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"Neural dynamics and personality traits under the predictive coding framework in Functional Neurological Disorders"

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“Neural dynamics and personality traits under the predictive coding framework in Functional Neurological Disorders”

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I would like to dedicate this doctoral thesis to Z. and C..
I am most thankful for their love, patience, and support which truly
paved the way for me to develop into the person who could enthusiastically
pursue and diligently complete this work.

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Introduction

Functional Neurological Disorders (FND) comprise a group of conditions characterized by neurological symptoms that are incongruent with recognized structural or pathophysiological disease mechanisms, yet experienced as fully involuntary and often severely disabling (Gelauff & Stone, 2016; Stone et al., 2005). Clinical presentations are heterogeneous and include functional motor symptoms (e.g., weakness, tremor, dystonia, gait disturbances), non-epileptic seizures, sensory alterations, and cognitive or speech disturbances (Hallett et al., 2022). Far from being rare, FND represent one of the most common diagnoses encountered in neurology clinics, accounting for approximately 5–16% of outpatient consultations and up to one third of patients in specialized movement disorder services (Bennett et al., 2021). The associated burden is substantial: patients frequently experience chronic disability, reduced quality of life comparable to other major neurological conditions, and high healthcare utilization with repeated investigations and hospital admissions. Together, these features make FND not only a conceptual challenge for neuroscience but also a pressing clinical and societal priority, motivating the need for mechanistic models that can bridge subjective experience with measurable brain dynamics.

The present thesis grows out of a tension that has shaped the modern study of Functional Neurological Disorders (FND): although these conditions show no structural lesion, their symptoms are unmistakably real, often disabling, and embedded in the lived body. This tension has pushed researchers to move beyond the old dichotomies (organic versus psychogenic, neurological versus psychiatric) and toward a more nuanced view of how the brain constructs sensations, emotions, and movement. Over the last twenty years, the field has accumulated enough empirical weight to suggest that FND are neither conceptual leftovers nor historical curiosities. Instead, they represent a privileged vantage point from which to observe how large-scale neural networks interact over time, and how predictive processes shape the interpretation of bodily signals.

The structure of the thesis reflects this shift. It is composed of two major sections that examine the disorder from complementary angles. The first part focuses on altered brain dynamics, drawing on neuroimaging, electrophysiology, and network neuroscience to describe how the brains of people with FND operate. The second part turns to predictive coding and related theoretical frameworks, asking how the brain's inferential machinery contributes to the emergence and persistence of symptoms. These two strands are intentionally interwoven: neither is sufficient on its own. Network-level findings remain fragmentary unless interpreted through a model of how the brain hierarchically predicts and updates information; predictive theories, in turn, risk floating above empirical grounding unless anchored to measurable neural and behavioral data.

The first part of the thesis begins by revisiting the historical and diagnostic context of FND, not as a nostalgic exercise but as a necessary clarification of how current scientific questions emerged. The shift from psychodynamic conversion models to diagnosis based on positive clinical signs opened the door to a renewed investigation of the disorder's neurobiological underpinnings. Building on this foundation, the thesis follows the trajectory of contemporary research across multiple domains: studies of motor control and the sense of agency; work on emotional and interoceptive processing; investigations of static and dynamic connectivity; structural MRI and diffusion imaging; and preliminary genetic and epigenetic findings. Electrophysiology, too, has played a surprisingly central role, offering fine-grained temporal insights that help to distinguish what is preserved in FND (primary sensory pathways) from what is disrupted (higher-order integrative networks). The central contribution of this first part is the original study based on EEG microstates. Microstate analysis, with its ability to capture the rapid succession of quasi-stable brain states on the millisecond scale, offers a distinct window onto brain dynamics that conventional EEG metrics tend to overlook. In the context of FND, this method reveals that certain network activity configurations, particularly those associated with sensorimotor integration, show reduced temporal stability, while others display atypical transition tendencies. These findings suggest that the disorder involves not only altered activation of particular networks but also a disruption in the brain's spontaneous "syntax," its natural rules for moving from one functional state to another. This first section thus does more than summarize the field: it provides an empirical demonstration of the dynamic instability that characterizes FND at rest.

If the first part deals with "what" is altered, the second part asks "why" – or more precisely, what computational principles might drive these alterations. This section introduces predictive processing not as a fashionable overlay but as a coherent framework for understanding how symptoms arise when the balance between prior expectations and sensory evidence becomes skewed. Concepts such as allostatic regulation, interoceptive inference, and emotion as a constructive process help to illuminate how patients may come to perceive their motor output, bodily sensations, and emotional states through biased inferential filters. Within this theoretical architecture, the thesis presents additional empirical studies that examine behavioral and neural signatures of predictive styles in FND. These include a dimensional investigation of schizotypal and autistic-like traits as proxies for inferential biases; an exploration of frontal theta-alpha asymmetry as a potential neural marker of altered predictive weighting; and a study on individual differences in interoceptive prediction mechanisms. Together, these investigations illustrate how altered priors, noisy prediction errors, and disrupted precision-weighting may contribute to the phenomenology of FND, without reducing the disorder to any single trait or mechanism.

The value of the thesis lies in the integration of these perspectives. It combines high-resolution electrophysiological data with psychological measures, computationally informed theory with clinical relevance, and empirical observations with conceptual clarity. Rather than portraying FND as an absence – of lesions, of organic markers, of coherence – it reframes them as the product of specific and measurable alterations in how the brain organizes information over time and how it assigns weight to internal and external signals. The thesis argues that FND are not failures of will nor symbolic transformations of conflict, but disturbances in the brain's dynamic and inferential architecture: a condition in which certain predictions become too rigid, certain sensory channels insufficiently trusted, and certain networks unable to shift with the required flexibility.

By weaving together these strands, the thesis aims to offer not only new data but a coherent interpretative framework. In doing so, it contributes to the growing effort to understand FND as disorders of dynamic inference, in which biological, cognitive, and affective processes converge to shape a form of experience that is at once disrupted and deeply revealing of how the brain normally constructs the self.

PART 1

ALTERED BRAIN DYNAMICS IN FUNCTIONAL NEUROLOGICAL DISORDERS

From concept to connectivity

Functional neurological disorder (FND) sits in an awkward spot in neurology. Patients have symptoms that look and feel neurological (e.g. paresis, tremor, non-epileptic seizures, sensory loss) yet standard investigations fail to reveal a structural lesion or a conventional disease mechanism. For a long time, this absence of visible pathology was read as absence of disease. The diagnosis was used reluctantly, often as a label of exclusion, and was frequently entangled with ideas of hysteria, simulation, or "psychogenic" weakness of will. Even as evidence accumulated that symptoms are genuine and disabling, FND remained conceptually underdefined: too neurological for psychiatry, too psychological for neurology, and poorly anchored to any coherent model of brain function. Over the last two decades, this has begun to change. A wide range of studies – functional and structural MRI, PET/SPECT, EEG and MEG, TMS, as well as early work in genetics and epigenetics – have converged on a different picture. Rather than a "non-disease", FND appears to involve reproducible alterations in the way large-scale brain networks coordinate motor control, attention, emotion, and bodily self-awareness. At the same time, developmental and stress-related factors, together with individual differences in neurochemical and genetic profiles, seem to shape how these vulnerabilities emerge and stabilize over time. Taken together, these findings push us to abandon the simple question "is there a lesion or not?" and to ask instead what kind of dynamic dysfunction could give rise to the characteristic phenomenology of FND.

The aim of this chapter is to make that shift explicit and to provide a coherent neurobiological backdrop for the rest of the thesis. It does not try to offer an encyclopedic review of everything ever published on FND. Rather, it uses key strands of evidence to argue for a specific view: that FND is best understood as a disorder of network-level integration and predictive control, in which motor, limbic, salience, and interoceptive systems interact in atypical ways over time. The chapter therefore moves from past to present with a clear rationale. It starts by briefly revisiting the historical trajectory from Charcot's idea of a "functional lesion" to modern diagnostic criteria that emphasize positive signs and internal inconsistency instead of presumed psychogenesis. This historical detour is not just decorative: it shows that the current network-based view is not a complete rupture with the past, but a return (armed with better tools) to the intuition that something in the way the nervous system functions has gone awry, even in the absence of structural damage.

It then turns to contemporary mechanistic work. Task-based neuroimaging studies are used to illustrate how circuits involved in motor intention, sense of agency, emotion processing, and interoception behave differently in FND, particularly in regions such as the supplementary motor area, prefrontal cortex, temporoparietal junction, insula, and amygdala. Resting-state and dynamic connectivity approaches extend this picture from local responses to the temporal choreography of networks, suggesting that patients may be "stuck" in certain connectivity states and show reduced flexibility in switching between them. Structural MRI and diffusion imaging add another layer, indicating that some of these functional patterns may sit on top of longer-term changes in gray and white matter that reflect a mixture of developmental vulnerability and experience-dependent plasticity. Genetic and epigenetic findings, although still preliminary, are brought in not as isolated curiosities but as part of a broader argument: susceptibility to FND likely reflects an interaction between stress-sensitive molecular pathways (for example, serotonergic and oxytocinergic systems), early adversity, and the gradual shaping of limbic–prefrontal circuits. Finally, electrophysiological studies show that the same motifs reappear when the brain is examined at millisecond resolution: primary sensory pathways are intact, yet higher-order processing, oscillatory coordination, and network communication are often altered.

In sum, this chapter lays out a unifying reading of a heterogeneous literature, not meant to replace clinical observation or psychosocial formulations, but to provide a mechanistic scaffold on which they can rest. The subsequent sections will build directly on this scaffold, using EEG-based measures of network dynamics and predictive processing to test how far these ideas can be taken in explaining the cognitive-affective profile and symptom expression of people with FND.

1. Historical perspective

Functional Neurological Disorder (FND) encompasses a heterogeneous group of conditions in which patients experience genuine neurological symptoms, such as weakness, tremor, sensory loss, or cognitive disturbance, that cannot be explained by structural pathology or conventional disease mechanisms (Espay et al., 2018; Hallett et al., 2022). These symptoms often fluctuate, appearing inconsistently across contexts or tasks, and may involve complex interactions between voluntary control, attention, emotion, and expectation. The absence of clear structural abnormalities has long rendered FND an enigmatic entity, fueling controversy and diagnostic uncertainty. Historically viewed through the lens of malingering or hysteria, the disorder has occupied a shifting and often uncomfortable position at the interface between neurology and psychiatry.

Over the past decades, however, a growing body of evidence has begun to redefine this condition within a neurobiological and biopsychosocial framework. Converging findings from functional neuroimaging, electrophysiology, and behavioral paradigms suggest that FND reflects alterations in the brain's dynamic functioning rather than irreversible tissue damage. The biopsychosocial model provides a broader understanding of these findings, emphasizing that biological vulnerabilities, psychological processes, and social or interpersonal factors dynamically interact in the development and maintenance of symptoms. Still, the precise mechanisms underlying these disturbances remain a subject of active investigation, and no single neural biomarker has yet achieved diagnostic utility (Thomsen et al., 2020).

The conceptual origins of this modern view trace back to the late nineteenth century and the work of Jean-Martin Charcot at the Salpêtrière Hospital in Paris. Charcot's clinical and theoretical legacy marked the first attempt to approach what was then called hysteria with the same methodological rigor applied to neurological disease. Through meticulous observation and experimental methods ranging from hypnosis to early neurophysiological testing, he proposed that these puzzling symptoms must arise from a disturbance within the nervous system itself, though of a kind invisible to anatomical inspection. In his 1889 lectures, Charcot observed that "there is without doubt a lesion at some level of the nervous system, but certainly not of the nature of a circumscribed organic lesion of destructive nature." This notion of a functional lesion (e.g. a dysfunction of neural processes without structural damage) anticipated by more than a century the modern idea of network-level dysregulation as a substrate for functional neurological symptoms.

Despite Charcot's pioneering approach, the century that followed witnessed a profound conceptual drift (**Fig. 1**). The psychodynamic formulation of "conversion," developed by Freud and Breuer, proposed that emotional conflicts could be transformed into bodily symptoms. This theory, though influential, progressively lost empirical grounding, and by the late twentieth century the disorder (by then labeled "conversion disorder") had become marginalized and met with skepticism among clinicians, which doubted its legitimacy, often interpreting symptoms as intentional or exaggerated rather than genuine (Edwards et al., 2023; Kanaan & Craig, 2019).

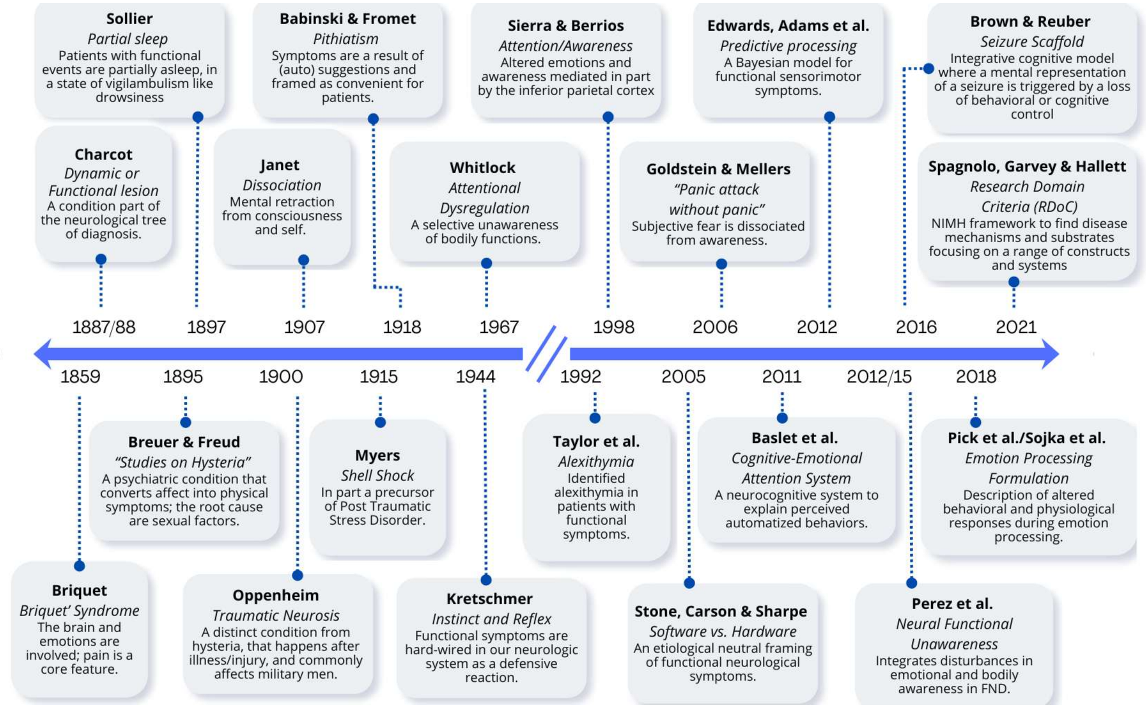

Figure 1 - Historical evolution of mechanistic models of Functional Neurological Disorders (FND)*. Timeline illustrating key theoretical frameworks proposed from the late nineteenth century to contemporary neuroscience accounts. The figure summarizes how interpretations of FND have progressively shifted from early notions of functional lesions and dissociation, through attentional and cognitive–affective models, to recent predictive-processing, interoceptive, and network-based perspectives. The schematic is intended as an illustrative overview rather than an exhaustive taxonomy. Adapted from Jungilligens et al. (2022) https://doi.org/10.1093/brain/awac204*

Diagnostic manuals such as ICD-10 and DSM-IV codified this uncertainty by requiring the exclusion of neurological disease and the demonstration of a psychogenic explanation (World Health Organization, 1992; American Psychiatric Association, 2013). However, these criteria were hard, if not impossible, to satisfy: the difficulty of conclusively ruling out malingering, together with the frequent absence of identifiable psychological conflict, left clinicians uncertain and patients stigmatized (Aybek & Perez, 2022; Hallett, 2006).

At the dawn of the new millennium, the disorder seemed to inhabit a conceptual no man's land: too "neurological" for psychiatry and too "psychological" for neurology. The diagnosis functioned more as a statement of diagnostic failure than as an affirmation of pathophysiological understanding. Despite its notable frequency and disabling impact, awareness of the disorder was almost nonexistent in the public sphere, where the term hysteria lingered only as a historical artefact of medical misogyny (Micale, 2019).

This situation began to change with the revision of diagnostic criteria in DSM-5 and ICD-11, in parallel with the terminological transition from "conversion disorder" to "functional neurological disorder", which marked a decisive shift toward a positive diagnostic model. Instead of relying on exclusion and presumed psychogenesis, the new framework emphasizes the identification of internal inconsistency or incongruence with known neurological patterns as features indicative of FND. Moreover, the presence of psychological trauma, once considered essential to the diagnosis, is no longer a necessary criterion. While adverse experiences may contribute to vulnerability in a subset of patients, they are neither ubiquitous nor explanatory in a univocal sense. The emphasis thus moved away from reconstructing a psychogenic narrative toward identifying mechanisms of aberrant neural integration, framing FND as a disorder of brain functioning rather than of character, intent, or hidden trauma.

This reframing had profound implications. For patients, it offered validation of their symptoms as real and grounded in brain dysfunction rather than moral or volitional weakness. For clinicians, it provided a coherent conceptual and practical framework, aligning FND more closely with other disorders of brain network dynamics. For researchers, it reopened long-closed avenues of investigation, stimulating the search for neurophysiological signatures that might capture how "functional" alterations unfold in the brain over time.

Within this renewed landscape, neuroimaging and electrophysiological studies have become central to elucidating the mechanisms of FND. They reveal distributed changes across cortical and subcortical systems implicated in motor control, attention, emotion, and interoception, yet the temporal dynamics through which these networks interact remain poorly characterized.

2. Neuroimaging evidence in FND: current state of the art

Initial indications that cerebral blood flow abnormalities in FND might be both reversible and dynamically modulated came from single-case investigations. Among the earliest of these, a study employing single-photon emission computed tomography (SPECT) documented a transient decrease in perfusion of the right parietal cortex occurring concomitantly with the manifestation of sensory-motor symptoms (Tiihonen et al., 1995). This early finding paved the way for a series of neuroimaging studies seeking to clarify the neural bases of FND. Some years later, a positron emission tomography (PET) investigation offered further evidence: when a patient with FND attempted to move the affected limb, increased activity was detected in the orbitofrontal and anterior cingulate cortices, whereas the primary motor cortex (M1) remained notably inactive (Marshall et al., 1997). These results began to shed light on the altered experience of volition and agency characteristic of the disorder, suggesting that symptoms arise from disturbances in neural control processes rather than from intentional simulation. Despite such advances, for a long time individuals with FND continued to be mischaracterized as consciously producing their symptoms.

Expanding on this line of inquiry, neuroimaging research has progressively dismantled long-standing misconceptions that framed FND symptoms as products of feigning or malingering, that is, as intentionally generated phenomena (Edwards et al., 2023). Functional neuroimaging evidence has repeatedly demonstrated that the neural activation patterns observed in FND differ substantially from those seen in individuals deliberately simulating symptoms. Comparative studies have shown that, across patients and regardless of the side of symptom manifestation, there is a consistent reduction of activity in the left dorsolateral prefrontal cortex (a region implicated in intentional motor control and volitional effort), a feature not found among feigners (Spence et al., 2000). Further investigations using functional magnetic resonance imaging (fMRI) during motor execution and inhibition paradigms have reinforced these findings: although certain superficial similarities exist between functional weakness and its simulated counterpart, the overall neural configurations remain clearly distinct. The resulting evidence suggests that patients with FND recruit broader and more complex neural processes than individuals instructed to mimic weakness, underscoring that their symptoms stem from specific disruptions in brain function rather than from deliberate, conscious control (Cojan et al., 2009; Stone et al., 2007; Van Beilen et al., 2011).

2.1 Motor control and sense of agency in FND

Research over recent years has increasingly pointed to consistent alterations within neural circuits subserving self-monitoring, motor preparation, and the subjective sense of agency in individuals with FND (Baek et al., 2017; Delange et al., 2007; Diez et al., 2019; Seghezzi et al., 2021). A landmark investigation explored these processes through a motor imagery paradigm in patients with functional paralysis who were instructed to imagine, but not perform, hand movements while undergoing fMRI (Delange et al., 2007). During imagery involving the affected limb, patients displayed markedly increased activation in the ventromedial prefrontal cortex (vmPFC) relative to imagery of the unaffected hand. This area, an essential hub of the default mode network (DMN), known for its role in self-referential thought and internal monitoring (Buckner et al., 2008; Gusnard et al., 2001), appeared to engage more strongly in the context of impaired motor representation. The enhanced vmPFC recruitment has been interpreted as signaling intensified self-monitoring processes, potentially reflecting a compensatory or maladaptive response to disrupted motor control (Delange et al., 2007).

Subsequent investigations into motor preparation mechanisms in patients with FND have identified consistent alterations within the supplementary motor area (SMA). In particular, Voon and colleagues (2011), using paradigms that contrasted self-initiated and externally cued movements, observed a marked reduction of SMA activation accompanied by increased activity in limbic structures. Importantly, this attenuation of SMA engagement emerged independently of whether movements were internally or externally triggered. Beyond regional activation, the study also revealed disrupted functional coupling between the SMA and the dorsolateral prefrontal cortex (DLPFC) during self-generated actions. Such a breakdown of SMA–DLPFC connectivity points to a deficit in top-down executive modulation, whereby frontal control systems fail to effectively govern motor planning and execution. This impairment appears particularly pronounced for voluntary actions that demand attentional monitoring, while more automatic, externally prompted movements remain relatively preserved. Clinically, this neurofunctional dissociation resonates with the well-known phenomenon of “motor discordance,” whereby patients can perform automatic actions such as walking or bearing weight on a weak limb yet remain unable to voluntarily lift it during examination (Hallett et al., 2022).

Abnormalities in interoceptive processing may also contribute to altered sensorimotor integration in FND. A recent task-based fMRI investigation explored how patients process internal bodily signals, focusing on cardiac interoception (Sojka et al., 2024). During a heartbeat-counting task, individuals with FND exhibited reduced activity in key salience network nodes, specifically the right anterior insula, dorsal anterior cingulate cortex, and temporoparietal junction (TPJ) when attention was oriented toward interoceptive sensations. Notably, this hypoactivation was linked to poorer interoceptive accuracy and to deviations in the interoceptive trait prediction error (ITPE), which quantifies the mismatch between one's perceived interoceptive ability and actual performance (Koreki et al., 2020; Sojka et al., 2020). In parallel, lower vmPFC activity associated with heightened ITPE may signify an impaired capacity to attribute appropriate significance to bodily signals, a mechanism potentially bridging interoceptive dysfunction and disturbances of agency (Sojka et al., 2024). This overlap is conceptually significant, as precise monitoring of internal states is fundamental for adjusting motor output and for sustaining a coherent sense of agency (J. W. Moore, 2016; Wen et al., 2021).

Indeed, a substantial body of evidence now supports the presence of altered sense of agency in FND. In a fascinating experiment, Nahab and colleagues (2017) employed a cyber-glove paradigm that manipulated perceived control over hand movements, inducing a mismatch between participants' intended actions and the visual feedback they received. Whereas healthy controls showed flexible modulation of the right TPJ activity in response to this manipulation, patients with FND did not, indicating a breakdown in the integration of sensory and motor information required for accurate agency attribution. Consistent results emerged from Baek et al. (2017), who used a Libet clock paradigm to demonstrate similar disturbances in the neural correlates of voluntary action awareness, further corroborating the notion of impaired agency processing in FND.

Beyond task-based approaches, resting-state connectivity studies have deepened our understanding of TPJ involvement in the disorder. Maurer and colleagues (2016) reported reduced coupling between the right TPJ and somatomotor areas, suggesting a disruption in the communication between sensorimotor systems and higher-order integrative processes essential for constructing a coherent sense of agency. Conversely, more recent graph-theoretical analyses revealed increased centrality of the left TPJ in patients with functional weakness (K. Mueller et al., 2022), a finding possibly reflecting compensatory neural reorganization or a hemispheric imbalance in TPJ functioning. Together, these results delineate a complex and dynamic alteration of the neural mechanisms supporting agency in FND.

Apart from descriptive or correlational evidence, recent neuromodulation studies have begun to explore the causal involvement of specific neural circuits in FND. In one such investigation, Bühler (2024) applied excitatory transcranial magnetic stimulation (TMS) over the right TPJ, previously shown to exhibit hypoactivation during agency-related tasks. Stimulation led to a temporary normalization of TPJ activity, suggesting that the aberrant neural dynamics underlying agency disturbances can be experimentally modulated. Although this enhancement of TPJ responsiveness did not translate into immediate symptomatic improvement – and likely depends on broader network-level interactions to yield therapeutic effects – the findings provided direct evidence that neuromodulatory interventions can influence core mechanisms implicated in FND.

The findings presented in this section emerge from methodologically diverse approaches. However, some caution is warranted when interpreting task-based results. Experimental paradigms such as motor imagery, agency-judgment tasks, or heartbeat counting rely heavily on introspection and attention, and may therefore be influenced by additional cognitive or non-specific processes, potentially confounding the interpretation of disorder-specific neural alterations (Desmedt et al., 2018). Moreover, several studies involve relatively small clinical samples, a common feature of FND research that largely reflects the practical challenges of recruiting and carefully phenotyping this population, and which may limit statistical power and generalizability. Nevertheless, the convergence of results across different tasks, modalities, and analytic strategies provides increasing confidence that the reported alterations reflect genuine network-level dysfunction rather than isolated methodological artefacts.

Taken together, these results reinforce a paradigm shift in understanding FND: rather than reflecting localized structural pathology, the disorder emerges from dysfunctional coordination among distributed brain networks. Early neuroimaging studies laid the foundation for this conceptual transition, and contemporary approaches have since emphasized the dynamic coupling between circuits subserving motor control, emotion regulation, and self-monitoring. Dysregulation within top-down control pathways, particularly those encompassing the vmPFC, DLPFC, and SMA, appears pivotal to the disorder's neural architecture. Concurrently, disturbances in agency-related processing, involving the TPJ and insula, likely interact with these executive–motor circuits, shaping the characteristic motor phenomena observed in patients with FND.

2.2 Emotion processing in FND

Although early neuroimaging investigations in FND predominantly centered on motor control, more recent work has increasingly examined the neural substrates of emotional processing. This evolution in focus partly reflects the enduring influence of psychogenic theories that emphasize the role of affective dysregulation in symptom expression. At the same time, it broadens the theoretical landscape by revealing additional mechanisms and interactions beyond traditional psychosomatic models.

A seminal contribution in this line of research came from Voon (2010), who explored amygdala reactivity to emotional facial expressions. In that study, patients with FND displayed greater amygdala activation than healthy controls when viewing happy faces, whereas responses to fearful expressions did not differ significantly between groups. These findings point to a generalized hyper-responsiveness of the amygdala in FND, reflecting an amplified emotional arousal that can be triggered even by stimuli of neutral or positive valence.

An important extension of research on emotional processing in FND was introduced by Aybek and colleagues (2015), who expanded upon previous paradigms by including sad facial expressions. Their findings revealed a sustained and even progressively increasing activation of the amygdala in patients, indicative of persistent hyperarousal, in contrast to the habituation observed in healthy controls. Moreover, patients exhibited enhanced activation of the periaqueductal gray (PAG), a midbrain structure implicated in interoceptive and defensive processes, particularly in regulating respiratory and autonomic components of the freeze response (Aybek et al., 2015). These results underscore the importance of further investigating automatic defense mechanisms and their neurobiological underpinnings in FND.

Additional support for limbic involvement comes from an fMRI study examining cognitive reappraisal, a key process in emotion regulation that involves reinterpreting the meaning of an emotional stimulus or situation in order to modify its affective impact (Hassa et al., 2021). FND patients displayed exaggerated right amygdala responses during attempts to reappraise negative stimuli, reinforcing the notion of a hyperreactive limbic system underlying emotional dysregulation in this population.

Converging evidence also implicates the insula, a hub integrating interoceptive and affective information (Fermin et al., 2022; Simmons et al., 2013). In an emotion regulation paradigm, Sojka and colleagues (2019) observed reduced insular activation in FND patients, which correlated inversely with alexithymia scores, suggesting that individuals with greater difficulty identifying and articulating emotions showed the strongest neural hypoactivation. This association highlights a potential mechanistic bridge between altered interoceptive awareness and impaired emotion regulation.

Altogether, these studies indicate that beyond motor disturbances, FND involves marked alterations in limbic and interoceptive circuits (including the amygdala, PAG, and insula), supporting the view that emotional dysregulation and disrupted bodily self-awareness may constitute core features of the disorder's neurobiological profile.

2.3 Static and dynamic functional connectivity studies

The transition from asking "where is the lesion?" to "what is the nature of the lesion?" has marked a conceptual and methodological turning point in FND research. Neuroimaging studies have progressively moved beyond task-based localization to embrace network-level analyses, reflecting a broader systems neuroscience perspective. Resting-state fMRI investigations employing static functional connectivity approaches have been particularly informative, quantifying the average temporal coherence of blood-oxygen-level-dependent (BOLD) signals across brain regions (Smitha et al., 2017; van den Heuvel & Hulshoff Pol, 2010). These studies consistently report alterations within large-scale networks: for instance, decreased coupling between key hubs of the DMN, such as the posterior cingulate and medial prefrontal cortices, and subcortical structures involved in motor and affective processing. Similarly, disrupted connectivity within the salience network, notably involving the anterior insula and dorsal anterior cingulate cortex, suggests atypical coordination among systems mediating emotion, attention, and motor control (Diez et al., 2021; Rai et al., 2022).

More recent work has advanced this framework by adopting dynamic functional connectivity approaches, which capture the time-varying nature of neural interactions rather than assuming their stability throughout the scan. One such method involves identifying co-activation patterns (CAPs): transient, recurring configurations of synchronized regional activity (X. Liu et al., 2018). In FND, Weber, Bühler, and colleagues (2024) demonstrated that specific CAPs differ from those observed in healthy controls both in frequency and duration. Several of these patterns showed enhanced co-activation among salience-related regions (e.g., anterior insula, dorsal anterior cingulate cortex) and sensorimotor cortices, possibly reflecting an abnormal bias toward motor initiation or inhibition. Furthermore, some CAP alterations correlated with salivary amylase levels and symptom duration, implying that patients may persist in certain connectivity states that modulate emotional arousal or interfere with consistent motor control (Weber, Bühler, et al., 2024). This constellation of findings has been interpreted as evidence of reduced network flexibility, a phenomenon that could help explain the fluctuating and episodic presentation of FND symptoms.

While the direct clinical implications of these network-level abnormalities remain to be fully elucidated, the shift toward network and temporal dynamics represents a major evolution in the field, away from searching for localized dysfunction and toward understanding how disrupted interactions among large-scale neural systems give rise to the characteristic phenomenology of FND.

2.4 Structural neuroimaging studies

Although the recognition of FND as a *dynamic*, network-level disorder might seem to render the search for structural abnormalities paradoxical, structural neuroimaging has nonetheless revealed consistent anatomical differences between affected individuals and healthy controls. These findings suggest that alterations in gray and white matter architecture may contribute to the vulnerability and expression of FND symptoms.

Voxel-based morphometry (VBM) studies have identified several gray matter volume (GMV) deviations in FND populations. Maurer (2016) reported increased GMV in the left amygdala, caudate, putamen, cerebellum, fusiform gyrus, and bilateral thalami, accompanied by reduced GMV in the left sensorimotor cortex. Given the involvement of these structures in emotion regulation and motor control, such alterations could plausibly underpin the functional disturbances characteristic of the disorder. Importantly, these volumetric changes did not correlate with measures of anxiety or depression, indicating that they may reflect FND-specific neural traits rather than secondary effects of psychiatric comorbidity.

Complementary evidence from developmental samples points to a similar pattern. In a pediatric study, Kozlowska (2017) observed enlarged GMV in the left SMA, right superior temporal gyrus (STG), and dorsomedial prefrontal cortex (DMPFC) among children and adolescents with FND. The SMA, particularly its pre-SMA subdivision, plays a critical role in motor planning and in integrating cognitive and emotional information (de la Peña et al., 2020; Ruan et al., 2018), suggesting that structural enlargement in this region may influence the coordination of voluntary movement. Moreover, increased volumes in the SMA and DMPFC were linked to faster emotion-identification performance, hinting at a structural–functional coupling between emotional awareness and motor regulation in younger patients.

With regard to white matter organization, diffusion tensor imaging (DTI) studies have provided compelling evidence of microstructural alterations in FND. Although early work reported increased fractional anisotropy (FA) within the left uncinate fasciculus in patients with psychogenic non-epileptic seizures (S. Lee et al., 2015), subsequent research has revealed a broader pattern of predominantly reduced FA across multiple fronto-limbic pathways. Diez and colleagues (2019) documented widespread decreases in FA involving the stria terminalis/fornix, medial forebrain bundle, extreme capsule, uncinate fasciculus, cingulum bundle, corpus callosum, and striatal–postcentral projections, tracts critically implicated in salience detection, emotion regulation, and defensive behaviors (D. L. Perez et al., 2017). Moreover, reduced FA within the stria terminalis/fornix and medial forebrain bundle correlated with greater physical disability and longer illness duration, suggesting that disruptions in white matter integrity may parallel disease chronicity and symptom severity. A follow-up study employing probabilistic tractography confirmed these widespread alterations, including further reductions in the uncinate fasciculus, thereby reinforcing the view that impaired limbic–prefrontal connectivity constitutes a structural hallmark of FND (Diez et al., 2021).

Taken together, these findings underscore that even within a “dynamic” or network-based conception of FND, discernible structural variations are present. However, interpreting such differences requires careful distinction between *trait* and *state* markers. Trait markers may index predisposing vulnerabilities that precede symptom onset, whereas state markers are more likely to reflect plastic changes secondary to symptom manifestation. For example, several studies reported increased GMV in the amygdala and premotor regions (Kozlowska, Griffiths, et al., 2017; Maurer et al., 2018; D. L. Perez et al., 2017), which could be interpreted as predisposing traits, although this hypothesis remains unproven. More recent evidence, conversely, suggests reduced bilateral amygdala volume as a potential trait marker in FND (Weber et al., 2023). Such discrepancies may arise from developmental and experiential factors, particularly the amygdala's sensitivity to early life stress—a common antecedent in FND populations (L. Ludwig et al., 2018). Early adversity is known to shape amygdala morphology through cortisol-mediated mechanisms (Hanson & Nacewicz, 2021; Khoury et al., 2023). Specifically, while childhood trauma has been associated with transient amygdala hypertrophy (Evans et al., 2016; Pechtel et al., 2014), longitudinal studies indicate that chronic or repeated stress exposure may lead to volumetric reductions in adulthood, depending on the developmental timing and nature of the stressor (Hallett, 2024; Korgaonkar et al., 2013).

Overall, these converging lines of evidence suggest that structural changes in FND likely emerge from a complex interplay between early vulnerability factors, stress-related neurodevelopmental trajectories, and ongoing network-level dysfunctions that shape both symptom onset and maintenance.

White matter abnormalities, most notably reductions in FA within limbic-prefrontal pathways such as the uncinate fasciculus and stria terminalis, may represent *state-dependent* neural markers in FND. Their correlation with illness duration and the degree of functional impairment suggests that these alterations reflect dynamic, experience-driven neuroplastic processes that evolve alongside symptom progression and may be amenable to modification through targeted therapeutic interventions (C. Mueller & Szaflarski, 2023).

2.5 Diagnostic biomarkers

Although clinical signs currently used to FND already demonstrate high diagnostic reliability (Aybek & Perez, 2022; Espay et al., 2018; Stone et al., 2020), the integration of biological indicators could, in the near future, contribute to refining diagnostic accuracy. In this regard, resting-state functional MRI (rs-fMRI) combined with machine learning approaches has gained attention as a potential avenue for identifying objective neurobiological correlates that complement the precision of conventional clinical assessment (Arbabshirani et al., 2017). By training algorithms on extensive datasets, it becomes possible to extract distinctive connectivity "fingerprints" that reflect individual patterns of dysfunction, thereby allowing for the delineation of network-level signatures associated with specific symptom profiles (Drysdale et al., 2017; Finn et al., 2015).

Initial studies employing rs-fMRI–based classifiers in FND have yielded encouraging results. In a pioneering investigation, Wegrzyk and colleagues (2018) analyzed whole-brain resting-state data from a small cohort of patients and matched controls using an atlas-based functional connectivity approach. A support vector machine (SVM) algorithm trained on these data achieved a classification accuracy of over 68% ($p = 0.004$), successfully distinguishing FND participants from healthy individuals. The most discriminative features involved connections within the right caudate, amygdala, prefrontal, and sensorimotor regions. Follow-up analyses indicated that these areas displayed hyperconnectivity in patients, pointing to identifiable neural signatures even when using relatively small samples.

Despite limitations related to sample size and single-site acquisition, this early study provided a compelling proof of concept: resting-state connectivity patterns, when analyzed through machine learning, can capture disease-relevant alterations in brain networks and may thus serve as preliminary objective markers of FND pathophysiology (Bègue et al., 2019; Wegrzyk et al., 2018).

Building on these early results, a subsequent multicenter investigation broadened both the scope and generalizability of findings by acquiring rs-fMRI data from FND patients and healthy controls across four centers located in Switzerland, the Netherlands, and the Czech Republic (Weber et al., 2022). Using a multivariate machine learning framework applied to whole-brain functional connectivity, the study achieved accuracies ranging between 70% and 73% within individual sites. When data from all centers were combined in a pooled cross-validation scheme, the overall classification accuracy reached 71.5% in distinguishing patients from controls. The most discriminative features were localized in the angular and supramarginal gyri, the sensorimotor and cingulate cortices, as well as the insular and hippocampal regions. Nonetheless, cross-site validation sometimes yielded results at chance level, underscoring the persistent challenge of replicating classifier performance across heterogeneous scanning environments and populations (Bayer et al., 2022; Weber et al., 2022).

A related investigation focusing specifically on functional movement disorders (FMD) further substantiated these findings through a more targeted machine learning strategy (Waugh et al., 2023). In that study, 66 seed regions identified from prior literature were used to compute a full correlation matrix. Recursive feature elimination isolated the most predictive connections, which were subsequently entered into an SVM classifier achieving an accuracy of 80%. The strongest discriminative features involved connectivity between regions associated with emotion regulation, reward circuitry, and sensorimotor integration (networks consistently implicated in the pathophysiology of FMD), suggesting that distinct alterations in functional coupling may constitute a neurobiological signature of the disorder.

Expanding upon this functional framework, Westlin and colleagues (2024) incorporated structural MRI metrics alongside functional connectivity measures, thereby highlighting the interaction between anatomical and functional substrates in FND. In patients with mixed FND presentations, classification accuracy reached 66% ($p = 0.005$) against healthy controls and 60% ($p = 0.038$) against psychiatric controls. The cingulate gyrus, hippocampal subfields, and amygdala emerged as the main contributors to these classifications. Within the FMD subgroup, the classifier effectively distinguished patients from healthy controls with 72% accuracy ($p = 0.002$), though it failed to reliably separate them from psychiatric controls. Likewise, functional seizure subtypes could not be robustly differentiated from either control group, emphasizing the diagnostic complexity inherent to the disorder's heterogeneity.

Adding a complementary dimension, other scholars examined voxel-wise variability in BOLD signal as a potential marker of dynamic neural processes in FND (A. Schneider et al., 2024). Patients displayed increased signal variability across several key networks (i.e. somatomotor, salience, limbic, and dorsal attention) compared to healthy individuals. Longitudinal data collected from 47 patients over an eight-month interval revealed that greater variability within the SMA correlated with clinical improvement, suggesting that SMA fluctuations may index adaptive neural plasticity and function as a state biomarker. In contrast, higher baseline variability in the left insula predicted poorer outcomes, indicating a potential trait-like marker of vulnerability. Together, these findings point toward the relevance of dynamic neural variability as both a prognostic and state-dependent indicator of functional change.

In keeping with this integrative perspective, Gandolfi (2024) proposed a diagnostic framework that combines behavioral, neurophysiological, and MRI-derived indices across multiple domains, including motor, exteroceptive, and interoceptive processes, to refine both diagnostic specificity and prognostic assessment.

Despite these methodological advances, translating resting-state classifiers into clinical tools remains an ongoing challenge. The heterogeneity of FND manifestations, variability in scanning and preprocessing pipelines, and the influence of comorbid psychiatric factors all complicate replication and standardization. Future progress will likely depend on the establishment of large-scale, harmonized consortia and open-data initiatives capable of unifying acquisition protocols, expanding sample diversity, and enhancing the reproducibility of machine-learning–based biomarkers in FND.

2.6 Neurobiological, genetic and epigenetic contributions

The genetic foundations of FND remain only partially understood, with relatively few investigations addressing heritability or identifying specific gene variants associated with the condition. To date, no single genetic locus has been conclusively linked to FND; however, several polymorphisms in genes implicated in neurotransmission, stress regulation, and neurodevelopment have been tentatively associated with vulnerability to the disorder.

For example, the G-allele of the rs1800629 polymorphism in the tumor necrosis factor-alpha (TNF-α) gene has been reported to occur more frequently in healthy individuals than in those with somatoform disorders, suggesting a possible protective role for this variant (Harms et al., 2013). Conversely, studies examining genes involved in monoaminergic metabolism—including tryptophan hydroxylase and the catechol-O-methyltransferase (COMT) Val158Met polymorphism—have not observed significant allele frequency differences between patients with conversion disorder and healthy controls (Armagan et al., 2013; Jakobi et al., 2010).

Further evidence has emerged from research on oxytocin-related genetic regulation, which may provide a molecular link between stress responsiveness and social-affective processing in FND. Following an initial report of increased methylation of the oxytocin receptor (OXTR) gene in patients (Apazoglou et al., 2018), subsequent investigations explored the rs53576 polymorphism, a variant previously implicated in social cognition and emotional regulation (Bahji et al., 2021; Cataldo et al., 2018). Recent data indicate that individuals with FND show elevated OXTR methylation, which correlates positively with higher salivary oxytocin concentrations (Weber, Stoffel, et al., 2024). This inverse relationship may reflect a compensatory mechanism, whereby reduced OXTR expression prompts increased oxytocin release to preserve homeostatic balance. Given the strong association between oxytocin signaling and limbic circuitry, particularly the amygdala and hippocampus (Quintana et al., 2019; Triana-Del Rio et al., 2022), and the observation of altered amygdalar connectivity patterns linked to oxytocin levels in FND (Weber, Stoffel, et al., 2024), these findings support the hypothesis that dysregulation of the oxytocinergic system contributes to the emotional and affective disturbances frequently observed in the disorder.

Beyond single-gene associations, gene-environment interactions are increasingly recognized as pivotal in shaping susceptibility to neuropsychiatric conditions (Hollander et al., 2020; H. Zhang et al., 2022), FND included. Epigenetic processes such as DNA methylation and histone acetylation can induce persistent alterations in gene expression without modifying the underlying DNA sequence (Bannister & Kouzarides, 2011; Cusack et al., 2020; L. D. Moore et al., 2013), thereby mediating the biological consequences of early-life adversity on brain function (Cánepa & Berardino, 2024; Kundakovic et al., 2015; Rahman & McGowan, 2022). Among the best-characterized examples, methylation of the glucocorticoid receptor gene (NR3C1) has been linked to childhood trauma and smaller hippocampal volume (Rahman & McGowan, 2022), while hypermethylation of the serotonin transporter gene (SLC6A4) has been associated with reduced serotonergic signaling in the orbitofrontal cortex, a process potentially underlying emotional and interoceptive dysregulation in FND (H. Zhang et al., 2022).

The serotonergic system itself appears central to FND pathophysiology. Variations in serotonin-related genes may modulate both symptom severity and neurobiological phenotype. Notably, polymorphisms in the tryptophan hydroxylase 2 (TPH2) gene, a key enzyme in serotonin synthesis, have been associated with distinct patterns of limbic–frontal functional connectivity and greater clinical severity in patients with functional movement disorders (Spagnolo et al., 2020). Similarly, the rs1800532 polymorphism in TPH1 has been linked to symptom burden, with carriers of the T allele showing more severe manifestations and poorer outcomes over an eight-month follow-up period (Weber, Rey Álvarez, et al., 2024). Importantly, this same study identified an interaction between TPH2 (rs4570625) and OXTR (rs2254298) variants, pointing to a functional interplay between serotonergic and oxytocinergic signaling pathways in determining symptom expression and disease progression. These converging findings echo broader evidence implicating serotonin dysregulation in stress-related disorders and highlight its potential role in the neurobiological framework of FND.

Although research into the genetic and epigenetic architecture of FND remains in its infancy, the available data underscore the necessity of a multifactorial model integrating genetic susceptibility, environmental exposures, and functional neurobiological changes. Progress in this area will depend on studies employing larger cohorts and multi-omic, integrative approaches to clarify how these molecular mechanisms converge to shape the onset and course of the disorder.

2.7 Contributions from electrophysiological studies

From the earliest days of human electrophysiology, EEG offered one of the first means to directly observe brain activity, and as early as the 1960s and 1970s it was employed to study patients historically diagnosed with hysterical conversion. These early investigations can be broadly grouped into two aims: some sought to demonstrate that primary sensory and motor functions remained electrophysiologically intact despite patients' reported losses, while others attempted to uncover subtle functional anomalies that might correspond to the clinical symptoms.

Across several sensory modalities, studies consistently showed that elementary evoked responses remained normal in FND. Somatosensory evoked potentials (SEPs), visual evoked potentials (VEPs), and brainstem auditory evoked potentials typically displayed preserved amplitudes and latencies even when patients reported anesthesia, blindness, or deafness (Drake, 1990; Howard & Dorfman, 1986), confirming the structural and functional integrity of the primary sensory pathways. Magnetoencephalography (MEG) provided further evidence: in patients with functional sensory loss, Hoechstetter (2002) observed normal activation patterns in both primary (SI) and secondary (SII) somatosensory cortices (contralateral and ipsilateral to the affected side), closely resembling those of healthy participants. Indeed, some responses appeared even enhanced, suggesting paradoxical amplification of cortical reactivity rather than suppression, in line with earlier EEG reports showing exaggerated tactile evoked responses during hysterical anesthesia (Moldofsky & England, 1975).

These findings challenged early theories positing that conversion symptoms arise from attentional "gating" mechanisms that block sensory signals from reaching conscious awareness (Ludwig, 1972; Vuilleumier et al., 2001). Instead, they indicated that low-level cortical processing remains unaffected.

Nonetheless, more refined paradigms revealed subtle abnormalities at higher processing stages. For example, Levy and Mushin (1973) found that tactile stimuli presented near perceptual threshold sometimes failed to elicit normal SEPs in patients with sensory conversion symptoms, even though suprathreshold stimuli evoked typical responses. Abnormal habituation patterns were also described: unlike healthy individuals or anxious controls, patients with conversion symptoms did not show the expected reduction in SEP amplitude or skin conductance response across repeated stimuli, instead reacting to frequent inputs as though they were novel (Horvath et al., 1980; Moldofsky & England, 1975).

Event-related potential (ERP) studies in patients with functional symptoms have, in turn, highlighted some evidence of altered EEG components at later stages of sensory and motor processing. For instance, in a single-case EEG study using an "oddball" paradigm to elicit the P300 component (a marker of attentional orienting), the authors compared responses to deviant tactile stimuli applied to the affected and unaffected hands of a patient with functional anesthesia (Lorenz et al., 1998). While early SEPs were normal on both sides, the P300 was absent for deviant stimuli on the anesthetized hand, indicating intact sensory transmission but disrupted higher-level processing of stimulus significance. Crucially, when a healthy control was asked to simulate anesthesia and to deliberately ignore stimuli, the P300 remained intact, suggesting that the reduction observed in the patient could not be explained by voluntary suppression.

In visual and auditory modalities, higher-order electrophysiological alterations have been less frequently explored. Still, patients with functional blindness exhibited reduced-amplitude P300 responses to unreported visual stimuli despite preserved early potentials (Towle et al., 1985), and individuals with functional deafness showed diminished auditory P300 with intact earlier N100 and N200 components (Fukuda et al., 1996). Abnormally decreased auditory mismatch negativity (MMN) responses have also been described in somatization disorders (James et al., 1989), suggesting disruptions in automatic pre-attentive processing (Choi et al., 2015; Näätänen et al., 2014). Interestingly, more recent evidence has begun to challenge the idea that functional neurological disorders are invariably associated with attenuated late-cognitive components. In a study using an auditory oddball paradigm, it has been reported that children and adolescents with FND displayed increased amplitudes across several event-related potential components, including P50, N100, P200, N200, and P300, compared to matched healthy controls (Kozlowska, Melkonian, et al., 2017). The authors interpreted this pattern as reflecting a state of generalized cortical hyperarousal (as a result of cascades of stress-induced changes) rather than a deficit in cognitive engagement or mere alterations in sensory gating (Hermans et al., 2011; Kozlowska et al., 2011).

Concerning motor processing, a study in patients with upper limb conversion paresis showed by contrast an enlarged P300 component in response to cues for the symptomatic limb in a reaction time task, alongside the attenuation of earlier components such as N100 (Blakemore et al., 2013). This combination appears consistent with diminished conscious motor awareness related to active suppression of brain circuits involved in the attribution of self-agency, despite preserved early sensory-motor transmission (Blakemore et al., 2013; Voon, 2010).

Motor conversion symptoms have similarly been examined with transcranial magnetic stimulation (TMS). During motor imagery tasks, patients with functional paresis have been found to exhibit attenuated MEPs compared to healthy controls (Liepert et al., 2011). This pattern of diminished corticospinal excitability was interpreted as a possible electrophysiological correlate of their compromised capacity to initiate or sustain voluntary movements; however, reduced MEPs are not specific for FND (Liepert et al., 2008; Meyer et al., 1992).

In more recent years, EEG investigations in FND have gradually shifted from traditional time-locked analyses toward frequency-based and network-oriented approaches. The following evidence, however, pertains specifically to patients with functional seizures (FS), representing one of the most extensively studied FND subtypes within this electrophysiological framework. Quantitative EEG studies seems to indicate that oscillatory brain activity is not entirely preserved: several research groups have reported increased beta-band power (≈13-30 Hz) over fronto-parietal and sensorimotor regions, both during rest and in the peri-symptomatic phase in patients with FS (Arıkan et al., 2021; Meppelink et al., 2017; Pastötter et al., 2024). This phenomenon recalls the event-related desynchronization typically observed before voluntary movements, and may represent a maladaptive form of motor anticipation, potentially influenced by dopaminergic modulation (Jenkinson & Brown, 2011; Kannan et al., 2025).

Alterations in gamma-band activity (≈30-100 Hz) have also emerged as a salient electrophysiological feature in FS, pointing to disturbances in higher-order integrative processes. Increased gamma power in left parietal regions has been interpreted as a sign of excessive or dysregulated sensorimotor processing (Arıkan et al., 2021), possibly reflecting the overactivation of neural circuits involved in body representation and movement preparation. Such hyperexcitability may correspond to a heightened readiness for action in the absence of overt movement, echoing the paradoxical mixture of volition and inhibition that characterizes FS. Conversely, other studies have described marked reductions in gamma activity, particularly in the right superior temporal gyrus (Umesh et al., 2017) and in long-range connectivity between frontal and posterior cortical regions (Xue et al., 2013). Instability within prefrontal networks may impair executive regulation of motor output, consistent with evidence that frontal dysfunction represents a core mechanism underlying PNES and related conversion disorders (Knyazeva et al., 2011). Taken together, the inconsistent findings on gamma activity (regional hyperactivation versus inter-regional decoupling) suggest that alterations in this frequency range are not uniform across studies, possibly reflecting heterogeneous disruptions in the balance between local synchronization and large-scale network integration.

Disturbances in alpha-band connectivity (≈8-12 Hz) further highlight the network-level dysregulation characteristic of FS. Functional coupling within and between basal ganglia, limbic areas, and widespread cortical regions, including prefrontal, temporal, parietal, and occipital cortices, appears significantly altered compared with healthy controls (Barzegaran et al., 2016). These abnormalities indicate that FS may not be confined to isolated cortical dysfunctions but rather involve aberrant coordination across subcortico–cortical loops responsible for the regulation of motor readiness, emotion, and attention. Graph-theoretical analyses have refined this picture, revealing that the degree of network inefficiency in the alpha-band correlates with the frequency of monthly seizure episodes, and thus suggesting that the chronicity and severity of FS may reflect a progressive loss of optimal network organization (Barzegaran et al., 2012). Such findings underscore the importance of viewing FS as disorders of large-scale connectivity, in which impaired communication between key hubs compromises the flexible coordination of cognitive, emotional, and motor functions that normally sustain bodily self-agency.

Comparable alterations have also been described in pediatric FND. In children, increased theta, delta, and gamma activity has been observed within the default-mode, premotor, and salience networks, in association with abnormal patterns of arousal, pain processing, and motor-sensory integration (Kozlowska et al., 2018; Kozlowska & Scher, 2024; Radmanesh et al., 2020; Umesh et al., 2017).

Finally, in the context of seizure-like manifestations, recent EEG spectral and machine-learning studies have successfully differentiated functional (psychogenic) seizures from epileptic events based on characteristic profiles of band-limited power and signal complexity, underscoring the presence of a distinctive oscillatory signature in functional attacks (Fussner et al., 2024; Hinchliffe et al., 2024; Shokouh Alaei et al., 2025).

Overall, these findings traced the gradual evolution of electrophysiological research in FND, from early efforts demonstrating preserved primary sensory and motor responses to more recent evidence of subtle but widespread disruptions in cortical communication. Contemporary EEG and MEG data suggest that the disorder does not reflect a simple cortical failure, but rather an alteration in how distributed networks synchronize and exchange information across perceptual, salience, and motor systems – domains intimately tied to attention, and the sense of agency. However, the current body of evidence remains highly fragmented. Most studies have been conducted on specific phenotypes (most notably FS) and findings across frequency bands or analytical approaches are far from consistent. This heterogeneity likely mirrors the clinical and neurobiological diversity of FND itself. Despite increasing methodological sophistication, so far no reproducible EEG signature or biomarker has yet been identified that can reliably discriminate FND from other neurological or psychiatric conditions, or capture its full phenomenological spectrum.

Study 1: Uncovering altered neural dynamics in FND with resting state microstates

Introduction

As we have outlined, recent theoretical advances have reframed FND from a diagnosis of exclusion to a disorder rooted in the dynamic dysfunction of brain networks, implicating alterations in domains spanning motor control, emotional regulation, and the sense of agency (Aybek & Perez, 2022; Edwards et al., 2012; Edwards & Bhatia, 2012). Notably, growing evidence highlights the centrality of disrupted sensory processing and impaired integration of somatosensory information in the pathophysiology of FND (Diez et al., 2019; MacLean et al., 2022; Ranford et al., 2020; Rossi et al., 2025; Weissbach et al., 2023).

Electroencephalography offers a non-invasive window into the temporal dynamics of brain activity with high temporal resolution. However, conventional EEG analyses (e.g. spectral power measures) often provide only a static or averaged perspective, potentially overlooking the rapid and transient reconfigurations of large-scale neural networks that underpin complex behaviors and subjective experiences (Chivu et al., 2024; Khanna et al., 2015; Li et al., 2020). In this context, EEG microstate analysis has emerged as a transformative approach. By segmenting the continuous EEG signal into a sequence of brief, quasi-stable topographical patterns, so called “microstates”, this method enables the investigation of the brain’s millisecond-level functional states and their temporal organization (Michel & Koenig, 2018; Van de Ville et al., 2010). Remarkably, simultaneous EEG-fMRI studies have shown that these fleeting microstates correspond to several canonical resting-state networks, albeit unfolding at a far finer temporal scale than the sluggish networks observable with fMRI (Custo et al., 2017). In light of such findings, microstates have even been metaphorically described as the brain’s “atoms of thought” (i.e. the smallest constituting elements of cognition) reflecting how each microstate may encapsulate an elementary building block of mentation (Lehmann, 1990).

Beyond their purely topographical definition, the canonical microstates (typically labeled A through G in recent literature) have been consistently associated with distinct functional roles (Tarailis et al., 2024). For instance, microstate A is linked to arousal and auditory processing; microstate B to visual perception, self-visualization, and autobiographical memory; microstate C to mind-wandering and self-referential thought; microstate D to executive functioning (e.g. working memory); microstate E to interoceptive and emotional processing (associated with the salience network); microstate F to the processing of personally significant information (as part of the default mode network); and microstate G to somatosensory network activity (Milz et al., 2016; Tarailis et al., 2024). Unlike time-averaged EEG metrics, microstate analysis captures the brain's fleeting transitions and the dynamic interplay between neural networks, offering a more nuanced, in vivo portrayal of brain function in both health and disease (Asha et al., 2024; Schiller et al., 2024; Vass et al., 2025). This dynamic, network-oriented perspective has opened new frontiers in research, positioning microstate metrics as promising neurophysiological markers for a variety of brain disorders , which often show disorder-specific shifts in microstate dynamics: for example, imbalances in the prevalence or duration of particular microstate classes suggest that each illness leaves its own "fingerprint" in the temporal coordination of large-scale brain networks. (de Bock et al., 2020; Kim et al., 2021).

Indeed, aberrant microstate patterns have been documented across numerous neurological and psychiatric conditions. Characteristic alterations in microstate parameters are reported in schizophrenia, mood and anxiety disorders, attention-deficit/hyperactivity disorder (ADHD), autism spectrum disorder, and others (Chivu et al., 2024; Das et al., 2022; Iftimovici et al., 2023).

In schizophrenia, for instance, patients consistently exhibit an overrepresentation of microstate class C alongside a marked reduction of class D, a disturbance so pronounced that the abnormally short duration of microstate D has been directly linked to the occurrence of auditory verbal hallucinations (da Cruz et al., 2020; Rieger et al., 2016; Q. Sun et al., 2022). Likewise, individuals with major depression and anxiety disorders have shown altered microstate duration times and transition patterns compared to healthy controls (Chivu et al., 2024; M. Murphy et al., 2020; Yan et al., 2021), reflecting the pervasive impact of these disorders on the brain's spontaneous activity flow. Importantly, microstate metrics such as their average durations, frequencies of occurrence, and fractional coverage display high test–retest reliability over both short and long intervals (Antonova et al., 2022; Kleinert et al., 2024; J. Liu et al., 2020). This trait-like stability reinforces the notion that microstates can serve as reliable biomarkers of underlying neural network organization, which is a compelling feature not only for research but also for clinical applications.

Nevertheless, to date no studies have specifically applied microstate analysis to FND. Considering that FND is increasingly conceptualized not as a focal neurological deficit but as a disorder of large-scale brain network organization, microstate analysis may offer a particularly suitable lens for capturing these dynamic alterations. The present study addresses this gap by examining resting-state EEG microstates to investigate temporal brain dynamics in individuals with FND, compared against matched healthy controls. Building on previous evidence of altered large-scale network dynamics and atypical sensorimotor and interoceptive processing in FND, we hypothesized that patients might show differences in the temporal organization of resting-state microstates compared with healthy controls. We additionally explored whether these temporal characteristics might relate to clinical symptom severity. In contrast, analyses of transition probabilities and the overall sequential organization of microstates were considered exploratory, as no specific directional predictions were formulated for these measures.

Methods

Participants

A total of ninety-two adults participated in the study, including 45 individuals diagnosed with FND and 47 healthy controls (HC). Following data quality control, six FND participants were excluded: two due to symptom exacerbation that prevented task completion, two for excessive EEG artefacts, one because of technical malfunction, and one identified as a statistical outlier in the microstate dataset. The final sample thus comprised 39 patients with FND (29 female; mean age = 38.5 ± 11.6 years) and 47 healthy controls (34 female; mean age = 37.8 ± 13.1 years). FND participants were recruited through the Clinical Neurology Unit of the Cantonal Hospital Fribourg and the Psychosomatic Medicine Unit of Inselspital Bern, while healthy controls were enrolled via community flyers, word of mouth, and online advertisements. Eligibility required participants to be over 18 years of age, cognitively able to provide informed consent, and where applicable, to meet DSM-5 and ICD-11 diagnostic criteria for FND (ICD-11 codes F44.4–F44.7), encompassing motor, cognitive, or mixed symptom profiles.

Exclusion criteria included a history of major psychiatric disorders, neurological surgery or the presence of implants, substance abuse, inability to comply with study procedures, or pregnancy. The clinical severity of functional motor symptoms was quantified using the Simplified Functional Movement Disorder Rating Scale (S-FMDRS; Nielsen et al., 2017).

The sample analyzed here forms part of a broader research project (Stoffel et al., 2025) conducted at the University of Fribourg and received ethical approval from the Ethics Committee of the Canton of Bern, registered on ClinicalTrials.gov (identifier NCT06084325), and was therefore not determined by an a priori power analysis specific to the present microstate outcomes.

EEG acquisition and preprocessing

Resting-state EEG recordings were obtained using a 64-channel Biosemi ActiveTwo system (BioSemi, Amsterdam, Netherlands), sampled at 2048 Hz. Each session lasted six minutes and consisted of alternating 20-second eyes-open and 40-second eyes-closed periods, designed to limit fluctuations in arousal and maintain a stable vigilance level (Baumgartner et al., 2019; Schiller et al., 2019). For the present analyses, only the eyes-closed segments (amounting to approximately five minutes of artifact-free data) were retained.

Preprocessing was conducted with EEGpal, a custom MATLAB-based toolbox built on EEGLAB 2023.1 (Delorme & Makeig, 2004) and openly available at https://github.com/DePrettoM/EEGpal. EEG signals were band-pass filtered between 0.3 and 40 Hz. Noisy or bridged electrodes were identified through visual inspection and temporarily excluded. Independent Component Analysis (ICA) was then performed (Onton et al., 2006; L. Sun et al., 2005), and artifactual components (e.g., ocular, muscular, or cardiac sources) were detected and removed using the ICLabel classifier (Pion-Tonachini et al., 2019). The excluded channels were subsequently interpolated, and the data were re-referenced to the common average. The cleaned continuous EEG was segmented into epochs of 2000 ms for subsequent microstate analyses.

Microstate Analysis

Microstate segmentation was performed in MATLAB using the MICROSTATELAB toolbox and following a standard pipeline summarized in **Fig. 2** (Nagabhushan Kalburgi et al., 2024). EEG topographies were extracted at peaks of Global Field Power (GFP), representing moments of maximal global synchrony (Parameshwaran & Thiagarajan, 2019; Skrandies, 1990). These maps were subjected to a modified k-means clustering algorithm to define subject-level microstate classes. To increase sensitivity to potentially subtle patterns relevant to clinical populations, a seven-class solution was adopted (Custo et al., 2017; Milz et al., 2016).

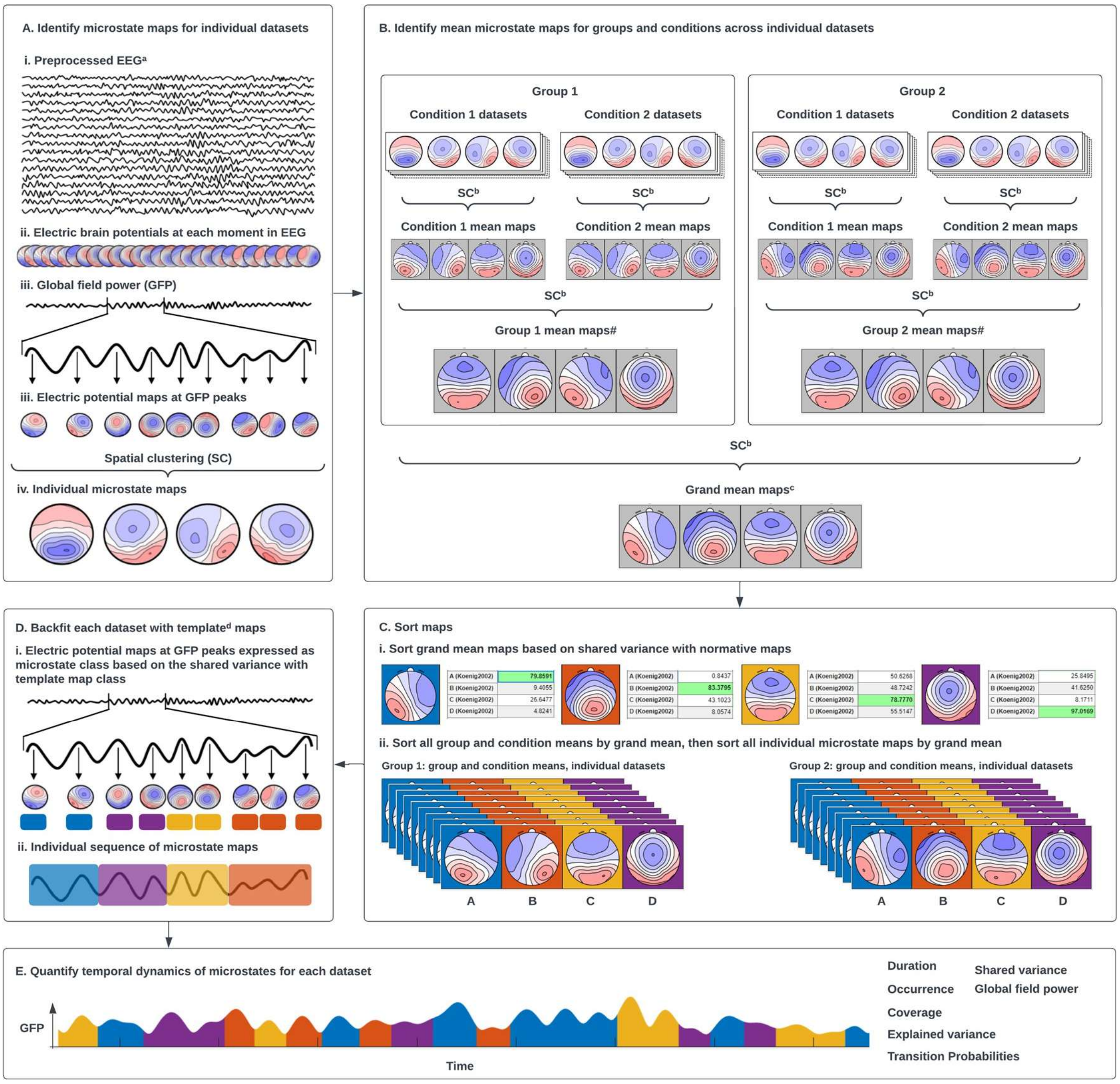

Figure 2 - Microstate segmentation and analysis pipeline. *Schematic overview of the procedure including subject-level extraction of GFP-peak topographies and clustering into individual microstate maps, computation of mean and grand-mean templates through hierarchical aggregation and sorting, back-fitting of the resulting templates to continuous EEG signals, and extraction of temporal parameters at the individual level. Reproduced from Kalburgi et al. (2024), Open Access licensed under CC-BY 4.0 https://doi.org/10.1007/s10548-023-01003-5*

Individual-level maps were then hierarchically clustered to produce group-average maps, which were further aggregated into grand-mean templates to ensure one-to-one correspondence across hierarchical levels and to maximize shared explained variance. Resulting templates were reordered and labeled according to established conventions (Khanna et al., 2015; Michel & Koenig, 2018), accounting for 94.79% of global variance; no outliers were identified.

These grand-average templates were then back-fitted to each participant's continuous EEG, assigning at each time point the microstate class exhibiting the highest spatial correlation with the instantaneous scalp potential map. For each class, we computed key temporal and spatial metrics: mean duration, occurrence rate (events per second), coverage (percentage of total recording time), mean GFP, and explained variance (per class and overall). Additionally, transition probabilities between microstates were quantified both as raw empirical frequencies (percentage of direct X→Y transitions among all transitions) and as adjusted probabilities, indicating deviations from chance and thus the relative tendency or avoidance for specific class-to-class transitions.

Statistical analysis

To address possible group-related variations in the temporal characteristics of individual microstate configurations, we compared the main microstate parameters – mean duration, occurrence rate, coverage, mean GFP, and explained variance – between groups using independent-samples t-tests. These between-group comparisons of temporal parameters were defined a priori as the primary analyses. To control for multiple comparisons, Bonferroni correction was applied separately for each parameter across the seven microstate classes, yielding a corrected significance level of $\alpha = .05/7 \approx .007$. Only results below this threshold were deemed statistically significant. Parameters that differed significantly between groups were subsequently entered into logistic regression models to test their ability to predict group membership. Among patients, those same parameters were further correlated with S-FMDRS symptom severity scores to explore their clinical relevance.

All subsequent analyses were considered exploratory. In particular, to assess potential group effects on the temporal "syntax" of microstates – that is, the likelihood of transitioning into or out of specific classes (Lehmann et al., 2005), we examined pairwise transition probabilities using a series of fourteen repeated-measures ANOVAs (rmANOVAs). For each microstate class, one set of seven "FROM" ANOVAs evaluated transitions originating from that class toward all others, while another set of seven "TO" ANOVAs analyzed transitions entering that class from all remaining classes (Antonova et al., 2022). In each case, transition direction ("FROM" or "TO") served as the within-subjects factor, and GROUP (FND vs. HC) as the between-subjects factor. When sphericity assumptions were violated, the Greenhouse–Geisser correction was applied, and Bonferroni adjustments were used for post hoc comparisons. All statistical analyses were conducted in JASP (version 19.3).

Results

Temporal parameters

Among all examined parameters, only the mean duration of microstate class G showed a significant difference between groups (**Fig. 3**). Patients with FND displayed a shorter mean duration compared to healthy controls ($t = 3.12$, $p = 0.005$, Cohen's $d = –0.619$). No other microstate class differed significantly in duration, occurrence rate, coverage, mean GFP, or explained variance after applying the correction for multiple comparisons (all $p > 0.007$).

Across the patient group, a trend-level negative correlation emerged between the duration of microstate G and F-MDRS symptom severity (Pearson's $r = –0.337$, $p = 0.052$).

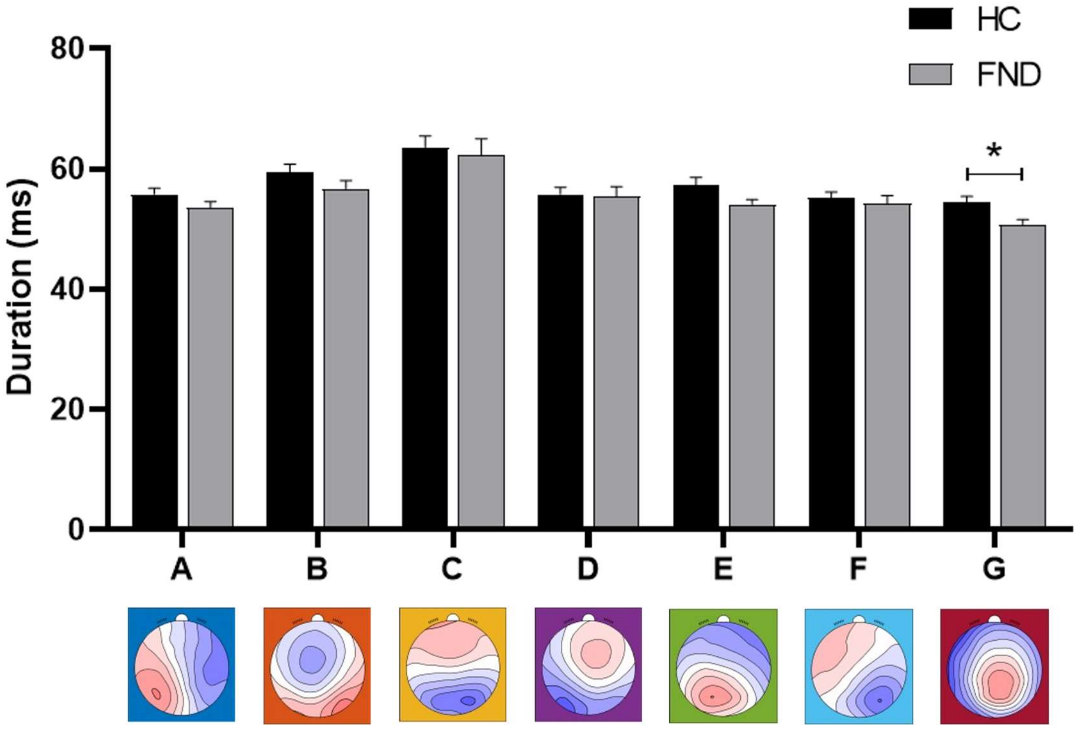

Figure 3 - Mean microstates duration across the seven classes*. Mean duration (ms) of the seven canonical EEG microstate classes (A–G) in patients with Functional Neurological Disorders (FND, grey) and healthy controls (HC, black). Bars represent group means ± standard error. Scalp topographies displayed below each label illustrate the corresponding prototypical microstate maps. Among all classes, only microstate G showed a significant between-group difference, with shorter durations in FND compared to controls.*

To evaluate whether this parameter could discriminate between groups, we performed a logistic regression using group membership (FND vs. HC) as the dependent variable. The duration of microstate G significantly predicted classification ($\chi^2 = 7.1$, $p = 0.009$), accounting for 11.6% of the variance (Nagelkerke $R^2 = 0.116$). The model showed a moderate discriminative ability (AUC = 0.65, 95% CI [0.54-0.76]), with an optimal cutoff yielding 48.7% sensitivity and 70.2% specificity.

Transition probabilities

The repeated-measures ANOVA on the adjusted transition probabilities from class A showed a robust main effect of transition direction, F(5, 420) = 29.35, $p < .001$, $\eta p^2 = .259$, as well as a significant interaction between transition direction and group, F(5, 420) = 2.83, p = .029, $\eta p^2 = .033$ (**Fig. 4**). Post hoc analyses were then performed to clarify this interaction. The adjusted transition probability from A to B was significantly lower in the FND group than in healthy controls (p = .007). A similar group effect was found for the A→C transition (p = .030). For the remaining transitions (A→D, A→E, A→F, A→G), no significant differences emerged between groups (all p > .19). Likewise, none of the other "FROM" transition series (originating from classes B, C, D, E, F, or G) yielded significant group effects.

Regarding transitions to each microstate class, the rmANOVA on the adjusted probabilities to class A revealed a main effect of transition direction, F(5, 400) = 23.21, $p < .001$, $\eta p^2 = .225$, together with a significant interaction between transition direction and group, F(5, 400) = 2.46, p = .032, $\eta p^2 = .030$. Subsequent post hoc comparisons indicated that the transition from class B to A occurred less frequently in FND patients than in healthy controls (p = .006). No other transitions leading to class A (from C, D, E, F, or G) differed significantly between groups, and the analyses conducted for transitions toward the remaining classes also showed no significant group interactions.

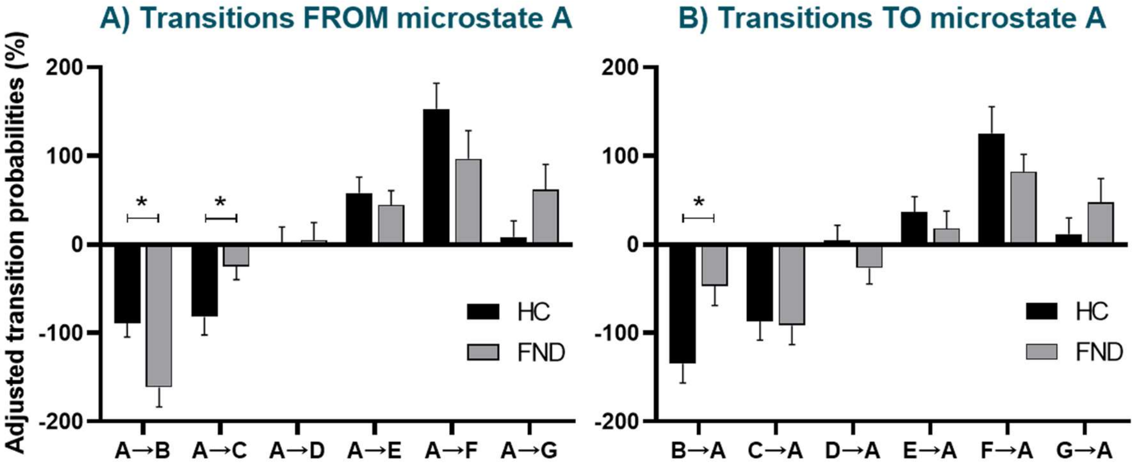

Figure 4 - Group differences in adjusted transition probabilities involving microstate A*. Adjusted transition probabilities (%) between microstate A and the remaining classes during resting-state EEG in healthy controls (HC, black) and patients with Functional Neurological Disorders (FND, grey). Panel A shows transitions originating from microstate A (A→X), whereas Panel B shows transitions entering microstate A (X→A). Values represent deviations from chance-level transitions, plotted as mean ± standard error. Significant between-group differences are marked with asterisks (*p < corrected α).*

Discussion

This work represents the first systematic exploration of resting-state EEG microstate dynamics in individuals with FND, offering novel evidence on both the temporal persistence of discrete brain states and the probabilistic rules governing transitions among them. Beyond the reduced duration of microstate G, previously reported as implicated in somatosensory integration (Tarailis et al., 2024), our results reveal an atypical configuration of transitions among microstates A, B, and C. This pattern delineates a potential framework for future investigations linking the temporal organization of neural states with core functional symptoms.

The shorter duration of microstate G observed in FND, in the absence of differences in coverage, suggests that patients briefly recruit this configuration but fail to maintain it, indicating possible temporal instability (Al Zoubi et al., 2019). This finding gains particular significance in light of microstate G's established relationship with somatosensory and sensorimotor networks (Stoffers et al., 2015; Tarailis et al., 2024). Microstate G is a relatively recent addition to the canonical repertoire, with estimated generators in the right inferior parietal lobule, superior temporal gyrus, and cerebellum; its consistent cerebellar engagement has been interpreted as indexing sensorimotor network activity (Custo et al., 2017), and its temporal features have been associated with cerebello-parietal connectivity (Stoffers et al., 2015). In healthy individuals, this microstate appears to support the contextualization of bodily information and the integration of sensory input into motor plans (Custo et al., 2017; Tarailis et al., 2024; Zanesco et al., 2021). Accordingly, the reduced stability of microstate G in FND may reflect impaired maintenance of coherent sensorimotor representations, implying that bodily input is less effectively sustained in neural processing. The specificity of this alteration to microstate G, rather than a generalized disruption across all classes, further points to a selective mechanism. Although the effect size was moderate, the association between microstate G and somatosensory processes underscores its potential clinical relevance. The observed trend toward an inverse relationship with motor symptom severity, as measured by the S-FMDRS, lends additional, albeit preliminary, support to this interpretation.

Replication in larger samples will be essential before considering microstate G dynamics as a prognostic marker of somatosensory network dysfunction. Even so, such parameters could complement current clinical assessments. The specificity of 70.2% indicates that, while a considerable proportion of controls can be correctly classified, false positives remain, suggesting that microstate G instability is not exclusive to FND but may intersect with more general mechanisms of somatosensory integration also perturbed in other neuropsychiatric conditions.

The analysis of transition probabilities adds a distinct and complementary perspective. Even in the absence of overt stimulation, patients with FND displayed an endogenous bias in the sequencing of their microstates. Alterations in transitions among microstates A, B, and C occurred despite comparable duration, occurrence, and coverage, implying that the individual state properties were preserved. What differed was the temporal syntax, in other words the way the brain moved between these states, rather than the states themselves.

A first key finding concerns the reduced probability of transitioning from microstate A to B, indicating that FND patients are less likely than controls to shift from an arousal-related network toward systems involved in visual perception and the conscious construction of imagery or autobiographical scenes (Tarailis et al., 2024). Microstate A is associated with auditory processing and heightened arousability, whereas microstate B engages occipital-parietal regions and supports internally guided perceptual integration (Bréchet et al., 2019; Britz et al., 2010; Tarailis et al., 2024). A reduced A→B transition may therefore reflect a tendency toward sustained sensory vigilance that limits access to imagery-based and integrative modes of processing (Bréchet et al., 2019; Chivu et al., 2024; Damborská et al., 2019). A second alteration concerns the A→C transition, where patients exhibited reduced resistance, suggesting an easier drift toward self-referential and introspective states. In healthy individuals, spontaneous transitions into microstate C—whose generators overlap with the default mode network—are thought to facilitate mind-wandering and the integration of bodily and autobiographical information (Bréchet et al., 2019; Custo et al., 2017). In FND, this facilitated access may instead signal a dysregulated interaction between arousal-related and DMN-mediated regulatory functions, potentially leading to fragmented or less coherent self-referential processing (Britz et al., 2010; Milz et al., 2016).

Finally, the higher transition probability from microstate B to A in FND points to an altered interplay between sensory-imagery and arousal systems. Among controls, this shift typically occurs with greater inertia, allowing visual or imagery-based processes to stabilize before reverting to vigilance states. In FND, by contrast, the facilitated B→A transition implies a quicker resetting of imagery or perceptual activity toward arousal-dominated dynamics.

Taken together, these findings suggest two complementary abnormalities: an instability within a sensorimotor-related state (microstate G) and a disruption in the temporal grammar that organizes transitions between arousal, imagery, and self-referential networks (microstates A, B, and C). Such combined disturbances may reflect a broader disorganization of brain dynamics in FND, in which both the persistence of bodily representations and the flexible sequencing of large-scale networks are compromised.

Limitations

The choice of a seven-class microstate solution, adopted to maximize sensitivity to clinically relevant configurations such as microstate G, inevitably limits direct comparability with the broader literature, which often relies on four canonical classes. Although this approach aligns with recent methodological developments, it complicates cross-study integration and makes it difficult to determine whether the observed alterations reflect a consistent signature of FND or a feature emerging from model-specific granularity. Moreover, the absence of psychiatric and neurological comparison groups constrains the interpretation of specificity. Conditions characterized by altered somatosensory processing, heightened arousal, or disrupted self-referential dynamics, such as anxiety disorders, chronic pain, or other functional somatic syndromes may show partially overlapping microstate patterns. Without such controls, it remains uncertain whether the reduced stability of microstate G and the atypical transitions among A, B, and C index mechanisms are distinctive to FND or reflect broader transdiagnostic perturbations in large-scale network dynamics.

Conclusions

This study provides the first direct evidence that FND is characterized by a selective disturbance in the temporal stability of a sensorimotor-related microstate, accompanied by an alteration in the intrinsic transition patterns that govern spontaneous brain activity. The reduced duration of microstate G points to difficulties in sustaining coherent somatosensory representations, while the atypical oscillatory tendencies among microstates associated with arousal, self-visualization, and integrative processes suggest a broader disruption in self-related neural dynamics. Further research in larger and independent cohorts is needed to replicate these results and to include suitable psychiatric and neurological comparison groups. Future work should also clarify whether the observed microstate alterations are specific to FND or reflect a more general mechanism shared across different neuropsychiatric conditions.

PART 2

PREDICTIVE CODING IN FUNCTIONAL NEUROLOGICAL DISORDERS

Predictive coding in FND: from emotions to predictive construction of the experience

Over more than a century, models of FND have oscillated between psychogenic and neurological poles, often placing "emotion" at the center – as a causal agent, a trigger, or a latent conflict seeking expression (Edwards et al., 2012; Pick et al., 2019; Raynor & Baslet, 2021). Yet, most of this discussion proceeded without a clear account of what emotions are. Classic views framed emotions as biologically hard-wired programs: discrete neural circuits produce stereotyped patterns of physiological arousal, facial expression and subjective feeling (Anderson & Adolphs, 2014; Tracy & Randles, 2011). FND research inherited this framework, implicitly or explicitly, when interpreting "emotion dysregulation", "fear processing abnormalities" or "conversion of affect" (Pick et al., 2019; Sojka et al., 2019).

Over recent decades, however, a parallel strand of work in affective and cognitive neuroscience has challenged this traditional view. Large-scale meta-analytic evidence and multi-method studies demonstrate that instances of "anger", "fear" or "sadness" do not map onto fixed physiological signatures or single, specialized brain circuits; instead, they display considerable within-category heterogeneity and cross-category overlap (Barrett & Satpute, 2019; Kober et al., 2008; Raz et al., 2016; Wager et al., 2015). These findings have encouraged a shift toward more constructionist accounts of emotion (Barrett, 2017), in which the brain is understood as a predictive organ: continuously modelling the body in the world, anticipating its needs, and assembling subjective experience from interoceptive, exteroceptive, and conceptual cues (Katsumi et al., 2022; Santamaría-García et al., 2025; Sennesh et al., 2022).

Against this theoretical backdrop, recent proposals have argued that FND may be best understood through the lens of predictive and interoceptive dysfunction (Drane et al., 2021; Edwards et al., 2012; Jungilligens et al., 2022). Within this view, emotional experiences are not fixed outputs that “leak” into motor or sensory systems, but emergent constructions that depend on the efficient integration of bodily signals and conceptual priors. When these processes become chronically biased or inefficient, the resulting mismatch between predictions and bodily states may contribute to the unstable symptoms, altered agency, and sensory–motor disturbances characteristic of FND. Such accounts frame FND not as the somatic expression of some hidden affect, but as a disorder of predictive regulation and emotion construction.

In this chapter, I summarize contemporary accounts of allostasis, predictive coding, interoception, and emotion construction and show how they converge to depict FND as a disorder of predictive regulation rather than emotional discharge. I outline how symptoms likely arise from the interaction between imprecise interoceptive modelling, high-precision illness priors, and a constrained capacity to differentiate affective states, and I draw on this framework to motivate the subsequent examination of cognitive-perceptual traits linked to predictive biases in adults with FND.

3. Allostasis, predictive processing, and interoception in FND

3.1 Allostasis: the brain as an energetic regulator

In multicellular organisms, neural systems emerged to coordinate the regulation of energy intake, storage and expenditure across time and context. The concept of allostasis captures this proactive regulation: the brain forecasts the body's metabolic needs and prepares responses before homeostatic imbalance becomes critical (Barrett, 2017; Sterling, 2012). Allostasis is not merely returning to set points, but budgeting energy based on predictions.

At the neural level, this implies a continuous stream of top–down predictions about bodily and environmental states, informed by past experiences and conceptual knowledge. These predictions shape autonomic, endocrine and behavioral responses (Katsumi et al., 2022; Migeot et al., 2022; Schulkin & Sterling, 2019). The brain's success or failure in performing allostasis is therefore central to understanding chronic fatigue, hyperarousal, and other pervasive features of FND (Butler et al., 2021; Jungilligens et al., 2022). In terms of predictive coding, these allostatic processes correspond to a hierarchical system of generative models that anticipate interoceptive and exteroceptive states and constantly compare them with sensory input. The regulation of bodily energy thus becomes a problem of inference: minimizing prediction error about what the body is doing and what it should be doing in a given context (Edwards et al., 2012; Friston, 2010). In FND, part of the chronic metabolic cost may stem precisely from the attempt to maintain consistency between strong symptomatic beliefs and bodily signals that, in reality, do not fully justify them (Adams et al., 2013; Fiorio et al., 2022; Jungilligens et al., 2022). From this perspective, these beliefs can be regarded more simply as expectations about the body and its likely states, built through prior experience and contextual learning (Espay et al., 2018; Hallett et al., 2022). When such expectations acquire excessive weight or are not readily revised, they may end up guiding perception more than the sensory input itself. Under these conditions, ambiguous bodily signals are more easily interpreted in ways that confirm the expected symptom pattern, allowing those experiences to persist even in the absence of clear physiological support (Edwards & Bhatia, 2012; Fiorio et al., 2022).

3.2 Predictive processing: priors, prediction errors, and precision

Predictive-processing accounts describe perception and action as forms of probabilistic inference under uncertainty (Knill & Pouget, 2004; Spratling, 2017). This framework turns on few tightly interwoven principles. First, the nervous system is engaged in a ceaseless stream of continuous prediction, drawing on learned generative models to anticipate the likely structure of upcoming sensory states (Friston, 2010; Hohwy, 2012). These predictions are constantly confronted with incoming data, which provide the likelihood term against which model expectations are evaluated, and any mismatch – the so called prediction error – marks a potential inaccuracy in the model and may prompt its revision (**Fig. 5**).

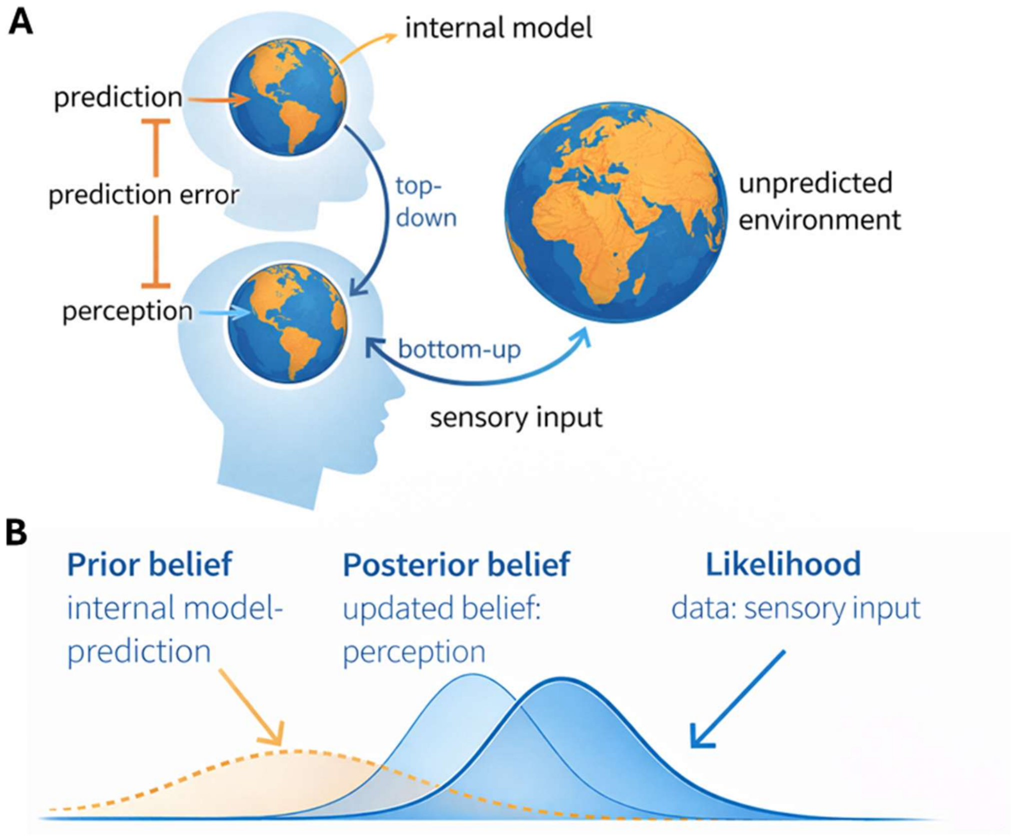

Figure 5 - Conceptual scheme of predictive brain-environment interactions*. (A) Schematic illustration of the reciprocal exchange between internal models and the external environment. Top–down expectations generated by the internal model are compared with bottom–up sensory signals; discrepancies between the two give rise to error signals that update perceptual states and guide adaptive action. (B) Distributional representation of prediction updating. Prior expectations (dashed orange) are combined with sensory evidence (blue likelihood), yielding an updated estimate (posterior, light blue) that reflects the relative influence of internal assumptions and incoming data. Adapted from Haker et al. (2016) https://doi.org/10.3389/fpsyt.2016.00107*

Also, precision weighting determines the relative impact of priors and prediction errors: the brain estimates the reliability of each source of information, a computation in which the salience network plays a central role. Highly trustworthy priors can dominate perception and action, whereas sharp, unambiguous sensory signals can shift the balance toward error-driven updating (Friston, 2010; Friston et al., 2016; Rao et al., 2023; Sarasso et al., 2024).

These operations are thought to unfold along a graded cortical architecture in which limbic, less laminated regions – such as the anterior insula, anterior cingulate, and orbitofrontal cortex – send predictions to more laminated sensory cortices, while prediction errors propagate upward (Bastos et al., 2012; Chao et al., 2018; Mikulasch et al., 2023). This gradient maps neatly onto canonical resting-state networks: the default mode network supports abstract generative models, salience regions estimate precision and orchestrate resource allocation, and sensorimotor circuits instantiate predicted actions (Murphy et al., 2018; Stawarczyk et al., 2021; Zhang et al., 2025).

Attention becomes, in this light, a dynamic modulation of gain: a mechanism that determines whether, at any moment, bodily priors or peripheral sensory evidence will carry more weight (Edwards et al., 2012; Feldman & Friston, 2010). When attention becomes narrowly and inflexibly directed toward a particular sensation or a "weak" limb, symptom-related priors can acquire excessive precision, leading to a systematic down-weighting of contradictory sensory evidence.

Active inference follows naturally from this scheme. Motor commands can be understood as predictions about the proprioceptive signals the body should generate next. These predictions are then "fulfilled" via spinal reflexes and descending pathways that act to minimize the gap between expected and actual states. If the system assigns high precision to a prior of weakness or tremor, the resulting behavior may resemble exactly that (i.e. an underperforming or irregularly moving limb) despite the structural integrity of the motor pathways (Edwards et al., 2012; Feldman & Friston, 2010; Fiorio et al., 2022).

2.3 Interoception and affect as outcomes of prediction

For FND, interoception is crucial. Interoception refers to the brain's modelling of the body's internal state across multiple channels (i.e. cardiovascular, respiratory, gastrointestinal, immune) and its continuous comparison of predicted and actual interoceptive input (Barrett & Simmons, 2015; Khalsa et al., 2018).

Affect, meaning the felt qualities of valence and arousal, has been defined as the conscious consequence of this modelling (Jungilligens et al., 2022). Affect is a low-dimensional summary of ongoing allostatic computations, not a separate "emotion system". It colors perception, influences predictions, and provides a barometer of the body's energetic state (Barrett, 2017; Russell et al., 2003).

In this setting, errors in interoceptive prediction can have wide-ranging consequences. If the interoceptive model is noisy, imprecise, or biased, the organism may chronically misestimate its internal condition. This can foster persistent negative affect, fatigue, and hyperarousal, features that are highly prevalent in FND and strongly linked to quality of life (Apazoglou et al., 2017; Jirásek et al., 2025; Pick et al., 2024).

Bayesian formulations of FND – that is, formal computational implementations of predictive processing in which perception and action are modeled as probabilistic inference combining prior beliefs and sensory evidence through Bayesian updating – treat these interoceptive errors as a borderline case of processes that are also observed, in a less extreme form, in placebos and nocebos: expectations, associative learning, and context modulate the weight attributed to certain bodily signals, so that the final experience reflects predictions rather than raw input (Fiorio et al., 2022). In other words, the functional symptom can be read as a "spontaneous nocebo," in which maladaptive beliefs about the body and illness are continually confirmed by the perceptual experience they themselves generate.

4. The theory of constructed emotion and symptom formation in FND

4.1 Emotion as conceptual categorization

The theory of constructed emotion (Barrett, 2017) marks a significant departure from classical models. Instead of positing dedicated neural "modules" for fear, anger, or sadness, it argues that the brain draws on learned, context-sensitive concepts to make sense of interoceptive and exteroceptive patterns. When a predictive concept aligns sufficiently with ongoing sensory input, the system constructs a situated emotional category as an instance of "fear," "shame," or "relief" (Barrett, 2017; Hoemann & Feldman Barrett, 2019). Furthermore, several features likely define this constructive process. One is degeneracy: no single neural configuration uniquely corresponds to a given emotion, as multiple patterns can support the same instance (Edelman & Gally, 2001). Another domain is generality, reflected in the fact that regions such as the amygdala, insula, and anterior cingulate participate in a wide range of psychological operations well beyond emotion per se (Šimić et al., 2021). A third property is perceiver dependence, meaning that identical bodily states may be categorized differently by different individuals, or even by the same individual at different moments, depending on contextual cues, conceptual knowledge, and goals (Barrett, 2017).

Individuals further vary in their level of emotional granularity, that is, the capacity to construct fine-grained emotional categories rather than collapsing diverse affective states into undifferentiated labels such as “bad.” Higher granularity has been linked to greater flexibility in both physiological responses and behavioral regulation (Jungilligens et al., 2022; Kashdan et al., 2015).

4.2 Emotion construction in FND

Against this theoretical background, Jungilligens and colleagues (2022) argue that FND symptoms can be understood as arising from disruptions at multiple points along the emotion-construction stream. One line of reasoning concerns chronic energy mismanagement (i.e. allostatic overload) which is frequently reported in FND and marked by persistent fatigue and hyperarousal (Butler et al., 2021). The authors suggest that when individuals rely on coarse-grained emotion concepts such as generic “fatigue” or “feeling unwell,” their allostatic predictions become imprecise. In predictive-coding terms, this corresponds to bodily priors that are overly precise yet conceptually impoverished, leaving them resistant to updating. States of alertness, depletion, and tension are repeatedly labeled in the same way, limiting opportunities for physiological recalibration. The body then expends considerable resources maintaining coherence with a poorly tuned internal model instead of using interoceptive variability to refine its energetic “budget” (Edwards et al., 2012; Jungilligens et al., 2022).

A second mechanism concerns aberrant emotion construction and the substitution of symptoms for emotions. High-arousal states triggered by interpersonal conflict or acute physical threat (e.g. a car accident) may never be categorized as “fear” or “shock” if the relevant concepts are absent or not recruited. Instead, the system may default to bodily or illness-related categories such as “weakness” or “shaking,” which can crystallize into functional motor or sensory symptoms. The same predictive machinery is at play: priors shape the interpretation of ambiguous interoceptive signals, but the priors themselves are biased toward disease rather than emotion. Bayesian models of FND describe such episodes as “primary failures of inference,” where a specific pattern of sensations or movements is encoded as the expected outcome of a salient event (“the arm won’t respond,” “the legs give way”) and is thereafter reproduced whenever similar contexts arise (Edwards et al., 2012). Lacking a robust emotional framework, the system gravitates toward illness-related categories that are further reinforced by clinical encounters, received explanations, and interpersonal responses (Fiorio et al., 2022).

A third proposal focuses on altered prediction-error learning. Individuals with FND show atypicalities in sensory processing, attentional orienting, interoception, and motor learning (Diez et al., 2019; MacLean et al., 2022; Ranford et al., 2020; Rossi et al., 2025; Weissbach et al., 2023), all of which can hinder the effective use of prediction errors to refine internal models. When errors are ambiguous, metabolically costly, or conflict strongly with entrenched priors, they may be discounted or misinterpreted, preventing corrective updating. Literature on placebo and nocebo effects illustrates a parallel mechanism: repeated associations between certain contexts (clinical settings, specific postures, particular movements) and symptoms can lead the organism to treat those contexts as predictors of negative bodily outcomes (Fiorio et al., 2022; Van den Bergh et al., 2017). Each recurrence of the symptom then appears to confirm the prior, further narrowing the space for prediction-error-based learning.

A fourth dimension involves reconceptualizing alexithymia, "panic without panic," and dissociation. High rates of alexithymia observed in FND (Ricciardi et al., 2015; van Dijl et al., 2024) may reflect a sparse or inflexible repertoire of emotion concepts. Episodes sometimes termed "panic attacks without panic" in functional seizures (Goldstein & Mellers, 2006) can be viewed as intense autonomic surges that fail to be categorized as fear. Dissociation, common in FND and closely linked to trauma and interoceptive deficits (Pick et al., 2020; Van Der Hart et al., 2004; Woelk & Garfinkel, 2025) may arise when higher-order generative models of self become temporarily decoupled from interoceptive predictions, leaving affective meaning out of sync with ongoing experience. From an inferential perspective, these states involve highly salient bodily signals that lack an adequate emotional label and are therefore explained via alternative models (e.g., "I'm having a neurological crisis"). Attention becomes locked onto symptom-related priors at intermediate hierarchical levels, amplifying their precision without enriching their conceptual content. Subjectively, this produces the sense that something is happening *to* the body from the outside, eroding agency and the unity of the self (Edwards et al., 2012).

A further source of disruption concerns the well-known clinician–patient discrepancy regarding "anxiety." Clinicians often perceive patients as anxious, while patients insist they do not feel anxious at all. Within this framework, both perspectives may be accurate.

Observers rely on their own emotion concepts to interpret visible cues, whereas the patient's predictive machinery may never construct an emotional category for that episode. As Barrett (2017) notes, emotional experience is inferred rather than observed. The placebo/nocebo lens adds nuance: traits such as anxiety and hypervigilance heighten the salience of bodily sensations and amplify certain medical messages, strengthening illness expectations even in the absence of an explicit subjective feeling of anxiety (Bakvis et al., 2009; Fiorio et al., 2022). The clinician detects peripheral signs of hyperarousal; the patient registers symptoms and exhaustion. The perspectives remain misaligned because they are tracking different levels of the same predictive architecture.

Finally, Jungilligens and colleagues (2022) emphasize the formative role of adverse life experiences, maltreatment, and insecure attachment: factors more prevalent in FND and linked to symptom severity and dissociation (Ludwig et al., 2018). Concepts that are adaptive under threat (e.g. heightened vigilance to bodily danger or rapid motor readiness) may later become maladaptive when contexts shift. On a Bayesian reading, early adversity and insecure attachment tune the system toward highly precise priors of threat and bodily vulnerability, while access to differentiated emotion concepts remains limited. In adulthood, this configuration may facilitate both the emergence of functional symptoms and the development of nocebo responses when ambiguous bodily signals or alarming medical information is encountered.

Taken together, these converging proposals portray FND not as a failure to express emotion but as a disruption in the predictive construction of experience itself. Bodily signals are entirely real, yet they are categorized and interpreted through models that favor symptom formation over emotional meaning.

5. Neurobiological correlates of predictive processes in FND

5.1 Network-level findings: default mode, salience, and sensorimotor systems

Neuroimaging studies in FND and related functional disorders have repeatedly identified alterations in default mode, salience, and sensorimotor networks (Aybek & Perez, 2022; Baizabal-Carvallo et al., 2019; D. L. Perez et al., 2017). For instance, differences in connectivity and activity have been reported in medial prefrontal cortex, posterior cingulate, anterior insula, dorsal anterior cingulate, and supplementary motor areas (Aybek et al., 2015; Diez et al., 2021a; Maurer et al., 2016). Within a predictive framework, these findings are not incidental. Default mode regions are implicated in high-level generative models, including self-related processing and concept formation. Salience regions (anterior insula, dorsal anterior cingulate) estimate precision and guide attentional reallocation, including interoceptive attention (Menon & Uddin, 2010; Seeley et al., 2007). Sensorimotor networks implement predictions about movement and its sensory consequences. Perturbations across this ensemble are consistent with the idea that concepts, predictions and precision estimates are atypical in FND.

Some studies have offered an even more detailed interpretation of these network processes: intermediate motor areas, such as the supplementary motor area, are considered the level at which specific priors on movement patterns are represented, while medial prefrontal and posterior parietal regions contribute to the formation of intentions and the sense of agency (Cojan et al., 2009; Edwards et al., 2012; Voon, 2010). The abnormal activation of these regions during symptoms, together with altered connectivity with the insula and anterior cingulate, is consistent with the idea of high-precision symptomatic priors, modulated by attention and emotional evaluation.

In parallel, work on placebo and nocebo shows the recurrent involvement of the anterior insula, dorsal anterior cingulate, ventromedial prefrontal cortex, and amygdala in mediating expectations about pain and other bodily sensations (Craggs et al., 2007; Fiorio et al., 2022; Wittkamp et al., 2024). The partial overlap between these networks and those involved in FND suggests that mechanisms of prediction and re-evaluation of bodily signals may be shared, even though they express themselves in clinically different ways.

5.2 Interoception, insula, and white matter integrity

One of the few imaging studies directly linking interoception to brain structure in FND found that ITPE (measure of the mismatch between cardiac interoceptive beliefs and accuracy) correlated with reduced white matter integrity in tracts originating from the insula and temporoparietal junction (Sojka et al., 2020). This suggests that the physical infrastructure supporting interoceptive prediction and error signalling may be compromised. More broadly, alterations of the insula reported in FND (functional and structural) are compatible with a disturbance of interoceptive predictive coding (Diez et al., 2021b; D. L. Perez et al., 2017). If insula-based interoceptive models are imprecise, the system may either overreact to ambiguous bodily cues or fail to integrate them, both of which can foster chronically elevated affect and aberrant symptom constructions.

In functional sensory models, intermediate levels (e.g., secondary somatosensory cortex and insula) represent the meeting point between ascending prediction errors and descending priors: when a prior of “anesthesia” or “pain” receives too much accuracy, the final estimate tends to reflect expectation rather than the afferent signal, while the residual error is “pushed” to higher levels that must explain why the subjective experience does not match the integrity of the sensory pathways. The alterations in insular connectivity and microstructure observed in FND fit into this scheme, providing anatomical support for the idea of disturbed interoceptive predictive coding.

5.3 Autonomic and neuroendocrine evidence: allostatic strain

Meta-analytic work on autonomic and endocrine markers in FND indicates increased heart rate, reduced heart rate variability, and cortisol alterations associated with trauma burden and threat processing (Bakvis et al., 2009; Paredes-Echeverri et al., 2022). These findings have often been framed as indices of “emotion dysregulation”. Within an allostatic account, they instead reflect inefficient energy management and chronic activation of stress-related systems (M. Schneider & Schwerdtfeger, 2020; Shaffer et al., 2022). Cortisol, in particular, is reinterpreted as primarily a metabolic hormone facilitating gluconeogenesis rather than a “stress hormone” per se (Barrett, 2017; Jimeno et al., 2018; Khani & Tayek, 2001).

These autonomic and endocrine features dovetail with self-reported fatigue and hyperarousal in FND cohorts (Butler et al., 2021) and fit the proposal that some individuals with FND operate under a chronically miscalibrated interoceptive-allostatic model.

Evidence from nocebo paradigms reinforces this picture: negative expectations and threatening signals can amplify pain, dyspnea, and other sensations through increased sympathetic-adrenaline activity and hypothalamic-pituitary-adrenal axis activity (Colloca & Benedetti, 2007). In individuals with FND, whose stress response systems are already unbalanced, these mechanisms may contribute not only to the severity of symptoms but also to their resistance to reassurance and negative investigations, maintaining high disease prior accuracy despite the absence of structural lesions.

Study 2: Predictive styles in FND

Introduction

The theoretical background outlined so far suggests that predictive processing provides a unifying framework for understanding FND and for situating it among other neuropsychiatric conditions that involve atypical inference. Yet, while the role of interoception, allostasis, and emotion construction in FND has been repeatedly emphasized, much less is known about how these patients differ in the way they typically balance expectations and sensory evidence across domains, and whether this "predictive style" aligns with broader, transdiagnostic continua already described in other conditions.

Bayesian inference and diametric predictive styles in schizophrenia and autism spectra

Recent work approached predictive processing in psychopathology from a particular vantage point (Tarasi et al., 2022). Although not focusing on FND, they deeply investigated how the same computational principles can clarify the relationship between the predictive styles of two disorders with very different clinical characteristics: schizophrenia and psychosis-spectrum disorders (SSD, including schizotypy, the subclinical expression of psychosis-like traits in the general population) and autism spectrum disorders (ASD). Psychometric research examining autistic and schizotypal characteristics side by side has repeatedly shown that the two spectra share a zone of overlap, particularly in the domain of social and communicative functioning (Dinsdale et al., 2013; Nenadić et al., 2021; Zhou et al., 2019). Beyond this shared territory, however, the profiles tend to diverge in a patterned way.

SSD have been conceptualized as paradigmatic examples of altered precision weighting (Adams et al., 2013; Fletcher & Frith, 2009; Horga & Abi-Dargham, 2019; Sterzer et al., 2018), within a framework linking subjective perceptual alterations to neurobiological dysfunctions, such as those related to dopaminergic and glutamatergic systems (Knolle et al., 2025; Millard et al., 2022). In some computational accounts of SSD, abstract beliefs and expectations can become pathologically precise, such that top-down models dominate inference and impose structure on ambiguous input, supporting hallucinations, delusions, and a tendency to impose meaning on random patterns (Ashinoff et al., 2022; Corlett et al., 2009; Powers et al., 2025); in other words, excessively precise priors might dominate sensory processing, biasing perception toward internally generated expectations and away from actual sensory data (Fletcher & Frith, 2009; Katthagen et al., 2022; Stuke et al., 2021).

Focusing on a lower sensory stage in SSD, there is extensive evidence for impoverished, noisy, or unstable representations and reduced use of learned regularities (Avissar et al., 2018; Koshiyama et al., 2020; Petrovic & Sterzer, 2023); high-level priors may sometimes "compensate" for unreliable sensory channels, but at the cost of becoming rigid, self-confirming, and resistant to disconfirmatory evidence (Koshiyama et al., 2020; Sterzer et al., 2018). Both scenarios describe psychosis not in terms of isolated heterogeneous symptoms, but as the result of a breakdown in inferential balance between expectations and sensations which can distort perceptual inference, producing symptoms such as hallucinations or delusions.

Conversely, in ASD, several converging proposals describe under-exploited priors and/or overweighted prediction errors: the "hypo-priors" hypothesis (Pellicano & Burr, 2012), accounts that focus on overly precise sensory prediction errors (Cannon et al., 2021; Goris et al., 2018; Karvelis et al., 2018; van Laarhoven et al., 2020) (Van de Cruys et al., 2014; Karvelis et al., 2018), and related work on weak central coherence (Happé & Frith, 2006). Phenomenologically, this manifests as detail-biased perception, reduced contextual modulation, and a world experienced as fragmented and hard to predict, with repetitive behaviors and insistence on sameness interpreted as attempts to carve out locally predictable niches (Cannon et al., 2021; Goris et al., 2018; Lawson et al., 2014).

Tarasi and colleagues (2022; 2023) build on this psychometric and clinical work by explicitly recasting SSD and ASD as endpoints of a predictive continuum. A further contribution of this model is to place environmental volatility estimation at the centre of that continuum. Both ASD and SSD struggle with learning in changing environments, but in opposite directions. Individuals on the ASD tend to treat the world as excessively volatile, assigning too much weight to momentary fluctuations and too little to stable regularities; prediction errors remain chronically high-precision, preventing the consolidation of robust, context-dependent expectations (Goris et al., 2018; Lawson et al., 2014; Tarasi, Martelli, et al., 2023). In contrast, psychosis-prone and SSD populations often behave as if the environment were more stable than it really is, clinging to initial hypotheses and under-weighting new evidence, a profile that maps neatly onto "jumping to conclusions" and bias against disconfirmatory evidence (Dudley et al., 2016; Mikus et al., 2025; Powers et al., 2025). In Tarasi et al.'s formulation, these complementary misestimates of volatility modulate the precision of priors and prediction errors across the hierarchy, further sharpening the diametric contrast between ASD and SSD. Against this backdrop, ASD and SSD can be understood as opposite predictive styles anchored at the poles of a continuum defined by how strongly internal models versus sensory data drive inference.

Situating FND along dimensions of predictive inference

Certain features of FND evoke the prediction-error-centered profile often described in autism research (i.e. heightened arousal, sensory overload, and a subjective sense of internal unpredictability) (Butler et al., 2021; Lawson et al., 2014). Findings of elevated ITPEs and reduced interoceptive accuracy in functional seizures (Koreki et al., 2020; Sojka et al., 2020, 2024) also imply noisy or unstable bodily predictions. However, many hallmark characteristics of FND point more convincingly toward a dominance of priors. Persistent symptom beliefs that resist correction despite repeated negative investigations, the habitual misinterpretation of benign bodily sensations, and the apparent over-precision of illness-related expectations all suggest a system in which top–down models exert greater influence than bottom–up signals (Edwards et al., 2012; Fiorio et al., 2022; Jungilligens et al., 2022). This is consistent with findings of altered probabilistic reasoning in patients with functional motor symptoms, who exhibit a "jumping to conclusions" bias and tend to draw inferences from insufficient information (Pareés, Kassavetis, et al., 2012).

This likely does not place FND midway between autistic and psychosis-spectrum profiles. Instead, it highlights FND as a distinct expression of predictive imbalance, one that, in our interpretation, leans toward an over-reliance on priors, particularly within the interoceptive and sensorimotor hierarchies. Illness-related concepts, learning histories, and biographical factors such as trauma or attachment might further shape the precision and content of these priors, rendering them particularly resistant to updating. Individual with FND appear primed to privilege certain expectations over prediction errors, such that tightly held priors continue to shape perceptual experience long after contradictory sensory evidence becomes available. The present study therefore aims to characterize autistic-like and schizotypal-like traits in adults with FND, using the Schizotypal Personality Questionnaire (SPQ; Raine, 1991) and the Autism Spectrum Quotient (AQ; Baron-Cohen et al., 2001) scores as dimensional proxies of predictive style. Within this framework, we expected that patients might exhibit a somewhat distinct trait configuration compared with healthy controls, broadly consistent with a relatively greater reliance on prior beliefs over sensory evidence. The advantage of this approach lies in its ability to move beyond disorder-specific descriptions and to situate

FND within a broader computational landscape. Rather than treating functional symptoms as an isolated clinical entity, mapping FND onto established dimensions of predictive-inference style allows us to test whether individuals with FND exhibit trait-level biases that could parallel (partially or divergently) those observed in other neuropsychiatric conditions. In doing so, it provides a structured lens through which to interpret heterogeneity and to refine mechanistic accounts of how altered inference may shape symptom experience. This approach builds directly on the theoretical integration outlined above and constitutes, to our knowledge, the first attempt to empirically map FND onto the broader dimensional space of predictive-inference strategies.

Methods

Participants

A total of 194 adults took part in the study, comprising 97 patients with FND (76 female, ≈ 78%; mean age = 42.62, s.d. 13.9) and 97 age- and gender-matched healthy controls (HC, 73 female, ≈ 75%; mean age = 45.66 s.d. 10.7). All participants were between 18 and 65 years of age and provided written informed consent prior to enrolment. The study was approved by the local institutional ethics committee and conducted in accordance with the Declaration of Helsinki. FND patients were recruited from specialized outpatient neurology and had received a diagnosis of FND from trained neurologists and according to current internationally accepted criteria based on positive clinical signs (e.g., inconsistency, incongruity). The sample included individuals with mixed FND symptomatology, such as functional motor symptoms, functional seizures, sensory disturbances. Inclusion criteria were: (i) a confirmed FND diagnosis; (ii) sufficient language proficiency to complete questionnaires; and (iii) absence of acute neurological or psychiatric conditions that could interfere with assessment. Exclusion criteria included: history of major neurological disease (e.g., epilepsy, multiple sclerosis, traumatic brain injury), intellectual disability, or unstable medical conditions.

Procedure

Data collection took place at the Department of Neuroscience, Biomedicine, and Movement Sciences of the University of Verona. Participants were asked to complete two self-report questionnaires to evaluate individual differences potentially linked to predictive coding styles; specifically, the SPQ and the AQ were used as measures of schizotypal and autistic traits, respectively, both of which have been associated with distinct predictive processing biases (Tarasi, Borgomaneri, et al., 2023; Tarasi et al., 2022).

Schizotypal traits were measured with the Schizotypal Personality Questionnaire (SPQ; Raine, 1991), a 74-item self-report measure that uses dichotomous "Yes"/"No" responses. The questionnaire is designed to capture a broad constellation of personality and cognitive features, spanning behavioral tendencies, perceptual experiences, and belief-related dimensions. These characteristics are organized into nine subscales: ideas of reference, magical thinking, social anxiety, unusual perceptual experiences, constricted affect, lack of close friendships, odd or eccentric behavior, peculiar speech, and suspiciousness. Each subscale contains between 7 and 9 items, producing scores that range accordingly. The subscale scores can be summed to derive a total SPQ score, which indexes the overall expression of schizotypal traits. In the literature, total scores of 41 or higher are commonly regarded as clinically meaningful (Compton et al., 2009; Raine, 1991).

Autistic traits were assessed using the Autism Spectrum Quotient (AQ; Baron-Cohen et al., 2001), a 50-item self-report questionnaire that employs a four-point response format (from "definitely disagree" to "definitely agree"). The items are distributed across five subscales reflecting core domains associated with autism spectrum conditions: imagination (capacity for imaginative thinking), communication (difficulties in communicative abilities), social skills (challenges in social interactions), attention to detail (heightened focus on perceptual or factual details), and attention switching (rigidity in shifting attention or a tendency toward strong, sustained focus). Each subscale includes 10 items scored from 0 to 10, and the subscale scores are summed to obtain a total AQ score. Higher total scores reflect a greater presence of autistic traits, with values of 32 or above typically considered clinically significant.

Statistical analysis

Because Study 2 relied on an available clinical cohort rather than prospective recruitment, no a priori sample-size calculation was performed. Instead, a post hoc sensitivity analysis was conducted in G*Power (v3.1) to estimate the minimum detectable effect size given the achieved sample size, assuming two-tailed tests, $\alpha = .05$, and 80% power (Faul et al., 2007).

All statistical analyses were conducted using two-tailed tests with an alpha level set at 0.05. Prior to group comparisons, the distributional properties of all variables were assessed.

Normality was evaluated through Shapiro-Wilk tests, while homogeneity of variance was examined using Levene’s test. As several variables showed deviations from equal variances, group differences between patients with FND and HC were examined using Welch’s *t*-tests, which provide robust estimates under heteroscedasticity.

Group matching was verified on key demographic variables. A chi-square test of independence was used to confirm that sex distribution (F/M) did not differ between groups. Equivalence in age was assessed by comparing mean age across groups using a Welch’s *t*-test. To control for multiple testing across subscales of the SPQ and AQ, *p*-values were adjusted using Bonferroni correction for 16 pairwise comparison. All analyses were performed using JASP (version 19.3).

Results

The sensitivity analysis indicated that, with n = 97 participants per group, an independent-samples t-test would provide 80% power to detect standardized group differences of approximately Cohen’s d = 0.40 at α = .05. Under more conservative thresholds used for multiple-comparison correction (e.g., α = .005), the minimum detectable effect increased to approximately d = 0.53. Thus, the available sample was adequately powered to detect small-to-moderate effects at the primary outcome level, while smaller subscale effects should be interpreted more cautiously.

Group comparisons between patients with FND and HC revealed significant differences on schizotypy and autistic traits (**Tab. 1**). Regarding schizotypal personality features, patients with FND obtained significantly higher scores on the SPQ total scale compared with healthy participants (p = 0.048) (**Fig. 6**). This effect was primarily driven by the SPQ Odd speech subscale, which showed robust group differences (p = 0.016).

Questionnaire/subscale	**Mean (s.d.)**		**t**	**df**	**p**	
	FND	**HC**				
SPQ_Total	19.23 (12.9)	14.35 (9.2)	3.008	171.960	0.048	*
SPQ_Ideas of reference	1.89 (2.1)	1.67 (1.6)	0.839	176.924	1	
SPQ_Magical thinking	1.33 (1.5)	1.27 (1.6)	0.480	190.981	1	
SPQ_Social anxiety	3.17 (2.4)	2.35 (2.2)	2.508	188.488	0.208	
SPQ_Unusual perceptual experience	1.50 (1.8)	1.34 (1.5)	0.649	183.246	1	

Questionnaire/subscale	Mean (s.d.)		t	df	p	
SPQ_Odd behavior	1.37 (1.8)	1.06 (1.4)	1.324	181.340	1	
SPQ_No close friends	2.09 (2.2)	1.43 (1.7)	2.286	179.774	0.368	
SPQ_Odd speech	3.56 (2.3)	2.11 (1.9)	4.688	186.700	0.016	*
SPQ_Constricted affect	2.07 (2.1)	1.48 (1.6)	2.201	175.672	0.464	
SPQ_Suspiciousness	2.22 (2.1)	1.67 (1.5)	2.047	168.939	0.672	
AQ_Total	18.22 (6.5)	21.82 (9.1)	-3.167	174.185	0.032	*
AQ_Imagination	4.13 (1.8)	4.15 (2.2)	-0.065	183.965	1	
AQ_Social skills	3.16 (2.2)	3.92 (2.7)	-2.138	183.467	0.544	
AQ_Attention switching	4.54 (2.2)	4.81 (2.1)	-0.876	189.862	1	
AQ_Attention to details	3.95 (2.1)	5.40 (2.2)	-4.723	190.869	0.016	*
AQ_Communication	2.43 (2.1)	3.53 (3.4)	-2.728	159.951	0.112	

Table 1 - Independent-samples t-tests (Welch's t-tests) comparing patients with FND and healthy controls on Schizotypal Personality Questionnaire (SPQ) and Autism Spectrum Quotient (AQ) total and subscale scores*. Reported are t values, degrees of freedom (df), and Bonferroni-corrected p-values (for 16 pairwise comparisons).*

Additional group differences were observed at the uncorrected level in the SPQ *Social anxiety* (p = 0.013) and SPQ *Constricted affect* (p = 0.029) subscales; however, these comparisons did not remain significant after correction for multiple testing. All other SPQ subscales – *Ideas of reference*, *Magical thinking*, *Unusual perceptual experiences*, *Odd behavior*, *No close friends*, and *Suspiciousness* – showed no significant differences between groups (all corrected p > 0.05).

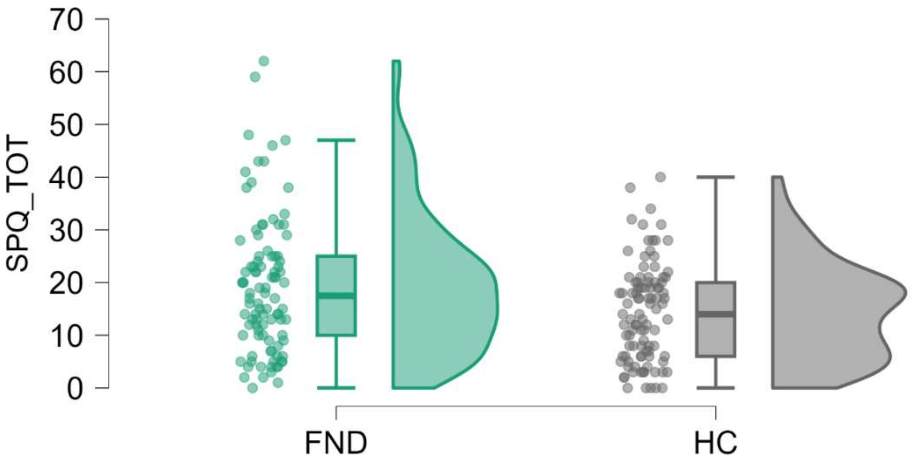

Figure 6 - SPQ total scores in FND and healthy controls*. Violin and box plots illustrate the distribution of Schizotypal Personality Questionnaire total scores. Boxes represent median and interquartile range; dots indicate individual observations. Higher scores were observed in the FND group..*

With respect to autistic traits, the AQ total score was significantly lower in the FND group compared with healthy controls (p = 0.032) (**Fig. 7**). At the subscale level, the only domain that reached significance after correction was AQ Attention to detail (p = 0.016), with patients showing reduced scores relative to controls. Differences observed in the AQ Social subscale at the uncorrected level (p = 0.034) did not survive multiple-comparisons correction, and no significant group effects emerged for Imagination or Attention subscales (all p > 0.05).

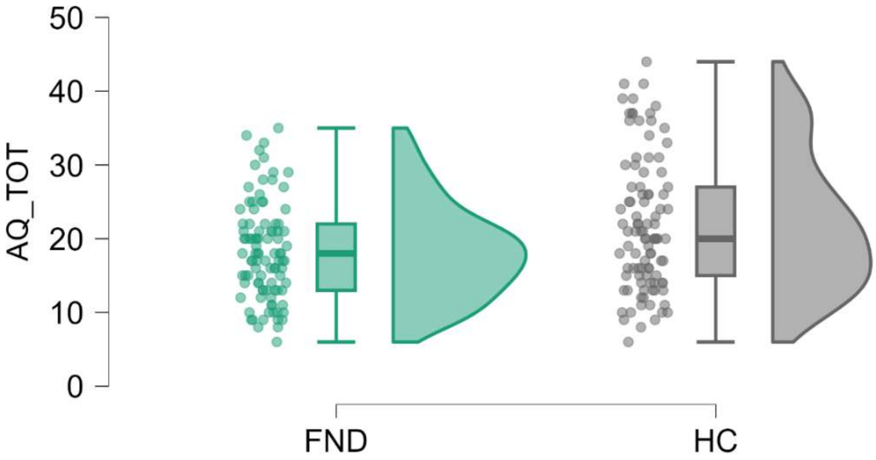

Figure 7 - AQ total scores in FND and healthy controls*. Violin and box plots illustrate the distribution of Autism Spectrum Quotient total scores. Boxes represent the median and interquartile range; dots indicate individual observations. Higher scores were observed in the healthy control group.*

Discussion

The present findings provide an initial empirical foothold for positioning FND within the broader landscape of predictive-inference styles. Although the theoretical sections of this paper outlined in detail how disruptions in allostasis, interoception, and concept-based emotion construction might bias inference in FND, the behavioral expression of these biases had remained largely speculative. By examining autistic-like and schizotypal-like traits, we aimed to determine whether FND carries a recognizable trait-level signature along this axis. The pattern that emerged aligns in meaningful ways with the mechanistic proposals discussed earlier.

The most robust difference concerned schizotypal features. Patients with FND scored higher on the total SPQ, with the Odd speech subscale driving this effect. Other subscales showed small, uncorrected elevations (particularly Social anxiety and Constricted affect) but these did not survive correction and should be interpreted cautiously. The narrowness of the effect is noteworthy. It suggests that FND is not broadly permeated by schizotypal traits, nor does it exactly overlap with the more diffuse profile often observed in psychosis-prone samples. Instead, the elevation in Odd speech may capture a subtler phenomenon: difficulties in articulating internal states, shifts in communicative pragmatics, or atypical ways of framing bodily experience. This is consistent with the emotion-construction framework outlined earlier, where impoverished concept use and coarse-grained affective categories can shape both subjective experience and its verbal expression (Barrett, 2017; Hoemann & Feldman Barrett, 2019; Jungilligens et al., 2022). In prediction-coding terms, reduced emotional granularity and imprecise interoceptive concepts may push individuals to rely on idiosyncratic or illness-colored interpretations of sensations, which in turn surface as irregularities in speech or narrative coherence.

At the same time, the SPQ profile does not resemble the pattern typical of psychosis spectrum conditions. We did not observe elevations in Ideas of reference, Magical thinking, Unusual perceptual experiences, or Suspiciousness – domains that, computationally, map onto overly precise high-level priors and aberrant belief updating (Adams et al., 2013; Corlett et al., 2009; Fletcher & Frith, 2009; Sterzer et al., 2018). If FND involves an over-reliance on priors, as our theoretical discussion argued, these priors likely appear to be specific to bodily threat, vulnerability, and symptom-specific templates rather than the more abstract or cosmological priors characteristic of SSD. This aligns with clinical observations: patients cling to illness-related expectations, but do not generally impose meaning on random stimuli or endorse implausible explanatory systems. The probabilistic-reasoning bias reported in functional motor symptoms, specifically a tendency to jump to conclusions based on minimal evidence (Pareés, Kassavetis, et al., 2012) dovetails with this picture: inference is skewed, but within a restricted semantic field defined by bodily concepts and symptom expectations.

The results for autistic traits pull in the opposite direction. Patients with FND showed lower AQ total scores and, more specifically, reduced Attention to detail compared to controls. The ASD profile is characterized by strong, precise sensory prediction errors, difficulty using priors to stabilize perception, and a perceptual style dominated by local features (Goris et al., 2018; Karvelis et al., 2018; Pellicano & Burr, 2012). In contrast, the diminished Attention to detail observed here hints at the opposite configuration: a reduced sensitivity to fine-grained sensory cues and a tendency to rely on broader, more abstract expectations. In computational terms, this would amplify the weight of priors relative to moment-to-moment sensory input, exactly the predictive mechanism proposed to underlie symptom persistence in FND (Edwards et al., 2012; Fiorio et al., 2022; Jungilligens et al., 2022).

This divergence between FND and ASD-like profiles also echoes the interoceptive findings discussed earlier. Elevated ITPEs, reduced interoceptive accuracy, and disturbances in insula-mediated prediction networks point to noisy, low-precision bodily signals (Koreki et al., 2020; Sojka et al., 2020). When sensory channels are unstable or ambiguous, the system may default to conceptually rigid priors that "explain away" this noise, particularly if illness-related concepts have acquired excessive precision through learning histories or repeated reinforcement. From this perspective, a reduced attention to detail is not an isolated cognitive trait but a downstream expression of a more global inferential style that de-emphasizes fine sensory evidence in favor of stable interpretative templates.

Importantly, the combination of higher schizotypal traits (in a limited set of domains) and lower autistic traits does not imply that FND sits between ASD and SSD, nor that it manifests a simple blend of their predictive signatures. Instead, it reinforces the argument advanced in the introduction: FND represents its own configuration of predictive imbalance: one marked by strong, symptom-centered priors and diminished reliance on granular sensory evidence, most probably lying within interoceptive and sensorimotor hierarchies.

The current findings offer preliminary support for this claim. They suggest that individuals with FND may occupy a region of the predictive continuum closer to the "priors-dominated" pole than to the "likelihood-dominated" one, but without the cognitive or belief-based distortions typical of psychosis-prone profiles. Finally, these results underscore the value of adopting a dimensional and mechanistic lens when studying FND. By situating functional symptoms within broad computational continua rather than disorder-specific silos, it becomes easier to link heterogeneous clinical presentations to concrete inferential processes, whether they relate to concept use, attention, sensory precision, or prior formation. Although the effects reported here are modest and require replication, they provide a first demonstration that FND is not psychologically agnostic with respect to predictive style. Trait-level tendencies in how individuals weigh priors and sensory evidence may shape vulnerability to symptom formation and influence how bodily signals are interpreted, maintained, and resisted.

In sum, the selective elevation of certain schizotypal traits, together with reduced autistic-like traits, coheres with a broader model in which FND reflects an over-reliance on high-level priors within a system struggling to integrate fine-grained sensory and interoceptive information. The profile uncovered here does not mirror that of ASD or SSD, but it aligns with the idea that altered predictive balance is a meaningful dimension along which FND can be mapped.

Limitations

Several limitations of the present study should be acknowledged. First, the design was cross-sectional and relied exclusively on self-report questionnaires, which restricts causal inference and may be influenced by response styles, insight, or current mood. SPQ and AQ scores are best understood as dimensional proxies of predictive style rather than as direct readouts of underlying computational parameters, and they cannot substitute for formal modelling or experimental tasks that probe learning and inference more directly. Second, although the achieved sample size provided sufficient sensibility for detecting group differences of small-to-moderate magnitude at the primary outcome level, it may have been underpowered to capture subtler effects at the subscale level, particularly after conservative Bonferroni correction for multiple comparisons. The absence of significant findings in several domains should therefore not be taken as definitive evidence of equivalence between groups.

Third, we did not systematically assess comorbid psychiatric conditions, medication use, or symptom severity, all of which might modulate questionnaire scores and interact with predictive styles. In particular, depressive and anxiety symptoms, trauma history, and dissociative phenomena are highly relevant in FND and could partially account for the observed trait pattern. Relatedly, patients were recruited from specialized neurology services, which may limit the generalizability of the findings to community or primary-care populations with functional symptoms. Finally, our approach focused on autistic-like and schizotypal-like traits as anchors on a predictive continuum derived from work on ASD and SSD (Tarasi et al., 2022). This necessarily narrows the lens: other dimensions (e.g. anxiety sensitivity, intolerance of uncertainty, or interoceptive beliefs) may capture complementary aspects of predictive imbalance in FND and warrant integration in future studies.

Conclusions

The study offers preliminary support for viewing FND through the lens of predictive processing. Within a framework that emphasizes allostatic regulation, interoceptive prediction, and concept-based emotion construction, patients with FND did not simply display a generic burden of psychopathology. Instead, they showed a selective profile marked by modest elevations in specific schizotypal traits and reduced autistic-like traits, consistent with a relative over-reliance on high-level priors and diminished weighting of fine-grained sensory evidence. While the effects are subtle, they converge with existing neurobiological and theoretical accounts in portraying FND as a distinct configuration of predictive imbalance, situated toward the “priors-dominated” end of the continuum but clearly separable from classical psychosis- or autism-spectrum profiles. Mapping FND onto this computational landscape may help to refine mechanistic models of symptom formation and, in the longer term, inform more targeted approaches to assessment and intervention.

Study 3: Theta–alpha asymmetry in FND: mapping trait profiles onto neural dynamics

Introduction

The previous chapter demonstrated that autistic-like and schizotypal-like traits provide a useful dimensional framework for characterizing predictive tendencies in FND. In a large cohort of patients with FND and matched controls, we observed a selective profile marked by low autistic-like traits and higher schizotypal features compared to healthy controls, suggesting a trait-level inclination toward high-level, concept-driven interpretations of bodily signals. These findings supported the broader theoretical perspective developed earlier in the thesis, in which altered interoception, allostasis, and conceptual processing bias inference toward stable priors rather than fine-grained sensory data.

A natural extension of this work is to ask whether these behavioral tendencies correspond to measurable physiological patterns. Resting-state EEG, and particularly frontal alpha and theta asymmetry, is widely used to probe neural processes underlying attention, cognitive control, and emotion regulation (Gordon et al., 2018; V. Perez et al., 2024; R. Smith, Badcock, et al., 2021), mechanisms that while not direct markers of predictive coding, are nevertheless relevant to how individuals integrate internal models with incoming evidence. Alpha asymmetry (difference in alpha power between hemispheres) is thought to primarily reflect cortical inhibitory control and sensory gating, indexing how efficiently neural systems suppress, filter, or prioritize incoming information at rest (Buchholz et al., 2014; E. E. Smith et al., 2017; Yoon & Kim, 2023). This regulatory role in shaping perceptual and attentional flow coexists with, but is conceptually distinct from, its well-documented associations with motivational and affective processes such as approach/avoidance tendencies, emotion regulation, and impulse control (Berretz et al., 2022; Kaur et al., 2020; J. Zhang et al., 2020). Although not diagnostically decisive on its own, frontal alpha asymmetry has also been linked to motor and cognitive symptoms in neurological disorders (da Rocha et al., 2025), as well as in some psychopathological conditions (Kaiser et al., 2018; Mathersul et al., 2008; Postema et al., 2019). Theta activity on the other hand, has been closely associated with cognitive control, error monitoring, and is thought to reflect the engagement of a common neural mechanism that signals the requirement for control and coordinates the updating of internal models, especially in the medial-frontal region (Cavanagh & Frank, 2014; Magosso et al., 2021; Tan et al., 2024).

To date, little is known about oscillatory dynamics in FND and whether (and if so, how) they relate to dimensional personality traits. Resting-state asymmetry appears able to capture narrow cognitive-behavioral dimensions within a diagnostic group, revealing relationships that would be obscured by broader categorical comparisons. Recent EEG research in autism, for instance, has shown that frontal alpha and theta asymmetry correlate with specific subdomains assessed through dedicated symptom-rating questionnaires, with each behavioral facet mapping onto a partly distinct, site-specific asymmetry profile (Bitsika et al., 2024). Although their study is grounded in traditional psychophysiology rather than predictive coding, the methodological strategy of linking oscillatory asymmetries to fine-grained behavioral indices appears suitable for the present investigation.

Building on this dimensional approach, the current study explores whether the trait configuration identified in the previous chapter has electrophysiological correlates in FND. We tentatively expected that individual differences in trait configuration might be accompanied by corresponding variations in resting-state oscillatory asymmetry, broadly consistent with differences in how sensory evidence and prior beliefs are weighted, while refraining from formulating strong directional predictions. Using resting-state theta and alpha asymmetry in a subsample of patients from the original AQ-SPQ cohort, we conducted an exploratory analysis to assess whether individual differences in trait-level tendencies (previously interpreted through a predictive lens) are mirrored by variations in oscillatory patterns that index how sensory input is gated or how uncertainty is monitored. Our objective is not to identify diagnostic biomarkers or to reduce EEG asymmetry to computational parameters. Instead, we aim to determine whether the behavioral expressions of predictive style observed in FND are reflected in resting physiology in a manner that complements the theoretical framework of this thesis. This integrated approach, linking dimensional traits and electrophysiology under a computational framework, may provide preliminary insight into the neurophysiological architecture that accompanies altered inference in FND.

Methods

Participants and procedure

For this investigation, we focused on a subsample of 22 FND patients (15 female, ≈ 68%; mean age = 36.55, s.d. 9.7) drawn from the original cohort described in the previous study and representing a mixed-symptom FND population, including individuals with functional motor symptoms, functional seizures, and functional sensory disturbances. These patients had completed the SPQ and AQ assessments and agreed to participate in an additional session consisting in a 5-minute resting-state EEG recording at the Department of Neuroscience, Biomedicine and Movement Sciences of the University of Verona. The session was scheduled after questionnaire completion, approximately within the same assessment period, ensuring that trait measures and EEG were temporally adjacent and derived from the same testing phase.

All participants from this subsample met the same inclusion and exclusion criteria applied to the original study. The EEG study received approval from the local institutional ethics committee and was conducted in accordance with the Declaration of Helsinki.

EEG acquisition and processing

EEG data was recorded using a 64-channel BrainVision ActiChamp system (BrainProducts GmbH, Munich, Germany) with a sampling rate of 1 kHz. Active electrodes were positioned according to the international 10–20 system. Electrode impedances were maintained at or below 5 kΩ throughout the recording. Given the sensory sensitivities frequently observed in this patient group, the experimenter minimized scalp abrasion and ensured that electrode preparation remained as gentle and non-invasive as possible. Recordings were obtained during a 5-minute eyes-open resting-state session, performed in a quiet, dimly lit room with participants seated comfortably and instructed to remain still, stay relaxed, and avoid excessive blinking or movements. No task was presented during the recording.

Preprocessing and data extraction were performed using BrainVision Analyzer 2. Continuous EEG data were band-pass filtered between 0.5 and 40 Hz. Ocular artefacts were corrected using the Gratton and Coles regression-based method (Gratton et al., 1983). The data were then segmented into non-overlapping 2-second epochs, and segments contaminated by residual artefacts were excluded from further analysis. Spectral power (μV^2) was computed using a fast Fourier transform (FFT) without windowing, and spectral power was extracted for the frequency bands of interest: theta (4-8 Hz) and alpha (8-12 Hz).

Asymmetry indices were then calculated by subtracting natural logarithm (base e)-transformed power obtained at the right side from that obtained at the left side (L-R formula) for each homologous electrode pair in frontopolar (FP1-FP2), frontal (F3-F4), frontocentral

(FC3-FC4), inferior frontal (F7-F8), central (C3-C4), centroparietal (CP3-CP4) and parietal (P3-P4) sites. Low or negative asymmetry scores indicate greater relative left-hemispheric activity in the alpha band, given that alpha power is inversely related to cortical activation (Metzen et al., 2022; Scheeringa et al., 2012). In contrast, asymmetry in the theta band does not follow this inverse relationship: higher theta power reflects greater functional engagement of medial and lateral frontal circuits (Asanowicz et al., 2021; van Noordt et al., 2022). Theta asymmetry scores therefore index relative differences in monitoring-related activity across hemispheres, rather than differences in cortical inhibition.

Statistical analysis

All statistical analyses were conducted using JASP (version 19.3). Associations between participants' age, EEG asymmetry indices and dimensional traits were examined using Spearman's rank-order correlations, given the non-parametric distribution of several questionnaire subscales as well as EEG data.

Because a large number of correlation coefficients were computed across multiple electrode pairs and questionnaire subscales, there was an increased risk of Type I error. At the same time, applying strict corrections for multiple comparisons in small samples can inflate Type II error and obscure potentially informative patterns. To balance these concerns, we adopted a combined criterion for identifying correlations of interest, following an approach previously applied in similar exploratory EEG-behavior studies (Bitsika et al., 2024). Specifically, associations were considered "meaningful" when they met both of the following conditions: i) a significance threshold of $p < .05$, and ii) an effect size of at least a medium magnitude ($|\rho| \geq .30$). This approach does not replace formal correction procedures but provides a pragmatic compromise for detecting interpretable patterns in exploratory correlational designs.

No formal a priori power analysis was performed. Power calculations for correlational EEG designs typically require assumptions about effect size and variance that are difficult to specify reliably in preliminary work, especially when no prior studies have examined SPQ-AQ dimensional traits and EEG asymmetry in FND specifically. In this context, the present analyses were conceived as an initial exploratory step, aimed at identifying potential relationships to be tested and refined in larger, confirmatory studies.

Results

Asymmetry data

Mean alpha and theta asymmetry data were calculated for each of the seven pairs of EEG sites and presented in **Fig. 8**. For the FP1-FP2 pair, both theta and alpha activity showed negative values, indicating greater power over the right hemisphere relative to the corresponding left-hemisphere site. A similar rightward dominance was observed for F3-F4, where asymmetry in both frequency bands again fell below zero. In contrast, FC3-FC4 exhibited a small positive theta asymmetry, reflecting slightly greater left-hemisphere theta activity, whereas alpha activity at the same sites showed a moderate negative value, pointing to relatively lower alpha power in the left hemisphere. For the F7-F8 and CP3-CP4 pairs, theta asymmetry values were modest and negative, with alpha activity likewise indicating right-greater-than-left power. At C3-C4 alpha activity displayed a substantial negative value, suggesting markedly greater right-hemisphere alpha power compared with the left. Finally, the P3-P4 pair showed consistent negative asymmetry for both theta and alpha bands, again reflecting rightward dominance.

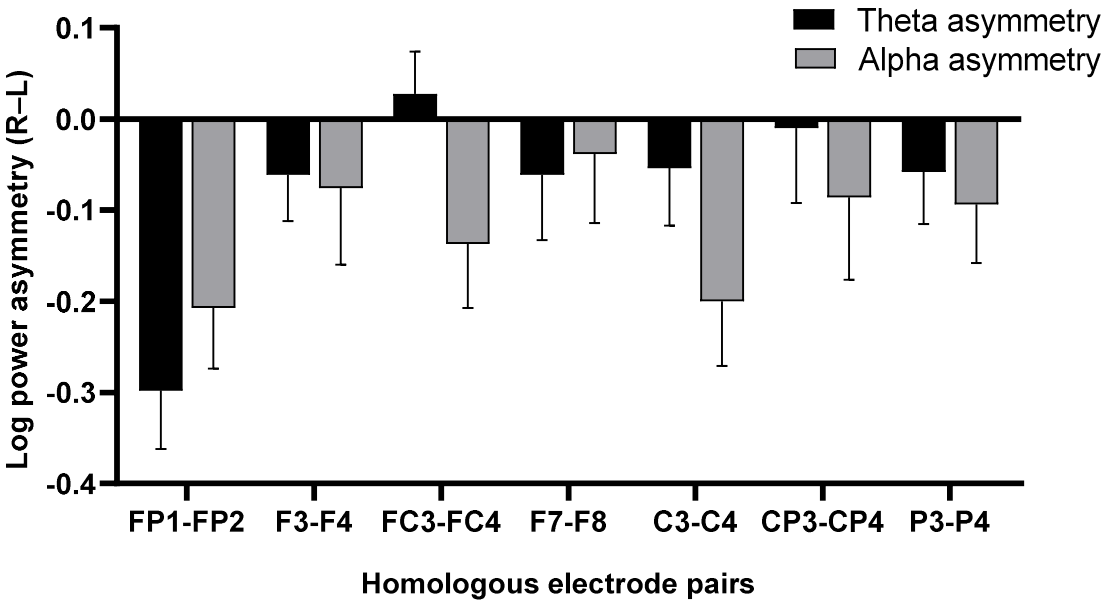

***Figure 8 - Mean EEG alpha- and theta-band power asymmetry indices across homologous electrode pairs**. Asymmetry was computed as log-transformed right minus left power (L–R). Positive values indicate relatively greater left-hemispheric power, whereas negative values indicate greater right-hemispheric power. Error bars represent ± SEM.*

Overall, asymmetry indices were predominantly negative, indicating greater right-hemispheric power for both bands. This pattern corresponds to relatively greater right-hemispheric activity for theta, whereas for alpha it reflects relatively greater left-cortical activation due to the inverse relationship between alpha power and neural activity (Metzen et al., 2022).

Associations between asymmetry indices and SPQ/AQ subscale scores

No meaningful associations were found between participants' age and any SPQ or AQ subscale or EEG asymmetry indices, indicating that age did not confound the variables of interest and that the sample could be treated as a single analytical group.

When examining the associations between dimensional traits and resting-state EEG asymmetry in FND patients, a distinct pattern emerged for autistic-like and schizotypal-like features (**Fig. 9-10**). Regarding the AQ, no subscale showed meaningful correlations with theta asymmetry in FND patients; all coefficients were below the effect-size threshold and nonsignificant. In contrast, several AQ dimensions were related to alpha asymmetry. Imagination was positively negatively associated with FC3-FC4 alpha asymmetry ($\rho = 0.465$, $p = 0.029$). The Attention to details subscale displayed the most robust pattern, correlating with F3-F4 ($\rho = 0.488$, $p = 0.021$), FC3-FC4 ($\rho = 0.584$, $p = 0.004$), and C3-C4 ($\rho = 0.474$, $p = 0.026$). Communication skills were also associated with FC3-FC4 asymmetry ($\rho = 0.466$, $p = 0.029$). No additional AQ–alpha relationships were detected.

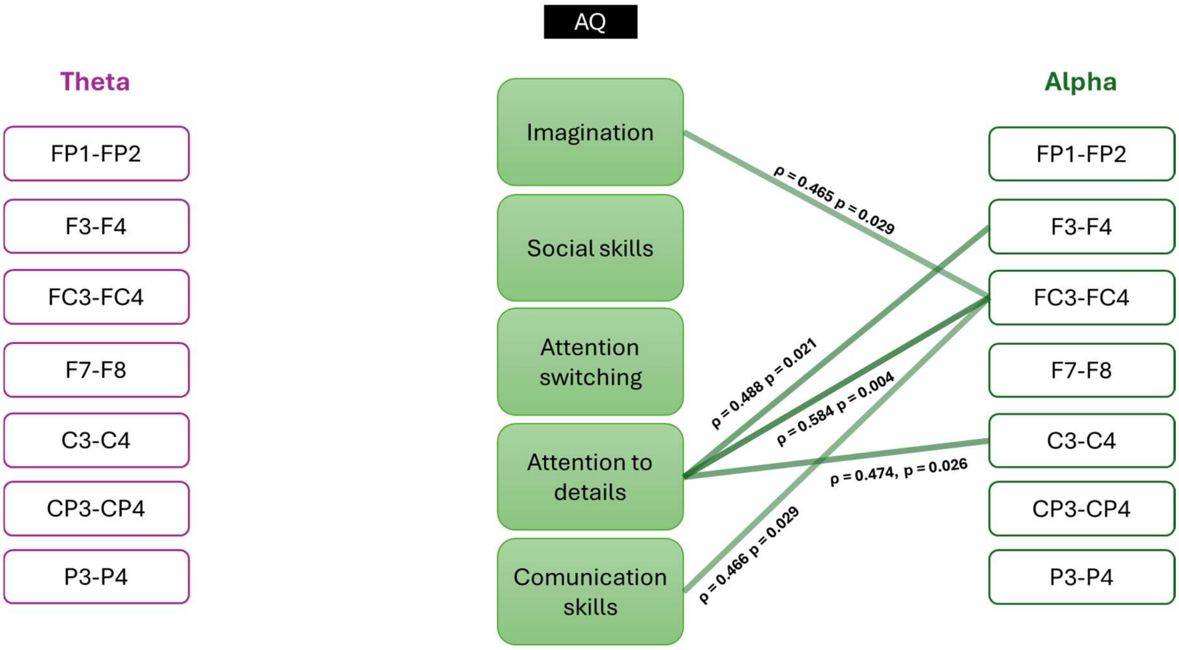

Figure 9 - Associations between AQ subscales and resting-state EEG asymmetry*. This diagram summarizes the pattern of statistically meaningful associations between AQ subscales and resting-state EEG asymmetry. Nodes represent questionnaire dimensions and electrode pairs, while connecting lines indicate significant correlations that exceeded both the significance threshold ($p < .05$) and a medium effect size ($|\rho| \geq .30$). Associations were observed in the alpha band only.*

A different profile was observed for the SPQ. Several schizotypal features were associated with theta asymmetry in FND. At the C3-C4 site, theta asymmetry correlated positively with Ideas of Reference (ρ = 0.451, p = 0.040), Odd behavior (ρ = 0.485, p = 0.026), Odd speech (ρ = 0.448, p = 0.042), and Suspiciousness (ρ = 0.524, p = 0.015). Schizotypal traits also showed a smaller set of associations with alpha asymmetry: Suspiciousness correlated with both FC3-FC4 (ρ = 0.527, p = 0.012) and F7-F8 (ρ = -0.444, p = 0.038). No other SPQ subscales were significantly related to alpha asymmetry.

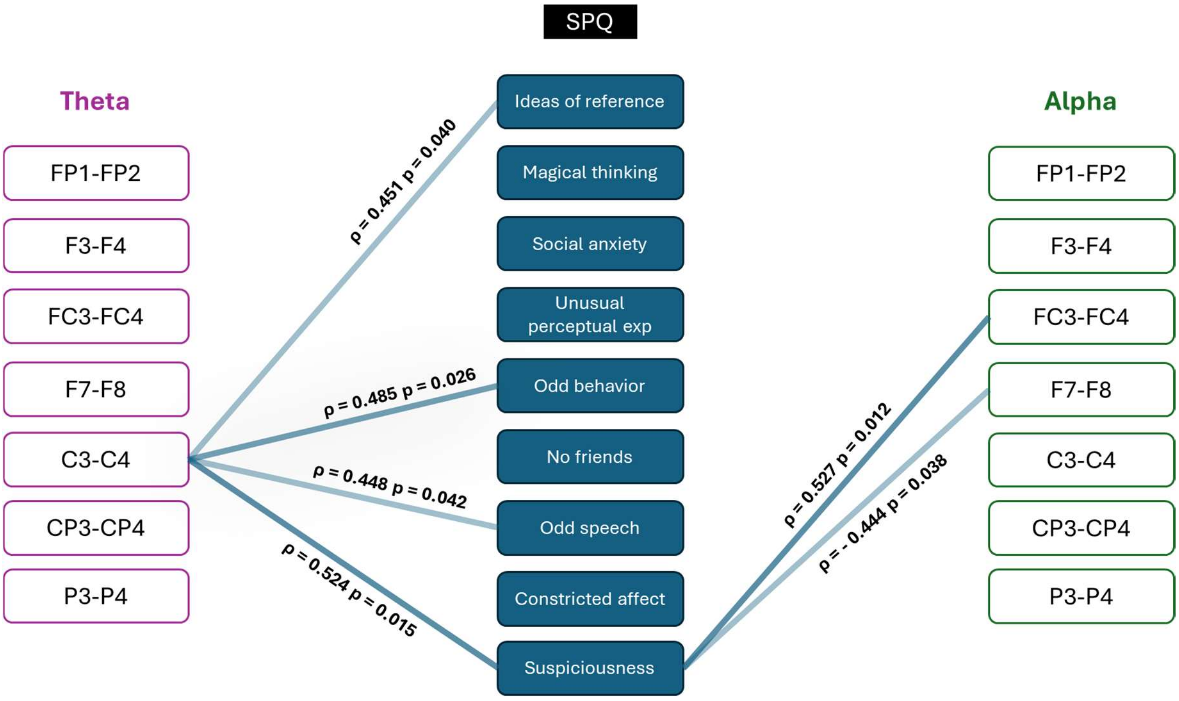

Figure 10 - Associations between SPQ subscales and resting-state EEG asymmetry*. Nodes represent questionnaire dimensions and electrode pairs, and connecting lines indicate correlations meeting the predefined criteria ($p < .05$, $|\rho| \geq .30$). Associations were observed in both the theta and alpha bands.*

Discussion

The present study investigated, in an exploratory and preliminary manner, whether the dimensional tendencies identified in the previous chapter (i.e. specifically, higher schizotypal traits and reduced autistic-like traits) find correspondence in resting-state oscillatory asymmetry within FND. By examining correlations between SPQ and AQ subscales and alpha/theta asymmetries in a subsample of the original cohort, our aim was not to identify definitive physiological markers but to explore whether these trait variations map onto distinct patterns of spontaneous neural organization. Although the findings should be interpreted cautiously given the modest sample size and intentionally exploratory nature of the analyses, a remarkably coherent and informative pattern emerged, revealing differentiated oscillatory signatures for autistic-like and schizotypal-like traits within the FND group that might offer preliminary physiological support for the broader predictive interpretation developed throughout the thesis.

It is noteworthy that the asymmetry estimates both in the alpha and the theta frequency bands were consistently negative, indicating a predominance of right-hemispheric power across sites, a pattern not previously described in the context of FND; however, the interpretation of this finding is far from straightforward. For theta, greater power likely reflects relatively greater right-hemispheric oscillatory activity, whereas for alpha, higher right-hemispheric power corresponds to relatively lower cortical activation due to the well-established inverse relationship between alpha power and neural activity (Metzen et al., 2022). Hemispheric alpha asymmetries have been reported across a wide range of affective and arousal states, including acute stress and hyperarousal (Berretz et al., 2022; Crost et al., 2008), but also in contexts associated with positive affect and adaptive emotional regulation (Hannesdóttir et al., 2010; M. Lee et al., 2020), with findings varying markedly across populations and experimental conditions. Given this heterogeneity, the current asymmetry pattern cannot be taken as a disorder-specific marker. It therefore remains an interesting descriptive feature of our dataset, but one that warrants cautious interpretation and should be revisited in future studies with larger samples and finer-grained affective and contextual measures.

The most robust effects involved significant positive correlations between different schizotypal subscales (Ideas of Reference, Odd Behavior, Odd Speech, and Suspiciousness) and theta asymmetry at C3-C4. Fronto-central theta rhythms are widely implicated in the detection of mismatch, the evaluation of ambiguous or uncertain stimuli, and the engagement of cognitive monitoring systems (Cavanagh & Frank, 2014; Magosso et al., 2021; Tan et al., 2024). Their asymmetric expression at rest in individuals with higher schizotypal tendencies suggests a baseline propensity to engage in interpretive vigilance or to allocate disproportionate monitoring resources to internal or external cues. This resonates with the psychological profile of these SPQ domains, all of which involve, in different ways, difficulties contextualizing information, managing ambiguity, or modulating salience attribution; indeed, this pattern is consistent with the behavioral findings of the previous chapter, which showed selective elevations in SPQ domains associated with interpretive and attentional biases. The oscillatory signature observed here provides a physiological complement to that behavioral profile, suggesting that even modest schizotypal tendencies in FND may shape how neural systems organize themselves at rest when evaluating internal and external cues.

Within a predictive framework, such a pattern may mirror the subtle differences in how individuals with FND balance top-down expectations and the monitoring of potentially unexpected inputs. Although theta asymmetry cannot be equated with prediction-error signalling, resting differences in monitoring-related rhythms may nonetheless reflect how comfortable (or uncomfortable) an individual is with uncertainty. The finding that referential ideation, behavioral oddity, linguistic disorganization, and suspiciousness converge on the same theta-based signature could reflect a shared underlying tendency: a predisposition toward interpretive vigilance, increased sensitivity to signal-noise ambiguity, or a baseline tilt toward monitoring rather than sensory openness.

The association between Suspiciousness and both theta and alpha asymmetry (respectively, at central and fronto-central sites) points toward an especially interesting mechanism. Whereas theta effects imply altered monitoring of ambiguous or potentially threatening cues, alpha asymmetry likely reflects differences in sensory gating and inhibitory control. Their combination may indicate that individuals high in suspiciousness exhibit both an interpretive bias and an altered way of filtering incoming information. In the context of FND, where bodily sensations are frequently misinterpreted or imbued with exaggerated salience, this dual oscillatory profile may be particularly relevant. It suggests that suspiciousness, even when expressed dimensionally and not pathologically, may influence how sensory inputs and internal bodily signals are weighted and integrated. An asymmetric configuration of both monitoring and sensory gating mechanisms could, in principle, contribute to the heightened salience assigned to ambiguous sensations that is characteristic of the disorder.

The AQ results painted a markedly different picture. In contrast to the SPQ results, autistic-like traits showed associations exclusively with alpha asymmetry at frontal and central sites. This profile was consistent across multiple AQ subscales, including Imagination, Attention switching, Attention to detail, and Communication skills. Alpha-band activity is strongly implicated in selective inhibition, sensory gating, and the allocation of attentional resources. Associations between AQ traits and alpha asymmetry therefore point toward differences in how sensory information is filtered and how attentional channels are opened or closed at rest. Importantly, no AQ subscale showed any meaningful association with theta asymmetry. This dissociation suggests that, within FND, autistic-like tendencies are not expressed through alterations in conflict monitoring or mismatch detection but instead through variability in sensory gating and attentional inhibition.

This is particularly consistent with the behavioral profile described earlier, where compared to healthy controls, FND patients showed reduced Attention to detail, an AQ dimension typically associated with heightened sensory precision. The alpha-based signature observed here reinforces the notion that autistic-like traits in FND, when present, manifest as subtle differences in sensory regulation rather than as cognitive-monitoring biases.

Taken together, the oscillatory findings reveal a structured and theoretically meaningful dissociation: on the one hand, schizotypal traits align with theta asymmetry, indicating differences in monitoring, interpretation of ambiguity, and salience attribution, with additional alpha contributions in Suspiciousness. On the other hand, autistic-like traits align exclusively with alpha asymmetry, suggesting differences in sensory gating and attentional allocation, but no involvement of mismatch-monitoring processes.

This dissociation likely echoes the dimensional profile obtained in the full cohort and provides converging physiological evidence that autistic-like and schizotypal-like traits within FND reflect genuinely distinct cognitive tendencies rather than two expressions of a single underlying dimension. The fact that schizotypal traits engage rhythms associated with the evaluation of ambiguous information, while autistic-like traits engage rhythms tied to gating and inhibition, offers a physiological complement to the behavioral distinctions observed earlier. Furthermore, although resting-state oscillations cannot be straightforwardly interpreted as neural correlates of priors or prediction errors, this pattern is compatible with the broader predictive account developed in the thesis. If individuals with FND tend to rely more heavily on high-level expectations and less on fine-grained sensory detail, then differences in monitoring asymmetry (theta) could shape how ambiguous bodily signals are interpreted, while differences in alpha asymmetry could shape how those signals are filtered in the first place. Although speculative, this interpretation may hold theoretical relevance, situating the oscillatory findings in interaction with the broader predictive tendencies outlined in previous chapters and contribute to the heterogeneity observed in FND phenomenology.

Limitations

Several methodological considerations are worth outlining in order to contextualize the present findings and anticipate potential concerns about interpretability, design, and statistical approach. Although the study adopts a dimensional framework inspired by predictive coding accounts, the link between resting-state alpha/theta asymmetry and inferential processes should be interpreted cautiously. Oscillatory asymmetries are not direct readouts of precision weighting or prior–likelihood balance, and their functional interpretation remains partly inferential. We do not claim that alpha or theta asymmetry instantiate predictive coding parameters per se; instead, we conceptualize them as low-level physiological indicators of processes (e.g. sensory gating, attentional and emotional regulation) that have been repeatedly implicated in predictive-inference models.

With respect to the decision to restrict the analysis to the patient group, this choice follows directly from the nature of the research question. The trait deviations of interest are specific to individuals with FND, and the mechanistic aim is to determine whether these traits align with characteristic oscillatory patterns within this population. Examining correlations in healthy controls would not meaningfully inform this question, as controls do not display the same trait profiles and therefore cannot serve as a theoretically valid comparison group for investigating trait–physiology coupling. The EEG patients' group was a subsample of adequate representativeness of the full behavioral cohort. However, the reduced sample size limits sensitivity to small effects and introduces the possibility of confounding from affective symptoms, trauma history, dissociation, and medication use, factors common in FND and known to affect resting-state dynamics. Accordingly, null findings should be interpreted with caution.

Conclusions

This exploratory study suggests that subtle variations in autistic-like and schizotypal-like traits within FND correspond to distinct patterns of resting-state oscillatory asymmetry. Autistic-like traits are associated with sensory–attentional dynamics indexed by alpha asymmetry, whereas schizotypal traits involve both monitoring-related theta asymmetry and sensory gating alterations. Although preliminary, these findings offer a physiological complement to the behavioral dissociation identified earlier and provide a basis for future work to examine how oscillatory regulation, attentional mechanisms, and interpretive biases shape bodily inference in FND. Findings of this kind may ultimately prove valuable in clarifying the heterogeneity of FND presentations and identifying pathways through which cognitive–perceptual tendencies influence symptom formation.

Study 4: Individual differences in predictive mechanisms: implications for interoception

Introduction

As discussed in the previous chapters, interoceptive inference occupies a central position in contemporary accounts of FND because it likely constitutes the foundation upon which the brain constructs affect, agency, and bodily experience (Jungilligens et al., 2022; Khalsa et al., 2018). As we have outlined, in the constructionist and predictive frameworks interoception is not conceived as a passive registration of visceral signals, but as an active, hierarchical process in which the brain continually generates predictions about the internal state of the body and tests them against ascending sensory input (Barrett & Simmons, 2015; Katsumi et al., 2022). When this inferential loop becomes biased by overly precise priors, conceptually impoverished, or insufficiently updated, the resulting mismatch between expected and actual bodily states can produce the unstable affective tone, persistent fatigue, and hyperarousal so characteristic of FND (Edwards et al., 2012; Fiorio et al., 2022; Jungilligens et al., 2022).

In studies 2 and 3, I have suggested that FND may be understood as a disorder of predictive regulation, arising from chronic mismatches between internal predictions, bodily signals, and the conceptual models through which the organism constructs affective meaning. Patients with FND showed elevated schizotypal traits and reduced autistic-like traits relative to healthy controls, suggesting a disposition toward prior-weighted perceptual strategies. Furthermore, analyses of resting-state EEG asymmetry revealed associations between these trait measures and neural markers of affective-motivational processing, further supporting the presence of a characteristic bias in the way predictive models and sensory evidence are integrated.

In this sense, symptoms would emerge not from the “conversion” of latent emotions but from the failure to construct accurate internal models. Such distortions also explain why functional symptoms resemble a spontaneous nocebo responses: symptom-related expectations shape the interpretation of ambiguous bodily cues, those interpretations reinforce the priors from which they arose, and prediction errors that could correct the model are either down-weighted or misinterpreted (Bakvis et al., 2009; Fiorio et al., 2022).

Moreover, disruptions in interoceptive inference help account for the high rates of alexithymia, dissociation, and “panic without panic” episodes observed in FND, all of which reflect compromised alignment between visceral signals and conceptual categories (Butler et al., 2021; Goldstein & Mellers, 2006; Pick et al., 2020).

Given this theoretical scaffolding, a natural next step is to ask whether trait-level differences in predictive style such as those linked to schizophrenia-spectrum disorders (SSD) and autism-spectrum disorders (ASD) dispositions are reflected in how interoceptive predictions are generated and updated. This question is situated within the wide body of work conceptualizing schizotypal traits as indexing a tendency toward prior-dominated inference, whereas autistic traits are thought to align with a sensory-weighted style of processing, as extensively covered in the Introduction to Study 2. However, this evidence comes almost entirely from studies of exteroception; far less is known about whether these predictive tendencies extend to the interoceptive domain.

If SPQ and AQ truly index opposite inferential strategies, they should be accompanied by distinct patterns also in the discrepancy between expected and perceived bodily states. A recently proposed metric, the Interoceptive Trait Prediction Error (ITPE), offers precisely the possibility to test this, capturing the mismatch between subjective beliefs about one’s interoceptive ability and objective performance on interoceptive accuracy tasks (Koreki et al., 2020).

In light of these considerations, the present study builds directly on the pattern observed in FND and sets out to examine whether individual differences in schizotypal and autistic traits (as proxies of predictive styles) map onto systematic variability in interoceptive prediction error even in the absence of clinical symptoms: if higher schizotypal traits in FND coincide with a prior-weighted predictive style, then healthy individuals with similarly elevated schizotypy might exhibit larger interoceptive prediction errors, reflecting the same inferential bias. Autistic traits, in turn, being positioned at the sensory-weighted end of the continuum, are not expected to exhibit the same association with ITPE.

To test this hypothesis, we measured schizotypal and autistic traits in a non-clinical sample using the SPQ and the AQ alongside a behavioral task assessing cardiac interoceptive accuracy. Together with the administration of the Body Perception Questionnaire (Porges, 1993), this allowed us to compute the ITPE and ultimately to explore whether such discrepancy relates to broad dispositional tendencies in predictive style.

Methods

Participants

Forty-six healthy individuals participated in the study (12 female; mean age = 41.3 years, s.d. = 16.3). The sample size was determined via a priori power analysis conducted with G*Power3 (Faul et al., 2007) based on two-tailed Pearson's correlation, indicating a minimum of 46 participants was required assuming a medium effect size ($\rho = 0.40$), an alpha level of 0.05, and a desired statistical power of 0.80. The study was approved by the Human research approval committee of the University of Verona and conducted in accordance with the Declaration of Helsinki.

Procedure

Data collection was conducted within the Department of Neuroscience, Biomedicine, and Movement Sciences at the University of Verona. Each participant completed a behavioral task and a set of questionnaires in a fixed order (**Fig. 11**).

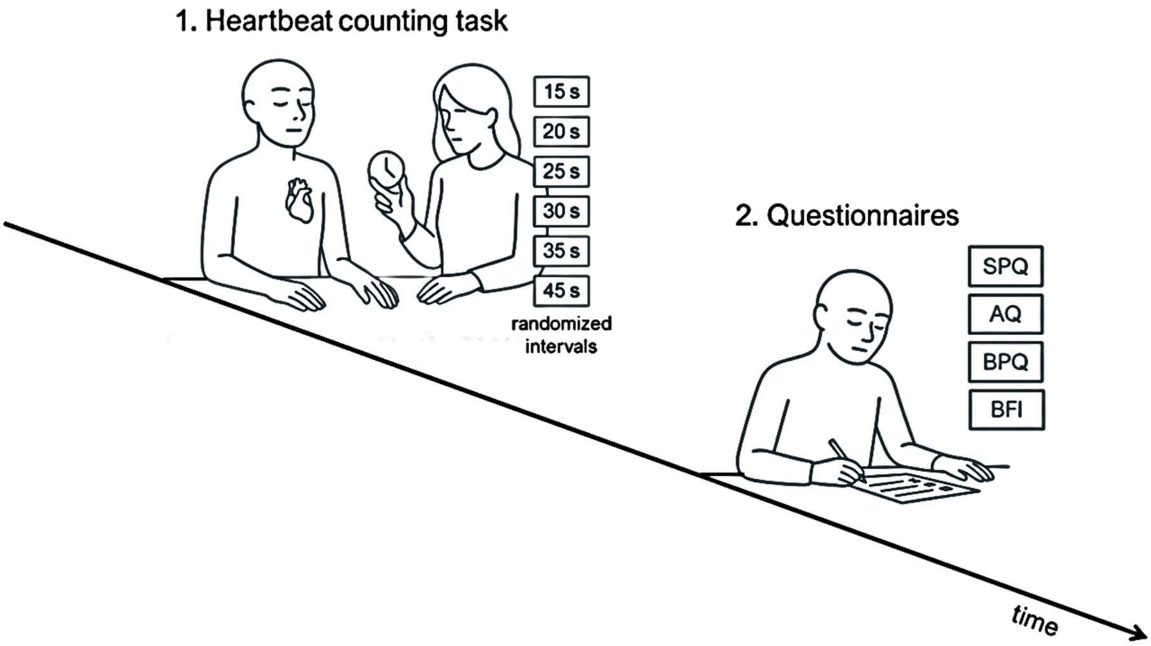

Figure 11 - Experimental procedure *– Participants first completed a heartbeat counting task, estimating their heartbeats across randomized time intervals (15–45 s), followed by the administration of self-report questionnaires assessing schizotypal traits (SPQ), autistic traits (AQ), body perception (BPQ), and personality dimensions (BFI).*

The session opened with the Heartbeat Counting Task (HCT), used here to obtain an index of objective interoceptive accuracy (IAcc) (Schandry, 1981). The task comprised six intervals of varying lengths, presented in random order and ranging from 15 to 45 seconds (Schandry, 1981; Sojka et al., 2020). Participants sat comfortably and were instructed to count their heartbeats internally, without touching their body or using external cues of any kind. At the end of each interval, they reported the number of heartbeats they believed they had perceived and rated their confidence on a four-point scale: 1 ("I did not feel any heartbeat; I guessed completely"), 2 ("I felt something, but I am not sure it was my heartbeat; confidence is low"), 3 ("I perceived my heartbeat occasionally or weakly; the count is only partly based on sensation"), and 4 ("I clearly perceived my heartbeat and am confident in my estimate"). Actual cardiac activity was monitored continuously with a Polar H10 chest sensor.

After the HCT, participants completed a questionnaire battery. The subjective interoceptive component of ITPE was derived from the Body Awareness subscale of the Body Perception Questionnaire (BPQ; Porges, 1993), which taps into trait-like beliefs about sensitivity to internal bodily signals (Koreki et al., 2020). Developed within the framework of polyvagal theory and later refined in a shorter form (Cabrera et al., 2018), the BPQ consists of 22 items covering awareness of autonomic stress-related sensations, rated from 1 ("never") to 5 ("always").

Schizotypal and autistic traits were assessed with the SPQ and AQ, measures already introduced in the previous chapters and included here because of their established links with predictive-processing styles (Tarasi et al., 2022; Tarasi, Martelli, et al., 2023).

Finally, the Big Five Inventory (BFI; Fossati et al., 2011; O. P. John et al., 2012) was administered as a control measure to account for potential contributions of broad personality traits . The BFI includes 44 items rated from 1 ("strongly disagree") to 5 ("strongly agree") and yields scores for Extraversion, Agreeableness, Conscientiousness, Neuroticism, and Openness to experience. Because the Big Five shows no consistent association with interoceptive accuracy or predictive-processing tendencies (Erle et al., 2021; Pearson & Pfeifer, 2022), its inclusion allowed us to verify that any relationships emerging for ITPE were not reducible to general personality dimensions.

Derivation of interoceptive measures

Interoceptive Accuracy (IAcc) was computed for each participant using the standard heartbeat perception formula, which quantifies the discrepancy between perceived and actual cardiac counts across the six intervals:

$$\mathrm{IAcc} = \frac{1}{6}\sum_{i=1}^{6}(1 - \frac{\mathrm{reported}_i}{\mathrm{actual}_i})$$

where *reported*$_{(i)}$ denotes the number of heartbeats estimated for interval i, and *actual*$_{(i)}$ corresponds to the objectively recorded heartbeats for that same interval. The resulting index ranges from 0 to 1, with higher values reflecting greater correspondence between perceived and actual heartbeats. Scores above approximately 0.5 are typically interpreted as indicating performance above chance (Schandry, 1981).

The interoceptive trait prediction error (ITPE) captures the mismatch between individuals' objective interoceptive performance and their subjective beliefs about bodily sensitivity (Koreki et al., 2020). To compute this index, both the HCT IAcc scores and the Body Awareness subscale of the BPQ were first transformed into standardized Z-scores. For each participant, ITPE was then derived by subtracting the objective score from the subjective one:

$$\mathrm{ITPE} = \mathrm{BPQ}_{\mathrm{awareness}} - \mathrm{IAcc}$$

Higher, positive values indicate that participants perceive themselves as more interoceptively sensitive than their actual performance suggests. Within an active inference framework, such overestimation is viewed as evidence for overly rigid top–down priors that fail to adjust appropriately to incoming bodily signals (Sojka et al., 2020).

Statistical analysis

In line with the theoretical framework guiding our hypotheses, the primary analyses examined the relationships among ITPE, IAcc, and the total scores of the SPQ and AQ. Because these correlations were specified a priori, we used Spearman's rank-order coefficients and applied a Bonferroni correction based on the six planned comparisons, yielding an adjusted significance threshold of $\alpha = 0.008$. Variables showing significant associations in this first step were subsequently entered into linear regression models, with

SPQ or AQ total scores treated as separate dependent variables. Age was included as a covariate in these models, given its documented association with schizotypal traits (Bora & Baysan Arabaci, 2009) and to ensure that any observed effects could not be attributed to demographic confounding.

We then carried out additional analyses exploring the relationships between ITPE, AQ, SPQ, the five BFI personality dimensions, and IAcc. These correlations served an exploratory or control function and were therefore corrected independently for multiple comparisons using the Bonferroni method across 28 tests (adjusted $\alpha = 0.0018$). This separation between confirmatory, theory-driven analyses and broader exploratory tests was determined in advance so as to limit Type II errors in the former, while maintaining an appropriate false-positive rate for the latter. Effect sizes were evaluated as Fisher's *z*. All statistical tests were two-tailed, and analyses were conducted using JASP version 19.3.

Results

Spearman's rank correlations revealed a clear association between schizotypal traits and interoceptive prediction errors. Higher SPQ total scores were reliably linked to larger ITPE values ($\rho = 0.399$, $p = 0.006$, Fisher's $z = 0.423$) (**Fig. 12A**). In practical terms, participants with more pronounced schizotypal features showed greater mismatches between actual and perceived interoceptive abilities, overestimating the latter. No analogous relationship emerged for autistic traits: ITPE did not correlate with AQ total scores ($\rho = 0.105$, $p = 0.486$, Fisher's $z = 0.106$) (**Fig. 12B**), with any of the BFI dimensions, or with interoceptive accuracy obtained from the heartbeat counting task (all $p > 0.05$).

Given this pattern, we examined whether schizotypal traits could prospectively account for variability in ITPEs. A linear regression model with ITPE as the outcome and SPQ total scores as the sole predictor reached significance ($F(1,43) = 6.49$, $p = 0.014$), accounting for 13.1% of the variance ($R^2 = 0.131$; adjusted $R^2 = 0.111$). The effect of SPQ scores was positive and statistically robust (unstandardized coefficient = 0.061, SE = 0.024, $\beta = 0.369$, $t = 2.55$, $p = 0.014$), indicating that each additional SPQ point corresponded, on average, to a 0.061-unit increment in ITPE. Importantly, introducing age as a covariate did not attenuate the effect, implying that the association was not explained by age differences within the sample. In contrast, when AQ total scores were entered into an equivalent model, the regression did not approach significance (F(1,44) = 0.88, p = 0.355; R^2 = 0.020; adjusted R^2 = –0.003).

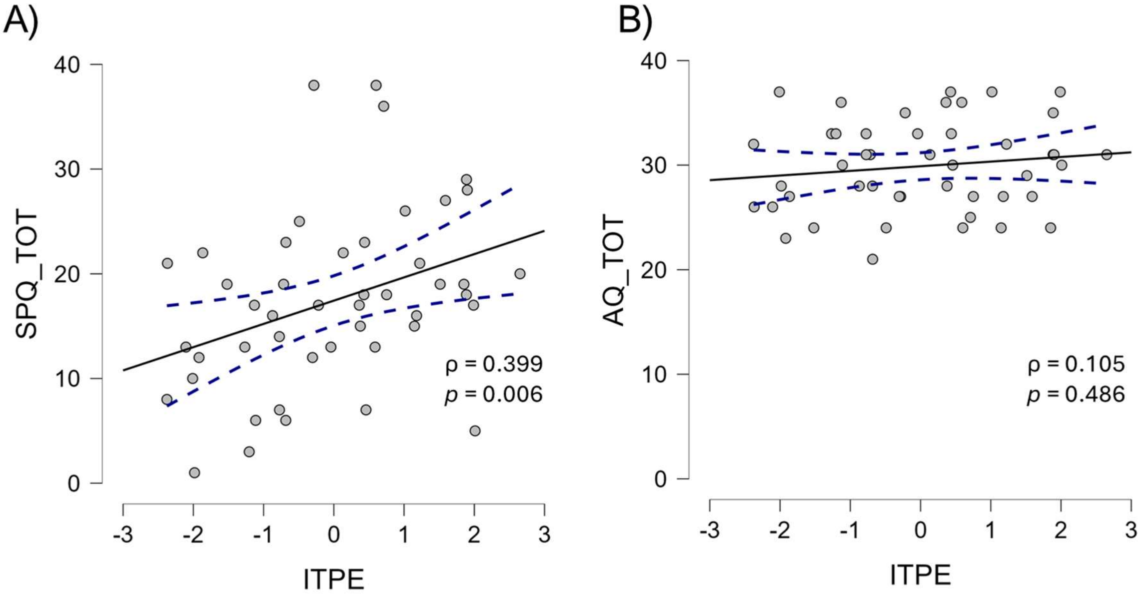

Figure 12 - Associations between ITPE and SPQ/AQ total scores*. Scatterplots show the relationships between Interoceptive Trait Prediction Error (ITPE) and total scores of the Schizotypal Personality Questionnaire (SPQ_TOT) and the Autism Spectrum Quotient (AQ_TOT), assessed using Spearman's correlations. Solid lines indicate fitted regression trends, and dashed lines represent 95% confidence intervals.*

Discussion

The study revealed a robust positive association between schizotypal traits and ITPE, an effect that remained evident when examined through linear regression. Interpreted within a predictive-coding perspective, this pattern suggests that individuals with higher schizotypy scores show an imbalance in how prior expectations and sensory evidence are weighted during the interpretation of bodily signals. In practical terms, they appear to place disproportionate confidence in their preconceived beliefs about their interoceptive abilities (typically an overestimation) while giving comparatively less weight to the actual afferent input from the body. This mismatch between expected and perceived cardiac signals results in larger interoceptive prediction errors.

Such an interpretation sits comfortably alongside computational approaches to psychosis, which describe SSD conditions as states in which priors become excessively precise or sensory evidence is down-weighted, leading to maladaptive inference and chronically unresolved prediction errors (Fletcher & Frith, 2009; Sterzer et al., 2018; Stuke et al., 2021). In these frameworks, many positive symptoms (e.g. hallucinations, delusional meanings imposed on neutral events) are conceptualized as the behavioral outcome of an overreliance on internal predictions relative to sensory data (Baker et al., 2019; Horga & Abi-Dargham, 2019). Our findings extend this reasoning to the internal bodily domain: elevated schizotypy was associated with greater interoceptive prediction error, implying that a prior-dominated perceptual style may also characterize how these individuals interpret their own visceral signals.

This reading resonates with recent proposals situating schizophrenia-spectrum and autism-spectrum traits at opposite poles of a predictive-processing continuum (Tarasi et al., 2022; Tarasi, Martelli, et al., 2023). On this continuum, the SSD end is marked by overly influential priors (Fletcher & Frith, 2009; Haarsma et al., 2020; Tarasi et al., 2025), whereas the ASD end is characterized by stronger adherence to sensory evidence and relatively weaker contextual predictions (Andersen, 2022; Pellicano & Burr, 2012). The positive correlation between SPQ and ITPE observed here aligns neatly with this framework, indicating that a similar prior-weighted pattern may extend to interoception.

In contrast, autistic traits did not show any detectable relationship with ITPE. This absence of association warrants caution in interpretation, particularly given the diametric model that partly motivated the study. If autistic traits genuinely reflect a tendency toward sensory-weighted inference, one might expect lower or negative ITPE scores (i.e. underestimation of interoceptive ability). Yet ITPE, as it is typically operationalized, is probably more responsive to positive deviations (overestimation) than to negative ones (underestimation), and the possibility of negative values has received little systematic attention (Koreki et al., 2020; Sojka et al., 2020). It is therefore plausible that the metric itself is better suited to capturing overconfidence than underconfidence, which may obscure any relationship with ASD traits.

A methodological consideration further supports this interpretation. Variability in AQ scores was markedly lower than in SPQ scores, as reflected in their respective coefficients of variation (SPQ = 0.49; AQ = 0.14). Such constrained variance substantially limits the ability to detect meaningful correlations, offering a plausible statistical explanation for the null result. This is consistent with previous work showing that, in non-clinical samples, autistic traits often exert subtle or heterogeneous influences on interoceptive processes, with mixed findings across studies examining interoceptive accuracy (DuBois et al., 2016; Williams et al., 2023).

A further element emerging from our data concerns the selectivity of the link between schizotypal traits and interoceptive predictive processing. The association was confined to ITPE, the composite measure capturing the mismatch between subjective estimations and behavioral performance, and did not generalize to interoceptive accuracy or to broader personality indices. SPQ scores showed no relationship with objective IAcc derived from the heartbeat counting task, nor with any of the BFI dimensions. This pattern argues against a generic tendency among individuals high in schizotypy to report bodily sensations more intensely or to display a diffuse personality configuration. Instead, it points toward a more circumscribed alteration affecting the integration of predictive models with interoceptive input. In other words, an SSD-like predictive strategy may distort the weighting assigned to interoceptive signals without necessarily undermining the capacity to detect them at a perceptual level. This view is congruent with accounts describing schizophrenia-spectrum conditions as disorders of inference rather than impairments in perceptual fidelity (Uhlhaas & Mishara, 2007). It also highlights that the trait-level prediction error assessed by ITPE taps a higher-order cognitive dimension (i.e. the integrity of one's internal model) rather than basic perceptual accuracy.

The regression analyses strengthened this interpretation. SPQ scores emerged as a significant predictor of ITPE, explaining close to 13% of the variance in interoceptive prediction error. Although modest, this effect size indicates that schizotypal disposition contributes meaningfully to individual differences in interoceptive predictive integration.

Taken together, these findings demonstrate that schizotypal traits are linked to, and help account for, increased interoceptive prediction errors, consistent with a prior-weighted predictive style characteristic of the SSD end of the continuum. Framed in this way, even subclinical levels of schizotypy reveal measurable differences in how the brain negotiates sensory information, including bodily ones. The divergence observed between actual and perceived interoceptive performance in individuals with higher SSD traits suggests that enlarged prediction errors stem from a rigid internal model that resists updating in light of sensory evidence, rather than from broadly imprecise priors.

Limitations

Some methodological aspects should be kept in mind when interpreting these findings. Interoceptive accuracy was derived from the Heartbeat Counting Task; although this task remains one of the most commonly used tools in the field, it cannot be considered a direct or process-specific index of cardiac afferent sensitivity, as performance may partly reflect expectations about heart rate, time estimation abilities, or broader cognitive strategies rather than interoceptive precision alone (Desmedt et al., 2025; Ring & Brener, 2018). For this reason, raw accuracy values are best treated with caution. At the same time, the use of self-report questionnaires introduces the possibility of shared-method variance and subjective biases (Brenner & DeLamater, 2016).

Conclusions

Overall, the results support the broader claim that variability in predictive coding strategies, likely present even at the level of personality traits, shapes systematic differences in how bodily states are interpreted and experienced. Importantly, although the present study was conducted in healthy individuals, establishing these relationships in the absence of clinical symptoms provided a useful interpretive baseline for the findings reported in FND in earlier chapters. If trait-level predictive tendencies already leave a measurable signature in interoception under typical conditions, this strengthens the rationale for examining the same inferential processes directly in FND in future work, where additional factors such as illness-related priors, attentional biases, and allostatic strain may further shape interoceptive prediction.

General conclusions and final remarks

Functional neurological disorders have long resisted unifying explanations. Their clinical presentation has historically encouraged dichotomous interpretations: either the symptoms reflect an invisible neurological lesion or they arise from psychological conflict. The work developed across this thesis suggests that a different approach is needed, one that considers brain function not as a collection of isolated modules but as a dynamic, inferential system that operates across multiple temporal and computational scales.

The first part of the thesis showed that FND are associated with a pattern of neural alterations that is neither random nor confined to a single module. Neuroimaging studies document consistent deviations in motor control circuits, emotion and salience systems, interoceptive hubs, and their reciprocal connectivity. Crucially, these findings extend beyond static measures. The EEG microstate study demonstrated that the temporal architecture of resting brain activity itself is altered in FND. Microstate G, associated with somatosensory and sensorimotor networks, exhibited reduced temporal stability; microstates corresponding to arousal, imagery, and self-referential processing displayed atypical transition tendencies. These results likely suggest that the spontaneous flow of neural activity in FND is subtly but distinctly reorganized compared to healthy controls: the system spends less time in configurations that support coherent sensorimotor integration, and moves between cognitive-affective modes in ways that could shape how bodily signals are processed and experienced. This observation resonates strongly with broader accounts of FND that describe altered agency (Maurer et al., 2016; Pareés, Saifee, et al., 2012), disrupted sensory attenuation (Pareés et al., 2014), and inconsistent integration of proprioceptive signals (Hallett et al., 2022). The temporal dysregulation revealed by microstates supports the idea that the generative models underlying sensorimotor experience in FND are not merely imprecise or misguided, but dynamically constrained.

At first glance, these observations might appear only loosely connected to the second part of the thesis, which provides the inferential counterpart to these neurophysiological observations. Yet the bridge between them becomes clear when we consider the core assumptions of predictive processing: the brain continuously generates predictions about sensory input; compares them to incoming evidence; and updates its internal models to minimize prediction error. This updating depends on precision-weighting, the assignment of confidence to priors and sensory signals. When priors become overly precise, or when sensory evidence is down-weighted or noisy, the system may become locked into self-fulfilling predictions. In such cases, even accurate sensory evidence may fail to revise entrenched models. The empirical studies examining schizotypal and autistic-like traits, theta–alpha asymmetry, and interoceptive prediction errors in healthy individuals show that individuals with FND might occupy a point on the predictive landscape characterized by over-commitment to particular expectations and reduced capacity to adjust these expectations in response to sensory contradictions.

When finally considering together both neural dynamics and predictive inference, a coherent picture emerges. The temporal instability of microstates suggests a system whose landscape has become overly deep in certain regions and flattened in others. From the perspective of the free-energy principle (Feldman & Friston, 2010; Friston, 2010), this corresponds to a state of pathologically low model entropy: the system has fewer viable hypotheses available to explain sensory input, and it sticks rigidly to the ones it has (Parr & Friston, 2017). In Bayesian terms, priors become too precise; symptom persistence, variability, and context-dependence are all natural consequences of such a configuration.

Implications for clinical practice

This perspective also opens a space for considering how these mechanisms might intersect with clinical practice. What physiotherapists, psychologists, occupational therapists, and neurologists do in practice appears heterogeneous: they shift attention, elicit novel movements, challenge beliefs, modify sensory expectations, encourage interoceptive focus, introduce reframing, or provide contextual novelty. The theoretical framework adopted in this thesis suggests beneath this surface-level diversity lies a shared computational logic. They might act by modulating the inferential landscape in which symptoms are maintained. Nearly all these interventions operate by loosening the precision of the maladaptive priors that maintain symptoms, or by amplifying the sensory prediction errors that could revise them. The result is a temporary but crucial increase in the entropy of the generative model: not in the colloquial sense of chaos, but in the formal sense used within information theory and predictive processing: an increase in the range of possible hypotheses the system is willing to consider (Friston & Kiebel, 2009).

Therapeutic change involves the deliberate introduction of a controlled amount of entropy into predictive hierarchies that have become excessively constrained. What makes this concept compelling is that it emerges naturally from the free-energy framework without requiring any new theoretical machinery. Entropy, within predictive processing, is simply the measure of uncertainty or distributional breadth over possible states or explanations (Parr & Friston, 2017). A system with excessively precise priors has artificially low entropy; a system able to revise those priors has higher entropy in precisely the domains where updating is needed. Thus, interventions that loosen rigid predictions, diversify prior beliefs, or heighten the precision of incoming sensory evidence all work by modulating entropy (Chamberlin, 2019; Nair et al., 2020; Sheffield et al., 2024).

This reframes the therapeutic process as one in which clinicians help patients re-establish flexibility, meaning the capacity of the generative model to move, explore, and update (Sarasso et al., 2022, 2024). The flexibility is not imposed from the outside; rather, it emerges because the therapeutic context provides a safe space for uncertainty, a temporary expansion of the inferential "degrees of freedom" available to the system. The most effective treatments may therefore be those that optimize this flexibility without overwhelming the system: those that carefully titrate uncertainty, rather than imposing it abruptly.

Physiotherapy exemplifies this mechanism particularly well. Effective movement retraining for FND does not merely strengthen muscles or correct flawed movement patterns. Instead, it creates a stream of carefully structured prediction errors: unexpected ease of motion, novel sensory feedback, movements performed automatically rather than via explicit monitoring, and tasks that shift attention away from the symptomatic limb (Espay et al., 2018; Gandolfi et al., 2024; Macías-García et al., 2024). Each of these manipulations undermines the precision of the prior that "the limb cannot move" or "movement will fail," while simultaneously increasing the precision of sensory evidence that contradicts that prior. The implicit nature of these tasks, often involving dual attention, external focus, or goal-directed actions, likely further reduces the influence of rigid top-down predictions. Psychotherapeutic interventions could operate through analogous mechanisms, challenging overly precise beliefs about symptoms, redirecting attention, and encourage behavioral experiments that yield disconfirmatory prediction errors (Sarasso et al., 2022, 2024; R. Smith, Moutoussis, et al., 2021). Even the act of explanation, when offered in clear, mechanistic terms, may play a role within this framework. Providing patients with a simple model, such as the "wire and lightbulb" metaphor commonly used in FND (where the wiring is intact but the current flow is disrupted), introduces an alternative account that can compete with their pre-existing interpretations (Bailey et al., 2024; Cope et al., 2021). By offering a coherent, non-damage-based explanation, such metaphors may reduce the precision of entrenched catastrophic beliefs and create conditions more favorable to updating prior expectations and supporting new learning.

What connects all these interventions is not their method but their function. In a sense, therapy becomes a structured opportunity for the brain to "relearn how to learn", in other words to re-engage the mechanisms of exploration, updating, and flexibility that are fundamental to adaptive behavior. Viewed in this way, the therapeutic mechanisms outlined above simply reflect, in the clinical domain, the broader argument developed across the thesis, translating its inferential principles into concrete therapeutic strategies.

Taken together, the empirical work and theoretical synthesis presented in this thesis support a reconceptualization of FND as conditions emerging from the interplay between altered network dynamics and disrupted predictive inference. Their symptoms are neither arbitrary nor symbolic, but reflect the computational principles through which the brain structures experience. The microstate findings, behavioral data, and dimensional measures converge on the view that FND do not stem from a single deficit but from a particular configuration of perceptual, motor, and interoceptive processing. By grounding these observations within a predictive-processing framework, the thesis shows how subtle differences in neural temporal structure and predictive integration might generate the complex phenomenology characteristic of the disorder. This perspective highlights the multidimensional nature of FND and frames them as a distinct mode of brain–body inference that is dynamic, interpretable, and amenable to a deeper scientific understanding. Ultimately, the conclusions presented here suggest that recovery in FND depends less on symptom suppression and more on restoring flexibility within the predictive system itself. When the brain regains the capacity to explore and revise its models, new trajectories of perception and action become available. It is the re-opening of the inferential system (and its capacity to explore, revise, and accommodate new evidence) that permits new trajectories of perception and action. This perspective not only integrates neuroscience, psychology, and clinical practice but also provides a new foundation for developing refined, mechanistically informed treatments.

Several considerations temper the interpretation and clinical scope of the present findings. Although the alterations in neural dynamics, interoceptive inference, and trait-related predictive styles observed across the studies are consistent with contemporary models of

FND, they are unlikely to constitute disorder-specific signatures. Similar disruptions in large-scale network coordination, bodily self-monitoring, and precision weighting have been documented across a range of psychiatric and neurological conditions. From this standpoint, the mechanisms described here may be better understood within a transdiagnostic framework, reflecting dimensional variations in brain–body inference rather than categorical markers uniquely tied to FND. Conceptualizing FND along such continua – for instance in terms of relative reliance on prior beliefs, sensory evidence, and interoceptive prediction – may offer a more parsimonious account of symptom formation while situating the disorder within a broader landscape of shared neurocognitive vulnerabilities. Future research should therefore move beyond comparisons with healthy controls alone and incorporate both psychiatric and neurological clinical groups to disentangle general vulnerability processes from features that are relatively specific to FND.

From a clinical perspective, the implications of this work should be considered primarily at a mechanistic and conceptual level. The measures examined here are not intended as diagnostic tools or immediate markers for individual decision-making. Rather, they contribute to a more fine-grained understanding of the processes that may shape symptom expression – such as altered bodily inference, attentional allocation, or network instability – and may help guide future translational research aimed at developing targeted assessment strategies and interventions. In this sense, the principal contribution of the thesis lies in offering an explanatory framework that bridges neurophysiology, behavior, and clinical phenomenology, thereby providing a foundation for subsequent studies explicitly designed to test clinical utility.

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