the results indicated greater psychological distress, anxiety, depression, PTSD, and lower quality of life. This was further supported by positive associations between trauma-exposure and fearful attachment, psychological distress, depression, anxiety, and PTSD. Alexithymia was a likely mediating factor between childhood trauma-exposure and levels of depression. Scores on physical anxiety and somatisation were mixed between subgroups. The trauma-exposed subgroup was further characterised by maladaptive personality traits like demoralisation, negativism, harm avoidance and lower self-directedness.

Summary of findings

Is there a difference in psychological characteristics between individuals with FND based on trauma-exposure?

The trauma-exposed subgroup was characterised by more psychoform dissociation, psychological distress, depression, anxiety, emotion processing difficulties, emotion-focused coping styles, and maladaptive personality traits marked by negative affect, and a lower quality of life. Furthermore, the trauma-exposed group was more likely to meet the criteria for PTSD and reported more severe PTSD symptoms. However, no differences were found between subgroups on somatoform dissociation or alexithymia.

What are the psychological correlates of trauma-exposure?

The reviewed studies found that trauma-exposure, especially during childhood, was associated with greater psychoform and somatoform dissociation, PTSD status and severity, alexithymia, and mental health difficulties. Childhood adversities, especially emotional abuse, had the most robust link with dissociation, PTSD, and alexithymia. Alexithymia and psychological distress were found to partially mediate the relationship between trauma-exposure and somatoform dissociation; and PTSD was a unique predictor of somatoform dissociation. Alexithymia was a possible mediator in the relationship between trauma-exposure and PTSD.

Methodological critique

Sample

All the studies included in this review did not fully meet the criteria of a representative sample. All participants were recruited from tertiary and specialist services, such as outpatient psychiatric services, FND clinics, epilepsy clinics, and neurological rehabilitation services. This limits the generalisability of findings, as individuals seen in such services may be more likely to present with greater symptom severity and/or co-occurring mental health difficulties. Furthermore, most of the studies recruited participants with FS, and excluded those with IQs of less than 70, or those with co-occurring neurological conditions (which affects approximately 20% of cases (Bennett et al., 2021)); and three studies excluded participants with historical or current mental health difficulties. Most participants were female (71%-91%), which is consistent with the higher prevalence of FND reported in women; however, these findings may not be fully comparable or applicable to men. Finally, only two studies reported on the ethnicity of their sample which was 73-80% Caucasian. Three studies mentioned non-responders, however, no descriptive information on them was provided, which prevented any comparison between these groups. Finally, it is important to note, that in 2013, DSM-5 had removed the requisite psychological stressor/trauma criteria for the diagnosis of FND, which may have resulted in an over-reporting of trauma-exposure in studies conducted up until that point.

The participant recruitment strategy was unclear in half of the studies, and two studies did not provide any information on inclusion/exclusion criteria of eligible participants beyond a positive FND diagnosis. In non-randomised research, consecutive recruitment can reduce sampling bias by including all eligible individuals into the study (Thewes et al., 2018). It is

possible, therefore, that bias was introduced through the inclusion of participants that were demographically or clinically different.

The sample size of FND participants across studies was relatively small (M=72, Mdn=60), and only three studies reported a power analysis for determining the sample size, all of which were powered to a detect a large effect only. According to Cohen's (1992) guidelines, six studies were powered to detect medium effect sizes, and the rest were powered to detect large-to-very large effect sizes only for their primary analyses. Inadequately powered studies lack precision to address the research question, inflate the risk of Type II error, and can lead to spurious conclusions (Maxwell, 2004). Moreover, underpowered studies are more likely to inflate effect sizes, and report greater discrepancies and departures from the null hypothesis that are not there (Rochefort-Maranda, 2021). Not all studies adjusted for the use of multiple comparisons in their analyses, which raised the risk of Type I error; and one study did not provide a statistical analysis plan for their results. Furthermore, studies rarely reported confidence intervals or effect sizes, which limits the interpretation of results, and their generalisability and clinical utility.

Design

All the studies included in this review used cross-sectional and case-control designs. Given that data were collected at a single time point, causality cannot be assumed, nor the direction of associations between variables confirmed. All studies relied of self-report measures which may have introduced response bias, such as the over/under-reporting of symptoms or events due to social desirability or inaccurate recall. Most of the studies controlled for demographic variables such as age, gender, and years of education in their analyses, however, only two studies controlled for psychological distress, which resulted in some associations being reduced or non-significant. Failure to control for key factors, such as psychological distress, limits the specificity of results and their interpretation.

Measures of trauma-exposure and psychological factors

The findings reported in this review must be carefully considered alongside the quality of the assessment and analysis of exposure and outcome variables. There was considerable methodological diversity in the measurement of variables, which raises concern about measurement error, internal validity, and the robustness of studies.

Trauma-exposure was assessed by validated self-report measures in ten studies, unvalidated clinical interviews in six, and a mixture of both in two studies. Measurement of key trauma-exposure characteristics such as trauma type, age at exposure, and frequency/duration varied across studies. For example, some studies did not specify or differentiate between trauma types or initial age of trauma-exposure in their analysis, whereas others only included firstperson experiences, and excluded events that were witnessed, learnt about, or part of someone's job. Minimal information was available on unvalidated clinical interviews that were used to assess trauma-exposure. Differences in the definition and criteria of trauma-exposure were apparent; for example, some studies did not assess for emotional abuse or neglect, one study did not report on events that only seldom occurred, and others only included events that were rated as 'severe'. Overall, the assessment of trauma-exposure was limited by inconsistent definitions and measurement of trauma and trauma characteristics, and therefore may not be fully representative of the relationship between trauma-exposure and psychological variables in FND.

Outcome measures of psychological factors also varied across studies. Most studies used validated self-report measures or structured clinical interviews to assess variables which increased the reliability of findings. Three studies, however, dichotomised psychological difficulties by recording the presence/absence of psychiatric diagnoses obtained from validated scales or self-reports in unvalidated interviews. Dichotomising variables can lead to loss of information (e.g., dimensions and levels of symptoms), reduced statistical power, and alterations in the association between variables (Peacock et al., 2012).

Another methodological issue worth highlighting is the validity and specificity of some of the outcome measures used. Studies that investigated dissociation predominantly used the DES and the SDQ-20. One shortcoming of the DES is that it assumes a unidimensional construct of dissociation, whereas at least two conceptually distinct constructs have been identified: detachment and compartmentalisation (Brown, 2016). Although the DES mainly measures detachment, some items also relate to compartmentalisation (Briere et al., 2005), however, an overall score conflates any differences. As such, the DES lacks specificity to identify differences between symptoms which may be unique to FND (Lawton et al., 2008). Similarly, the SDQ-20, which measures somatoform dissociation has limited specificity to differentiate between symptoms, as some of the items resemble detachment phenomena (Lawton et al., 2008), and others possibly reflect aspects of FND itself rather than the tendency to dissociate (Brown et al., 2013).

Discussion

This review aimed to investigate the impact of trauma-exposure in FND by a) examining psychological correlates of trauma-exposure and b) by examining differences on psychological characteristics between individuals with and without a history of trauma. Eighteen eligible studies were identified. Results will be discussed with reference to relevant research, after which clinical and research implications, and limitations of the review will be considered.

Studies that explored mental health and personality difficulties found that individuals with a history of trauma were characterised by both more prevalent and severe difficulties, including depression, anxiety, PTSD, suicide attempts, and personality traits marked by harm avoidance and pervasive negative affect. Trauma-exposure was associated with fearful attachment, and greater severity of mental health difficulties. Current models posit that psychological distress may a precipitating and perpetuating factor in FND which leads to the activation of FND symptoms (e.g., Brown & Reuber, 2016; Pringsheim & Edwards, 2017), however, findings on psychological difficulties in FND have been inconsistent. Given current findings, it may be that previous studies reported on mean group scores that possibly obscured the subgroup effect of trauma-exposure.

All studies that explored psychoform and somatoform dissociation reported a positive association with trauma-exposure. Childhood adversity, especially emotional abuse and neglect had the most robust link. This contradicted some previous findings that suggested somatoform dissociation was predominantly associated with physical injury (Nijenhuis et al., 2004; Waller et al., 2001). While the trauma-exposed participants reported psychoform dissociation scores to be in the upper average or clinical range, somatoform dissociation scores were clinically elevated across studies regardless of trauma-exposure, and no significant differences on somatoform

dissociation were found. There are several possible explanations for this finding. Studies that examined differences on somatoform dissociation only compared subgroups on lifetime sexual abuse or cumulative lifetime trauma, which may have obscured any differences between subgroups based on childhood trauma, especially emotional abuse, and neglect. Conversely, unanimously high scores may reflect a common underlying mechanism in FND, which supports Brown's (2016) hypothesis that FND is essentially a compartmentalisation phenomenon which can be generated by both trauma-exposure and other more subtle emotional events.

Although psychoform (i.e., detachment) and somatoform (i.e., compartmentalisation) dissociation are distinct constructs (van der Hart, 2021), research converges on their defensive function in response to overwhelming threat and danger (Spitzer et al., 2006). There is growing evidence which suggests that repeated traumatisation, especially in early life, can lead to the overactivation of dissociative responses (Cavicchioli et al., 2021), which could explain the association between childhood trauma-exposure and dissociation in this review. Furthermore, dissociation in FND has been associated with the over-modulation of anxious arousal and negative affect (Brown & Reuber, 2016b; Roberts & Reuber, 2014), some of which may be related to traumatic experiences.

Studies identified three possible mediators between trauma-exposure and somatoform dissociation: PTSD, alexithymia, and psychological distress. Regarding PTSD, it may be that somatoform dissociation reflects the re-experiencing of sensorimotor components of trauma material within a PTSD presentation (Schauer & Elbert, 2010); or a coping strategy for PTSDrelated distress. Regarding alexithymia and psychological distress, somatoform dissociation may be a coping strategy to alleviate associated negative affect, which reflects ICM, somatisation, and stress-system hypotheses about the function of FND; however, some authors hypothesise that

dissociation and psychological distress may be conflated in the measurement of dissociation. For example, some suggest that dissociation is in itself a distressing experience, and high dissociation scores may actually reflect psychological distress than dissociation (Williams et al., 2018). Overall, it is important to emphasise that cross-sectional research cannot ascertain causality or the direction of relationships, therefore it remains unclear whether dissociation is the sequalae of FND itself, or whether trauma-exposure had predisposed individuals towards more dissociative experiences.

As previously mentioned, alterations in emotion processing and regulation are considered important factors in the aetiology and maintenance of FND symptoms (Williams et al., 2018). The expression of emotions through physical symptoms may be either due to danger associated with emotional experiences or due to a deficit in the ability to identify emotions and differentiate them from physical sensations (Brown & Reuber, 2016). Results from the reviewed studies offer support to both ideas. Emotional processing difficulties were primarily indexed by alexithymia, which was associated with both PTSD and childhood emotional abuse and neglect, but not with lifetime physical or sexual abuse. Existing research suggests that emotional maltreatment is especially pernicious to the development of emotion processing and regulation skills (Kuo et al., 2015; Zdankiewicz-Ścigała & Ścigała, 2020).

In subgroup analyses, 'possible alexithymia' scores were reported across studies irrespective of trauma-exposure or PTSD. This may suggest that alexithymia is ubiquitous across FND individuals, and that trauma-exposure is not a helpful specifier of alexithymia, however, these findings need to be considered alongside the operationalisation and measurement of alexithymia. Research distinguishes between primary and secondary alexithymia, where the former is a marker of underdeveloped emotional processing due to early life adversity, while the

latter is a consequence of psychological distress and poor coping that occurs in later life (de Vente et al., 2006). Although the measurement of alexithymia in the reviewed studies does not allow for this distinction to be explored, the positive association with childhood trauma-exposure suggests that some of the elevated alexithymia scores may related to primary alexithymia, while others may be a function of secondary alexithymia, and relate to psychological difficulties such as PTSD. Regarding emotion regulation, subgroup analyses found that both the trauma-exposed and PTSD subgroups relied more on emotion-focused coping such as suppression, avoidance, and blaming others. Trauma-exposure was not associated with any coping style, however, these studies only included measures of sexual and physical, but not emotional, trauma-exposure.

Studies that examined PTSD found that childhood emotional abuse and neglect was associated with an increased risk of a PTSD diagnosis and greater PTSD symptom severity, and it was the only trauma type to differentiate individuals with and without PTSD. In addition to underdeveloped emotion regulation capabilities, childhood trauma-exposure is linked to enhanced threat processing (McLaughlin & Lambert, 2017) and alterations in neurobiological stress systems (Delahanty & Nugent, 2006) which confer vulnerability towards both PTSD and FND. There has been some speculation whether FND is related to the dissociative or complex subtype of PTSD given overlap in some symptoms (Fiszman et al., 2004), however, further research is required to explore and clarify this hypothesis. Overall, findings on PTSD indicated that for a subset of trauma-exposed individuals, concurrent PTSD is associated with a more severe symptom profile.

Clinical implications

While previous reviews highlighted the prevalence and risk of trauma-exposure in developing FND (Jones & Rickards, 2021; Ludwig et al., 2018), the current review added to the literature by describing the relationship between trauma-exposure and psychological features in FND, which may be helpful both in clinical assessment and treatment planning.

Individuals with a history of trauma reported more prevalent and severe psychological difficulties than individuals without such histories. As such, screening for a history of trauma during assessment should be an important task, which may also orient the clinician towards the assessment of related psychological features such PTSD, emotional dysregulation, and dissociation. Furthermore, the review highlighted elevated suicide attempts and personality difficulties such as pervasive negative affect and hopelessness in individuals with trauma-exposure which should inform clinical risk assessment and management. The presence of PTSD and childhood trauma, especially emotional maltreatment, should alert clinicians to a potentially more complex psychological profile, characterised by greater dissociative tendencies and emotional dysregulation.

Overall, these results emphasise the importance of a formulation-driven approach to treatment. The psychological difficulties identified in this review suggest an import role for psychological therapies in supporting this client group. A history of trauma should be integrated within a person-centred formulation, and where needed, trauma and trauma-related symptoms such as dissociation and PTSD may be the most appropriate treatment targets. There is emerging evidence that trauma-focused interventions such as Eye Movement and Desensitisation Reprocessing (EMDR) might be useful in reducing both FND symptoms and associated psychological distress (Cope, 2020).

Research implications

Many of the results in this review were based on FS-only participants, which may not be entirely applicable to other types of FND, and future research is needed to clarify this. Future studies should include psychiatric control groups to ascertain whether these findings are unique to FND or whether there are overlapping patterns, which would help to further elucidate the aetiology and mechanisms of FND. Specifically, it would be useful to explore clinical profiles between FND and dissociative/complex subtypes of PTSD based on trauma-exposure.

Future research would benefit from more multivariate designs that control for key psychological features such as depression and anxiety. Studies would also benefit from larger sample sizes and participants recruited from non-specialist settings to ascertain the generalisability of the relationships and clinical profiles described in this review. Furthermore, prospective cohort studies may offer greater certainty on the impact of trauma-exposure and clarify the direction of variables, such as whether trauma-related psychological distress is a precipitating factor in FND or whether it is a consequence of it.

It would be important for future research to incorporate an assessment of childhood emotional maltreatment in their trauma-exposure measures. Furthermore, given that no subgroup differences were identified on somatoform dissociation and alexithymia, but both had a significant relationship to childhood emotional maltreatment, further research is needed to clarify their relationship to both this and other trauma types.

Future research would benefit from investigating the effectiveness of evidence-based, trauma-focused treatments such as CBT for trauma and EMDR in alleviating both FND symptoms and associated psychological distress.

Limitations of the review

There are several limitations of this review which need to be considered. Firstly, there is no single accepted definition of 'trauma-exposure' in the literature, or what qualifies as a traumatic event. Given the exploratory nature of this review, all studies that included a measure of 'trauma-exposure' were included; however, studies differed in their definition and measurement of trauma-exposure, which made it difficult to compare results across studies. Furthermore, the prevalence, nature, and extent of trauma-exposure is a matter of ongoing debate, and some authors speculate that individuals may be unwilling or unable to recall traumatic experience or that the measures currently used are not adequate (Reuber, 2018). Finally, small sample sizes, uncontrolled variables (such as psychological distress), and crosssectional design only allowed for tentative conclusions to be made.

Conclusion

This review contributed to the existing literature by synthesising available research on the psychological correlates of trauma-exposure, and by examining psychological differences between individuals based on trauma-exposure. Notwithstanding methodological limitations, the review found that exposure to traumatic events, especially in childhood, was associated with greater psychological distress, such as depression, anxiety, PTSD, dissociation, and emotional dysregulation. Significant differences were found for most factors between subgroups based on trauma, except for somatoform dissociation and alexithymia, which may reflect a similar underlying mechanism for all individuals with FND. This review highlighted the relevance of trauma-exposure in this population, which should be integrated within treatment and inform future research.

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SECTION B: EMPIRICAL PAPER

EMDR FOR FUNCTIONAL NEUROLOGICAL DISORDER: A SINGLE-CASE EXPERIMENTAL DESIGN

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Keywords: EMDR, Functional Neurological Disorder, trauma, single-case experimental design
Abstract

Background: Psychological interventions are frequently the main treatment offer for individuals with FND. Their effectiveness, however, is often limited, which may be due to clinical and psychological differences in this population. The examination of clinical subgroups based on psychological factors, such as exposure to trauma, has been encouraged to determine more targeted interventions. This study explored the benefit Eye Movement Desensitisation and Reprocessing (EMDR) in alleviating FND symptoms and psychological distress in individuals with FND and a history of trauma.

Methodology: A multiple baseline single-case experimental design was implemented to examine the effectiveness of EMDR in reducing FND symptomatology, functional impairment, and psychological distress in a community FND sample (N=6) with a history trauma. Participants completed a 2-, 4-, or 6- week baseline, received 8 sessions of EMDR, and completed a 2-, 4-, and 6-week follow-up phase.

Findings: Four participants reported significant treatment gains on all outcome measures after completing EMDR, which was mostly maintained at follow-up. Two participants, however, reported no significant improvement on any outcome measure, and both participants reported more adverse events during the study than others.

Conclusion: Findings suggest that EMDR may be a promising treatment option for individuals with FND who also have a history of trauma. However, EMDR may not be helpful, or requires protocol modification, for individuals with ongoing social stressors, dissociation, and long-standing emotional dysregulation. Further replication of the study effect is needed to clarify findings. Future studies would benefit from inclusion of broader FND subtypes and participants from non-specialist settings.

Introduction

Definition of Functional Neurological Disorder

Functional neurological disorder (FND) is a complex and heterogenous condition that is not compatible with known organic pathology but attributed to a complex interplay of biopsychosocial risk factors (Kola & LaFaver, 2022). Symptoms of FND resemble neurological abnormalities, such as tremors, seizures, and sensory anomalies. The most common types of FND include functional seizures, functional motor symptoms, and functional sensory symptoms.

FND terminology had gone through many iterations since it was first coined as *hysteria* in mainstream neurological and psychological research in the late 19th century (Raynor & Baslet, 2021). Since then, FND had been termed conversion disorder, dissociative neurological disorder, and psychogenic neurological disorder, which reflects the changes in theoretical assumptions (Freedman, 2022). Given both the historical and current ethical issues associated with this diagnostic label, such as the pathologizing of female experiences, and dismissal of symptoms by healthcare providers, this paper uses the prefix 'functional' to describe symptoms, which has been more widely accepted by the patient community (Ding & Kanaan, 2017).

Conceptualisation of FND

Interest in FND was most prominent in the late 19th and early 20th century within the neurological and later psychoanalytic community. After the First World War, however, interest in this complex and multi-faceted presentation waned and lay mostly dormant until the turn of the last century. Early theories of conversion and dissociation –which emphasised the aetiological role of trauma– dominated the conceptualisation of FND for much of the 20th century, such that preceding stressors or trauma were a prerequisite for the diagnosis of FND in

major classificatory systems such as the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) and International Classification of Diseases (ICD-10). Pierre Janet (Janet, 1889, 1907) who pioneered the early dissociation theory, posited that traumatic experiences could overwhelm the person's mental functioning and integrative capacities, and lead to the compartmentalisation of mental, emotional, and physical components of traumatic material. In this way, FND was understood as dissociated somatic fragments of trauma. Sigmund Freud's conversion theory (Breuer & Freud, 1893-1895/1982), however, suggested that FND was a result of repressed stressful or traumatic material that was converted into physical symptoms. Like dissociation, conversion was believed to be a psychological defence mechanism, which alleviated emotional distress by converting it into physical symptoms.

More recently, the clinical and psychosocial heterogeneity across FND presentations suggested that FND may be a result of a complex interplay of biopsychosocial factors, including biological vulnerability, exposure to traumatic events, alternations in cognitive and affective functioning, and disruptions within structural and functional neurobiological processes. The Integrative Cognitive Model (ICM) (Brown, 2004; Brown & Reuber, 2016b) which built on both dissociation and conversion theories, proposed that the overactivation of specific, automatic illness beliefs termed 'rogue representations' can initiate and maintain FND through alterations in attention, perception, and threat processing. Other accounts highlighted difficulties with emotional processing and dysregulation, such as higher rates of alexithymia (difficulty with identifying and differentiating between feelings, and externally oriented thinking) (Sojka et al., 2018), poor interception sensitivity (difficulty connecting physical sensations to emotions) (Williams et al., 2021), and the social modulation of FND symptoms, where FND can offer an 'escape' from adverse social contexts (Aybek et al., 2014). Finally, neuroimaging research found

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an abnormal limbic-motor activation in individuals with FND in response to negative emotional stimuli (Voon et al., 2011). Some authors posit that this may be due to a conditioned threat response following a triggering event, such as physical injury (Nijenhuis et al., 1998). While there is no consensus about the aetiology or mechanism of FND, some authors argue this may reflect the multitude of possible pathways to FND (Kanaan et al., 2017).

Treatment of FND

It has been widely reported that clinical outcomes for individuals with FND are poor, especially without targeted interventions (Carson, 2003). Interest in the understanding, management, and treatment of FND in the last two decades has spurred advancements in clinical research across psychology, psychiatry, and neurobiology. More recently, a multidisciplinary approach in treating FND was highlighted, including input from neurology, psychology, physiotherapy, and other allied health professions (Demartini et al., 2014). Nevertheless, psychological therapy is still considered the core intervention for FND. Psychological treatments that target both FND symptoms and/or co-occurring mental health difficulties were linked to favourable outcomes in FND symptoms, mental health, and quality of life (Espay et al., 2019; Gutkin et al., 2021a). Nevertheless, a recent meta-analysis and systematic review on the efficacy of psychological therapies for functional seizures found the quality of studies to be mixed, and therapeutic effects to be limited, with the most common therapies offered being cognitivebehavioural therapy, paradoxical intention therapy, psychodynamic psychotherapy, and hypnosis (Carlson & Perry, 2017; Ganslev et al., 2020) One reason for this modest success may be related to the heterogeneity of symptoms and psychosocial characteristics, which may require a greater understanding of specific clinical subgroups (Gutkin et al., 2021b).

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Trauma and FND

FND has a long history of being associated as a trauma-related disorder, in part due to the early psychogenic models described, and in part due to the high rates of trauma reported by individuals. More recently however, research indicated abnormalities in brain activity and connectivity (Bègue et al., 2019), emotion processing (Pick et al., 2019), and higher order executive dysfunction (Hamouda et al., 2021). Symptoms can now be objectively observed, and a positive diagnosis made, without the prerequisite of trauma (Espay et al., 2018). This was an important scientific and clinical advancement that helped to somewhat de-stigmatise FND for both patients and clinicians, as many individuals with FND do not report a history of trauma. Nevertheless, exposure to trauma is still considered an important risk factor in the development of FND. A recent meta-analysis found that individuals with FND reported trauma and adverse life events eight times more than healthy controls, and twice as much as neurological and psychiatric controls (Ludwig et al., 2018). Therefore, setting aside the argument of causation, the presence of a trauma history is likely to be an important indicator of the treatment needs for a large proportion of individuals with FND (Reuber, 2018).

Eye Movement Desensitisation and Reprocessing (EMDR) and FND

EMDR therapy is an evidence-based gold standard treatment for posttraumatic stress disorder (PTSD) with promising effects in sensory and somatic difficulties such as chronic pain (Grant & Threlfo, 2002) and somatic symptom disorder (Demirci et al., 2017). EMDR is guided by the Adaptive Information Processing (AIP) model (Shapiro & Laliotis, 2011) which posits that many mental health difficulties are the result of unprocessed traumatic memories that remain isolated from the adaptive and contextual autobiographical memory that is necessary to process and assimilate such experiences. Furthermore, unprocessed memories are sometimes referred to as being stored in a state-specific form, whereby cognitive, emotional, and somatosensory components of the original event continue to be re-experienced (van der Hart et al., 2010) until successful reprocessing and resolution of the memory occurs.

EMDR is an integrative psychological therapy that aims to facilitate effective processing of traumatic memories, alleviate psychological distress, and promote adaptive cognitions related to the traumatic event (Shapiro, 2014). While EMDR draws on several psychological therapies such as CBT and psychodynamic psychotherapy, its use of alternating bilateral stimulation (BLS) (e.g., eye movements), is unique. While the mechanism of BLS is unknown, it has been posited that the dual-attention necessary for engaging with both trauma memories and BLS promotes emotional processing and adaptive assimilation of traumatic material into the existing memory network (Amano & Toichi, 2016).

Over the last two decades, a small but growing number of case studies have explored the use of EMDR in the treatment of FND symptoms in individuals with a history of trauma (Cope, 2020; Demirci & Sagaltici, 2021; Kelley & Benbadis, 2007). In their recent review of this evidence base, Cope et al. (2018) found that EMDR showed promising effects in reducing the symptomology of FND, however, the review only consisted of three studies, and a total of five patients, four of whom had functional seizures. The review reported that the quality of studies was relatively poor due to the use of unsystematic case studies, inconsistencies in delivering EMDR, lack of standardised outcome measures, and no assessment of functional impairment.

Aims and rationale

FND is a heterogenous presentation which may be explained by different causal pathways and mechanisms, all of which are likely to affect the success of treatment. The examination of clinical subgroups in FND has been widely encouraged in recent years (Bodde et al., 2013; Gray et al., 2020). As such, the current study aimed to investigate the effectiveness of EDMR in reducing FND symptoms, functional impairment, and psychological distress in individuals with FND who also report a history of trauma.

The current study sought to address some of the limitations outlined by Cope et al. (2018) and to contribute to the small, but growing, evidence base. Firstly, this study included a standardised measure of function impairment to assess the interference of FND symptoms in daily life. Secondly, the study implemented a single-case experimental design (SCED), which is more methodologically robust than case studies. Finally, the study included participants with different FND symptoms to increase generalisability and ecological validity. This study aimed to address the following six hypotheses:

- 1: FND Frequency, severity, and distress will be reduced following EMDR.
- 2: Functional impairment will be reduced following EMDR.
- 3: Trauma-related distress will be reduced following EMDR.
- 4: Anxiety symptoms will be reduced following EMDR.
- 5: Depression symptoms will be reduced following EMDR.
- 6: Global psychological distress will be reduced following EMDR.

Method

Design

A concurrent multiple baseline single-case experimental design (SCED) was implemented. This study had three distinct phases: baseline (A) treatment (B) and follow-up (C). The baseline phase served as a benchmark for assessing the intervention effect. Participants were consecutively assigned to a 2-, 4-, or 6-week baseline phase. In SCED research, repeated measures, or replication, of the dependent variable across time and participants allows for inferences about intervention effect to be made. Staggering the baseline across participants and introducing treatment at different times can further control for extraneous variables such as history, maturation, and regression to the mean (Engel & Schutt, 2016). Furthermore, participants in lower tiers (longer baseline) act as a control for participants in the tiers above. If therapeutic changes occur for participants in the intervention phase, but not for those in the baseline phase, conclusions about intervention effects are more robust (Hawkins et al., 2007).

Following the 2-, 4-, or 6- week baseline phase, participants received eight, 90-minute, weekly EMDR sessions using the standard eight-phase protocol, and completed follow-up measures at 2-, 4-, and 6- weeks post treatment.

Recent guidelines on SCED methodology stipulate that there should be at least three attempts to demonstrate an intervention effect at three different time points (What Works Clearinghouse, 2020). Therefore, this study aimed to recruit at least six participants, and sought permission to recruit nine to account for possible attrition.

Participants

Participant recruitment occurred between August and September 2020. All participants were recruited from a tertiary neuropsychiatry service in England, where they were awaiting EMDR assessment.

Individuals were recruited to the study if they 1) were 18-65 years old; 2) had a diagnosis of FND; 3) had a history of trauma-exposure; 4) had capacity to provide informed written consent; 5) had sufficient knowledge of English. Individuals were excluded if 1) they had a diagnosis of learning disability; 2) they had significant current alcohol/substance misuse that would negatively impact their ability to engage in EMDR; 3) they lacked capacity; 4) they had a high-risk presentation that required input beyond what could be offered by the study therapist. Participant demographic and clinical characteristics are presented in Table 1.

Table 1

Participant characteristics

	Age, gender, ethnicity	Comorbidi	ties Trauma characteristics	Previous therapy	FND subtype	FND symptoms	Duration of symptoms	Events prior to FND onset
P1	27, Female, White British	 Anxiety Depress Self-har Comple PTSD 	 Frequent medical procedures in childhood. Emotional abuse in childhood. Sexual assault in adolescence. 	None	Functional seizures with absences	 Absences, multiple times a day: "glazes over" and eyes flicker Functional seizures 2-3 times a month 	8 years	Stressful life events, including university exams, illness, and death in the family
P2	45, Female, White British	 Depress Anxiety Epilepsy Migrain 	sion - Early childhood sexual abuse. y - Sexual assault in adolescence and adulthood.	d CBT for depression and anxiety	Mixed FND; motor and sensory symptoms; functional seizures	 Functional seizures (twice a year) Limb jerking, up to 30 times a day Pain in limbs Headaches Daily absences 	8 years	Memory of sexual abuse triggered in a work context.
Р3	49, Female, White British	 Low mo Anxiety PTSD sympton (intrusive experien nightmatic) 	bod - Childhood sexual and emotional ms abuse. ve nces, arres).	None	Functional motor symptoms	- Heaviness and involuntary movement in legs	1 year	Stressful life events including death of family member.
P4	34, Male, White British	 Social anxiety OCD 	- Childhood sexual, physica	- CBT l,	Functional seizures	- Functional seizures 4-7 times a week	9 years	N/A

		-	Depression Diabetes	-	and emotional abuse. Witnessed sudden and violent death of parent in adulthood.	-	FND group (dropped out)					
P5	56, Female, White British	- -	Autism Epilepsy Agoraphobia	-	Childhood sexual abuse. Recent loss of parent.	- -	EMDR for agoraphobia CAT CBT for anxiety	Functional seizures	-	Functional seizures 1-3 times a day. Functional absences 2-3 a week: unresponsive.	7 years	N/A
P6	23, Female, White British and Caribbean	-	Anxiety Depression Self-harm Cerebral palsy	-	Early childhood sexual abuse, emotional abuse, and neglect. Exposure to violent criminal activity in childhood.	-	CAT Supportive counselling	Functional seizures	-	Functional seizures, including eye rolling, limb shaking, partial awareness of surroundings during seizure.	1.5 years	Stressful life events: university exams and contact with her perpetrator

EDMR therapy

An eight-stage standard EMDR protocol was administered in this study (Shapiro, 2018; Shapiro & Laliotis, 2011). This protocol involves eight distinct phases of therapy, with a specific aim in each phase. Brief information on each phase and its aim is presented in Table 2. In this study, participants received eight, 90-minute sessions of EMDR, and one follow-up session six weeks later to evaluate treatment effects and plan for discharge.

Table 2

EMDR standard protocol

Dhaga	A :
rnase	Aim
1) History-taking	Identify presenting difficulties, specific events, and memories ("targets") for reprocessing and determine treatment goals.
2) EMDR Preparation	Psychoeducation on the impact of trauma and the rationale, aims and techniques of EMDR therapy.
	Stabilisation: development of 'safe place', mindfulness, grounding techniques.
3) Assessment	In-depth assessment of target memories that were identified in the first phase. This includes eliciting images, emotions, and sensations of the target memory, and associated negative cognitions and subjective distress. This also involves identifying an alternative cognition that the individuals would like to have about the target memory.
4) Desensitisation	Reprocess identified targets through sets of bilateral stimulation until subjective distress related to the target is gone or significantly reduced.
5) Installation	Installation of adaptive and helpful cognitions (such as those identified in phase 3). Individual is asked to bring into awareness

	both the target memory and adaptive cognition while therapist administers bilateral stimulation and checks the validity of new cognition.
6) Body Scan	Identify and reprocess any residual bodily distress that may be associated with the target memory and strengthen adaptive material.
7) Closure	Focus on debrief and grounding at the end of the session to promote coping and stability between sessions.
8) Re-evaluation	Re-assess therapeutic effect from previous sessions, both in relation to global functioning and target memory.

Materials

Both standardised and idiographic 'target' measures were used in this study. The use of idiographic measures is traditional to SCED research, which are designed to assess changes in specific problems/behaviours, that are the 'target' of the intervention (Morley, 2018). Participants were asked to complete all outcome measures with reference to the previous 7 days only.

Primary outcome measures

FND symptoms

An idiographic measure was designed to assess and monitor changes in FND symptoms throughout the study (Appendix I). The measure consisted of three items which assessed the frequency, intensity, and distress of FND symptoms. Each item was measured on a 10-point scale, where higher scores indicated greater severity. This study used the following cut-off scores: 0-2 (low severity), 3-6 (moderate severity) and 7-10 (high severity).

Functional impairment

The Work and Social Adjustment Scale (WSAS; Marks, 1986) is a 5-item self-report measure that assesses functional impairment due to an identified problem in the following areas: work, home, social, and private life, and relationships. Each item is rated on a 0-8 scale, with higher scores indicating greater impairment, with a total possible score ranging from 0 to 40. Scores of 10-20 suggest significant impairment, while scores above 20 are considered indicative of moderate-to-severe psychopathology (Mundt et al., 2002). WSAS demonstrated good internal consistency, test-retest reliability, and post-treatment change-sensitivity (Cella et al., 2011; Mataix-Cols et al., 2005).

Secondary outcome measures

Trauma-related distress

The Impact of Events Scale-Revised (IES-R; Weiss & Marmar, 1997) is a 22-item selfreport measure of psychological distress following exposure to a traumatic across three domains: hyperarousal, avoidance, and intrusive/re-experiencing symptoms. Each item is rated on a 0-4 scale, with higher scores indicating greater distress. A total possible score ranges from 0 to 88, with scores of 33 considered to be indicative of PTSD. IES-R demonstrated have good internal consistency, sensitivity, and specificity (Beck et al., 2008; Rash et al., 2008).

Anxiety

The Generalised Anxiety Disorder (GAD-7; Spitzer et al., 2006) is widely used 7-item self-report screening measure of general anxiety symptoms such excessive worry, trouble relaxing, and irritability. Items are scored on a scale of 0-3, with total scores ranging from 0 to

21, where higher scores indicate greater anxiety. A score of ≥ 10 has been suggested as a clinical cut-off point. GAD-7 has been used in both primary and secondary care settings, and has shown good internal consistency, construct validity, reliability, and sensitivity to change (Beard & Björgvinsson, 2014; Byrd-Bredbenner et al., 2021).

Depression

The Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001) is a 9-item self-report screening measure for depression, that assesses for symptoms such a low mood, lack of motivation, and alterations in energy levels and appetite. PHQ-9 is scored on a 0-3 scale with total scores ranging from 0 to 27, where higher scores indicate more severe depression. A score of \geq 10 has been suggested as a clinical cut-off point. PHQ-9 has been validated across multiple clinical samples, and has demonstrated good internal consistency, reliability, and sensitivity to change (Kroenke et al., 2010; Rathore et al., 2014).

Global psychological distress

The Clinical Outcomes in Routine Evaluation Outcome Measure (CORE-OM; Evans et al., 2002) is a 34-item self-report measure that was designed to assess and monitor global psychological distress. CORE-OM measures distress across four domains: subjective well-being, specific problems, functioning, and risk. Items are scored on a 0-4 scale, with total scores ranging from 0 to 40, where higher scores indicate greater psychological distress. A score of ≥ 10 has been suggested as a clinical cut-off point. CORE-OM has demonstrated good internal consistency, reliability, and sensitivity to change (Barkham et al., 2015; Jenkins & Turner, 2014).

Procedure

Any individual that met the inclusion criteria was invited to participate in the study during an EMDR screening appointment by the study therapist. Participants were given a participant information sheet and encouraged to ask any questions. Written informed consent was obtained from all participants by the study therapist and checked regularly throughout the study (Appendix C).

After informed consent was obtained, participants were allocated to one of three baselines (2, 4, 6 weeks). Due to practical limitations, participants were not randomised to the baseline, but allocated to the baseline consecutively, whereby the first person recruited was allocated to the shortest baseline length and so on. SCED guidelines stipulate that a degree of concurrence in baselines is necessary to establish treatment effect (What Works Clearinghouse, 2020), therefore all participants started the baseline phase in September 2020.

Following the baseline phase, participants were seen face-to-face by the study therapist for eight, 90-minute, weekly EMDR sessions. After therapy completion, participants were followed-up at 2-, 4-, and 6- weeks. The last follow-up session was completed in person with the study therapist for a clinical review prior to discharge from the service. Throughout the study, participants were asked to complete weekly outcome measures via an online platform (Qualtrics), on the phone, or in person with study therapist.

Procedural changes

The start of the study was delayed by several months due to the COVID-19 pandemic. When restrictions were lifted, the study timeline coincided with the Christmas period and study therapist's annual leave, which resulted in 2-4 weeks interruption between therapy sessions for

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all participants except P3. No outcome data were available for P3 and P4 during this time, as both opted to complete measures in person. For clinical reasons, session frequency for P2 and P3 were changed to fortnightly, and P3 was only able to complete outcome measures on a fortnightly basis.

Quality assurance

To ensure treatment fidelity, EMDR was delivered by a Senior Clinical Psychologist with over 10 years of qualified clinical experience, whose EMDR training (parts 1-3) was accredited by the EMDR International Association. EMDR was delivered using a manualised, eight-stage protocol, and all participants received the same number of sessions. The study therapist received clinical supervision throughout the study. To ensure SCED quality, the lead researcher consulted relevant SCED standard guidelines (e.g., Tate et al., 2016; What Works Clearinghouse, 2020) and used supervision as needed.

Ethical considerations

The study protocol was reviewed and approved by the Health Research Authority (HRA), local NHS Research Ethics Committee (REC) and the Research and Development (R&D) department of the host NHS trust (Appendices F-H).

Data analysis

The data in this study was analysed using both visual and statistical methods. Visual analysis is the primary method of data analysis in SCED, and is considered a relatively conversative method, as it can only detect moderate-to-large changes that are easily seen

(Kazdin, 2011). As such, visual analysis is especially useful in detecting functional relationships between variables that are likely to be clinically meaningful (Ledford et al., 2018). Nevertheless, reliance on visual analysis alone can increase Type II error (McClain et al., 2014; Ninci et al., 2015), and statistical methods can provide additional objectivity and precision (Parker & Hagan-Burke, 2007).

Visual analysis

Data were visually analysed using the guidelines and recommendations set out by Lane and Gast (2014) and Ledford et al., (2018). First, data were visually graphed, and then assessed for changes in level, trend, stability, immediacy of effect and consistency of patterns within and between participants. Level was assessed by calculating central tendency (median), relative and absolute change scores between and within phases. Trend celeration was calculated using the split-middle method, and stability was calculated using the stability envelope, where stability was defined as \geq 80% of data falling within 25% of the median or trend line. Consistency of patterns was analysed by assessing level and trend across participants, and immediacy of effect was analysed by assessing level and trend change following the introduction of the intervention.

Statistical analysis

Tau-U is a nonparametric measure of non-overlapping data that was used to assess changes in FND symptoms across adjacent phases. Tau-U is especially useful in SCED research because it is relatively robust with autocorrelated data, does not rely on a normal distribution, and is able to detect medium-to-large effects in small samples (Brossart et al., 2018; Parker et al., 2011, 2014). Tau-U is also able to control for undesired baseline trend if needed (Tarlow, 2017).

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Values of ≤ 0.20 may be considered as a small effect size, 0.20-0.60 as a moderate effect size, and ≥ 0.80 as a large effect size (Vannest & Ninci, 2015).

Standardised outcome measures were analysed using the Reliable Change Index (RCI) and the Clinically Significant Change (CSC) criteria (Jacobson & Truax, 1991). The RCI determines whether the change is statistically reliable or due to random fluctuation that is expected within a specific measure (Guhn et al., 2014). In addition, the CSC criterion can be calculated to establish whether change is also clinically meaningful (Jacobson et al., 1986). In this study, change was deemed clinically meaningful if post-treatment scores were within ±1.96 standard deviations of the mean of the non-clinical population (Criterion B; Jacobson & Truax, 1991) The RCI and CSC were calculated for each outcome measure using psychometric data from published research and presented in Table 3.

Table 3

Outcome measure	RCI	CSC	
WSAS	4.95	17.02	
IES-R	13.91	34.45	
GAD-7	4.99	10.06	
PHQ-9	6.55	9.97	
CORE-OM	5.90	6.03	

RCI and CSC scores

Note. Psychometric data was obtained from the following studies: WSAS (Jamalamadaka et al., 2020; Tchanturia et al., 2013); IES-R (Beck et al., 2008; Suzuki et al., 2014); GAD-7 (Johnson et al., 2019; Ruiz et al., 2011), PHQ-9 (Beard et al., 2016); CORE-OM (Connell et al., 2007).

Results

Retention and adherence

Nine participants were recruited to this study, but only six are included in the results . Two participants dropped out after the first session due to finding therapy too emotionally demanding (n=1), and due to difficult family circumstances (n=1). One participant completed seven sessions of EMDR and dropped out due to a stressful live event. Results for this participant are not presented due to the large amounts of missing data. Non-completers did not differ from the completers on demographic or clinical characteristics.

Missing data

There was some missing data for participant (P) 2, P3, and P6 in the intervention phase. Furthermore, P3 did not provide data for 2- and 6-weeks follow-up but did provide follow-up data for 4, 7, 8 and 11 weeks. Guidelines on SCED research have not provided recommendations for handling missing data, however, a recent review suggested that imputation methods should be considered if there is minimal to moderate (<30%) missing data (Peng & Chen, 2021). The proportion of missing data for each participant (20-25%) met this criterion and was therefore imputed using the 'last observation carried forward' method, which substitutes missing data with the participant's previous score.

Notable and adverse events

Table 4 presents information about notable and adverse events that occurred during the study.

Table 4

Notable and adverse events during the study

Participant	Event	Time
P1	Family crisis/overdose /referral to secondary care mental health services	Session 2
	Overdose	Session 3
	Overdose	Session 5
	Short-term hospital stay	Session 6
	Terminal illness diagnosis in the family	6-week follow-up
P2	Work stress	Session 4
	Life event that triggered trauma memory	6-week follow-up
Р3	Increase in intrusive memories	Session 2
	Increase in functional symptoms and intrusive memories / referral to physiotherapy	Session 3
	Home life stress	Session 8
P4	Chronic illness diagnosis in the family	Session 6
Р5	Physical injury	Session 1
	Seizure during session	Session 2
	Seizure during session	Session 3
	Home life stress	Session 4
	Increase in seizures following trauma disclosure	Session 5
	Social care needs/home life stress	Session 7
P6	Death in the family	Session 2
	Relationship breakdown	Session 8

Hypothesis 1: FND frequency, severity, and distress will be reduced following EMDR

Frequency of FND symptoms

Results were graphed and presented in Figure 1. Visual and statistical analyses offered some support for this hypothesis.

Visual analysis

Data were stable at baseline and Tau- $U_{A vs A}$ found no significant trend. There was some variability in the intervention (P2, P4) but not at follow-up. Trend direction varied across participants in the baseline and follow-up. During the intervention, data were decelerating—improving for all except P1 and P5, who had zero-celerating—stable data. All participants reported high scores during the baseline. Summary of individual changes in level and trend across phases is presented in Table 5.

Table 5

FND frequency scores

Participant	Baseline			Int	Intervention			Follow-up		
	Median	Range	Last	Median	Range	Last	Median	Range	Last	
			score			score			score	
P1	81	7-9	9	$6.5 \rightarrow$	6-8	5	$7 \rightarrow$	7-8	7	
P2	8↓	7-9	7	2↓	1-10	1	1↑	1-2	2	
P3	10→	9-10	10	7.5↓	2-10	2	3.5↑	2-5	5	
P4	81	7-9	9	3↓	1-10	1	2↓	1-2	1	
Р5	10→	8-10	10	10→	10	10	8↓	7-8	7	
P6	<u>10</u> →	8-10	19	5.5↓	1-8	1	11	1-4	4	

Note: \downarrow = Decelerating trend, \uparrow =Accelerating trend, \rightarrow = Zero-celerating trend.

Assessment of the A>B contrast revealed an overall therapeutic change in level and trend for P2, P3, P4 and P6. P1, P2 and P6 had an immediate decrease in level (session 1) which continued to improve for P2 and P6, but not for P1, who had a contra-therapeutic change after session 1. There was a steep decrease in level for P4 after session 2, and for P3 after session 4. There was no change in level or trend for P5.

Assessment of the B>C contrast indicated an overall improvement in level for P2, P3, P4, P5 and P6, and a deterioration for P1. There was a contra-therapeutic trend change for P2, P3, and P6, and a change from a zero-celerating to a decelerating trend for P5.

Tau-U analyses

Individual and combined Tau-U_{A vs B} analyses are presented in Table 6. The A>B contrast indicated a significant reduction in FND frequency for P2 (-0.90, p<.05) and P6 (-0.96, p<.01). The B>C contrast found a significant improvement for P5 (-1.00, p<.05). Combined Tau-U_{A vs B} analyses found a significant moderate effect size across both contrasts (p<.01).

Table 6

Phase contrast	Participant	Tau-U A vs B	<i>p</i> -value	90% CI
A>B	P1	-0.63	0.19	-1.00, 0.16
	P2	-0.90	0.04*	-1.00, -0.18
	P3	-0.46	0.26	-1.00, 0.21
	P4	-0.53	0.15	-1.00, 0.07
	P5	0.33	0.30	-0.20, 0.86
	P6	-0.96	0.003*	-1.00, 0.43
Weighted average		-0.50	0.002*	-0.76, 0.24
B> C	P1	0.42	0.31	-0.26, 1.00
	P2	-0.45	0.212	-1.00, 0.15
	P3	-0.63	0.09	-1.00, -0.02
	P4	-0.63	0.13	-1.00, 0.05
	P5	-1.00	0.014*	-1.00, -0.33
XX 7 • 1 4 1	P6	-0.75	0.062	-1.00, 0.08
weighted average		-0.51	0.001*	-0.77, 0.24

Tau-U_{A vs B} analyses of FND frequency

Note. * *p*≤0.05, ** *p*≤0.001

Figure 1



FND frequency scores across baseline (A), treatment (B), and follow-up (C)

Note. B= baseline, T= treatment, F= follow-up, NT= no treatment. Shaded regions indicate the range of baseline scores

Intensity of FND symptoms

Results were graphed and presented in Figure 2. Visual and statistical analyses offered some support for this hypothesis.

Visual analysis

Data were stable at baseline and Tau-UA vs A found no significant trend. There was some variability in the intervention (P2, P4, P6) but not at follow-up. Trend direction varied across participants in the baseline and follow-up. During the intervention, data were deceleratingimproving for all except P1 and P5, who had zero-celerating-stable data. All participants reported high scores during the baseline. Summary of individual changes in level and trend across phases is presented in Table 7.

Table 7

FND intensity scores

Participant	Ba	Baseline	
	Median	Range	Last

Participan	t Ba	seline		Intervention				Follow-up			
	Median	Range	Last score	Median	Range	Last score	Median	Range	Last score		
P1	7.5↓	7-8	7	5.5→	4-8	5	81	7-8	8		
P2	$7 \rightarrow$	7	7	2↓	1-8	1	1↑	1-2	2		
P3	9↑	9-10	10	7↓	2-10	2	2↑	2-3	3		
P4	7↑	6-9	8	2↓	1-10	1	2↓	1-2	1		
P5	10→	10	10	$10 \rightarrow$	10	10	$7 \rightarrow$	7	7		
P6	8.5↓	7-10	7	5↓	2-8	2	1→	1	1		
Note: - Do	a lorating tr	and T-A	agalarat	ing trand	$\lambda - Zara$	alarati	na trand				

Note: \downarrow = Decelerating trend, \uparrow =Accelerating trend, \rightarrow = Zero-celerating trend.

Assessment of the A>B contrast revealed a therapeutic change in level and/or trend for all participants except P5, who had no change. After the introduction of treatment, there was an immediate improvement in level and trend for P2 and P6 that continued until the end of the phase. P1 also had an immediate improvement in level but a slight accelerating-deteriorating trend thereafter. After session 2, P3 had a therapeutic change in level which continued to improve, whereas P4 had a steep decrease in level after which data mostly stabilised.

Assessment of the B>C contrast revealed a therapeutic change in level for P2, P3, P5, and P6. There was a contra-therapeutic accelerating trend change for P1, P2 and P3. For others, the trend had either stabilised (P6) or maintained direction at follow-up (P4, P5).

Tau-U analyses

Individual and combined Tau-U_{A vs B} analyses are presented in Table 8. The A>B contrasts found a significant improvement for P2 (-0.90, p<.05) and P6 (-0.87, p=.01). The B>C contrasts revealed a significant deterioration for P1 (0.79, p=.05), and an improvement for P5 (1.00, p=.01) and P6 (-1.00, p=.01). Combined Tau-U_{A vs B} analyses found a significant moderate effect size across both contrasts (p<.01).

Table 8

Phase contrast	Participant	Tau-U _{A vs B}	<i>p</i> -value	90% CI
A>B	P1	-0.75	0.12	-1.00, 0.04
	P2	-0.90	0.04*	-1.00, -0.18
	P3	-0.71	0.08	-1.00, -0.04
	P4	-0.53	0.15	-1.00, 0.07
	P5	0	1.00	-0.53, 0.53
	P6	-0.87	0.01*	-1.00, 0.34
Weighted average		-0.60	<0.001**	-0.92, -0.29
B>C	P1	0.79	0.05*	0.12, 1.00
	P2	-0.52	0.16	-1.00, 0.08
	P3	-0.69	0.06	-1.00, -0.08
	P4	-0.25	0.54	-9.20, 0.42
	P5	-1.00	0.01*	-1.00, -0.33
	P6	-1.00	0.01*	-1.00, -0.33
Weighted average	0.001	-0.45	0.005*	-0.77, 0.13
<i>Note.</i> * $p \le 0.05$, ** $p \le$	0.001			

 $Tau-U_{A vs B}$ analyses of FND intensity

Figure 2



FND intensity scores across baseline (A), treatment (B), and follow-up (C)

Note. B= baseline, T= treatment, F= follow-up, NT= no treatment. Shaded regions indicate the range of baseline scores.

Distress of FND Symptoms

Results were graphed and presented in Figure 3. Visual and statistical analyses offered some support for this hypothesis.

Visual analysis

Data were stable at baseline and Tau- $U_{A vs A}$ found no significant trend. There was some variability in the intervention (P1, P2, P4) and follow-up (P3). Trend direction varied across participants in the baseline and follow-up. During the intervention, data were decelerating—improving for all except P1 and P5, who had zero-celerating–stable data. All participants reported moderate-to-high scores at baseline. Summary of individual changes in level and trend across phases is presented in Table 9.

Table 9

Participant	Baseline			Int	Intervention			Follow-up		
	Median	Range	Last score	Median	Range	Last score	Median	Range	Last score	
P1	6↑	5-6	7	$5 \rightarrow$	2-10	5	6 ↑	5-8	8	
P2	6↓	5-7	5	1↓	1-8	1	1↑	1-2	2	
P3	9↓	9-10	9	7.5↓	1-10	2	2↑	1-3	3	
P4	6.5↑	5-8	8	1↓	0-9	1	0.5↓	0-1	0	
P5	$10 \rightarrow$	10	10	$10 \rightarrow$	10	10	4↓	3-4	3	
P6	2↑	1-4	3	1.5↓	1-3	1	$1 \rightarrow$	1	1	

FND distress scores

Note: \downarrow = Decelerating trend, \uparrow =Accelerating trend, \rightarrow = Zero-celerating trend.

Assessment of the A>B contrast revealed an overall therapeutic change in level and trend for P2, P3, P4, and P6. P1 and P6 had a small initial improvement, and data continued to improve for P6, but not P1, who had an accelerating–deteriorating change after session 2. P2 had variable but decelerating data in the first half of the intervention which stabilised around session 4 and continued to improve thereafter. Data were stable in level and trend for P3 until session 4, after which there was steep decrease in level followed by a gradual improvement. For P4, there was a steep decrease in level after session 2, after which data stabilised. No change was found for P5.

Assessment of the B>C contrast, revealed an improvement in level for P3, P4 and P5; a slight deterioration for P1 and no change for P2 and P6. There was a contra-therapeutic accelerating change for P1, P2 and P3; a therapeutic-decelerating change for P5, and a zero-celerating trend for P6.

Tau-U analyses

Individual and combined Tau-U_{A vs B} analyses are presented in Table 10. The A>B contrasts revealed a significant improvement for P6 only (-0.65, p=.05). The B>C contrasts found a significant improvement for P3 (-0.75, p<.05) and P5 (-1.00, p=.01). Combined Tau-U_A vs B analyses found a significant moderate effect size across both contrasts (p<.01).

Table 10

	Participant	Tau-U A vs B	<i>p</i> -value	90% CI
A>B				
	P1	-0.31	0.51	-1.00, 0.48
	P2	-0.79	0.07	-1.00, 0.07
	P3	-0.29	0.48	-0.96, 0.38
	P4	-0.63	0.09	-1.00, 0.02
	P5	0.00	1.00	-0.53, 0.53
Weighted average	P6	-0.65	0.05*	-1.00, 0.12
		-0.43	0.006*	-0.75. 0.12
B> C	P1	0.38	0.36	-0.30, 1.00
	P2	-0.23	0.52	-0.83, 0.37
	P3	-0.75	0.04*	-1.00, 0.15
	P4	-0.54	0.18	-1.00, 0.13
	P5	-1.00	0.01*	-1.00, 0.33
	P6	-0.50	0.22	-1.00, 0.17
Weighted average		-0.44	0.006*	-0.76, 0.13

 $Tau-U_{A vs B}$ analyses of FND distress

Note. * *p*≤0.05, ** *p*≤0.001

Figure 3



FND distress scores across baseline (A), treatment (B), and follow-up (C)

Note. B= baseline, T= treatment, F= follow-up, NT= no treatment. Shaded regions indicate the range of baseline scores.

Hypothesis 2: Functional impairment will be reduced following EMDR

Results were graphed and presented in Figure 4. Visual and statistical analyses offered some support for this hypothesis.

Visual analysis

RCI calculations found a significant deterioration in symptoms during the baseline for P1, P2, P3 and P4, however, data were stable. There was some variability in data during the intervention (P2, P3, P4) and follow-up (P3). Trend direction varied across participants in the baseline and follow-up. During the intervention, however, data were decelerating–improving for all except P1 and P5. All participants reported severe functional impairment at the end of baseline (scores >20). Summary of individual changes in level and trend across phases is presented in Table 11.

Assessment of the A>B contrast revealed an improvement in level for all participants, except P5 who had no change in level or trend. There was a therapeutic decelerating trend change for P2, P3, and P4, and no change for P1 and P6. There was improvement for P2 after session 1 that continued until scores stabilised at session 5. For P3, improvement was gradual until session 4 where there was a steep decrease in level and little variability thereafter. P4 had an abrupt decrease in level after session 2 that continued to improve. For P6, the improvement was gradual throughout the intervention. P1 had an immediate, but small, improvement in level, but data were accelerating–deteriorating after session 1.

Assessment of the B>C contrast revealed an overall improvement in level for P2, P3, P5, and P6; a deterioration for P4; and no change for P1. P2 and P3 had a slight contra-therapeutic

accelerating trend change; P5 had a therapeutic decelerating trend change; and P1, P4 and P6 had no trend changes.

Reliable and clinical change

RCI results are summarised in Table 12. The A>B contrast revealed that all participants, except P5, had a reliable reduction in functional impairment scores, which was also clinically significant for P2, P3, P4, and P6. Nevertheless, P4 and P6 still reported significant functional impairment at the end of the intervention (scores >10). The B>C contrast showed a significant deterioration for P3 and P4, a reliable improvement for P5, and a clinically significant improvement for P6. No changes were found for P1 and P2.

Table 11

	P1	P2	P3	P4	P5	P6
Last Baseline						
WSAS	27↑	22↑	33↑	38↑	38→	33↓
IES-R	50↓	55↓	63→	54→	66↑	66↓
GAD-7	15↓	12↓	19↑	$18 \rightarrow$	18→	20↓
PHQ-9	13↑	14↓	20↑	23↑	25↑	20↑
CORE-OM	16↓	15↓	20↑	29↑	30 →	33↑
End of Treatment						
WSAS	22↑	0↓	2↓	14↓	40→	13↓
IES-R	53↓	4↓	12↓	10↓	74↑	17↓
GAD-7	12↓	1↓	1↓	5↓	18→	5↓
PHQ-9	16↑	2↓	2↓	6↓	25↓	3↓
CORE-OM	27↑	1↓	4↓	12↓	31→	9↓
Last Follow-up						
WSAS	22↑	2↑	9↑	22↓	20↓	6↓
IES-R	67↑	11↑	12↓	30↓	8↓	11↑
GAD-7	17↑	0↓	$2 \rightarrow$	14→	6↓	5↑
PHQ-9	18↑	$2 \rightarrow$	$4 \rightarrow$	14→	$11 \rightarrow$	6↑
CORE-OM	24↑	3↑	9→	20↓	12↓	10↑

Scores on standardised outcome measures

Note. \downarrow = Decelerating trend, \uparrow =Accelerating trend, \rightarrow = Zero-celerating trend.
Table 12

Phase contrast	WSAS (95% $CI = RCI \pm 4.95$)	IES-R (95% CI = RCI ± 13.91)	GAD-7 (95% CI = RCI \pm 4.99)	PHQ-9 (95% CI = RCI ± 6.55)	CORE-OM (95% $CI = RCI \pm 5.90$)
P1					
А	-7 [-11.95, -2.05]^	13 [-0.91, 26.91]	2 [2.99, 6.99]	-2 [-8.55, 4.55]	9 [3.10, 14.90]*
A>B	5 [0.05, 9.95]*	-3 [-16.91, 10.91]	3 [1.99, 7.99]	-3 [-9.55, 3.55]	-11 [-16.90, -5.10]^
B>C	2 [-2.95, 6.95]	-14 [-27.91, -0.09]^	-5 [-9.99, -0.01]^	-2 [-8.55, 4.55]	3 [-2.90, 8.90]
P2					
А	-8 [-12.95, -3.05]^	23 [9.09, 36.91]	4 [-0.99, 8.99]	4 [-2.55, 10.55]	4 [-1.90, 9.90]
A>B	22 [17.05, 26.95]**	51 [37.09, 64.91]**	11 [6.01, 15.99]**	12 [5.45, 18.55]**	14 [8.10, 19.90]**
B>C	-2 [-6.70, 2.95]	-7[-20.91,6.91]	1 [-3.99, 5.99]	0	-2 [-7.90, 3.90]
P3					
А	-11 [-15.95, 6.05]^	0	-7 [-11.99, 2.01]^	-8 [-14.55, -1.45]^	-3 [-8.90, 2.90]
A>B	31 [26.05, 35.95]**	51 [37.09, 64.91]**	18 [-22.99, -13.01]**	18 [11.45, 24.55]**	16 [10.10, 21.90]**
B>C	-7 [-9.00, -2.05]^	0	-1 [-5.99, 3.99]	-2 [-8.55, 4.55]	-5 [-10.90, 0.90]

Change scores with 95% Confidence Intervals (CI) for standardised measures

P4					
А	-6 [-10.95, -1.05]^	-6 [-19.91, 7.91]	1 [-3.99, 5.99]	-8 [-14.55, -1.45]^	-4 [-9.90, 1.90]
A>B	24 [19.05, 28.95]**	44 [30.09, 57.91]**	13 [8.01, 17.99]**	17 [10.45, 23.55]**	17 [11.10, 22.90]*
B>C	-8 [-12.95, -3.05]^	-20 [-33.91, -6.09]^	-9 [-13.99, -4.01]^	-8 [14.55, 1.45]^	-8 [-13.00, 2.10]^
P5					
А	-3 [-7.95, 1.95]	-14 [-27.91, -0.09]^	-1 [-4.99, 3.99]	-5 [-11.55, 1.55]	-1 [-6.90, 4.90]
A>B	2 [-2.95, 6.95]	-8 [-21.91, 5.91]	0	0	-1 [-6.90, 4.90]
B>C	20 [15.05, 24.95]*	66 [52.09, 79.91)**	12 [7.01, 16.99]**	14 [26.20, 7.45]**	19 [13.10, 24.90]*
P6					
А	3 [-1.95, 7.95]	4 [-9.91,17.91]	1 [-3.99, 5.99]	3 [-3.55, 9.55]	-2 [-7.90, 3.90]
A>B	20 [15.05, 24.95]**	49 [35.09, 62.91]**	15 [10.01, 19.99]**	17 [10.45, 23.55]**	24 [18.10, 29.90]*
B>C	7 [2.05, 11.95]**	6 [-7.91, 19.91]	0	-3 [-9.55,3.55]	-1 [-6.90, 4.90]

Note. ^ Reliable deterioration. * Reliable improvement.** Clinically significant improvement.

Figure 4



WSAS scores across baseline (A), treatment (B), and follow-up (C)

Note. B= baseline, T= treatment, F= follow-up, NT= no treatment. Shaded regions indicate the range of baseline scores.

Hypothesis 3: Trauma-related distress will be reduced following EMDR

Results were graphed and presented in Figure 5. Visual and statistical analyses somewhat supported this hypothesis.

Visual analysis

Baseline data were stable, however, RCI calculations found a significant deterioration in trauma symptoms during the baseline for P5. There was some variability in data during the intervention (P2, P3, P4, P5) and follow-up (P2, P3). Trend direction varied across participants in the baseline and follow-up. During the intervention, however, data were decelerating– improving for all except P5 (accelerating–deteriorating). At the end of baseline, all participants scored above the clinical cut-off point (scores >33). Summary of individual changes in level and trend across phases is presented in Table 11.

Assessment of the A>B contrast revealed a therapeutic change in trend for P3 and P4, a therapeutic change in level for all participants, except P5, who reported an improvement in the first half of the intervention but deteriorated to baseline level in the second half. There was an immediate decrease in level for P2 that continued to improve throughout the intervention. P3 had a steep decrease in level after session 4 and data continued to gradually improve, while P4 and P6 had a steep decrease in level after session 3 which continued to improve for P6 but stabilised for P4.

Assessment of the B>C contrast indicated an improvement in level for P2, P3, P5, and P6, and a deterioration for P1 and P4. There was no change in trend for P1, P3, and P4; a contratherapeutic accelerating trend change for P2 and P6; and therapeutic decelerating trend change for P5.

Reliable and clinical change

Results on the IES-R are summarised in Table 12. There was a reliable increase in trauma-related symptoms for P5 during baseline phase. The A>B contrast found a clinically significant improvement for all participants except P1, who had a non-significant increase in symptoms. The B>C contrast found a clinically significant improvement only for P5. A reliable increase in symptoms was found for P1 and P4.

Figure 5



IES-R scores across baseline (A), treatment (B), and follow-up (C)

Note. B= baseline, T= treatment, F= follow-up, NT= no treatment. Shaded regions indicate the range of baseline scores.

Hypothesis 4: Anxiety symptoms will be reduced following EMDR

Results were graphed and presented in Figure 6. Visual and statistical analyses somewhat supported this hypothesis.

Visual analysis

There was some data fluctuation in the baseline (P1) and RCI calculations found a significant deterioration in anxiety symptoms during the baseline for P3. Variability was also found in the intervention (P1, P2, P4) and follow-up (P6). Trend direction varied across participants in the baseline and follow-up. At the end of baseline, all participants scored above the clinical cut-off point (scores>10). Summary of individual changes in level and trend across phases is presented in Table 11. During the intervention, however, data were decelerating–improving for all except P5 (zero-celerating–stable).

Assessment of the A>B contrast revealed an improvement in level for all participants except P5, who had no change in level or trend. There was a therapeutic decelerating trend change for P3 and P4, and no change for others. There was an immediate, but small improvement in level for P1, however data were very variable throughout the phase. P2 also had an immediate reduction in symptoms, which continued to improve throughout the intervention. A steep decrease in level was found for P3 after session 4, which continued to gradually improve, while P4 had a steep decrease in level after session 2 that remained mostly stable thereafter. There was a stepwise improvement for P6, where the level decreased after session 3 and again after session 7. Analysis of the B>C contrast revealed an improvement in level for P2, P3, P5 and P6. There was a change to an accelerating–deteriorating trend for P1 and P6, a zero-celerating–stable change P3 and P4, and a decelerating–improving trend change for P5.

Reliable and clinical change

Results are summarised in Table 12. The A>B contrast found a clinically significant improvement for P2, P3, P4 and P6. No significant changes were found for P1 and P5. The B>C contrast found a statistically, but not clinically, significant deterioration for P1 and P4, and no changes were found for P2, P3, P5, and P6.

Figure 6



GAD-7 scores across baseline (A), treatment (B), and follow-up (C)

Note. B= baseline, T= treatment, F= follow-up, NT= no treatment. Shaded regions indicate the range of baseline scores.

Hypothesis 5: Depression symptoms will be reduced following EMDR

Results were graphed and presented in Figure 7. Visual and statistical analyses somewhat supported this hypothesis.

Visual analysis

Baseline data were mostly accelerating–deteriorating (expect P2) for participants and RCI calculations found a significant increase in depression for P3 and P4. There was some variability in data during the intervention (P2, P4, P6) and follow-up (P2). At the end of the baseline, all participants scored above the clinical cut-off point (scores >10). Summary of individual changes in level and trend across phases is presented in Table 11. During the intervention, data were decelerating–improving for all except P1 (accelerating–deteriorating).

Assessment of the A>B contrast revealed an improvement in level for P2, P3, P4 and P6; a deterioration for P1; and no overall change for P5. Data for P3, P4, P5 and P6 changed from an accelerating to a decelerating trend; and no change was found for P1 and P2. There was an immediate improvement in level for P1 and P6, but data gradually deteriorated after session 1 for P1, and continued improve for P6. A steep decrease in level was visible for P3 after session 4, and for P4 after session 2, after which data continued to gradually improve for P3, but mostly stabilised for P4.

Assessment of the B>C contrast revealed a change to a zero-celerating trend for P2, P3, P4 and P5; an accelerating trend for P6; and no change for P1. There was improvement in level for P3, P5, and P6; a deterioration for P1 and P4; and no change for P2.

Reliable and clinical change

A summary of scores is presented in Table 12. The A>B contrast found a clinically significant improvement for all participants except for P1, who had no change. The B>C contrast found a clinically significant improvement for P5 only, and a significant deterioration for P4.

Figure 7



PHQ-9 scores across baseline (A), treatment (B), and follow-up (C)

Note. B= baseline, T= treatment, F= follow-up, NT= no treatment. Shaded regions indicate the range of baseline scores.

Hypothesis 6: Global psychological distress will be reduced following EMDR

Results were graphed and presented in Figure 8. Visual and statistical analyses somewhat supported this hypothesis.

Visual analysis

Baseline data were mostly accelerating–deteriorating (expect P2) and RCI calculations found a significant increase in depression for P3 and P4. There was some variability in data during the intervention (P2, P4, P6) but not at follow-up. At the end of the baseline, all participants scored above the clinical cut-off point (scores >10). Summary of individual changes in level and trend across phases is presented in Table 11. During the intervention, data were decelerating–improving for P2, P3, P4 and P6; accelerating–deteriorating for P1; and zerocelerating–stable for P5.

Analysis of the A>B phase revealed an overall therapeutic change in level and trend for P2, P3, P4, and P6; a contra-therapeutic change in trend but a small reduction in level for P1; and no change in level or trend for P5. P1, P2, and P6 had an immediate decrease in level that was maintained for P2 and P6, but not P1, where an accelerating–deteriorating change in data were visible from session 4 onwards. A gradual improvement in level was found for P3 after session 4, and a steep decrease in level for P4 after session 2, after which data remained mostly stable.

Analysis of the B>C contrast revealed an overall improvement in level for P2, P3, P5, and P6; and a deterioration for P1 and P4. There was a change to an accelerating trend for P2 and P6, a decelerating trend for P5, a zero-celerating trend for P3, and no change for P1 and P4.

Reliable and clinical change

A summary of scores across phases is presented in Table 12. The A>B contrast found a reliable improvement in general psychological distress for everyone except P1, however, changes were only clinically significant for P2 and P3. The B>C contrast found a significant deterioration for P4.

Figure 8





Note. B= baseline, T= treatment, F= follow-up, NT= no treatment. Shaded regions indicate the range of baseline scores.

Discussion

To the author's knowledge, this is the first study to implement a concurrent multiple baseline design to investigate the effectiveness of EMDR in FND. Primary outcomes were a reduction in FND symptom severity, frequency, distress, and functional impairment. Secondary outcomes were posttraumatic distress, anxiety, depression, and global psychological distress.

Summary of findings

In the baseline phase, all participants reported moderate-to-high scores on FND symptoms, and clinically significant scores on functional impairment, posttraumatic distress, anxiety, depression, and global psychological distress. Visual and combined Tau-U_{A vs B} analyses a found a moderate intervention effect size in reducing FND symptom frequency, severity, and distress for four participants. Clinically significant improvements were also found on all standardised outcome measures for these four participants, except for two that reported reliable but not clinically significant improvement on functional impairment and global psychological distress. For three of these four participants, treatment gains were mainly maintained or extended at follow-up, however, one participant (P4) reported a significant deterioration at follow-up on all standardised measures but not in their FND symptoms. Regarding immediacy of effect, visual analysis found that initial treatment gains occurred within the first half of the intervention which were consistent within and between participants, and across all measures.

Two participants, however, did not report similar changes. P1 reported a small improvement in functional impairment and FND symptoms during the intervention, however, FND symptoms returned to baseline level at follow-up. P5 reported an improvement on all

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measures at follow-up up, however, these could not be reliably attributed to the intervention, as the participant reported mostly ceiling scores during baseline and intervention phases.

Interpretation of findings

For the participants that reported an improvement across outcomes, the relative stability of data in the baseline, therapeutic change in level and/or trend, and consistent timing of effect suggests that these gains were unlikely to be due to random fluctuation, regression to the mean, maturation, or symptom monitoring. Furthermore, although it is unclear why P4 reported a significant deterioration on all standardised measures at follow-up, it is encouraging that P4 was able to maintain treatment gains related to FND symptoms, despite an increase in other difficulties. As such, these findings offer some support to the existing evidence-base on the use of EMDR in alleviating FND symptoms in those with trauma-exposure (Cope, 2020; Demirci & Sagaltici, 2021; Kelley & Benbadis, 2007).

The concurrent improvement on all outcome measures reflects the theoretical premise of EMDR, whereby the reprocessing of traumatic memories leads to a simultaneous improvement in cognitive, affective, and somatosensory functioning (Maxfield, 2003; Shapiro, 2014). These improvements also indicate a link between trauma and FND (Keynejad et al., 2019). Previous research found abnormal neural connectivity between the amygdala and motor activation centres in individuals with FND following exposure to negative emotional stimuli, which was posited to be a result of previous triggering events (Voon et al., 2011). As such, if the traumatic events that triggered this abnormal limbic-motor interaction were successfully resolved with EMDR, then it is possible that a reduction in FND symptoms was due to the amelioration in this connectivity. Future research, however, is needed to explore and clarify this further.

When results on FND symptoms and functional impairment were compared, it appeared that despite a reduction in FND symptoms, functional impairment remained a significant issue for two participants. Similar findings were reported in previous studies using other treatment approaches in FND such as CBT (e.g., Goldstein et al., 2020), which suggests that factors outside of symptom severity may be important in maintaining functional impairment in FND. A recent review of functional impairment in somatic disorders reported that the development of coping skills, increased self-efficacy, and symptom acceptance was associated with more positive outcomes (Pourová et al., 2020). As such, these treatment factors may be important to incorporate into FND treatment in the future.

Nevertheless, confidence in the treatment effect is limited due to the lack of improvement in two participants (P1, P5). There are several possible reasons for this limited effect. First, both participants had more adverse events and life stressors during the study than others. P1 reported several overdoses, a short-term hospital stay, and required a referral to secondary care mental health services. P5 experienced functional seizures and dissociation several times during sessions and reported an increase in intrusive traumatic memories, distress, and seizures between sessions. It is possible that these events are reflective of pre-treatment emotional regulation difficulties that were exacerbated in therapy, and limited the participants' ability to access, tolerate, and fully engage in the reprocessing of traumatic material during EMDR (González et al., 2017). While a degree of emotional distress is expected in trauma-focused therapy, individuals with longstanding difficulties, such as complex PTSD (P1) and dissociation (P5) may require additional resource installation and preparation (Korn & Leeds, 2002), and extended stabilisation work (de Jongh et al., 2016). Some authors suggest that individuals with early childhood trauma and emotional dysregulation may require multi-modal interventions and longer

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treatment length (Korn, 2009). Therefore, it may both participants would have benefited from additional support or a longer treatment phase.

The ability to tolerate trauma-related distress may also be relevant for the participants that dropped out of the study. A recent review found that interventions with a trauma-focus have a significantly higher dropout rate than those without, largely due to difficulties with distress tolerance and an initial increase in symptoms (Lewis et al., 2020). Although two participants in this study cited family/relational difficulties as reasons for dropout, one participant cited feeling too emotionally overwhelmed to engage, and may have therefore benefitted form an extended preparatory phase.

Confidence in the therapeutic effect of EMDR was also limited due to data variability in the intervention and follow-up phases, and discrepancy between the functional relationships established by visual analysis and largely non-significant individual Tau-U results. Notwithstanding, poor concordance between Tau-U and visual analysis have been previously reported (Brossart et al., 2018) and Tau-U has been criticised for failing to take into account replication logic and with-in case variability of SCED data (Fingerhut et al., 2021; Ledford & Gast, 2018) which may have influenced the significance of results in this study.

Strengths and Limitations

This study benefitted from the use of SCED, which is more methodologically robust than case studies, and can be used to investigate the effectiveness of an intervention while requiring fewer participants and resources than traditional group studies. A multiple baseline design in this study controlled for effects of maturation and regression to the mean (Engel & Schutt, 2016). Direct replication across participants allowed for causal inferences about EDMR effectiveness and insight into the variability of treatment effects within and between participants (Byiers et al., 2012; Janosky, 2005). This study also benefitted from the inclusion of a follow-up phase to determine if treatment effect was maintained over time.

Nevertheless, several limitations of this study must be considered. Firstly, the sample in this study may not be representative of the wider FND population for several reasons: all participants were recruited from a specialist service; all reported multiple traumatic experiences and co-occurring mental health issues; the majority were White British and female; and most participants had functional seizures. As such, these the results may not be generalisable to individuals with less severe histories or other FND types. The study therapist recruited and consented all participants, assisted with completing outcome measures, and administered the intervention; therefore, therapist-specific effects and the social desirability bias cannot be ruled out. Furthermore, the lack of baseline randomisation, blind assessors, treatment fidelity measures, and interruptions between therapy sessions precludes causal claims about EMDR in this study. Furthermore, given the dropout rate and lack of improvement for some participants, this study would have benefited from measures of treatment fidelity, acceptability, and satisfaction.

Clinical implications

Findings from this study have several important clinical implications. Firstly, they highlight the importance of assessing for a history of trauma in this client group, as it may be aetiologically linked to FND, and serve as an important treatment target. Secondly, results indicate that EMDR may be a promising treatment option for this client group, and that despite the severity or complexity of trauma or the duration of FND symptoms, improvements can be

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obtained within relatively few sessions. Nevertheless, findings also indicate that EMDR cannot be assumed to uniformly lead to better outcomes, and that some individuals, especially with emotional dysregulation and dissociation, may struggle to engage and make meaningful changes. While research on the use of EMDR in complex presentations suggests that it may be important to extend the preparation phase to upskill clients in self-regulation, there is not enough evidence from the current study to inform decision-making for these clients, except to highlight, that until further research, these clinical characteristics may be important contraindicators for EDMR effectiveness.

Research implications

To examine the generalisability and applicability of this study's results, further systematic replication of the intervention effect is needed across contexts, individuals, and therapists. Specifically, it would be important to include more males, FND subtypes other than functional seizures, and individuals from non-specialist services. Future research should address the methodological limitations of this study to include baseline randomisation, treatment fidelity and acceptability measures, as well as qualitative feedback from participants. To address nonresponders and dropouts in this study, it would be important to assess whether modification to the treatment protocol, to include greater distress tolerance and stabilisation skills, would yield more positive outcomes for individuals with concurrent dissociation and emotional regulation difficulties.

Conclusion

This study examined the effectiveness of EMDR in alleviating FND symptomatology, functional impairment, and psychological distress for participants with FND and a history of

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trauma. The findings presented a mixed picture regarding treatment effectiveness, with most participants showing significant treatment gains with relatively few sessions on all outcome measures, while some participants had little-to-no improvement. Despite methodological limitations and inconclusive findings, this study offers tentative evidence for the effectiveness of EMDR in this client group that warrants further empirical investigation.

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SECTION C: APPENDICES

A thesis submitted in partial fulfilment of the requirements of Canterbury Christ Church University for the degree of Doctor of Clinical Psychology

April 2022

Salomons Institute for Applied Psychology Canterbury Christ Church University

Appendix A

AXIS Quality Appraisal Tool

	Bodde et al., 2013	Boesten, Myers & Wijnen, 2019	Haringay et al., 2011	Haringay et al., 2017	Martino et al., 2021	Myers et al., 2013c	Myers et al., 2013b	Rosales et al., 2020	Sar, Islam & Öztürk, 2009	Spinhoven et al., 2004	Williams et al., 2019	Zeng, Myers & Lacman, 2018
Introduction												
1. Were the aims/objectives of the study clear?	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
Methods												
2. Was the study design appropriate for the stated aim(s)?	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
3. Was the sample size justified?	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
4. Was the target/reference population clearly defined? (Is it clear who the research was about?)	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES

5. Was the sample frame taken from an appropriate population base so that it closely represented the target/reference population under investigation?	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
6. Was the selection process likely to select subjects/participa nts that were representative of the target/reference population under investigation?	Partly	Partly	Partly	Partly	Partly	Partly	Partly	Partly	Partly	Partly	Partly	Partly
7. Were measures undertaken to address and categorise non- responders?	No missing data reported	No missing data reported	No missing data reported	No missing data reported	No missing data reported	NO (no informat ion on non- responde rs who left clinic soon after diagnosi s)	No missin g data reporte d	No missing data reported	No missing data reported	No missing data reported	No missing data reported	NO (no informati on on non- responder s who did left clinic soon after diagnosis)
8. Were the risk factor and outcome variables measured	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES

appropriate to the
aims of the
study?

9. Were the risk factor and outcome variables measured correctly using instruments/meas urements that had been trialled, piloted or published previously?	Trauma: YES Others: YES	Trauma: NO (Unvalid ated clinical intervie w) Others: YES	Trauma: YES Others: YES	Trauma: Somewhat (Validated tool used for childhood trauma but clinical interview for adulthood traumas)	Trauma: YES Others: YES	Trauma: NO (Unvalid ated clinical Intervie w) Others: YES	Traum a: NO (Unval idated Clinica l Intervi ew) Others: YES	Trauma: NO (Unvalid ated clinical intervie w) Others: YES	Trauma: YES Others: YES	Trauma: YES Others: YES (Not all samples were administere d the same measures)	Trauma: YES Others: YES	Trauma: NO (Unvalida ted clinical interview) Others: YES
				Others: YES						,		
10. Is it clear what was used to determine statistical significance and/or precision estimates? (e.g. p-values, confidence intervals)	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
11. Were the methods (including statistical methods) sufficiently described to	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	NO

enable them to be repeated?

Results												
12 Were the basic data adequately described?	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
13 Does the response rate raise concerns about non- response bias?	NO	NO	YES	NO	NO	YES	No missin g data reporte d	No missing data reported	No missing data reported	No missing data reported	No missing data reported	YES
14 If appropriate, was information about non- responders described?	No missing data reported	No missing data reported	YES	No missing data reported	NO	NO	No missin g data reporte d	No missing data reported	No missing data reported	No missing data reported	No missing data reported	NO
15 Were the results internally consistent?	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
16 Were the results presented for all the analyses described in the methods?	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
Discussion												

17 Were the authors' discussions and conclusions justified by the results?	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
18 Were the limitations of the study discussed?	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
Other												
Were there any funding sources or conflicts of interest that may affect the authors' interpretation of the results?	Not stated	Not stated	Not stated	NO	NO	NO	Not stated	NO	Not stated	Not stated	YES, only for the last author	NO
Was ethical approval or consent of participants attained?	Not stated	YES	YES	YES	YES	YES	Not stated	YES	YES	Not stated	YES	YES

Appendix B

Joanna Briggs Quality Assessment Tool

	Kienle et al., 2017	Lally et al., 2010	Myers et al., 2013a	Pick, Mellers, Goldstein, 2017	Steffen et al., 2015	Steffen-Klatt et al., 2019
1. Were the groups comparable other than the presence of disease in cases or the absence of disease in controls?	YES	YES	YES	YES	YES	YES
2. Were cases and controls matched appropriately?	Partly - healthy controls had higher level of education to FND and PTSD groups	YES	YES	Partly – FND cases had lower education and more comorbid medical diagnoses	Partly – FND cases had lower education than healthy controls	YES
3. Were the same criteria used for identification of cases and controls?	No - HC controls excluded if they had co-occurring mental health issues or used psychiatric medication	YES	YES	YES	NO - HC controls excluded if there was a history of mental health difficulties	NO - HC controls excluded if there was a history of mental health difficulties
4. Was exposure measured in a standard, valid and reliable way?	NO - unvalidated clinical interview	YES	NO - unvalidated clinical interview	YES	YES	YES
5. Was exposure measured in the same way for cases and controls?	YES	YES	YES	YES	YES	YES
6. Were confounding factors identified?	YES	NO	YES	YES	YES	NO
7. Were strategies to deal with confounding factors stated?	NO - did not control for level of education	N/A	YES	YES	NO - did not control for education or PTSD	N/A
8. Were outcomes assessed in a standard,	YES	YES	YES	YES	YES	YES

valid and reliable way for cases and controls?							
9. Was the exposure period of interest long enough to be meaningful?	YES	YES	YES	YES	YES	YES	
10. Was appropriate statistical analysis used?	YES	YES	YES	YES	YES	YES	

Appendix C

Consent Form



Salomons Institute for Applied Psychology Lucy Fildes Building One Meadow Road, Tunbridge Wells, Kent TN1 2YG www.canterbury.ac.uk/appliedpsychology

CONSENT FORM

Title of Project: EMDR for Functional Neurological Disorder and trauma: an investigation using a single-case experimental design

Participant Identification Number (PIN):

Please initial each box

1. I confirm that I have read and understand the information sheet dated 10/06/2020 (version 1.01) for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.	
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 understand that data collected during the study, will be looked at by lead researcher (Tanya Suhalitka) and clinical psychologist/project supervisor (Dr Nadine Bearman). I give permission for these individuals to have access to this data.	
4. I give permission for my contact details to be shared with the lead researcher (Tanya Suhalitka) for the purpose of completing questionnaires.	
5. I give permission for a short, anonymised summary of my clinical presentation to be included in the study report.	
6. I give permission for a copy of this consent form to be kept confidentially and securely for 5 years by the Canterbury Christ Church University and my anonymised data for 10 years.	
7. I would like to receive a summary of the results of this study	
8. I agree to take part in the above study.	
	. <u> </u>

Name of Participant	Date	_
Signature		
Name of Person taking consent	Date	_
Signature		

Appendix D

Participant information sheet



Salomons Institute for Applied Psychology Lucy Fildes Building One Meadow Road, Tunbridge Wells, Kent TN1 2YG www.canterbury.ac.uk/appliedpsychology

INFORMATION ABOUT THE RESEARCH

Title: EMDR for Functional Neurological Disorder and trauma: a single-case experimental design

Hello. My name is Tanya Suhalitka and I am a trainee clinical psychologist at Canterbury Christ Church University. I would like to invite you to take part in a research study. Before you decide whether to take part, it is important that you understand why the research is being done and what it would involve for you.

Talk to others about the study if you wish. (Part 1tells you the purpose of this study and what will happen to you if you take part. Part 2 gives you more detailed information about the conduct of the study).

What is the purpose of the study?

The aim of this study is to understand if a psychological therapy called Eye Movement Desensitization and Reprocessing (EMDR) is effective in helping people with Functional Neurological Disorder (FND). FND is a term used to describe various neurological conditions that don't have a medical explanation but can cause significant burden to those affected. Examples include problems with walking, 'mental fogginess', and non-epileptic seizures.

Although research about FND is growing, we still know little about it and how best to treat it. A history of traumatic/frightening experiences is common in a lot of people with FND, and it is thought that physical symptoms can occur if traumatic experiences have not been properly psychologically processed. If so, treatment that focuses on helping people to deal with their traumatic events, might help to reduce symptoms of FND and improve their quality of life.

EMDR is a brief and effective psychological therapy for people struggling with traumatic experiences. We think that this therapy might also be helpful in treating FND for those who have had a traumatic/frightening experience in the past. However, we need to conduct research studies to find out for sure.

Why have I been invited?

We are looking to recruit nine people to this study. We are looking for people with a diagnosis of FND and a history of traumatic/frightening experiences who are waiting to receive EMDR treatment. We are approaching everyone who meets these criteria in the service.

You have been invited to take part in this study because you have a diagnosis of FND and a history of traumatic/frightening experience/s. You are currently waiting to start EMDR therapy to help with these difficulties.

Do I have to take part?

It is up to you to decide whether to join the study. If you agree to take part, you will be asked to sign a consent form. 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?

If you decide to take part in the study, you will be given the exact same treatment as you would if you weren't taking part, with the exception of completing additional questionnaires. The purpose of the questionnaires is to track any changes in your physical symptoms, mood, and daily function. Data collected from the questionnaires will be used to investigate if EMDR is successful or not.

As you are currently on the waiting list to start therapy, you will normally have to wait between 2-6 weeks for treatment. During this time, you will be asked to complete weekly questionnaires by your clinical psychologist. These should take you approximately 15-20 minutes to complete. They can be completely over the phone with the researcher, online, or through post.

EMDR therapy normally runs for 8 sessions, with the clinical psychologist seeing you every two weeks for 90 minutes. During treatment, you will be asked to complete questionnaires. These are the same questionnaires that you had to do whilst waiting for therapy to start. Completing questionnaires might take an additional 15-20 minutes of your time.

After you finish treatment, you will be asked to complete the same questionnaires 2, 4 and 6 weeks afterwards. This is important in helping us to understand the effect of treatment after it has been completed.

In total you will be asked to complete between 24-28 questionnaires, depending on how long you wait for treatment to start.

We will also ask to use a short, anonymised summary of your clinical presentation (i.e. reason for referral) in the study report. This is to ensure that the research is transparent and that others can replicate it in the future.

What are the possible disadvantages and risks of taking part?

It is possible that you may find completing questionnaires repetitive and tedious. It's possible that completing these questionnaires might make you feel distressed at times.

What are the possible benefits of taking part?

We hope that EMDR treatment will be effective in helping you with your difficulties, and that by completing questionnaires you will be able to see positive change over time.

By taking part in research you are helping to further our understanding of FND and the best way to treat it, which we hope will be helpful for others in the future.

This completes part 1. If the information in Part 1 has interested you and you are considering participation, please read the additional information in Part 2 before making any decision.

Part 2 of the information sheet

What will happen if I don't want to carry on with the study?

You are free to withdraw from this study at any point without giving a reason by contacting the researcher whose details are given at the bottom of the consent form or by telling your clinical psychologist who is administering treatment. Withdrawing from the study will not affect your treatment and care in any way.

Should you wish to withdraw from the study, the anonymous data collected so far will be used in the final report of the study.

Concerns and Complaints

If you have a concern about any aspect of this study, you should ask to speak to me or you clinical psychologist, and we will do our best to address your concerns. You can contact me by leaving a message on the 24-hour voicemail phone number 01227 927070. Please leave a contact number and say that the message is for Tanya Suhalitka and I will get back to you as soon as possible. If you remain dissatisfied and wish to complain formally, you can do this by contacting Dr Fergal Jones, Clinical Psychology Programme Research Director, Salomons Institute for Applied Psychology <u>fergal.jones@canterbury.ac.uk</u>

Will information from or about me from taking part in the study be kept confidential? All personal information about you will be kept strictly confidential. You will be given a study number so that all information about you will be anonymous and cannot be identified in any research reports that are produced from this study.

All information collected will be stored in secure locations and on an encrypted, password protected NHS USB key and secure university server. We will keep your personal information (i.e. how you might be identified, e.g. consent form) separate from the other information and we collect (i.e. questionnaires). Research information will be kept securely at Canterbury Christ Church University for 10 years after the study ends; after 10 years this information will be securely destroyed.

What will happen to the results of the research study?

The results of the study will be written up as part of my doctoral dissertation, and potentially published in an academic research journal. Results might also be presented at relevant academic conferences.

If you would like a summary of results from this study, I will be happy to provide you with this once the study is completed.

Who is sponsoring and funding the research?

Canterbury Christ Church University.

Who has reviewed the study?

All research in the NHS is looked at by an independent group of people, called a Research Ethics Committee, to protect your interests. This study has been reviewed and given favourable opinion by the Surrey Research Ethics Committee, and Kent and Medway NHS and Social Care Partnership Trust Research and Development Department.

If you would like to speak to me and find out more about the study of have questions about it answered, you can leave a message for me on a 24-hour voicemail phone line at 01227 927070. Please say that the message is for Tanya Suhalitka and leave a contact number so that I can get back to you.

Appendix E

Summary letter to NHS ethics and study participants



SUMMARY OF RESEARCH STUDY

Title: EMDR for Functional Neurological Disorder and trauma: a single-case experimental design

Dear participant,

Thank you for taking part in the above study conducted between 2020-2021. You are receiving this email because you have requested a summary of results and findings following completion of this study.

Aim of the study

The overarching aim of this study was to evaluate the effectiveness of EMDR therapy for individuals with FND and a history of traumatic experiences in reducing the following difficulties: 1) frequency, severity, and distress of FND symptoms 2) functional impairment associated with FND 3) trauma-related symptoms 4) anxiety symptoms 5) depression symptoms 6) general psychological distress.

EMDR is an effective, evidence-based treatment for trauma-related conditions. The model of EMDR propose that exposure to traumatic events can have a negative and lasting impact on thoughts, feeling, behaviours, and physical sensations. EMDR proposes that when traumatic memories are not fully processed, they exist in a 'frozen state' and the person can continue to re-experience the thoughts, feelings, and sensations that occurred during the traumatic event long after. Although, there are many theories put forward for the development of FND, research and clinical practice indicate that many individuals with FND present with a history of trauma, and therefore, the two may be connected. If so, this connection can have important implications for treatment.

Method

To investigate the effectiveness EMDR, this study recruited nine participants and allocated each participant to a 2-, 4-, or 6- week waiting-list period. By introducing treatment at different times, it allows for greater clarity about the effect of the intervention, especially if symptoms improve for those

receiving treatment but not for those who are waiting. After the waiting-list period, all participants received eight 90-minute session of EMDR. To track progress, all participants completed weekly outcome measures online or in with person with the study therapist. All participants were followed up at 2-, 4-, and 6-weeks after treatment for a clinical review.

Results

The findings from this study offer some preliminary evidence of the effectiveness of EMDR. Four participants in this study reported a significant reduction in FND symptoms, functional impairment, trauma-related distress, anxiety, depression, and global psychological distress. However, there was no improvement for two participants. One reason for the lack of changes may related to individual characteristics and ability to tolerate therapy, however, this cannot be known for sure and further research is needed.

Conclusion

These results suggest that EMDR may be a promising treatment option for individuals with FND and trauma. Although not everyone in the study improved, the participants that did reported clinically significant improvement on all measures. Future research is needed to further understand who might benefit from EMDR and under what conditions.

King regards, **Tanya Suhalitka Trainee Clinical Psychologist** For any queries about this study, please email t.suhalitka423@canterbury.ac.uk

Appendix F

Research Ethics Committee (REC) letter of favourable opinion

Appendix G

Health Research Authority (HRA) Approval Letter

Appendix H

Research & Development (R&D) Letter of Access

Appendix I

Outcome measures