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Not Defined by FND

Functional Neurological Disorder as a Disorder of Network Integration and Predictive Inference

Functional Neurological Disorder (FND) presents with genuine, often disabling neurological symptoms—e.g., limb weakness, tremor, seizures—without a structural lesion that accounts for them. Modern practice emphasizes positive clinical signs (e.g., Hoover's sign, tremor entrainment) rather than a "rule-out-everything" diagnosis (Hallett et al., 2022; Voon et al., 2016). Historically cast as "psychological," contemporary models frame FND as disrupted integration across large-scale brain networks involved in bodily awareness, movement planning, agency, attention/salience, and executive control (Stone et al., 2021; Perez et al., 2021). This paper argues that FND symptoms emerge from aberrant network interactions, not a single damaged node (Stone et al., 2021).

A useful unifying perspective is predictive processing: symptoms reflect maladaptive priors/expectations and attentional weighting applied to interoceptive and sensorimotor signals. In FND, abnormal precision (confidence) may be assigned to misleading internal cues, destabilizing perception of the body and control of action. Here, "precision" (or "confidence") refers to how strongly the brain weights a signal or prediction when deciding what is happening; too much precision on noisy internal cues can overpower corrective sensory evidence and bias experience toward non-movement or non-agency (Stone et al., 2021; Perez et al., 2021).

The Salience (Cingulo-Insular) Network (SN/CIN)

The salience network acts like the brain's "relevance filter," deciding what deserves attention right now and shifting resources accordingly. Its hubs, the anterior insula and anterior cingulate cortex (ACC), link body signals (like heartbeat, muscle tension, visceral sensations) with emotion and goal-directed control (Craig, 2009; Stone et al., 2021). In FND, this filter appears overreactive for internal threat cues: interoceptive sensations are marked as highly important, attention is drawn inward, and competing goals (like fluid movement) get crowded out. Empirically, this shows up as atypical coupling between salience hubs and

sensorimotor/control networks and aligns with common clinical features such as anxiety and difficulties identifying/labeling internal states (alexithymia) (Hallett et al., 2022; Perez et al., 2021; Aybek et al., 2015). The issue is not a linear cause-and-effect sequence but a broader miscalibration in how the system assigns importance to internal versus external cues, especially under uncertainty (Stone et al., 2021).

Supplementary Motor Area (SMA) and Movement Initiation

The supplementary motor complex—encompassing pre-SMA and SMA proper—coordinates the preparation, sequencing, and initiation of voluntary movement, particularly when actions are internally generated rather than purely stimulus-driven. In a predictive-processing frame, these regions help specify forward models of the intended action and issue efference copies that predict the sensory/proprioceptive consequences of the movement (Stone et al., 2021). Successful execution depends on a tight communication loop among pre-SMA/SMA, primary motor cortex, basal ganglia, cerebellum, and parietal comparators, with top-down precision signals determining which motor intentions come to fruition (Voon et al., 2016; Perez et al., 2021).

In FND, available evidence is most consistent with an instability at the interface between intention and enactment of movement. Preparatory activity can be present but expressed weakly in behavior, suggesting a decoupling between motor readiness and the subjective experience of willing the movement (Edwards et al., 2011; Maurer et al., 2016). Altered coupling between SMA/pre-SMA and parietal–temporal nodes (including agency-related regions) fits a picture in which prediction and feedback are not being integrated with the usual confidence. When the salience network biases attention toward internal threat signals, this mis-calibration is amplified: the system prioritizes monitoring and “checking” over fluid execution, and the intended movement is either inhibited or experienced as not truly self-generated (Hallett et al., 2022; Perez et al., 2021).

This account supports why physiotherapy that minimizes self-focused monitoring and restores automaticity can be effective. External cueing (rhythm, targets), dual-tasking, graded complexity, and an external focus of attention reduce the maladaptive precision assigned to interoceptive “error” signals and allow the SMA–parietal–cerebellar communication loop to

re-stabilize. When movement is shaped under conditions that discourage excessive internal error monitoring, individuals more readily re-experience actions as intended and voluntary (Stone et al., 2021; Hallett et al., 2022).

Temporoparietal Junction (TPJ) and Sense of Agency (SoA)

The temporoparietal junction (TPJ) contributes to the sense of agency (SoA) by integrating multisensory predictions (derived from efference copies) with the reafferent feedback, which is the sensory information that returns to the brain following actions. Positioned at the confluence of visual, vestibular, and somatosensory streams, the TPJ helps maintain a coherent body schema and evaluates whether outcomes match intentions (Perez et al., 2021). Agency, in this view, emerges when predicted and observed consequences are sufficiently aligned and weighted with appropriate precision (Baek et al., 2022; Stone et al., 2021).

Findings in FND indicate abnormal TPJ participation in these comparisons, with connectivity to sensorimotor and frontal control regions altered across cohorts and phenotypes. Rather than a single direction of effect, the pattern suggests a mismatch in how predictive and feedback signals are integrated and trusted. When salience circuitry up-weights bodily noise and control networks fail to recalibrate, TPJ computations are performed on noisy inputs with skewed precision, increasing the likelihood that self-generated actions feel alien. The clinical phenomenology of “I can’t make it move,” or “it moves but not by my will,” naturally follows from this imbalance: the comparator does not recognize the action as self-produced, even when the motor system can execute it (Weber et al., 2025; Baek et al., 2022; Hallett et al., 2022).

Therapeutic strategies that restore reliable prediction–feedback contingencies therefore make mechanistic sense. Techniques that provide accurate, synchronous visuo-proprioceptive feedback (e.g., action observation, video feedback, mirror-based tasks when appropriate) and that grade exposure to self-initiated movement under an external focus help re-calibrate TPJ computations. As the system repeatedly experiences consistencies between intended and observed outcomes without threat-biased monitoring, agency strengthens and symptoms subside (Stone et al., 2021; Hallett et al., 2022).

Dorsolateral Prefrontal Cortex (DLPFC) / Frontoparietal Control

The dorsolateral prefrontal cortex (DLPFC) is an important node within the frontoparietal control network that provides top-down control, helping the system decide how much weight to give interoceptive and sensorimotor signals at any moment. In a predictive-processing frame, this involves calibrating the precision of competing hypotheses about bodily states and actions. When this control is compromised, misleading internal cues (amplified by salience circuitry) retain excessive influence, and corrective evidence is underweighted. Clinically, that looks like persistent convictions of "something is wrong with my body" and a persistent sense of non-agency, even when motor pathways are capable of producing movement (Stone et al., 2021; Perez et al., 2021).

Converging work places the DLPFC as the link between attention allocation, conflict monitoring, and metacognitive updating, processes that should suppress maladaptive prior expectations and support re-estimation when predictions fail. In FND, alterations within this network can weaken the system's ability to down-regulate threat-biased interoception and to reconcile prediction errors arising from disrupted interactions among the salience network, supplementary motor regions, and temporoparietal nodes. The result is not simply a failure to move but a failure to re-experience movement as intended and self-generated. This model complements evidence of abnormal SMA–parietal/TPJ coupling by specifying how DLPFC-mediated control would ordinarily stabilize intention–action coherence and re-establish confidence in voluntary control (Baek et al., 2022; Perez et al., 2021).

Therapeutically, this positioning of DLPFC within the control network explains why explanatory models, attention-shifting physiotherapy, and CBT-informed strategies can be effective: they all work, in part, by retuning precision and re-weighting misleading bodily predictions under active, goal-directed control. It also motivates cautious exploration of adjunctive neuromodulation aimed at control or agency nodes as potential stabilizers of the network dynamics that support accurate bodily inference, as a complement to behavior-based retraining (Perez et al., 2015; Stone et al., 2021).

Discussion: A Systems-Level Account

FND appears to be an emergent property of disrupted integration among salience, sensorimotor/agency, and control networks. Explored through predictive processing, symptoms arise when maladaptive priors and attentional weighting are applied to interoceptive and sensorimotor evidence, producing compelling but misleading inferences about the body—for example, that a limb cannot move or that a tremor is entirely involuntary. Clinically, abnormalities within the salience network bias attention toward internal signals and threat, differences in coupling between SMA/pre-SMA and parietal/TPJ nodes destabilize the coherence between intention and action and erode the SoA, and alterations in frontoparietal control limit the system's capacity to re-weight misleading priors or to update beliefs in the face of contradictory evidence (Stone et al., 2021; Hallett et al., 2022). This account does not require the nervous system to be "normal" in every respect, but it emphasizes that the characteristic symptoms reflect network-level dysfunction rather than a focal lesion (Perez et al., 2021; Voon et al., 2016).

Future Directions and Conclusion

Progress depends on moving beyond static group comparisons toward methods that reveal how signals flow through the system. Dynamic connectivity analyses, coupled with computational models of predictive coding and precision weighting, can show when and where salience, sensorimotor, and control networks miscommunicate during symptom expression and recovery. Studies should also stratify participants by phenotype, such as functional weakness, tremor, or seizures, because connectivity patterns and treatment responses likely differ across these groups (Hallett et al., 2022). Practically, that means planning studies in advance with clear hypotheses, ensuring adequate sample sizes, and measuring both how the brain–behavior mechanisms change and whether patients actually improve (Perez et al., 2021).

Within this framework, neuromodulation can be tested as an adjunct to physiotherapy and CBT-informed rehabilitation rather than as an independent intervention, with protocols targeted to specific nodes (for example, rTPJ, motor, or control centers) and evaluated for their ability to stabilize network interactions that support accurate bodily inference and voluntary control (Perez et al., 2015; Stone et al., 2021).

References

Aybek, S., Nicholson, T. R., O'Daly, O., Zelaya, F., Kanaan, R. A., et al. (2015). Emotion–motion interactions in conversion disorder: An fMRI study. *PLOS ONE, 10*(4), e0123273. doi: 10.1371/journal.pone.0123273

Baek, K., Donamayor, N., Morris, L. S., Strelchuk, D., Mitchell, S., Mikheenko, Y., ... & Voon, V. (2022). The self-agency network: Neural correlates of sense of agency in healthy controls and patients with functional neurological disorder. *Brain and Behavior, 12*(9), e2733. doi: 10.1002/brb3.2733

Craig, A. D. (2009). How do you feel—now? The anterior insula and human awareness. *Nature Reviews Neuroscience, 10*(1), 59–70. doi: 10.1038/nrn2555

Edwards, M. J., Moretto, G., Schwingenschuh, P., Katschnig, P., Bhatia, K. P., & Haggard, P. (2011). Abnormal sense of intention preceding voluntary movement in patients with psychogenic tremor. *Neuropsychologia, 49*(9), 2791–2793. doi: 10.1016/j.neuropsychologia.2011.05.021

Hallett, M., Aybek, S., Dworetzky, B. A., McWhirter, L., Staab, J. P., & Stone, J. (2022). Functional neurological disorder: New subtypes and shared mechanisms. *The Lancet Neurology, 21*(6), 537–550. doi: 10.1016/S1474-4422(21)00422-1

Maurer, C. W., LaFaver, K., Ameli, R., Epstein, S. A., Hallett, M., & Horovitz, S. G. (2016). Impaired supplementary motor area inhibition in functional movement disorders: A preliminary study. *PLOS ONE, 11*(7), e0158556. doi: 10.1371/journal.pone.0158556

Perez, D. L., Dworetzky, B. A., Dickerson, B. C., Leung, L., Cohn, R., Baslet, G., & Silbersweig, D. A. (2015). An integrative neurocircuit perspective on psychogenic nonepileptic seizures and functional movement disorders: Neural functional unawareness. *Clinical EEG and Neuroscience, 46*(1), 4–15. doi: 10.1177/1550059414555905

Perez, D. L., Nicholson, T. R., Asadi-Pooya, A. A., Begue, I., Butler, M., Carson, A. J., David, A. S., Deeley, Q., Diez, I., Edwards, M. J., Espay, A. J., Gelauff, J. M., Hallett, M., Horovitz, S. G., Jungilligens, J., Kanaan, R. A., Tijssen, M. A., & Aybek, S. (2021). Neuroimaging in functional neurological disorder: State of the field and research agenda. *The Lancet Psychiatry, 8*(10), 826–841. doi: 10.1016/S2215-0366(21)00303-3

Stone, J., Carson, A., & Hallett, M. (2021). A framework for understanding the pathophysiology of functional neurological disorder. *The Lancet Psychiatry, 8*(9), 819–829. doi: 10.1016/S2215-0366(21)00343-4

Voon, V., Cavanna, A. E., Coburn, K., Sampson, S., Reeve, A., & LaFrance, W. C., Jr. (2016). Functional neuroanatomy and neurophysiology of functional neurological disorders (conversion disorder). *Journal of Neuropsychiatry and Clinical Neurosciences, 28*(3), 168–190. doi: 10.1176/appi.neuropsych.14090217

Weber, S., Cartoni, E., Gaggioni, L., Turri, F., Wiest, R., & Aybek, S. (2025). Altered brain network dynamics in functional neurological disorder: The role of the temporoparietal junction. *Brain, 148*(1), 1–15. doi: 10.1093/brain/awae999