Adverse Childhood Experiences and Symptom Trajectories in Functional Neurological Disorder: An Exploratory Analysis of Year-1 Outcomes.

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BACKGROUND

Adverse childhood experiences (ACEs) are common among individuals with functional neurological disorder (FND) and have been repeatedly linked to worsened psychiatric and somatic symptom burden[1]. Exposure to early life adversity disrupts normal emotional development and neurobiological systems, leading to persistent vulnerabilities in affect regulation, interoception, and stress responsiveness[2]. Neuroimaging and longitudinal studies suggest that ACEs can create lasting changes in brain areas responsible for emotional regulation and body awareness — mechanisms central to the pathophysiology of FND[3].

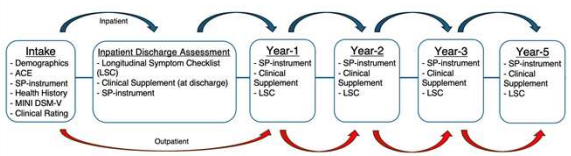
Patients with FND who report higher ACE exposure frequently demonstrate more severe and complex symptom presentations, including heightened somatic distress, functional impairments, and psychiatric comorbidity[4]. Research emphasizes that emotional neglect, physical abuse, and family dysfunction are particularly potent predictors of symptom severity and chronicity in this population[5]. Despite the recognition of ACEs as a major risk factor, the long-term symptom trajectories of FND patients with varying levels of early adversity remain poorly characterized.

The *Somatic Prospectives (SP) Project* seeks to monitor psychiatric and psychological changes prospectively while documenting the severity and types of somatic complaints for patients referred to the BC Neuropsychiatry Program, a tertiary publicly-funded provincial mental health program. The data in this brief report summarizes early findings from cases after initial assessment, initiation of treatment, and data at 1-year.

PROTOCOL

The summary protocol and clinical scales used for evaluations are as follows:

Prospective Study Timeline: Assessment Protocol for Inpatients and Outpatients with Functional Neurological Disorder



Note. SP-Instrument = battery of psychiatric self-report assessments, including PHQ-9, PHQ-15, W-14, BAI, CSI, IPDS, TAS-20, CD-RISC-10, QOLS-16, NQoL-Cog. Clinical Rating Form = physician's assessment of 1) chronicity of functional symptoms 2) nature of functional symptoms, and 3) multidimensional formulation. Clinical Supplement = physician's assessment of 1) changes to multidimensional diagnosis and 2) treatment summary.

Self-reported psychiatric assessments, including the Patient Health Questionnaire-9 (PHQ-9), Beck Anxiety Inventory (BAI), and Patient Health Questionnaire-15 (PHQ-15) are administered at each time point. At intake, patients complete an ACE questionnaire and undergo a MINI DSM-5[6] structured interview to screen for DSM disorders. In addition to the self-report scales, clinicians complete a Clinician Rating Form at intake, which documents the chronicity and nature of functional symptoms. Longitudinal symptom checklist (LSC), mental health changes, and multidimensional psychiatric diagnosis data are collected for each follow-up period to track changes over time. Due to the more intensive nature of inpatient treatments, those who were admitted for inpatient care were also assessed just prior to discharge from the hospital.

While this observational prospective study is ongoing, this report documents early findings for 30 patients diagnosed with Functional Neurological Disorder (FND) collected to year-1. This cohort includes both inpatients (n=16) treated in a multidisciplinary program and community outpatients (n=14) typically treated by individual clinicians. Data was collected at intake, discharge (inpatients only), and year-1 follow-up. Additional assessments are planned at 2, 3, and 5 years, to come.

RESULTS

Across the full sample, depression (PHQ-9) and anxiety (BAI) scores significantly improved ($p = 0.029$ and $p = 0.049$, respectively), while somatic symptoms (PHQ-15) showed no significant change ($p = 0.178$). Among outpatients, no statistically significant changes were observed for depression, anxiety, or somatic symptoms.

Inpatients demonstrated significant change across timepoints in all three domains. Post-hoc Wilcoxon test revealed that:

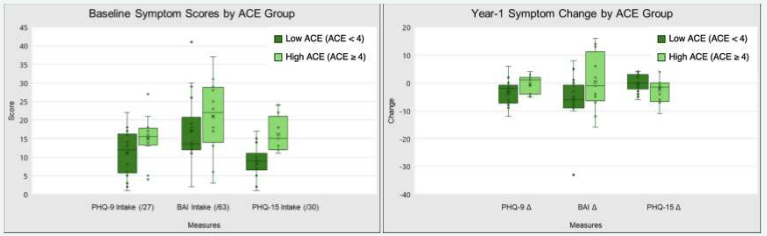
- For **PHQ-9**, median scores improved from 18.0 at intake to 12.0 at discharge ($p = 0.0021$, $r = 0.741$), then slightly declined to 13.0 at year-1 ($p = 0.0161$, $r = 0.578$), with a trend-level improvement when comparing intake to year-1 ($p = 0.0603$, $r = 0.463$).
- For **BAI**, median scores improved from 29.0 at intake to 20.0 at discharge ($p = 0.0404$, $r = 0.504$), with further improvement to 19.5 at year-1 ($p = 0.0465$, $r = 0.509$); no significant change occurred between discharge and year-1 ($p = 0.5619$, $r = 0.171$).
- For **PHQ-15**, median scores improved from 15.0 at intake to 11.0 at discharge ($p = 0.0443$, $r = 0.494$), with no significant changes between discharge and year-1 ($p = 0.4401$, $r = 0.248$) or intake and year-1 ($p = 0.2769$, $r = 0.332$).

Year-1 Comparison of Symptom Trajectories in Inpatients and Outpatients (N=30)

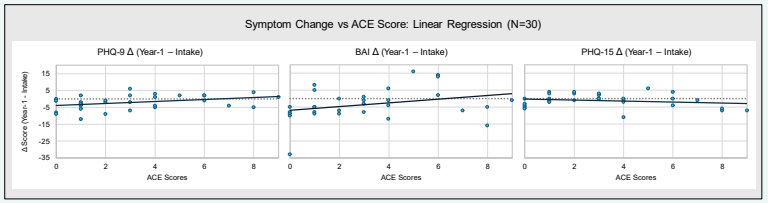
Scale	All Patients (N=30)	Inpatients (N=16)	Outpatients (N=14)
PHQ-9	↓ (Δ = -2.03, *r* = 0.47, *p* = .029)	↓↓ (χ^2 = 11.43, W = 0.357, *p* = .0033)	NS (Δ = –1.71, *r* = 0.11, *p* = .704)
BAI	↓ (Δ = -3.37, *r* = 0.35, *p* = 0.049)	↓ (χ^2 = 7.84, W = 0.245, *p* = .0199)	NS (Δ = –2.64, *r* = 0.23, *p* = .379)
PHQ-15	NS (Δ = –1.13, *r* = 0.29, *p* = .178)	↓ (χ^2 = 7.76, W = 0.242, *p* = .0207)	NS (Δ = –0.86, *r* = 0.18, *p* = .589)

Note. Δ = Mean change score (Year-1 – Intake); **r** = Wilcoxon effect size; **W** = Kendall's *W* (effect size); ↓↓ $p < .01$; ↓ $p < .05$; **NS** = Not significant. Wilcoxon signed-rank tests used for all patients and outpatients; Friedman test used for inpatients.

Patients with high ACE scores (≥ 4) reported significantly greater somatic symptom burden at intake, with median PHQ-15 scores of 14.0 compared to 8.0 in the low-ACE group ($p = .0005$). Intake scores for depression (PHQ-9) and anxiety (BAI) were also elevated in the high-ACE group, with median PHQ-9 scores of 15.5 vs. 12.0 ($p = .137$) and BAI scores of 22.0 vs. 13.5 ($p = .175$), though these differences did not reach statistical significance. Over one year, patients with high ACE scores showed less symptom improvement across multiple domains. Median PHQ-9 change scores were 1.0 for the high-ACE group compared to –2.0 in the low-ACE group ($p = .081$), while BAI scores improved by –1.0 vs. –6.0 ($p = .102$). Change in somatic symptoms (PHQ-15) did not differ significantly by ACE group (–1.5 vs. 0.0; $p = .194$).



Linear regression models were used to examine whether higher ACE scores predicted less symptom improvement over one year. For depression, higher ACE scores were significantly associated with reduced improvement on the PHQ-9 (slope = 0.59, $p = .047$, $r^2 = 0.13$). A similar trend was observed for anxiety, with higher ACE scores predicting smaller reductions on the BAI (slope = 1.08, $p = .105$, $r^2 = 0.09$), though this did not reach statistical significance. Somatic symptom change (PHQ-15) was not associated with ACE scores (slope = –0.30, $p = .272$, $r^2 = 0.04$).



Impact of ACE Scores on 1-Year Symptom Change: Inpatient vs Outpatient

Group	Outcome	Continuous ACE		Categorical ACE	
Inpatient	ΔPHQ-9	R^2 = 0.074, *p* = .213		r = 0.298, *p* = .254	
	ΔBAI	R^2 = 0.009, *p* = .624		r = 0.095, *p* = .744	
	ΔPHQ-15	R^2 = 0.020, *p* = .475		r = -0.285, *p* = .275	
Outpatient	ΔPHQ-9	R^2 = 0.240, *p* = .092	⇓	r = 0.345, *p* = .214	
	ΔBAI	R^2 = 0.331, *p* = .107	⇓	r = 0.483, *p* = .081	⇓
	ΔPHQ-15	R^2 = 0.042, *p* = .385		r = -0.242, *p* = .397	

Note. Δ = Year-1 – Intake; **R^2** = Proportion of variance explained by ACE scores; **r** = Wilcoxon effect size; ⇓ = trending significance.

Among inpatients, continuous ACE scores were not significantly associated with changes in depression, anxiety, or somatic symptoms. Categorical comparisons in inpatients also showed no significant differences between high and low ACE groups across outcomes.

Among outpatients, continuous ACE scores showed trend-level associations with reduced improvement in depression ($R^2 = 0.240$, $p = .092$) and anxiety ($R^2 = 0.331$, $p = .107$), while somatic symptom showed no significant relationship. Categorical comparisons among outpatients showed a trend-level difference for anxiety symptom change between high and low ACE groups ($r = 0.483$, $p = 0.081$), with no significant differences observed for ΔPHQ-9 and ΔPHQ-15.

DISCUSSION

Higher ACE exposure was associated with greater baseline symptom burden. Patients with ACE scores ≥4 reported significantly greater somatic symptom severity (PHQ-15) at intake, consistent with prior findings linking early adversity to greater somatic burden[2]. Although depression and anxiety scores were also higher at intake among high-ACE patients, these differences did not reach statistical significance, suggesting that somatic symptoms may be a more sensitive early marker of ACE burden in FND populations.

Over one year, higher ACE scores predicted reduced psychiatric improvement. Continuous ACE modeling indicated that patients with greater early adversity experienced less reduction in depression and anxiety symptoms, aligning with evidence that trauma histories may contribute to more treatment-resistant psychiatric trajectories[1]. Categorical comparisons further supported this pattern, with high-ACE patients showing trend-level reductions in anxiety symptom improvement relative to low-ACE patients. These findings highlight the importance of trauma-informed care approaches that specifically address the lingering psychiatric impacts of early adversity in FND.

When examining treatment settings, ACE effects were more pronounced among outpatients. Among outpatients, higher ACE scores showed trend-level associations with reduced improvement in depression and anxiety symptoms, and high-ACE outpatients showed a trend toward worse anxiety outcomes compared to low-ACE outpatients. In contrast, among inpatients, ACE exposure was not significantly associated with changes in depression, anxiety, or somatic symptoms, suggesting that intensive multidisciplinary inpatient care may buffer the negative psychiatric impacts of early adversity. This differential pattern supports the potential benefit of structured, higher-intensity interventions for FND patients with significant trauma histories, who may otherwise experience limited psychiatric recovery in outpatient settings.

REFERENCES

1. Mendizabal, A., Nathan, C. L., Khankhanian, P., Anto, M., Clyburn, C., Acaba-Berrocal, A., Breen, L., & Dahodwala, N. (2022). Adverse Childhood Experiences in Patients With Neurologic Disease. *Neurology. Clinical practice*, *12*(1), 60–67. https://doi.org/10.1212/CPJ.0000000000001134
2. Drane, D. L., Fani, N., Hallett, M., Khalsa, S. S., Perez, D. L., & Roberts, N. A. (2020). A framework for understanding the pathophysiology of functional neurological disorder. *CNS spectrums*, 1–7. Advance online publication. https://doi.org/10.1017/S1092852920001780
3. Hakamata, Y., Suzuki, Y., Kobashikawa, H., & Hori, H. (2022). Neurobiology of early life adversity: A systematic review of meta-analyses towards an integrative account of its neurobiological trajectories to mental disorders. *Frontiers in neuroendocrinology*, *65*, 100994. https://doi.org/10.1016/j.yfrne.2022.100994
4. Haczkiewicz, K. M., Shahid, S., Finnegan, H. A., Moninn, C., Cameron, C. D., & Gallant, N. L. (2024). Adverse childhood experiences (ACEs), resilience, and outcomes in older adulthood: A scoping review. *Child abuse & neglect*, 106864. Advance online publication. https://doi.org/10.1016/j.chiabu.2024.106864
5. Oral, R., Ramirez, M., Coohey, C., Nakada, S., Walz, A., Kuntz, A., Benoit, J., & Peek-Asa, C. (2016). Adverse childhood experiences and trauma informed care: the future of health care. *Pediatric research*, *79*(1-2), 227–233. https://doi.org/10.1038/pr.2015.197
6. Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., Weiller, E., Hergueta, T., Baker, R., & Dunbar, G. C. (1998). The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *The Journal of clinical psychiatry*, *59 Suppl 20*, 22–57.

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