



Trauma History Mapping and Family Psychoeducation Protocol (THMFP): A Structured Trauma-Informed Clinical Formulation Framework for Functional Neurological and Psychosomatic Disorders

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ABSTRACT

Functional Neurological Disorders (FND), dissociative conditions, and psychosomatic presentations remain clinically challenging due to fragmented care pathways, diagnostic uncertainty, and inconsistent integration of psychological and neurological perspectives. This paper presents the Trauma History Mapping and Family Psychoeducation Protocol (THMFP), a structured clinical framework designed to support trauma-informed formulation and psychoeducation in patients with functional and psychosomatic symptoms. THMFP integrates three core components: (1) systematic trauma history mapping, (2) neuropsychological and psychosomatic formulation, and (3) consent-based family psychoeducation. The protocol is grounded in contemporary models of FND emphasizing predictive processing, salience network dysregulation, autonomic nervous system imbalance, and trauma-related emotional processing. The model was developed through retrospective clinical application in a sample of approximately 28 patients presenting with functional and psychosomatic symptoms, alongside a comparison group of 15 individuals with stress-related but non-functional presentations. Observational outcomes suggested improved diagnostic clarity, enhanced patient understanding, and improved engagement with care; however, no standardized outcome measures were applied. THMFP is presented as a structured clinical formulation and psychoeducation framework requiring further validation through prospective and controlled studies.



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1. Introduction

Functional Neurological Disorders (FND), dissociative disorders, and psychosomatic conditions represent a significant proportion of presentations in neurology and psychiatry. These conditions are characterized by clinically significant distress and functional impairment without identifiable structural neurological pathology.

Despite increasing recognition of their neurobiological basis, patients frequently experience delayed diagnosis, fragmented care across specialties, and persistent stigma. Conventional biomedical models alone are often insufficient to explain the interaction between trauma exposure, emotional dysregulation, autonomic dysfunction, and symptom expression.

Contemporary research supports an integrative biopsychosocial understanding of FND involving predictive processing dysfunction, altered salience network activity, autonomic dysregulation, and trauma-related emotional processing abnormalities (Edwards et al., 2012; Hallett, 2016; Kozłowska et al., 2017). Within this context, structured clinical frameworks that integrate trauma history and family systems may enhance formulation clarity and treatment engagement.

This paper introduces THMFP as a structured clinical protocol designed to operationalize trauma-informed formulation and family psychoeducation in functional and psychosomatic disorders.

2. Theoretical Background

2.1 Functional Neurological Disorders and Contemporary Neurocognitive Models

Functional Neurological Disorder (FND) has undergone a profound conceptual transformation over the past two decades. Historically referred to as conversion disorder or hysteria, FND was traditionally conceptualized as a diagnosis of exclusion in which neurological symptoms occurred despite the absence of structural pathology. Contemporary neuroscience has fundamentally changed this perspective. FND is now recognized as a disorder of altered brain function involving abnormal interactions among neural networks responsible for motor control, emotion regulation, attention, salience detection, agency, and interoceptive awareness rather than structural neurological damage (Edwards et al., 2012; Hallett et al., 2022).

The predictive processing (predictive coding) framework has become one of the most influential theoretical models explaining FND. According to this model, the brain continuously generates hierarchical predictions about

incoming sensory information based on previous experiences and learned expectations. Perception and motor behavior emerge from the interaction between these top-down predictions and bottom-up sensory signals. Normally, prediction errors allow continuous updating of internal models. In FND, however, maladaptive prior beliefs become excessively precise or rigid, resulting in diminished weighting of incoming sensory evidence. Consequently, symptoms such as limb weakness, tremor, sensory loss, or psychogenic non-epileptic seizures may emerge because internally generated expectations override actual sensory information (Edwards et al., 2012; Friston, 2018).

This Bayesian framework explains several characteristic clinical observations. Functional symptoms often fluctuate according to attention, emotional state, suggestion, or contextual cues, phenomena difficult to explain using structural neurological models alone. Increased self-focused attention may amplify abnormal predictions, while reassurance, distraction, or therapeutic reframing may reduce symptom expression by modifying maladaptive expectations.

Functional neuroimaging studies further support this network-based conceptualization. Resting-state and task-based functional MRI investigations consistently demonstrate altered connectivity among the supplementary motor area, anterior cingulate cortex, insular cortex, temporoparietal junction, amygdala, prefrontal cortex, basal ganglia, and cerebellum. These regions collectively regulate emotional processing, motor initiation, self-agency, salience attribution, and autonomic regulation (Perez et al., 2021). Rather than identifying isolated lesions, these findings suggest widespread disturbances in communication across distributed neural networks.

Abnormalities within the salience network appear particularly important. The anterior insula and anterior cingulate cortex normally determine which internal or external stimuli deserve conscious attention. Hyperactivation of these regions may increase attentional allocation toward bodily sensations, facilitating symptom amplification and reinforcing illness expectations. Simultaneously, impaired executive regulation by the dorsolateral prefrontal cortex may reduce cognitive flexibility necessary to update maladaptive symptom beliefs.

Another emerging concept involves disturbances in the sense of agency. Patients frequently report that movements or symptoms are involuntary despite appearing physiologically possible. Neurophysiological studies suggest impaired integration between motor

intention and sensory feedback, leading patients to genuinely experience symptoms as beyond voluntary control (Voon et al., 2016). This distinction is clinically important because it differentiates FND from malingering or factitious disorders and reinforces the understanding that symptoms represent genuine dysfunction in brain network processing.

Recent consensus statements from international neurological organizations emphasize that FND should be diagnosed using positive clinical signs rather than by exclusion. Clinical features such as Hoover's sign, tremor entrainment, distractibility, and internal inconsistency provide positive evidence supporting the diagnosis while simultaneously facilitating therapeutic explanation (Aybek et al., 2023). This shift toward positive diagnosis reduces diagnostic delay, decreases unnecessary investigations, and enhances patient engagement with treatment.

Collectively, contemporary neurocognitive models position FND as a disorder arising from maladaptive predictive processing, abnormal attention allocation, altered self-agency, emotional dysregulation, and dysfunctional large-scale brain networks rather than structural neurological injury. These developments provide a robust theoretical foundation for trauma-informed clinical formulation models such as the Trauma History Mapping and Family Psychoeducation Protocol (THMFP).

2.2 Trauma, Stress Neurobiology, and Autonomic Dysregulation

Traumatic experiences represent one of the most consistently reported risk factors for Functional Neurological Disorder, dissociative disorders, and psychosomatic symptom presentations. Although not every individual with FND has experienced major trauma, adverse childhood experiences, chronic interpersonal stress, emotional neglect, attachment disruption, and cumulative life adversity significantly increase vulnerability to functional symptoms (Kozłowska et al., 2021).

Trauma influences the developing nervous system through prolonged activation of stress-response pathways. Chronic activation of the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system produces sustained elevations in cortisol, catecholamines, inflammatory mediators, and autonomic arousal. Over time, these physiological adaptations may alter neural development, emotional regulation, threat detection, and interoceptive processing. Individuals become increasingly sensitive to stress-related cues while

demonstrating reduced flexibility in autonomic regulation.

The autonomic nervous system plays a central role in understanding functional symptom generation. Polyvagal Theory proposes that adaptive responses to danger occur through hierarchical activation of ventral vagal, sympathetic, and dorsal vagal systems depending on perceived safety or threat (Porges, 2011). Under conditions of overwhelming stress, defensive responses may progress beyond fight-or-flight toward immobilization, dissociation, emotional numbing, or collapse. Many functional neurological symptoms—including psychogenic non-epileptic seizures, transient paralysis, fainting episodes, dissociative states, and functional sensory disturbances—can be conceptualized within this framework as maladaptive protective responses rather than deliberate behaviors.

Neuroimaging studies demonstrate increased amygdala reactivity, altered hippocampal functioning, reduced medial prefrontal regulation, and impaired connectivity between emotional and motor networks among trauma-exposed individuals. These alterations influence memory integration, emotional regulation, bodily awareness, and stress responsiveness. Importantly, trauma-related neural adaptations may persist long after the original threat has resolved, increasing susceptibility to symptom recurrence during subsequent psychosocial stress.

Recent research also highlights the importance of interoception—the brain's capacity to perceive internal bodily states. Trauma survivors frequently demonstrate altered interoceptive accuracy and heightened vigilance toward bodily sensations. Minor physiological fluctuations may therefore be interpreted catastrophically, reinforcing maladaptive predictive models and promoting functional symptom expression. These findings support integrated models linking trauma, autonomic dysregulation, predictive processing, and psychosomatic symptom formation.

Nevertheless, trauma should not be considered either necessary or sufficient for FND. Current evidence supports a multifactorial model in which biological vulnerability, personality characteristics, cognitive expectations, attachment experiences, sociocultural influences, and environmental stressors interact dynamically over time. Trauma-informed care therefore seeks to understand symptom development within an individual's life narrative while avoiding simplistic causal assumptions.

2.3 Psychosomatic Integration: Interoception, Emotion Regulation, and Brain–Body Communication

Psychosomatic medicine has increasingly moved beyond traditional mind–body dualism toward an integrated biopsychosocial neuroscience perspective. Functional symptoms are now conceptualized as genuine disturbances in brain–body communication arising from dynamic interactions among emotional processing, autonomic regulation, cognitive appraisal, interoceptive awareness, immune signaling, and environmental stress.

Interoception represents a central mechanism in this integration. The insular cortex continuously monitors internal physiological signals including heart rate, respiration, visceral sensation, pain, and autonomic activity. These signals contribute to emotional experience, self-awareness, and adaptive behavioral regulation. Disturbances in interoceptive processing may amplify normal bodily sensations or distort their interpretation, contributing to persistent symptom perception despite the absence of structural pathology.

Emotion regulation provides another important mechanism. Chronic suppression, avoidance, or dysregulated expression of emotional experiences increases physiological arousal while reducing cognitive flexibility. Emotional conflict may therefore be expressed somatically when adaptive emotional processing becomes impaired. Functional symptoms should not be interpreted as symbolic conversion alone but rather as neurobiological manifestations of dysregulated stress processing involving multiple interacting systems.

The concept of central sensitization further contributes to understanding persistent psychosomatic symptoms. Repeated activation of nociceptive and stress-related neural pathways increases central nervous system responsiveness, producing heightened sensitivity to pain, fatigue, dizziness, and other bodily sensations. This mechanism helps explain the overlap among chronic pain disorders, fibromyalgia, chronic fatigue syndrome, irritable bowel syndrome, and FND.

Contemporary models emphasize reciprocal rather than unidirectional relationships. Psychological stress alters physiological regulation, while persistent bodily symptoms increase anxiety, catastrophic interpretation, avoidance behavior, and social disability, creating self-reinforcing feedback loops. Effective clinical intervention therefore requires simultaneous attention to biological, psychological, and social processes rather than privileging one domain over another.

2.4 Family Systems, Social Learning, and Psychoeducation in Functional Disorders

Family systems exert profound influence on illness perception, symptom maintenance, treatment engagement, and functional recovery. Contemporary family systems theory conceptualizes symptoms not only as individual phenomena but also as occurring within broader interpersonal, cultural, and relational contexts. Family beliefs, communication patterns, caregiving behaviors, and emotional responses may either facilitate recovery or inadvertently reinforce disability.

Social learning theory suggests that illness behaviors can be strengthened through reinforcement, attention, accommodation, or avoidance of stressful situations. Family members who respond with excessive reassurance, overprotection, repeated medical consultation, or restriction of normal activities may unintentionally increase symptom persistence despite compassionate intentions. Conversely, dismissive or stigmatizing responses may intensify psychological distress and reduce treatment adherence.

Research consistently demonstrates that psychoeducation improves patient understanding, reduces fear, enhances therapeutic alliance, and increases adherence to multidisciplinary treatment in FND. Effective psychoeducation explains symptoms as genuine disorders of nervous system functioning rather than fabricated illness, emphasizing reversibility through rehabilitation, emotional regulation, and behavioral retraining.

Family involvement is particularly valuable in trauma-informed care because relatives frequently struggle to reconcile normal medical investigations with severe functional disability. Structured psychoeducation provides families with a shared explanatory framework that reduces blame, decreases misunderstanding, and encourages adaptive support. Educational interventions also help family members recognize reinforcement patterns that may unintentionally maintain symptoms while promoting gradual return to functional activities.

Cultural context should also be considered. In many collectivist societies, family members play central roles in health-care decision making, caregiving, and illness interpretation. Consequently, interventions targeting only the individual patient may overlook influential interpersonal dynamics that contribute to symptom persistence or recovery. Integrating family psychoeducation into trauma-informed clinical formulation therefore represents a theoretically coherent extension of contemporary biopsychosocial practice.

Taken together, current evidence suggests that successful management of Functional Neurological Disorder requires integration of neuroscience, trauma psychology, psychosomatic medicine, autonomic physiology, and family systems perspectives. The THMFP framework builds upon these complementary theoretical foundations by providing a structured clinical process through which trauma history, neuropsychological formulation, and family psychoeducation can be synthesized into individualized patient care.

3. Methods

3.1 Design

This study describes a retrospective clinical framework analysis based on structured application of THMFP in routine outpatient counseling and psychotherapy practice.

3.2 Participants

Approximately 28 individuals presenting with functional neurological symptoms, dissociative symptoms, or somatic symptom presentations were included in THMFP-informed clinical sessions. A comparison group of 15 individuals with stress-related complaints without functional neurological symptom presentation received brief supportive counseling.

No formal diagnostic instruments or structured rating scales were systematically applied.

3.3 Procedure

THMFP was implemented in three phases:

Phase 1: Trauma History Mapping

A structured clinical interview was conducted to reconstruct developmental, acute, and chronic stress exposure. Symptom onset and progression were mapped alongside psychosocial stressors.

Phase 2: Neuropsychological Formulation

Clinicians developed an integrative formulation linking emotional processing, autonomic regulation, cognitive appraisal, and somatic symptom expression.

Phase 3: Family Psychoeducation

With patient consent, family members were engaged in structured psychoeducation sessions focusing on functional neurological symptoms, stress-symptom relationships, and adaptive family responses.

3.4 Outcomes (Observational)

Clinical observations were recorded in case notes regarding:

- patient understanding of symptoms
- engagement with treatment
- perceived reduction in stigma
- subjective symptom improvement
- No standardized outcome measures were used.

4. Results

Across the clinical sample, THMFP implementation was associated with:

- Improved patient insight into symptom patterns
- Enhanced engagement in psychological treatment
- Reduced family misunderstanding and stigma in selected cases
- Subjective reduction in symptom distress reported in follow-up sessions

The comparison group receiving supportive counseling demonstrated stabilization of stress-related symptoms but did not require full THMFP implementation.

These findings are preliminary and descriptive in nature.

5. Discussion

This paper presents THMFP as a structured clinical framework for integrating trauma history mapping, neuropsychological formulation, and family psychoeducation in functional and psychosomatic disorders.

The model aligns with contemporary FND research emphasizing predictive processing dysfunction, salience network alterations, and autonomic dysregulation. It also extends existing trauma-informed care approaches by formalizing a structured workflow for clinical application.

5.1 Clinical Implications

THMFP may support:

- improved diagnostic clarity
- enhanced patient understanding
- reduced stigma and misattribution of symptoms
- improved family engagement in care

5.2 Comparison with Existing Models

THMFP overlaps with existing trauma-informed CBT approaches, psychosomatic medicine frameworks, and FND psychoeducation models. Its contribution lies primarily in the structured integration of trauma

mapping and family psychoeducation into a unified protocol.

5.3 Limitations

This study has several limitations:

- retrospective and observational design
- small sample size
- absence of standardized outcome measures
- lack of long-term follow-up
- potential clinician bias in outcome interpretation

5.4 Future Directions

Future research should evaluate THMFP using:

- prospective cohort designs
- standardized clinical scales

- randomized controlled trials
- cross-cultural validation studies

6. Conclusion

THMFP is a structured, trauma-informed clinical formulation and psychoeducation protocol designed for functional neurological and psychosomatic disorders. While preliminary clinical observations suggest potential benefits in diagnostic clarity and patient engagement, rigorous empirical validation is required.

Declarations

Ethics approval: Not applicable (retrospective clinical framework description). Conflict of interest: None declared. Funding : None.

Figure -1. APPENDIX A: MASTER CASE SERIES TABLE (n = 28)

Functional Neurological, Dissociative, and Psychosomatic Presentations

Case	Age / Sex	Clinical Context	Core Presenting Symptoms	Pain / Somatic Pattern	Medical Workup	Functional Impact	Psychosocial Context	Clinical Classification
1	18 / F	Student	Dissociation, fainting episodes	Episodic non-epileptic events	No structural cause	Academic disruption	Exam stress	Functional neurological symptom disorder (FND)
2	19 / F	Student	Motor weakness, fatigue	Limb weakness fluctuating	Normal investigations	Reduced school function	Emotional stress	FND
3	21 / M	Student	Tremors, anxiety	Tremor episodes	No neurological disease	Social withdrawal	Peer stress	FND
4	22 / F	Student	Non-epileptic seizures	Episodic body collapse	Normal EEG	Functional impairment	Acute stress trigger	FND (PNES type)
5	42 / F	Social Worker	Memory gaps, dissociation	Perceptual disruption	No pathology	Academic decline	Trauma history	Dissociative disorder
6	24 / M	Office worker	Chronic pain, fatigue	Diffuse body pain	No organic cause	Reduced work efficiency	Workplace stress	Somatic symptom disorder
7	25 /	Office	Panic + somatic	Chest/body	Cardiac causes	Functional	Anxiety	Somatic symptom

Cas e	Ag e / Sex	Clinical Context	Core Presenting Symptoms	Pain / Somatic Pattern	Medical Workup	Functional Impact	Psychosocial Context	Clinical Classificatio n
	M	worker	complaints	discomfort	excluded	limitation	disorder	disorder
8	23 / F	Student	Weakness episodes	Fluctuating limb symptoms	Normal neurology	Intermittent disability	Emotional overload	FND
9	36 / M	Homemaker	Somatic pain, emotional distress	Generalized pain	No pathology detected	Reduced household functioning	Marital stress	Somatic symptom disorder
10	28 / F	Homemaker	Dissociative episodes	Altered awareness states	No organic disease	Functional disruption	Trauma-related stress	Dissociative disorder
11	30 / F	Homemaker	Functional weakness	Limb weakness variability	No neurological disease	Partial disability	Family stress	FND
12	32 / M	Worker Daily Wages	Pain, anxiety	Diffuse pain	No pathology	Reduced coping	Emotional insecurity	Somatic symptom disorder
13	35 / F	Daily Wages Worker	Somatic + sleep disturbance	Body pain + insomnia	Normal investigations	Functional impairment	Chronic stress ,	Somatic symptom disorder
14	38 / F	Homemaker	Emotional dysregulation	Somatic distress/compulsive behavior	No organic cause/reported Daily hassles and Familial affects.	Reduced functioning	Trauma exposure	OCD
15	40 / M	Worker	Chronic pain syndrome	Musculoskeletal pain	No structural pathology	Reduced activity	Long-term stress	Somatic symptom disorder
16	42 / F	Homemaker	Functional weakness + fatigue	Limb heaviness	No neurological findings	Household difficulty	Care burden	FND
17	45 / M	Technician	Pain in hands	Bilateral hand pain	No nerve pathology	Difficulty coping	Emotional strain	FND (pain dominant)
18	47 / F	Homemaker	OCD-like symptoms + somatic distress	Pain + anxiety	No organic disease	Functional impairment	Family accommodation	OCD + somatic symptoms
19	50 / F	Homemaker	Chronic somatic	Diffuse pain	No pathology	Reduced functioning	Long-term stress	Somatic symptom

Cas e	Ag e / Sex	Clinical Context	Core Presenting Symptoms	Pain / Somatic Pattern	Medical Workup	Functional Impact	Psychosocial Context	Clinical Classificatio n
			complaints					disorder
20	52 / M	Homemake r	Somatic + depressive features	Generalized pain	Normal investigations	Functional decline	Emotional burden	Somatic symptom disorder
21	55 / F	Homemake r	Functional neurological symptoms	Episodic weakness	No neurological disease	Partial disability	Chronic stress	FND
22	58 / F	Homemake r	OCD contaminatio n type	Cleaning-related distress	No organic findings	Functional impairment	Family accommodation	OCD
23	60 / M	Homemake r	Chronic pain disorder	Diffuse musculoskeletal pain	No pathology	Reduced activity	Aging + stress	Somatic symptom disorder
24	62 / M	Retired	Somatic complaints + anxiety	Generalized pain	No structural disease	Reduced function	Loneliness/stres s	Somatic symptom disorder
25	32 / F	Wife of Gulf- employed spouse; homemaker	Insecurity, body pain	Diffuse unspecified pain	No pathology detected	Difficulty coping but functional	Emotional insecurity, separation stress	Functional somatic symptom disorder
26	45 / F	Wife of Gulf- employed spouse; homemaker	Hand pain, coping difficulty	Bilateral hand pain	No neurological abnormality	Functional but distressed	Psychosocial strain	FND (pain dominant)
27	27 / F	Wife of Gulf- employed spouse; homemaker	Generalized pain, stress intolerance	Diffuse pain	No pathology detected	Functional with distress	Emotional overload	Somatic symptom disorder
28	29 / M	Company Man/Newly appointed	Mixed somatic + anxiety symptoms	Variable body pain	No organic disease	Partial functional impairment	Chronic psychosocial stress	Somatic symptom disorder

Figure -2

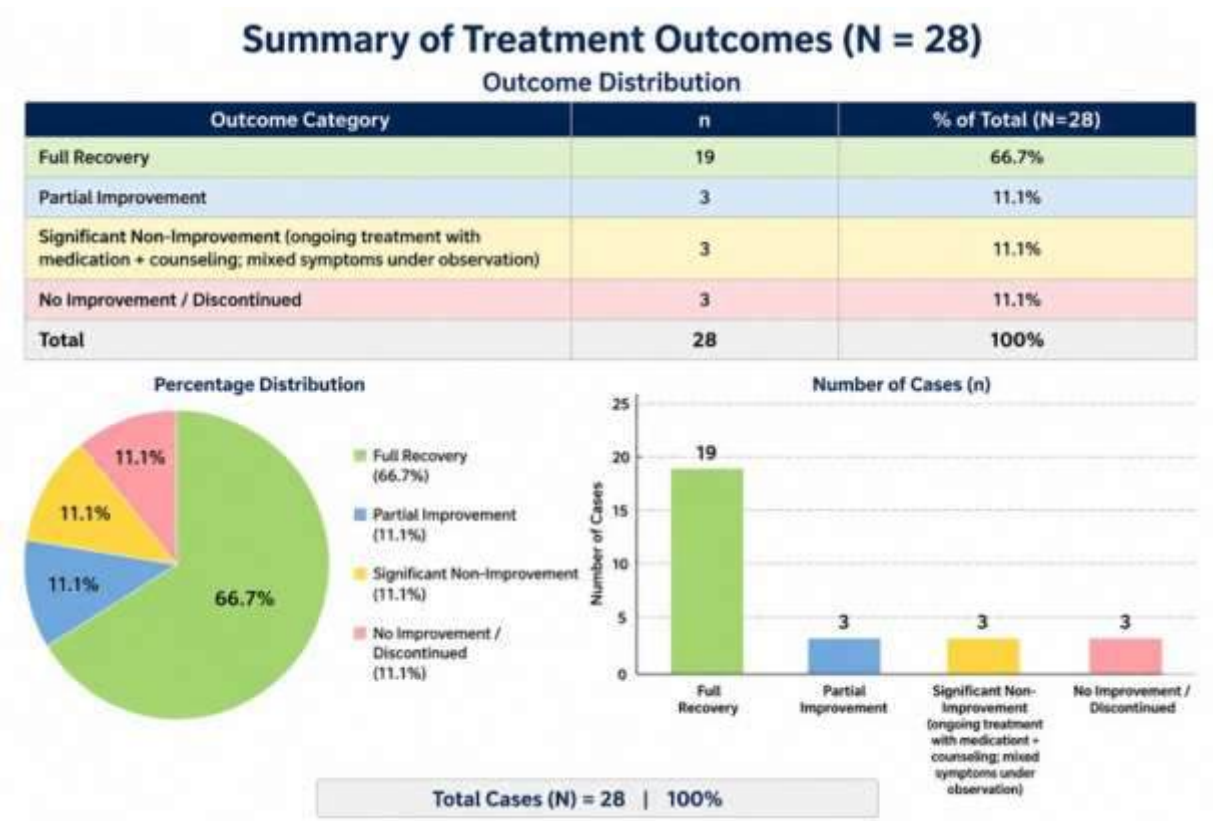
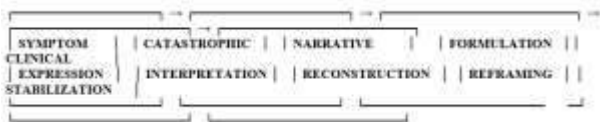


FIGURE 3. THMFP MICRO-PROCESS MODEL OF CLINICAL TRANSFORMATION (An Example)

Title

From Symptom to Meaning to Recovery: Micro-Process Pathway in THMFP Intervention

FIGURE LAYOUT (LEFT → RIGHT CLINICAL TRANSFORMATION FLOW)



STEPWISE MODEL (FIGURE CONTENT)

1. Symptom Expression

- Functional paralysis / seizures / pain / dissociation
- Sudden onset often after stress exposure
- Medical investigations normal



2. Catastrophic Interpretation

- “Am I schizophrenic?”
- Fear of permanent neurological disease
- Identity collapse
- High anxiety + medical uncertainty



3. Narrative Reconstruction (THMFP Mapping)

- Temporal anchoring (“When did it start?”)
- Event recall before onset
- Emergence of psychosocial stress story
- Emotional meaning revealed



4. Formulation Reframing

- Symptom = functional stress response
- Not structural disease
- Brain–body protective pattern
- Family reinforcement identified



5. Clinical Stabilization

- Anxiety reduction
- Improved insight
- Reduced stigma
- Functional engagement begins

FIGURE CAPTION

Figure 3. THMFP micro-process model of clinical transformation. The figure illustrates the proposed therapeutic

pathway observed during implementation of the Trauma History Mapping and Family Psychoeducation Protocol (THMFP). Patients initially present with functional neurological or psychosomatic symptoms that are frequently interpreted catastrophically. Guided trauma history mapping facilitates narrative reconstruction and identification of relevant psychosocial stressors. Neuropsychological formulation and structured psychoeducation subsequently reframe symptoms as functional brain-body responses rather than structural neurological disease, promoting anxiety reduction, improved illness understanding, family engagement, and progressive functional recovery.

A clinical transformation flow

B Neural network model (predictive processing)

Assessment

Symptoms



Functional Expression



Trauma Mapping



Clinical Formulation



Mechanism

Catastrophic Threat Prediction



Predictive Processing



Narrative Integration



Predictive Reframing



Outcome

Stabilization



Recovery



Self-Regulation & Adaptive Functioning

This structure is conceptually stronger because it tells a complete story:

1. **Assessment** – understanding the patient's symptoms and trauma.
2. **Mechanism** – explaining those symptoms through predictive processing and maladaptive threat predictions.
3. **Intervention and Outcome** – updating predictions through narrative reconstruction and predictive reframing, leading to stabilization, recovery, and improved self-regulation.

References

- Aybek, S., Perez, D. L., Nicholson, T. R., Stone, J., Carson, A., Gelauff, J., ... & Hallett, M. (2023). Functional neurological disorder: New directions in diagnosis and treatment. *The Lancet Neurology*, 22(2), 142–154.
- Baslet, G. (2011). Psychogenic non-epileptic seizures: A treatment review. What Have We Learned Since the Beginning of the Millennium? *Neuropsychiatric Disease and Treatment*, 7, 585–598.
- Baslet, G., Roiko, A., & Prensky, E. (2020). Heterogeneity in functional neurological disorders: Understanding the role of psychological trauma. *Epilepsy & Behavior*, 108, 107073.
- Brown, R. J., & Reuber, M. (2016). Psychological and psychiatric aspects of functional neurological disorders. *Current Opinion in Neurology*, 29(4), 507–512.
- Carson, A., Lehn, A., & Ludwig, L. (2016). Explaining functional disorders in the neurology clinic: A photo story. *Practical Neurology*, 16(1), 56–61.
- Carson, A., Stone, J., Hibberd, C., Murray, G., Duncan, R., Coleman, R., ... & Sharpe, M. (2011). Disability, distress and unemployment in neurology outpatients with symptoms unexplained by organic disease. *Journal of Neurology, Neurosurgery & Psychiatry*, 82(7), 810–813.
- Critchley, H. D., & Garfinkel, S. N. (2017). Interoception and emotion. *Current Opinion in Psychology*, 17, 7–14.
- Craig, A. D. (2009). How do you feel—now? The anterior insula and human awareness. *Nature Reviews Neuroscience*, 10(1), 59–70.
- Damasio, A. R. (1999). *The feeling of what happens: Body and emotion in the making of consciousness*. Harcourt Brace.
- Edwards, M. J., Adams, R. A., Brown, H., Pareés, I., & Friston, K. J. (2012). A Bayesian account of "hysteria."

Journal of Neurology, Neurosurgery & Psychiatry, 83(8), 842–850.

Espay, A. J., Aybek, S., Carson, A., Edwards, M. J., Goldstein, L. H., Hallett, M., ... & Morgante, F. (2018). Current concepts in diagnosis and treatment of functional neurological disorders. *JAMA Neurology*, 75(9), 1132–1141.

Fani, N., & Ressler, K. J. (2018). Imaging genetics of anxiety disorders. *NeuroImage*, 53, 822–831.

Friston, K. (2010). The free-energy principle: A unified brain theory? *Nature Reviews Neuroscience*, 11(2), 127–138.

Friston, K. (2018). Does predictive coding have a future? *Nature Neuroscience*, 21(8), 1019–1021.

Goldstein, L. H., & Mellers, J. D. C. (2019). Recent developments in our understanding of the semiology and treatment of functional neurological disorders. *Current Opinion in Neurology*, 32(4), 442–447.

Goldstein, L. H., Robinson, E. J., Mellers, J. D. C., Stone, J., Carson, A., Reuber, M., ... & Chalder, T. (2020). Cognitive behavioural therapy for adults with dissociative seizures. *The Lancet Psychiatry*, 7(6), 491–505.

Hallett, M. (2016). Functional neurological disorder: Is it a neurological condition? *Journal of Neurology*, 263(6), 1211–1213.

Hallett, M., Aybek, S., Dworetzky, B. A., McWhirter, L., Staab, J. P., & Stone, J. (2022). Functional neurological disorder: New subtypes and shared mechanisms. *The Lancet Neurology*, 21(6), 537–550.

Kozłowska, K., English, M., Savage, B., & Chudleigh, C. (2021). Functional somatic symptoms in children and adolescents: A stress-system approach to assessment and treatment (2nd ed.). Palgrave Macmillan.

Lanius, R. A., Vermetten, E., & Pain, C. (2010). The impact of early life trauma on health and disease: The hidden epidemic. Cambridge University Press.

Lanius, R. A., Frewen, P. A., Vermetten, E., & Yehuda, R. (2020). Fear conditioning and early life trauma. *American Journal of Psychiatry*, 177(7), 563–571.

LeDoux, J. E. (2012). Rethinking the emotional brain. *Neuron*, 73(4), 653–676.

McEwen, B. S. (2017). Neurobiological and systemic effects of chronic stress. *Chronic Stress*, 1, 1–11.

Nicholson, T. R., Carson, A., Edwards, M. J., Goldstein, L. H., Hallett, M., Mildon, B., ... & Stone, J.

(2020). Outcome measures for functional neurological disorder: A review. *Journal of Neurology, Neurosurgery & Psychiatry*, 91(10), 1032–1040.

Paulus, M. P., & Stein, M. B. (2010). Interoception in anxiety and depression. *Brain Structure and Function*, 214(5–6), 451–463.

Perez, D. L., Nicholson, T. R., Asadi-Pooya, A. A., Bègue, I., Butler, M., Carson, A., ... & Hallett, M. (2021). Neuroimaging in functional neurological disorder: State of the field and research agenda. *NeuroImage: Clinical*, 30, 102623.

Porges, S. W. (2011). The polyvagal theory: Neurophysiological foundations of emotions, attachment, communication, and self-regulation. W. W. Norton.

Price, C. J., & Hooven, C. (2018). Interoceptive awareness skills for emotion regulation. *Frontiers in Psychology*, 9, 798.

Schönenberg, M., Mares, L., & Jusyte, A. (2023). Trauma, dissociation, and functional neurological symptoms: A systematic review. *Journal of Psychiatric Research*, 161, 235–247.

Sharpe, M., Carson, A., & Stone, J. (2021). Functional neurological disorder: Clinical challenges and future directions. *The BMJ*, 373, n1356.

Stone, J. (2021). Functional neurological disorders: The neurological assessment as treatment. *Practical Neurology*, 21(1), 5–8.

Stone, J., Carson, A., Duncan, R., Roberts, R., Warlow, C., Hibberd, C., ... & Sharpe, M. (2010). Symptoms unexplained by organic disease in neurology outpatients. *Brain*, 133(5), 1537–1551.

van der Kolk, B. A. (2014). The body keeps the score: Brain, mind, and body in the healing of trauma. Viking.

Voon, V., Cavanna, A. E., Coburn, K., Sampson, S., Reeve, A., & LaFrance, W. C. (2016). Functional neuroanatomy and neurophysiology of functional neurological disorders. *Journal of Neuropsychiatry and Clinical Neurosciences*, 28(3), 168–190.

World Health Organization. (2022). World mental health report: Transforming mental health for all. World Health Organization.

Yehuda, R., & Lehrner, A. (2018). Intergenerational transmission of trauma effects. *World Psychiatry*, 17(3), 243–257.