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Structured clinical diagnostic assessment reveals autism spectrum disorder in adults with functional neurological disorder

Belen Gonzalez-Herrero[1,2]✉, Jan Coebergh[3,4], Javier Pagonabarraga[5,6], Francesca Morgante[7], Quinton Deeley[8,9] & Mark J. Edwards[10]✉

Emerging evidence suggests a link between Autism Spectrum Disorder (ASD) and Functional Neurological Disorder (FND), underscoring the importance of considering neurodevelopmental traits in neurological care. This study examined the prevalence of clinically probable ASD (CP-ASD) in a specialist FND clinic and explored its associations with symptom presentation, mental health, alexithymia and interoceptive awareness. Sixteen consecutively recruited adults with FND underwent comprehensive ASD assessment, including self-report questionnaires (RAADS-R, AdAS Spectrum), observational interview (ADOS-IV), and evaluation against DSM-5 criteria. Additional validated psychometric measures assessed anxiety (GAD-7), depression (PHQ-9), dissociation (Cambridge Depersonalization Scale, CDS), alexithymia (TAS-20), camouflaging (CAT-Q), and interoceptive sensibility (MAIA-2). Half of the participants ($n = 8$) met criteria for CP-ASD. Compared with the non-CP-ASD group, the CP-ASD group had a younger age at symptom onset and a longer interval from onset to FND diagnosis. After correction for multiple comparisons, significant group differences remained for anxiety (GAD-7), dissociation (CDS), and camouflaging behaviours (CAT-Q total, Compensation, and Assimilation subscales). Several further differences reached uncorrected significance with large effect sizes, including alexithymia (TAS-20) and the MAIA-2 Not Worrying and Emotional Awareness subscales, but did not survive correction and should be considered exploratory. Among functional symptom types, only sensory symptoms differed, being more prevalent in the CP-ASD group (62.5% vs 12.5%, p =.021), while treatment response did not differ between groups. . These findings suggest that ASD may frequently co-exist with FND but remain under-recognised. Incorporating routine screening and neurodevelopmentally informed care could improve diagnostic accuracy and support more personalised interventions. Larger, adequately powered studies are needed to confirm these preliminary results and to clarify further the role of neurodevelopmental factors in the onset, persistence, and treatment response of FND.

Keywords Functional Neurological Disorder, Autism Spectrum Disorder, Clinical overlap, Screening, Interoception.

Functional Neurological Disorder (FND) is a complex neuropsychiatric condition characterised by genuine neurological symptoms and signs which reflect difficulties in voluntary conscious access to movement and percepts. These symptoms, which can include motor and sensory disturbances, transient loss and alteration

[1]Departamento de Medicina, Universidad Autónoma de Barcelona (UAB), Bellaterra 08193, Spain. [2]Queen's Hospital, Havering and Redbridge University Hospitals, Barking, Romford RM7 0AG, UK. [3]Department of Neurology, Ashford St. Peter's Hospitals NHS Foundation Trust, Chertsey, England. [4]Department of Neurology, St. George's Hospital NHS Foundation Trust, London, England. [5]Instituto de Investigación Biomédica de Sant Pau, Barcelona 08041, Spain. [6]Centro de Investigación en Red-Enfermedades Neurodegenerativas (CIBERNED), Madrid 28031, Spain. [7]Neurosciences and Cell Biology Institute, Neuromodulation and Motor Control Section, St George's University of London, London SW17 0RE, UK. [8]Social, Genetic and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London SE5 8AF, UK. [9]National Autism Unit, South London and Maudsley NHS Foundation Trust, London, UK. [10]Department of Clinical and Basic Neuroscience, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London SE5 8AF, UK. ✉email: Belen.GonzalezH@autonoma.cat; mark.j.edwards@kcl.ac.uk

of consciousness, swallowing or cognitive difficulties, often lead to significant functional disability and psychological distress[1,2].

Although the exact aetiology of FND remains unclear, multiple risk factors have been identified, including early-life trauma or comorbid psychiatric conditions (e.g., anxiety, depression). Acute stressors, such as injury or illness, often act as triggers, while persistent stress, maladaptive coping, and challenging healthcare or social environments may perpetuate symptoms[3]. More recently, research has begun to explore the role of neurodevelopmental differences in FND, with a growing interest in a possible connection to Autism Spectrum Disorder (ASD)[4]. This aligns with a growing dimensional perspective in the field, suggesting that autistic traits are continuously distributed across the population and may manifest in diverse psychiatric contexts, including mood disorders, personality disorders, and somatic conditions[5,6].

ASD is a lifelong neurodevelopmental condition defined by difficulties in social communication, restricted interests, repetitive behaviours, and sensory sensitivities (ICD-11, DSM-5). However, its presentation varies widely, particularly in association with sex, intelligence quotient, personality traits, and coping styles, factors that may contribute to underdiagnosis in certain populations[7,8].

While the evidence on ASD-FND overlap is still emerging, existing studies and two recent systematic reviews have reported rates of diagnosed ASD in FND populations ranging from 8% to 17%[9–13] with even higher rates of elevated autistic traits identified through self-report questionnaires[11,14].

Although awareness is increasing among FND specialists, autism is still rarely considered and even less frequently assessed during routine neurological evaluations of individuals presenting with functional symptoms. FND is often primarily assessed by neurologists, who may not have specific training or familiarity with ASD. As a result, autistic traits, particularly those that are masked or present without a formal diagnosis, can be overlooked. This lack of recognition may lead to incomplete formulations, misattribution of symptoms, and missed opportunities for interventions that account for neurodevelopmental differences.

Conducted as a service development initiative, this project examined (i) how many adults in a consecutively recruited FND clinic cohort meet diagnostic criteria for ASD, and (ii) how ASD traits relate to comorbidities, treatment outcomes, and related dimensions such as interoception and alexithymia. To achieve this, we used structured clinical interviews and standardised diagnostic criteria, allowing for a detailed assessment of developmental history, behavioural patterns, and core features of ASD. In doing so, the project advances the field by moving beyond self-report autistic measures to provide systematic clinical evaluations of ASD in FND, with the goal of clarifying their relationship and guiding more personalised, neurodevelopmentally informed care.

Methodology

This observational, cross-sectional project was conducted as a service development initiative. It was reviewed and registered with the Clinical Audit Department at St George's University Hospitals NHS Foundation Trust (registration reference: AUDI003674). All methods were approved and carried out in accordance with relevant guidelines and regulations, including those outlined by the Declaration of Helsinki. The aim was to evaluate and improve local assessment practices for identifying autism spectrum traits within a Neuropsychiatric clinic for FND, as well as other psychiatric comorbidities. As this project constituted service evaluation and did not involve any deviation from standard clinical care, it did not require review by an Ethics Committee, in line with the UK Health Research Authority guidelines.

Consecutive patients diagnosed with FND attending specialist neurology services at St George's were invited to participate. From the outset, it was emphasised that the assessment was not a substitute for a formal clinical ASD evaluation through NHS neurodevelopmental services. Patients meeting criteria for a clinically probable ASD (CP-ASD) diagnosis were informed and offered referral to primary care to access appropriate pathways. All participants provided written informed consent for the use of their anonymised data and the dissemination of findings.

Participant recruitment and inclusion criteria

Eligible participants were identified consecutively from the specialist FND clinic and had a confirmed diagnosis of FND by at least two neurologists experienced in the condition. Inclusion criteria required participants to be aged 18–65, of any gender or socioeconomic background, and capable of completing project questionnaires in English. Patients with comorbid mental health or medical conditions were eligible as long as their condition did not preclude them from completing self-report questionnaires. Participants needed to be able to provide informed consent and could complete the assessments independently or with the assistance of a family member or friend.

Of the 41 eligible patients initially approached, 19 provided signed consent forms. No participants withdrew after consenting. However, three were subsequently excluded due to incomplete project questionnaires/interviews, resulting in a final sample of 16 participants.

Data collection and assessment measures

As part of routine practice within the FND clinic, all consecutive patients underwent a comprehensive assessment for ASD, conducted by a trained clinician (BGH). The evaluation included the *Interview Guide for the Diagnostic Assessment of Able Adults with Autism Spectrum Disorder*, a semi-structured tool based on DSM-5 criteria, and ADOS Module 4 (ADOS-IV)[15], which assesses social communication and restricted, repetitive behaviours. BGH had received specialised training and formal certification in administering the ADOS-IV, a standard instrument used to support adult ASD diagnostic formulation.

In addition to the clinical evaluation, individuals were routinely asked to complete supplementary self-report questionnaires to inform the overall formulation. These were delivered via a secure, university-affiliated Microsoft Form, with paper copies provided when needed. The assessing health professional (BGH), who

conducted both the interview and the ADOS-IV, remained blinded to participants' questionnaire responses throughout the assessment process.

These questionnaires included the *Ritvo Autism Asperger Diagnostic Scale-Revised* (RAADS-R)[16] and the Adult *Autism Subthreshold Spectrum* (ADAS Spectrum)[17], both self-reported screening tools designed to identify autistic traits. The ADAS Spectrum, in particular, is more female-focused and includes a specific cut-off to distinguish between autistic traits and clinically significant ASD. Furthermore, whenever a family member was available, they were approached to provide additional information on the patient's early developmental history. This was done using structured questions specifically focused on neurodevelopmental milestones and patterns.

The *Adult ADHD Self-Report Scale* (ASRS)[18] was used as a screening tool for identifying symptoms of Attention-Deficit/Hyperactivity Disorder (ADHD), given its shared neurodevelopmental features and the known high comorbidity rates with ASD[19]. In line with standard practice, Part A of the ASRS was considered, as it has the strongest predictive validity and is most used for case identification, whereas Part B is intended primarily for additional symptom exploration. The six items in Part A are rated on a five-point scale ranging from 0 (never) to 4 (very often). A positive screen was defined as endorsement of four or more items at the threshold level.

The *Camouflaging Autistic Traits Questionnaire* (CAT-Q)[20] assessed how much individuals mask autistic traits in social situations. It includes three subscales: *Compensation*, measuring strategies used to improve social interaction; *Masking*, assessing efforts to conceal autistic traits; and *Assimilation*, evaluating the degree to which individuals try to fit into social norms. Higher scores indicate greater camouflaging efforts, which have been associated with increased social exhaustion and mental health difficulties.

Additional psychological measures were employed to assess psychiatric comorbidities and symptom burden across various domains. The *Somatic Symptom Scale-8* (SSS-8)[21] uses a five-point Likert scale to evaluate the severity of physical symptoms linked to psychological distress, such as headaches, dizziness, and gastrointestinal discomfort. Higher scores indicate greater symptom burden, ranging from minimal to very high severity. The *Patient Health Questionnaire-9* (PHQ-9)[22] assesses depressive symptom severity over the past two weeks, with scores categorising depression as mild, moderate, or severe. Similarly, the *Generalised Anxiety Disorder-7* (GAD-7)[23] evaluates anxiety severity, with cutoff scores distinguishing between mild, moderate, and severe levels of anxiety.

To assess early-life trauma, the *Adverse Childhood Experiences* (ACE) *Questionnaire*[24] was included. Early trauma can lead to long-term mental and physical health outcomes, and research suggests that significant childhood adversity may lead to behaviours resembling autism as a response to trauma rather than innate neurodiversity[25,26]. The ACE Questionnaire comprises 10 items assessing exposure to abuse, neglect, and household dysfunction. Higher scores reflect greater cumulative adversity and are associated with increased risk for psychological and physical health outcomes. In line with established literature[27], a cut-off score of ≥ 4 was used to indicate a high level of early-life adversity.

Difficulties in emotional processing, or alexithymia, were assessed using the Toronto Alexithymia Scale-20 (TAS-20)[28]. Alexithymia is defined as a difficulty in identifying, describing, and differentiating one's own emotions, often accompanied by a tendency to focus attention externally rather than on internal emotional states[29]. The TAS-20 comprises 20 items, each rated on a 5-point Likert scale (1 = *strongly disagree* to 5 = *strongly agree*), with total scores ranging from 20 to 100. Higher scores indicate greater alexithymia. A total TAS-20 score of ≤ 51 suggests *low alexithymia*, 52–60 indicates *borderline alexithymia*, and ≥ 61 reflects *high alexithymia*. Some items are reverse scored before summing up. For the purposes of analysis, both the continuous TAS-20 total score and a dichotomous categorical variable were calculated, classifying participants as "alexithymia present" (≥ 61) or "alexithymia absent" (< 61).

The *Cambridge Depersonalization Scale* (CDS)[30] was also employed to measure the frequency and severity of depersonalisation symptoms, with higher scores reflecting clinically significant distress.

Interoceptive awareness, which refers to the ability to perceive and interpret bodily sensations and is known to be altered in some individuals with FND and ASD, was assessed using the *Multidimensional Assessment of Interoceptive Awareness – Version 2* (MAIA-2)[31]. This tool includes eight subscales that explore various dimensions of body awareness. These include: *Noticing*, which refers to the awareness of comfortable, uncomfortable, and neutral bodily sensations; *Not Distracting*, which reflects the tendency not to ignore or avoid sensations of pain or discomfort; and *Not Worrying*, which measures the extent to which individuals refrain from reacting with anxiety or distress to such sensations. *Attention Regulation* assesses the ability to sustain and control focus on bodily sensations, while *Emotional Awareness* refers to the recognition of the connection between bodily sensations and emotional states. *Self-regulation* captures the ability to regulate distress by attending to bodily sensations. *Body Listening* reflects the practice of actively listening to the body for insight, and *Trusting* assesses the extent to which one experiences the body as safe and trustworthy. Scores were analysed in quartiles, with lower quartiles indicating reduced interoceptive awareness and higher quartiles reflecting greater body awareness and were compared to normative values.

Statistical analysis

All statistical analyses were conducted using SPSS version 25. Normality was assessed using the Shapiro–Wilk test, skewness, and kurtosis values to determine the appropriate statistical tests for group comparisons. For between-group comparisons, independent samples t tests were used for normally distributed continuous variables, while Mann–Whitney U tests were applied to non-normally distributed data. Differences in categorical variables, such as symptom presentation and psychiatric comorbidities between groups, were analysed using chi-square tests or Fisher's exact tests, where appropriate. To examine associations, correlation analyses were conducted using Pearson's correlation for variables meeting normality assumptions and Spearman's rank correlation for non-normally distributed variables. Covariance (ANCOVA) was used to control for potential confounding variables

in group comparisons. Effect sizes were calculated for all psychometric measures (Cohen's d for t tests, r for Mann–Whitney U tests) to provide an estimate of the magnitude of observed effects, in addition to statistical significance. According to conventional benchmarks, Cohen's d values of 0.2, 0.5, and 0.8 represent small, moderate, and large effects, respectively. For r, thresholds of 0.1, 0.3, and 0.5 are typically interpreted as small, moderate, and large effects.

To account for the increased risk of Type I error from multiple between-group comparisons, we applied the Benjamini–Hochberg false discovery rate (FDR) procedure with a threshold of $q < 0.05$. This approach was selected over the more conservative Bonferroni correction because the latter can substantially inflate Type II error rates in small samples and when measures are correlated, as is common with psychometric scales. In contrast, the FDR method controls the expected proportion of false positives among statistically significant results while retaining greater sensitivity to detect true effects. This balance was considered important given the exploratory nature of the study and the clinical relevance of identifying potential associations that warrant follow-up in larger samples. Both unadjusted p-values and FDR-adjusted q-values are reported in the results tables, with significance determined by $q < 0.05$.

Results

To explore the potential coexistence of autistic traits and FND, we conducted detailed diagnostic assessments in a consecutive clinical sample. Below, we report the prevalence of CP-ASD and compare clinical, psychiatric, and interoceptive features across groups.

The final analysis included 16 participants with a confirmed diagnosis of FND. Half of the sample ($n = 8$; 50%) met criteria for clinically CP-ASD, based on converging evidence from the DSM-5, ADOS-IV, RAADS-R, and ADAS Spectrum assessments. The remaining eight participants did not meet criteria for CP-ASD and are referred to as the non-CP-ASD group throughout the analysis. Within this group, two participants (12.5%) tested positive on the ADOS-IV and exceeded thresholds on both the RAADS-R and ADAS Spectrum but met only two core DSM-5 criteria without fulfilling the accessory requirements, suggesting a potentially milder ASD phenotype. An additional two participants (12.5%) displayed mixed profiles, testing negative on the DSM-5 and ADOS-IV but positive on the RAADS-R and ADAS Spectrum. Three participants (18.75%) endorsed autistic traits exclusively on the ADAS Spectrum, while one participant (6.25%) tested negative across all assessment tools. A family history of established ASD diagnosis was reported by four participants (25%), although this was not significantly associated with CP-ASD classification ($p = .20$).

Sociodemographic and clinical features by group

Both the CP-ASD and non-CP-ASD groups were predominantly female, and all participants identified as white British. There were no statistically significant differences between groups in age, socioeconomic status, employment status, or educational background. Group sociodemographics data are summarised in Table 1.

The mean age at onset of FND symptoms was significantly lower in the CP-ASD group (M = 27.3 years, SD = 14.9; median = 24.5 years, IQR = 28.5) compared to the non-CP-ASD group (M = 43.5 years, SD = 12.5; median = 49.5 years, IQR = 22.5); $p = .03$. The time from symptom onset to FND diagnosis was significantly longer in the CP-ASD group (M = 7.38 years, SD = 7.2; Median = 5.5, IQR = 7) compared to the non-CP-ASD group (M = 1.8 years, SD = 1.4; Median = 1, IQR = 1.7); $p = .02$.

In terms of clinical features, sensory symptoms were significantly more prevalent in the CP-ASD group (62.5%) compared to the non-CP-ASD group (12.5%), $p = .021$. Other functional neurological symptoms, including movement disorders, functional seizures, cognitive difficulties, gait abnormalities, speech disturbances,

Characteristic	Category	CP-ASD Categorical: *N* (%) Numerical: mean (SD); median (IQR) years	Non CP-ASD (%) Categorical: *N* (%) Numerical: mean (SD); median (IQR) years	*p*-value
Sex	Female	6 (75.0)	5 (62.5)	1.0
	Male	2 (25.0)	3 (37.5)	1.0
Age		40.8 (15.6); 35 (26.5)	51.7 (13.2); 57 (24.5)	0.1
Education	Secondary school	2 (25.0)	3 (37.5)	1.0
	College	3 (37.5)	2 (25.0)	1.0
	University	1 (12.5)	2 (25.0)	1.0
	PhD/Master	2 (25.0)	1 (12.5)	1.0
Ethnicity	White British	8 (100.0)	8 (100.0)	1.0
Socioeconomic status	Working class	7 (87.5)	6 (75.0)	1.0
	Middle class	1 (12.5)	2 (25.0)	1.0
Active employment	No	5 (62.5)	6 (75.0)	1.0
	Yes	3 (37.5)	2 (25.0)	1.0

Table 1. Participant sociodemographics by group. Sociodemographic variables are presented as the number of participants (percentage within group) for categorical variables and as mean (standard deviation) and median (interquartile range) for numerical variables. CP-ASD = clinically probable autism spectrum disorder. No statistically significant differences were observed between groups (p-value).

and swallowing difficulties, were similarly distributed across groups. Swallowing difficulties, visual disturbances, and bladder issues were infrequently reported in both groups, with no statistically significant group differences identified.

Concerning psychiatric comorbidities, participants in the CP-ASD group self-reported a significantly higher total number of previous psychiatric diagnoses (M = 3.7, SD = 1.3) compared to those in the non-CP-ASD group (M = 2.1, SD = 1.2), $p = .02$. Although no statistically significant differences emerged when comparing individual diagnoses, the prevalence of each condition was consistently higher in the CP-ASD group. Anxiety disorders were reported by 87.5% of individuals in the CP-ASD group and 50% in the non-CP-ASD group. Depression was reported by 75% and 50%, respectively. PTSD and panic attacks were each reported by 50% of participants in both groups. OCD, bipolar disorder, and emotionally unstable personality disorder (EUPD) were reported exclusively in the CP-ASD group (12.5% each).

Concerning previous diagnoses of neurodevelopmental conditions, ASD was identified in four participants within the CP-ASD group, while no participants in the non-CP-ASD group reported a prior ASD diagnosis. No participants reported a previous diagnosis of ADHD. Dyslexia was reported at similar rates across both groups, with no significant difference observed ($p = .90$). These findings are visually represented in Fig. 1.

No statistically significant group differences were identified for chronic pain, chronic fatigue, migraine or headaches, sleep problems, dizziness, or gastrointestinal symptoms. Similarly, access to treatments such as CBT and physiotherapy did not differ between groups. Ratings of subjective improvement varied within both groups, ranging from none to moderate improvement, with no significant differences observed.

Figure 1. Percentage of participants in the CP-ASD and non-CP-ASD groups self-reporting psychiatric and neurodevelopmental comorbidities. Group differences in frequency are visualised for common diagnoses. See the main text for statistical comparisons.

Psychological measures and interoceptive profiles

All variables met the assumption of normality ($p > .05$) except the CDS, which demonstrated a significant deviation from normality ($p = .038$); group comparisons were conducted accordingly.

In the initial analysis, participants in the CP-ASD group reported significantly higher anxiety levels on the GAD-7 compared with the non-CP-ASD group ($p = .02$). No significant group differences were observed for the SSS-8 or PHQ-9.

Scores on the TAS-20 indicated significantly elevated alexithymia in the CP-ASD group ($p = .04$). When applying the categorical cut-off for alexithymia (TAS-20 ≥ 61), 75% (6/8) of participants in the CP-ASD group met criteria, compared with 25% (2/8) in the non-CP-ASD group ($p = .04$), demonstrating a significant association between alexithymia and CP-ASD classification.

Similarly, participants in the CP-ASD group reported significantly more frequent dissociative experiences, as reflected by higher scores on the CDS ($p = .02$).

Camouflaging behaviours were notably elevated in the CP-ASD group, with CAT-Q total scores significantly higher than those of the non-CP-ASD group ($p < .001$). Subscale analyses showed significantly higher scores in the Compensation ($p < .001$) and Assimilation ($p = .02$) domains. Sex classification influenced camouflaging, with females reporting higher CAT-Q scores than males ($p = .012$). A significant Sex × CP-ASD interaction was observed ($p = .031$), indicating that the difference between groups was larger in females than in males. Post-hoc

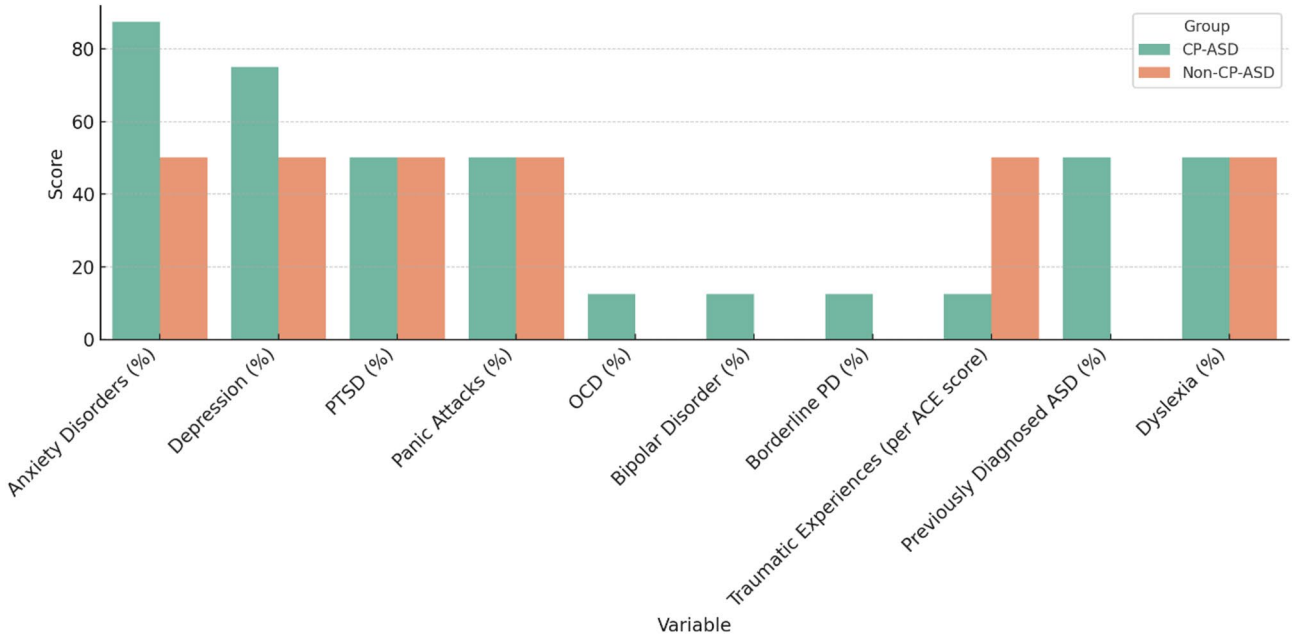

Fig. 1. Psychiatric and Neurodevelopmental Comorbidities by Group.

comparisons showed that females with CP-ASD had significantly higher CAT-Q scores compared with females without CP-ASD ($p < .001$), whereas the difference between CP-ASD and non-CP-ASD males did not reach statistical significance ($p = .15$).

High ACE scores (≥ 4) were observed in 12.5% of the CP-ASD group compared with 62.5% of the non-CP-ASD group, a difference that was not statistically significant ($p = .10$). Positive ADHD screening on the ASRS Part A (≥ 4 positive responses) were more frequent in the CP-ASD group (75%) than in the non-CP-ASD group (25%), though this difference was also not significant ($p = .13$).

When controlling for multiple comparisons using the FDR procedure ($q < 0.05$), significant group differences remained for GAD-7, CDS and CAT-Q. Group differences in continuous TAS-20 did not remain significant after correction. These data are summarised in Table 2, including effect sizes, which were generally large even where significance was lost after correction..

MAIA-2 subscale scores revealed a complex interoceptive profile among participants. These findings are visually represented in Fig. 2. While many individuals demonstrated relatively strong abilities in Noticing and Body Listening, several subscales highlighted areas of difficulty; a substantial proportion of participants scored in the lowest quartile on Not Distracting, Not Worrying, Self-Regulation, Emotional Awareness, and Trusting. Attention Regulation scores were variable.

When examined by group, distinct patterns of interoceptive awareness emerged. The largest between-group differences (large effect sizes) were observed for Not Worrying ($d = -1.27$), Emotional Awareness ($d = 1.11$), and Body Listening ($d = 0.87$). Specifically, the CP-ASD group scored lower on Not Worrying and higher on Emotional Awareness and Body Listening. A moderate effect was observed for Attention Regulation ($d = 0.48$), with the CP-ASD group tending to score higher. Other subscales showed small effects ($|d| < 0.3$), suggesting minimal differences.

Although initial uncorrected analyses indicated statistically significant differences for Not Worrying and Emotional Awareness, these did not survive false discovery rate (FDR) correction for multiple comparisons ($q > 0.05$), underscoring the need for cautious interpretation given the small sample size.

Figure 2. Participants in the CP-ASD and non-CP-ASD groups were distributed across MAIA-2 subscales by normative percentile ranges. Each bar represents the number of participants (maximum $N = 8$ per group) falling within the 0–25%, 26–50%, 51–75%, and 76–100% percentile bands relative to a normative population. CP-ASD bars are solid; non-CP-ASD bars are hatched. See the main text for statistical comparisons.

Correlation analysis

Correlation analyses were conducted to explore associations among key psychometric measures. Variables meeting normality assumptions were analysed using Pearson's correlation coefficients, while the CDS, which deviated from normality, was analysed using Spearman's rank correlations. Across the full sample, Pearson's r indicated significant positive correlations between GAD-7 and both PHQ-9 ($p = .001$) and CAT-Q ($p = .020$), as well as between PHQ-9 and SSS-8 ($p = .009$). Measures of autistic traits (RAADS-R, ADAS Spectrum, and ADOS-IV) were strongly intercorrelated. RAADS-R ($p < .001$ with GAD-7; $p = .001$ with CAT-Q), ADAS Spectrum ($p = .002$ with GAD-7; $p = .004$ with CAT-Q), and ADOS-IV ($p < .001$ with GAD-7; $p = .007$ with CAT-Q) all showed significant positive correlations with anxiety and camouflaging. Spearman's ρ for the Cambridge Depersonalization Scale indicated significant positive correlations with SSS-8 ($p = .036$), PHQ-9

Measure	CP-ASD Mean (SD), Median (IQR)	Non-CP-ASD Mean (SD)	*p*-value	*P* -adjusted (FDR)	Significant at q < 0.05	Effect size Cohen's d/Rank biserial *r*
ADOS-IV score	12.1 (3.3)	6.5 (3.5)	< 0.001	0.002	Yes	1.65
ADAS Spectrum	107.6 (13.7)	62.6 (13.3)	< 0.001	0.002	Yes	3.33
RAARS	158.7 (24.0)	65.1 (22.8)	< 0.001	0.002	Yes	4.0
GAD-7	14.7 (4.5)	7.2 (6.3)	0.02	0.03	Yes	1.37
PHQ-9	15.3 (7.2)	10.7 (6.3)	0.1	0.12	No	0.68
SSS-8	19.1 (7.1)	17.6 (6.7)	0.6	0.6	No	0.22
TAS-20	64 (14.3)	50 (9.3)	0.04	0.053	No	1.16
CDS	81 (7.2), 85.5 (65)	18 (14.3), 20.4 (18.4)	0.003	0.006	Yes	0.72
CAT-Q Total	121.5 (17.3)	83.2 (10.9)	< 0.001	0.002	Yes	2.65
CAT-Q Compensation	42.2 (9.8)	19 (6.3)	< 0.001	0.002	Yes	2.82
CAT-Q Masking	36.6 (9.3)	31.6(5.7)	0.2	0.218	No	0.65
CAT-Q Assimilation	42.6 (6.9)	32.3 (8.9)	0.02	0.03	Yes	1.29

Table 2. Psychometric measurements by group. Participants in the CP-ASD group scored significantly higher on measures of anxiety (GAD-7), depersonalisation (CDS), and autistic camouflaging behaviours (CAT-Q total, Compensation, and Assimilation subscales). They also showed higher scores on ADOS-IV, ADAS Spectrum, and RAARS assessments. No significant differences were observed in depression (PHQ-9), somatic symptom severity (SSS-8), alexithymia (TAS-20), or the CAT-Q Masking subscale after FDR correction but effect sizes were generally large, highlighting the robustness and relevance of these group differences. SD = Standard Deviation. Group differences with FDR-adjusted $q < 0.05$ were considered statistically significant.

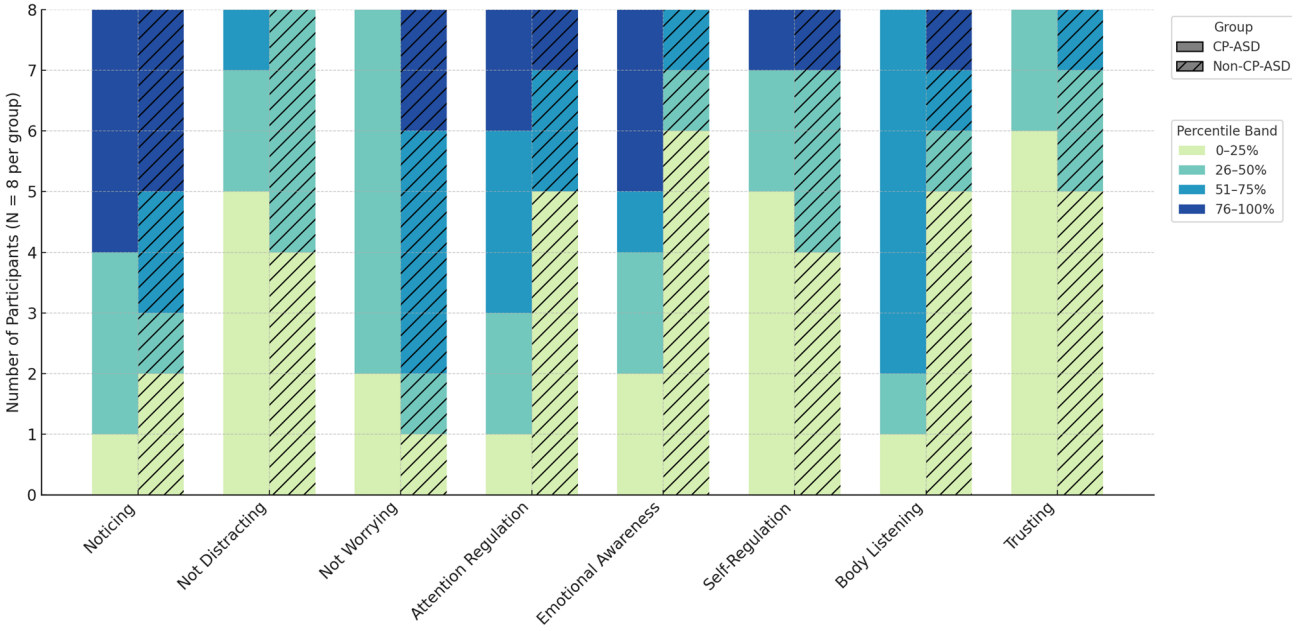

Fig. 2. Percentile-based distribution of MAIA-2 subscale scores by groups.

(p =.032), RAADS-R (p <.001), ADAS Spectrum (p =.001), and TAS-20 (p =.014). No significant associations were observed between MAIA-2 subscales and mood symptoms.

Discussion

Our results contribute to a growing body of research exploring the intersection between neurodevelopmental conditions and functional neurological symptoms. While prior research has relied largely on self-report screening tools, our use of gold-standard diagnostic methods allowed for a more in-depth identification of clinically probable ASD within this cohort and examined how these traits may interact with interoception, emotional processing, and psychiatric comorbidity.

We found a high prevalence of CP-ASD in our cohort of people attending a specialist FND clinic, with half of the participants meeting criteria across multiple assessment tools. This group included both individuals with a pre-existing ASD diagnosis and newly identified cases, suggesting that comprehensive, multi-method screening, including self-report measures, observational interviews, and DSM-5 criteria, can uncover previously unrecognised autistic traits in adult individuals with FND. We acknowledge, however, that while this rigorous, multi-step approach strengthened the validity of our findings, it may not be feasible in routine clinical practice due to limitations in time, expertise, and access to specialised diagnostic services. These challenges underscore the need for clearer clinical pathways and improved access to ASD assessment in individuals with FND to enable earlier identification and more personalised care.

Interestingly, only a subset of participants who self-reported a diagnosis of dyslexia (3 out of 7) met criteria for CP-ASD, suggesting that while comorbidity may exist, dyslexia alone does not reliably predict ASD classification within this cohort. Regarding ADHD, no participants in either group reported a prior diagnosis, although a higher proportion of individuals in the CP-ASD group screened positive on the ASRS compared to the non-CP-ASD group; however, this difference did not reach statistical significance. Importantly, no formal diagnostic assessment for ADHD was conducted during the interview, which focused exclusively on ASD. These findings highlight the need for further investigation into the broader spectrum of neurodevelopmental differences, including ADHD and dyslexia, as potential independent or interacting risk factors in the development of functional symptoms.

Participants with mixed or isolated positive findings on self-report ASD screening tools may represent individuals with elevated autistic traits who do not cross the diagnostic threshold for ASD. Alternatively, these profiles may reflect other conditions that are identified in neurodevelopmental questionnaires and assessments, but which do not represent a mild 'subthreshold' form of ASD. In some cases, sensory sensitivities, difficulties with emotional regulation, or atypical social cognition may arise from pathways distinct from core autistic traits but still lead to similar responses on self-report measures[32–34].

The comparison between participants with and without a CP-ASD classification revealed several interesting distinctions. Females were overrepresented in both groups, consistent with the demographic profile of specialist FND clinics, where women constitute the majority of referrals. This distribution reflected the characteristics of the population attending such services rather than sampling bias.

There was a significantly younger onset of FND within the CP-ASD group, suggesting that neurodevelopmental vulnerability may contribute to earlier manifestation of functional symptoms. The time from symptom onset to diagnosis was also significantly longer in the CP-ASD group, which may be related to diagnostic overshadowing

of autistic features by other co-occurring psychiatric or medical conditions or may also be related to autism-associated difficulties in communication and self-advocacy, including challenges in describing internal experiences and physical symptoms, navigating complex healthcare systems, and the lack of healthcare provider training in recognising autism in adults.

Core FND symptom types, associated complaints such as fatigue and pain, and self-reported treatment outcomes were broadly similar across groups, yet psychiatric profiles diverged significantly. This suggests that the mechanisms driving functional symptoms may be shared across individuals, regardless of autism status, whereas autistic traits may contribute specifically to an increased psychiatric burden, consistent with literature documenting greater psychiatric vulnerability in autistic populations[35].

There is extensive literature linking early life adversity, particularly abuse, neglect, and household dysfunction, to increased vulnerability to functional neurological symptoms[36,37]. These experiences may contribute to FND risk through long-term dysregulation of stress response systems, heightened bodily threat perception, and maladaptive emotion regulation strategies[38]. Given the frequent report of early life traumatic experiences in FND populations, the ACE scale was included to account for the potential influence of such experiences, which have in some cases been associated with autistic-like behaviours[25,26] and to explore whether this could act as a confounder in classifying individuals as autistic. For example, the longitudinal English and Romanian Adoptees study[39] found that early deprivation was linked to elevated autistic behaviours persisting into adulthood, although these traits were assessed with a shortened Social Communication Questionnaire rather than formal diagnostic tools[35]. Interestingly, in our sample, high ACE scores were more frequent in the non-CP-ASD group than in the CP-ASD group, but the difference was not statistically significant. In the context of FND without co-occurring autism, higher ACE scores are consistent with established evidence that early trauma can increase susceptibility to functional symptoms through its impact on stress regulation and emotional processing. However, while autistic populations generally report higher rates of trauma, our findings did not reflect this pattern. One possible explanation is that autistic individuals may perceive and be affected by a broader range of life events, beyond those captured by the ACE framework, as traumatic, thereby influencing vulnerability to PTSD and other stress-related conditions[40].

Camouflaging behaviours emerged as a prominent distinguishing feature of the CP-ASD group, with particularly marked differences in women. This finding aligns with broader autism research, which has consistently shown that autistic women engage in camouflaging at higher rates than autistic men[41,42]. Camouflaging may contribute to the underdiagnosis of ASD in individuals with FND and may also affect their engagement with healthcare services. Additionally, previous research has shown that sustained camouflaging is associated with increased psychological burden[43], which may further complicate the clinical picture and contribute to the observed higher psychiatric comorbidity in this group. From an allostatic load perspective, it could be argued that the chronic psychological effort required to maintain camouflaging behaviours contributes to chronic stress, lowering the threshold for developing functional neurological symptoms. Allostatic load refers to the cumulative physiological burden imposed by repeated or chronic activation of the body's stress response systems. Over time, this can lead to dysregulation of systems such as the hypothalamic-pituitary-adrenal axis and autonomic nervous system, contributing to both physical and mental health vulnerabilities. In this model, camouflaging in itself could be an active stressor, enhancing vulnerability for FND through its impact on stress regulation systems.

Participants in the CP-ASD group also scored significantly higher on the CDS, suggesting more frequent dissociative experiences. This may reflect underlying disruptions in emotional processing or self-awareness associated with neurodevelopmental differences[44], though it may also be a secondary effect of increased psychiatric comorbidity in this group.

Patterns of interoceptive sensibility, as measured by the MAIA-2, provided additional insight into bodily self-awareness in this population. While most participants showed elevated scores on the Noticing and Body Listening subscales, suggesting a heightened awareness of bodily sensations and a tendency to attend to them when making decisions, lower scores on Trusting, Not Distracting, Self-Regulation, and Emotional Awareness in all participants indicated difficulties in managing, interpreting, or trusting those signals. These findings may reflect challenges in filtering irrelevant sensory input or adaptively using interoceptive information.

Group comparisons revealed a distinctive profile within the CP-ASD group, characterised by lower Not Worrying scores (indicating greater distress in response to bodily sensations) and higher Emotional Awareness scores (suggesting a stronger subjective sense of the connection between bodily sensations and emotional states). Both differences showed large effect sizes and were independent of co-occurring anxiety or depression. Given that the MAIA-2 captures interoceptive sensibility (a subjective perception rather than objective accuracy), the observed trend toward higher Emotional Awareness in the CP-ASD group may reflect greater confidence in identifying bodily–emotional links rather than actual precision. This pattern mirrors findings in ASD populations, where individuals often report high interoceptive confidence despite reduced interoceptive accuracy[45]. A similar mismatch has also been observed in FND populations, although findings related to interoception in FND have been inconsistent and vary across studies[46–49]. It is important to note, however, that the initial uncorrected group differences for Not Worrying and Emotional Awareness did not remain significant after FDR correction, likely reflecting both the limited statistical power of the small sample and the increased stringency of multiple-comparison adjustment. Nonetheless, given that the direction and magnitude of these effects are theoretically meaningful, they should be regarded as exploratory and hypothesis-generating, warranting replication in larger, adequately powered studies.

Patterns across measures of emotional processing (TAS-20) suggested a tendency toward greater alexithymia in the CP-ASD group. Although these differences did not remain significant after correction for multiple comparisons, the effect size was large, and the convergence of findings across the TAS-20 and MAIA-2 highlights a potential profile marked by both difficulty identifying and expressing emotions and a heightened subjective

awareness of the link between emotions and bodily sensations. This combination is consistent with theoretical models in autism, proposing that physiological signals are experienced but poorly integrated into higher-order emotional frameworks[50]. This heightened yet imprecise awareness may contribute to somatic hypervigilance and emotional reactivity. In some cases, emotional experiences may be misinterpreted as physical symptoms, further blurring the boundary between affective and somatic states[51]. These mechanisms are supported by findings from both ASD and FND research, where intolerance of uncertainty and heightened vigilance to internal bodily cues are commonly reported[52,53], Such dynamics may help explain an increased vulnerability to developing and maintaining functional symptoms observed in individuals with autistic traits, and they warrant further investigation in larger, adequately powered samples.

Furthermore, autistic individuals appear to exhibit distinct neuroendophenotypes. Using large-scale imaging-transcriptomic analyses, Buch et al. (2023)[54] identified neurobiological subtypes within the autism spectrum. Their findings show that variability in gene expression, cortical connectivity, and cellular architecture is closely associated with divergent clinical presentations. These subtypes reflect differing patterns of brain organisation and are likely linked to specific functional outcomes and co-occurring conditions, suggesting that vulnerability to disorders such as FND may be mediated by particular ASD-related neurobiological pathways. Further research is needed to investigate this possibility and clarify its clinical implications.

Lastly, the strong intercorrelations between autistic trait measures (RAADS-R, ADOS-IV and ADAS Spectrum) confirm that these instruments capture overlapping constructs related to the autism phenotype, supporting the validity of the CP-ASD classification within the sample. The three of them showed significant positive correlations with anxiety and camouflaging, indicating that higher autistic trait severity was associated with greater anxiety symptoms and increased engagement in compensatory social strategies. In the context of FND, this pattern may reflect a compounded vulnerability, where elevated autistic traits lead to greater social effort and anxiety, potentially shaping both symptom expression and clinical trajectory toward an FND diagnosis. Anxiety was also positively correlated with depression and somatic symptom burden, consistent with evidence that physical and mental health conditions jointly contribute to symptom severity, regardless of autism status[33]. The absence of significant correlations between interoceptive sensibility (MAIA-2 subscales) and mood or alexithymia suggests that altered interoceptive processing in FND with CP-ASD may constitute a distinct dimension of functioning rather than being secondary to emotional distress. Further research in larger, well-controlled cohorts is warranted to clarify the relative contributions of ASD traits and co-occurring mental health conditions to the experience of functional somatic and neurological symptoms.

Limitations

Several limitations should be considered when interpreting the findings of this project. First, the small sample size limits the generalisability of the results and reduces statistical power, potentially increasing the risk of Type II errors. Although participants were recruited consecutively to minimise selection bias, the voluntary nature of participation may have introduced self-selection bias, particularly among individuals more motivated to explore a possible ASD diagnosis. The sample was also demographically homogeneous, with all participants identifying as White British, limiting the findings' applicability to other populations. Additionally, while the project employed validated diagnostic and self-report tools, the reliance on subjective measures, particularly for constructs such as interoception, emotional processing, and camouflaging, may be influenced by participant insight, recall bias, or social desirability. Psychiatric and previous neurodevelopmental diagnoses were also self-reported and, therefore, may be subject to misclassification or incomplete reporting, which could introduce bias. Additionally, while we controlled for multiple comparisons using the FDR procedure, which is widely used in exploratory studies to maintain statistical power, we acknowledge that this method is less conservative than the Bonferroni correction. As such, some findings that remained significant under FDR adjustment might not survive more stringent correction, and should therefore be interpreted with caution until replicated in larger, independent samples. Finally, the cross-sectional design precludes causal inferences; longitudinal studies are needed to examine the stability of these interoceptive-emotional profiles over time and their impact on symptom progression and treatment response in individuals with FND and ASD traits.

Conclusion

As awareness grows around the complexity and heterogeneity of FND, it is increasingly important to consider neurodevelopmental contributions. Our findings suggest that ASD may be substantially under-recognised in FND populations, and that interoceptive-emotional profiles may play a role in shaping symptom experience and response to care. These insights have meaningful implications for both diagnostic pathways and intervention strategies.

While this project provides valuable preliminary insights, further research is needed to explore the mechanisms underlying the ASD-FND relationship. Larger-scale studies with diverse populations could help determine whether specific ASD subtypes are more susceptible to developing FND and explore shared mechanisms and vulnerabilities. Longitudinal research is also necessary to assess how ASD traits influence long-term prognosis in FND patients.

Additionally, qualitative research could provide deeper insight into patient experiences, particularly regarding diagnostic pathways, desirability of ASD diagnosis and healthcare interactions. Understanding how patients with ASD navigate the healthcare system may help improve clinical communication and patient-centred care.

Despite its limitations, our findings add to the growing evidence of a substantial overlap between FND and ASD. Using a comprehensive assessment approach, we identified high rates of clinically probable ASD among FND patients alongside a possible distinctive interoceptive-emotional profile marked by heightened bodily awareness, somatic worry, and impaired emotional insight. These findings underscore a neurodevelopmental vulnerability that may influence symptom onset and persistence in FND. Routine screening for ASD in FND

services, along with tailored, neurodiversity-informed interventions, could play a critical role in understanding symptom production and mechanisms, helping to improve care and outcomes for this under-recognised group.

Data availability

The data supporting the findings of this study are not publicly available due to privacy and confidentiality considerations. Reasonable requests for access to the data may be directed to: Belén González HerreroDepartamento de Medicina, Universidad Autónoma de Barcelona (UAB), 08193 Bellaterra, SpainEmail: Belen.GonzalezH@autonoma.

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References

1. Stone, J. et al. Who is referred to neurology clinics?--the diagnoses made in 3781 new patients. *Clin. Neurol. Neurosurg.* **112** (9), 747–751 (2010).
2. Gendre, T. et al. Quality of life in functional movement disorders is as altered as in organic movement disorders. *J. Psychosom. Res.* **116**, 10–16 (2019).
3. Merritt Millman, L. et al. 13 Predisposing, precipitating and perpetuating factors in functional neurological disorder: a pilot study. *J. Neurol. Neurosurg. Psychiatry*. **94** (12), e2 (2023).
4. Gonzalez-Herrero, B. et al. Functional neurological disorder and autism spectrum disorder: A complex and potentially significant relationship. *Brain Behav.* **14** (12), e70168 (2024).
5. Demartini, B. et al. Prevalence of autistic traits and their relationships with other psychopathological domains in young adults seeking psychiatric attention: a cluster analysis. *Eur. Psychiatry*. **67** (1), e71 (2024).
6. Carpita, B. et al. Autistic traits and somatic symptom disorders: what is the link? *Brain Sci.* ;**14**(3), 274 (2024).
7. Matson, J. L. et al. Examining cross-cultural differences in autism spectrum disorder: A multinational comparison from Greece, Italy, Japan, Poland, and the united States. *Eur. Psychiatry*. **42**, 70–76 (2017).
8. Sarovic, D. A unifying theory for autism: the pathogenetic triad as a theoretical framework. *Front. Psychiatry*. **12**, 767075 (2021).
9. McWilliams, A. et al. Autism spectrum disorder in children and young people with non-epileptic seizures. *Seizure* **73**, 51–55 (2019).
10. Pun, P., Frater, J., Broughton, M., Dob, R. & Lehn, A. Psychological profiles and clinical clusters of patients diagnosed with functional neurological disorder. *Front. Neurol.* **11**, 580267 (2020).
11. González-Herrero, B., Morgante, F., Pagonabarraga, J., Stanton, B., & Edwards, M. J. Autism Spectrum Disorder May Be Highly Prevalent in People with Functional Neurological Disorders. *Journal of Clinical Medicine*, **12**(1), 299 (2022).
12. Tamilson, B., Poole, N. & Agrawal, N. The co-occurrence of functional neurological disorder and autism spectrum disorder: a systematic literature review and meta-analysis. *Cogn. Neuropsychiatry*. **29** (6), 358–385 (2024).
13. Vickers, M. L. et al. Comorbidity rates of autism spectrum disorder and functional neurological disorders: A systematic review, meta-analysis of proportions and qualitative synthesis. *Autism* **29** (2), 344–354 (2025).
14. Cole, R. H., Elmalem, M. S. & Petrochilos, P. Prevalence of autistic traits in functional neurological disorder and relationship to alexithymia and psychiatric comorbidity. *J. Neurol. Sci.* **446**, 120585 (2023).
15. Hus, V. & Lord, C. The autism diagnostic observation schedule, module 4: revised algorithm and standardized severity scores. *J. Autism Dev. Disord.* **44** (8), 1996–2012 (2014).
16. Ritvo, R. A. et al. The Ritvo autism asperger diagnostic Scale-Revised (RAADS-R): a scale to assist the diagnosis of autism spectrum disorder in adults: an international validation study. *J. Autism Dev. Disord.* **41** (8), 1076–1089 (2011).
17. Dell'Osso, L. et al. Defining the optimal threshold scores for adult autism subthreshold spectrum (AdAS spectrum) in clinical and general population. *Clin. Pract. Epidemiol. Ment Health*. **16**, 204–211 (2020).
18. Kessler, R. C. et al. Validity of the world health organization adult ADHD Self-Report scale (ASRS) screener in a representative sample of health plan members. *Int. J. Methods Psychiatr Res.* **16** (2), 52–65 (2007).
19. Rong, Y., Yang, C-J., Jin, Y. & Wang, Y. Prevalence of attention-deficit/hyperactivity disorder in individuals with autism spectrum disorder: A meta-analysis. *Res. Autism Spectr. Disorders*. **83**, 101759 (2021).
20. Hull, L. et al. Development and validation of the camouflaging autistic traits questionnaire (CAT-Q). *J. Autism Dev. Disord.* **49** (3), 819–833 (2019).
21. Gierk, B. et al. The somatic symptom scale-8 (SSS-8): a brief measure of somatic symptom burden. *JAMA Intern. Med.* **174** (3), 399–407 (2014).
22. Kroenke, K., Spitzer, R. L. & Williams, J. B. The PHQ-9: validity of a brief depression severity measure. *J. Gen. Intern. Med.* **16** (9), 606–613 (2001).
23. Spitzer, R. L., Kroenke, K., Williams, J. B. & Löwe, B. A brief measure for assessing generalized anxiety disorder: the GAD-7. *Arch. Intern. Med.* **166** (10), 1092–1097 (2006).
24. Felitti, V. J. et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: the adverse childhood experiences (ACE) study. *Am. J. Prev. Med.* **14** (4), 245–258 (1998).
25. Stavropoulos, K. K. & McPartland, J. C. *Differential Diagnosis of Autism* (Oxford University Press, 2022).
26. Stavropoulos, K. K.-M., Bolourian, Y., & Blacher, J. (2018). Differential Diagnosis of Autism Spectrum Disorder and Post Traumatic Stress Disorder: Two Clinical Cases. *Journal of Clinical Medicine*, **7**(4), 71.
27. Hughes, K. et al. The effect of multiple adverse childhood experiences on health: a systematic review and meta-analysis. *Lancet Public. Health*. **2** (8), e356–e66 (2017).
28. Leising, D., Grande, T. & Faber, R. The Toronto alexithymia scale (TAS-20): A measure of general psychological distress. *J. Res. Pers.* **43** (4), 707–710 (2009).
29. Hogeveen, J., Grafman, J. & Alexithymia *Handb. Clin. Neurol.* ;**183**:47–62. (2021).
30. Sierra, M. & Berrios, G. E. The Cambridge depersonalization scale: a new instrument for the measurement of depersonalization. *Psychiatry Res.* **93** (2), 153–164 (2000).
31. Mehling, W. E., Acree, M., Stewart, A., Silas, J. & Jones, A. The multidimensional assessment of interoceptive Awareness, version 2 (MAIA-2). *PLoS One*. **13** (12), e0208034 (2018).
32. South, M., Carr, A. W., Stephenson, K. G., Maisel, M. E. & Cox, J. C. Symptom overlap on the srs-2 adult self-report between adults with Asd and adults with high anxiety. *Autism Res.* **10** (7), 1215–1220 (2017).
33. Tonge, N. A., Rodebaugh, T. L., Fernandez, K. C. & Lim, M. H. Self-reported social skills impairment explains elevated autistic traits in individuals with generalized social anxiety disorder. *J. Anxiety Disord.* **38**, 31–36 (2016).
34. Lobregt-van Buuren, E., Hoekert, M., Sizoo, B. Autism, Adverse Events, and Trauma. In: Grabrucker, A. M. (ed.) Autism Spectrum Disorders. Exon Publications. (2021).
35. Copyright The Authors.; (2021).

36. Mosner, M. G. et al. Rates of Co-occurring psychiatric disorders in autism spectrum disorder using the mini international neuropsychiatric interview. *J. Autism Dev. Disord*. **49** (9), 3819–3832 (2019).
37. Paredes-Echeverri, S., Guthrie, A. J. & Perez, D. L. Toward a possible trauma subtype of functional neurological disorder: impact on symptom severity and physical health. *Front. Psychiatry*. **13**, 1040911 (2022).
38. Mavroudis, I., Kazis, D., Kamal, F. Z., Gurzu, I.-L., Ciobica, A., Păduraru, M., Novac, B., & Iordache, A. (2024). Understanding Functional Neurological Disorder: Recent Insights and Diagnostic Challenges. *International Journal of Molecular Sciences*, **25**(8), 4470.
39. Smith, K. E. & Pollak, S. D. Early life stress and development: potential mechanisms for adverse outcomes. *J. Neurodev Disord*. **12** (1), 34 (2020).
40. Sonuga-Barke, E. J. S. et al. Child-to-adult neurodevelopmental and mental health trajectories after early life deprivation: the young adult follow-up of the longitudinal english and Romanian adoptees study. *Lancet* **389** (10078), 1539–1548 (2017).
41. Rumball, F., Happé, F. & Grey, N. Experience of trauma and PTSD symptoms in autistic adults: risk of PTSD development following DSM-5 and Non-DSM-5 traumatic life events. *Autism Res.* **13** (12), 2122–2132 (2020).
42. Alaghband-Rad, J., Hajikarim-Hamedani, A. & Motamed, M. Camouflage and masking behavior in adult autism. *Front. Psychiatry*. **14**, 1108110 (2023).
43. Griffiths, S. et al. The vulnerability experiences quotient (VEQ): A study of vulnerability, mental health and life satisfaction in autistic adults. *Autism Res.* **12** (10), 1516–1528 (2019).
44. Khudiakova, V., Russell, E., Sowden-Carvalho, S. & Surtees, A. D. R. A systematic review and meta-analysis of mental health outcomes associated with camouflaging in autistic people. *Res. Autism Spectr. Disorders*. **118**, 102492 (2024).
45. Patil, O. & Kaple, M. Sensory processing differences in individuals with autism spectrum disorder: A narrative review of underlying mechanisms and Sensory-Based interventions. *Cureus* **15** (10), e48020 (2023).
46. Williams, Z. J. et al. Characterizing interoceptive differences in autism: A systematic review and Meta-analysis of Case-control studies. *J. Autism Dev. Disord*. **53** (3), 947–962 (2023).
47. Millman, L. S. M. et al. Interoception in functional motor symptoms and functional seizures: preliminary evidence of intact accuracy alongside reduced insight and altered sensibility. *Behav. Res. Ther.* **168**, 104379 (2023).
48. Pick, S. et al. Dissociation and interoception in functional neurological disorder. *Cogn. Neuropsychiatry*. **25** (4), 294–311 (2020).
49. Sojka, P., Bareš, M., Kašpárek, T. & Světlák, M. Processing of emotion in functional neurological disorder. *Front. Psychiatry*. **9**, 479 (2018).
50. Yogarajah, M. et al. *17 State and Trait Interoception Is Disrupted in Functional Seizures* (BMJ Publishing Group Ltd, 2019).
51. Kinnaird, E., Stewart, C. & Tchanturia, K. Investigating alexithymia in autism: A systematic review and meta-analysis. *Eur. Psychiatry*. **55**, 80–89 (2019).
52. Jungilligens, J., Paredes-Echeverri, S., Popkirov, S., Barrett, L. F. & Perez, D. L. A new science of emotion: implications for functional neurological disorder. *Brain* **145** (8), 2648–2663 (2022).
53. Jenkinson, R., Milne, E. & Thompson, A. The relationship between intolerance of uncertainty and anxiety in autism: A systematic literature review and meta-analysis. *Autism* **24** (8), 1933–1944 (2020).
54. Buch, A. M. et al. Molecular and network-level mechanisms explaining individual differences in autism spectrum disorder. *Nat Neurosci. 26*(4), 650–663.

Author contributions

1) Research project: Conception and organisation: BG-H, M.E, Q.DExecution: BG-H2) Statistical Analysis: Design and execution: BG-HReview and critique: J.C, F.M, J.P, Q.D, M.E3) Manuscript: Writing of the first draft: BG-H. Review and Critique: J.C, F.M, J.P, Q.D, M.E.

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Declarations

Competing interests

M.E. provides expert medicolegal testimony for people with neurological and psychiatric conditions, including FND. J.C. provides expert medicolegal testimony for people with neurological conditions, including FND.The rest of the authors do not have conflicts of interest in the current study.

Ethics statement

This observational, cross-sectional project was conducted as a service development initiative and was registered with the Clinical Audit Department at St George's University Hospitals NHS Foundation Trust (registration reference: AUDI003674). All methods were carried out in accordance with relevant guidelines and regulations, including those outlined by the Declaration of Helsinki. The aim was to evaluate and improve local assessment practices for identifying autism spectrum traits within a Neuropsychiatric clinic for FND, as well as other psychiatric comorbidities. As this project constituted service evaluation and did not involve any deviation from standard clinical care, it did not require review by an Ethics Committee, in line with the UK Health Research Authority guidelines.

Additional information

Correspondence and requests for materials should be addressed to B.G.-H. or M.J.E.